A DYNAMIC ANALYSIS OF LONG TERM IMPACTS OF GENETICALLY MODIFIED CROPS ON AGRICULTURE

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ABSTRACT

A DYNAMIC ANALYSIS OF LONG TERM IMPACTS OF GENETICALLY MODIFIED CROPS ON AGRICULTURE

In this study, two simulation models are built to study the long term usefulness of genetically modified agriculture via two GM crops: insect resistant (Bt-corn) and herbicide tolerant (HT canola). Using system dynamics modeling methodology, agricultural sustainability is analyzed under different policies and scenarios, by focusing on the fundamental feedback mechanisms in the environment. The most critical feedback mechanism is the evolution of resistance in pests and weeds via natural selection. The effect of Bt-crops on increasing the rate of pests' resistance evolution is found to be notable. Depending on the initial level of resistance in the pest population, resistance can evolve in ten seasons of Bt-corn farming whereas it would take fifty seasons with conventional pesticides under the same conditions. Refuge strategy is tested under different scenarios. Results show that benefits from the refuge strategy decline considerably if pest develops cross-resistance to the pesticide that is used to treat the refuge. Furthermore, refuge strategy becomes futile if heterozygote pests show some level of resistance or if the initial level of resistance alleles in the pest population is high and there is no 'fitness cost' of resistance. Agriculture with HT crops is analyzed by simultaneous comparison with conventional crops. Two cases of herbicide use are analyzed: in the first case, herbicide is sprayed as a function of weed density and in the second one it is used at a predetermined amount. It is found that superweed emergence increases the rate of resistance evolution in the weed population. Under the constant herbicide strategy, GM crop turns out to be more effective than the conventional one, in spite of superweeds. However, this strategy results in a higher rate of resistance development in weeds and more herbicide usage compared to the variable herbicide strategy. In terms of long term cumulative yield losses, rate of resistance development and herbicide usage, the best policy is discovered to be planting conventional crops with the variable herbicide application strategy.

ÖZET

GENETİĞİ DEĞİŞTİRİLMİŞ TARIM ÜRÜNLERİNİN UZUN ERİMLİ ETKİLERİNİN DEVİNGEN ÇÖZÜMLEMESİ

Bu çalışmada, genetiği değiştirilmiş tarım ürünlerinin uzun vadeli yararlığını değerlendirmek için böceğe dayanıklı (*Bt*-mısır) ve yabancı ot ilaçlarına dayanıklı (HT-kanola) ürünlerin dinamik benzetim modelleri kurulmuştur. Sistem dinamiği yöntemi kullanılarak, bu ürünlerle yapılan tarımın sürdürülebilirliği değişik senaryolar altında ve doğadaki geribildirim mekanizmaları göz önüne alınarak incelenmiştir. Bu mekanizmalardan en önemlisi doğal seleksiyon yoluyla böcek ve otlarda direnç evrimidir. *Bt* tarım ürünlerinin böcekte direnç kazanım hızını artırıcı etkisi kayda değer bulundu. Bu ürünler sonucu direnç evrimi, böcek populasyonunun başlangıçtaki direnç aleli seviyesine bağlı olarak on mevsimde gerçekleşebilirken, aynı koşullar altında geleneksel sprey pestisitlerle bu süre elli mevsimi bulmaktadır. “Barınak” stratejisi değişik senaryolar altında denenmiştir. Eğer böcek, barınakta kullanılan pestiside karşı çapraz direnç kazanmışsa, bu stratejinin olumlu etkilerinin ciddi düzeyde azaldığı görülmüştür. Heterozigot böceklerin bir miktar direnç göstermeleri ya da başlangıç direnç aleli seviyesinin yüksek olması ve direnç kazanımının başka yaşam dezavantajının olmaması durumlarında ise barınak stratejisi işe yaramamaktadır. Ilaçlamaya dayanıklı ürünle (HT kanola) yapılan tarım, geleneksel tarımla karşılaştırılırken iki ayrı ilaçlama yöntemi göz önüne alınmıştır: ilkinde kullanılan herbisit miktarı tarladaki yabancı ot yoğunluğuna bağlı olup (değişken herbisit yöntemi), ikincisinde ise hep aynı doz uygulanmaktadır (sabit herbisit yöntemi). HT ürün tarlalarında hibritleşmeyle oluşan transjenik yabancı otların direnç kazanım hızını artırdığı gözlenmiştir. Bu otların ürün kaybına etkisi ise, herbisit yöntemine bağlıdır. Sabit herbisit yöntemi altında HT ürünün geleneksel ürüne kıyasla daha verimli olduğu saptanmıştır. Fakat, bu yöntem değişken herbisit yöntemine göre daha hızlı ot direnci evrimine ve daha fazla herbisit kullanımına yol açmaktadır. Uzun vadede toplam ürün kayıpları, direnç oluşma hızı ve herbisit kullanımı göz önüne alınınca, normal ürün ile değişken herbisit yöntemini uygulamanın en iyi strateji olduğu görülmüştür.

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bf	Birth fraction
Bt	Bacillus thuringiensis
comp	Competition
df	Death fraction
DDT	Dichloro-diphenyl-trichloroethane
ECB	European corn borer
eff	Effect
EEC	European Economic Committee
EPA	Environmental Protection Agency
fr	Fraction
GM	Genetically modified
grm	Germination
HI	Harvest index
HT	Herbicide tolerant
IPM	Integrated Pest Management
IR	Insect resistant
max	Maximum
NRDS	Natural Resources Defense Council
OMAFRA	Ontario Ministry of Agriculture, Food and Rural Affairs
prob	Probability
ref	Reference
rgr	Regeneration
Sweed	Superweed
USDA	United States Department of Agriculture

1. INTRODUCTION

Insect pests and weeds have detrimental effects on the efficiency of agricultural production, which resulted in a history of pest control dating back to 2500s BC. Though this history passed through revolutionary steps, a general method, which is both efficient and sustainable, has not been fully developed. Genetic modification of the crop in order to provide an enhanced and efficient pest control is the latest step taken along this path.

A pest species is any species that is considered to be undesirable by humans. It may be competing with humans for food by damaging or consuming food crops; it may be feeding on domesticated animals and it may be transmitting diseases. In general it is a species that threatens human welfare, comfort and health (Begon *et al.,* 1996). Weeds are a type of pest. They are plants growing in a place where a human wants a different kind of plant or no plant at all and are usually successful competitors for the food crops. The aim of pest control is to reduce the pest population to an acceptable level, below which no further reductions are profitable (Begon *et al.,* 1996).

Pest control was revolutionized with the advent of organic insecticides such as dichloro-diphenyl-trichloroethane (DDT) during the Second World War. These chemicals were capable of killing hundreds of different kind of species at once and there was a general opinion that they brought enormous benefits with negligible side effects (NRDC, 1997). Though the invention was greeted with such strong optimism due to its effectiveness, low cost and ease of application, its hey day ended with the publication of Rachel Carson's *Silent Spring* in 1962. In her book, she described how DDT entered the food chain and caused serious health damages due to the accumulation of chemicals (Carson, 1962). This resulted in a reconsideration of the effects of pesticides, giving rise to the notion of the need to regulate industry in order to protect the environment. DDT was banned in the USA in 1977. Since then, many undesirable consequences of chemical pest control have been reported, even though more specific and less toxic pesticides have been and are being invented (NRDC, 1997).

Genetic modification brings forth a new means of pest control for insect pests. The insertion of a special protein which is harmful for the target pest prevents the genetically modified (GM) crop from being eaten by the pest. Hence, when a crop is modified to be insect resistant (IR), pesticide is no longer sprayed onto the crop but bred into it. On the other hand, the means for weed control is not altered in this GM technology; still herbicides are sprayed to kill the unwanted plant species. However, insertion of a herbicide tolerance gene enables farmers to spray wide-spectrum herbicides on their fields killing all the plants but the herbicide tolerant (HT) crop. Hence, genetically modified IR and HT crop varieties offer powerful pest control mechanisms. However, negative impacts of this technology on human health and the environment are yet to be discovered.

Because GM technology is powerful, it has the ability to make significant positive or negative changes in agriculture. However, many possible consequences of adopting GM technology on crop yield will stem from complicated interactions in the environment and will not be evident in the short run. Hence, analyzing these consequences requires a systemic view and a consideration of long timeframes. In the scope of this study, two simulation models are built using system dynamics methodology to make a dynamic analysis of the long-term effects of IR and HT type of GM crops. By focusing on the fundamental feedback mechanisms in the environment and the intervention of agricultural practices, we aim to evaluate the sustainability of this technology.

1.1. State-of-the Art in Genetic Modification

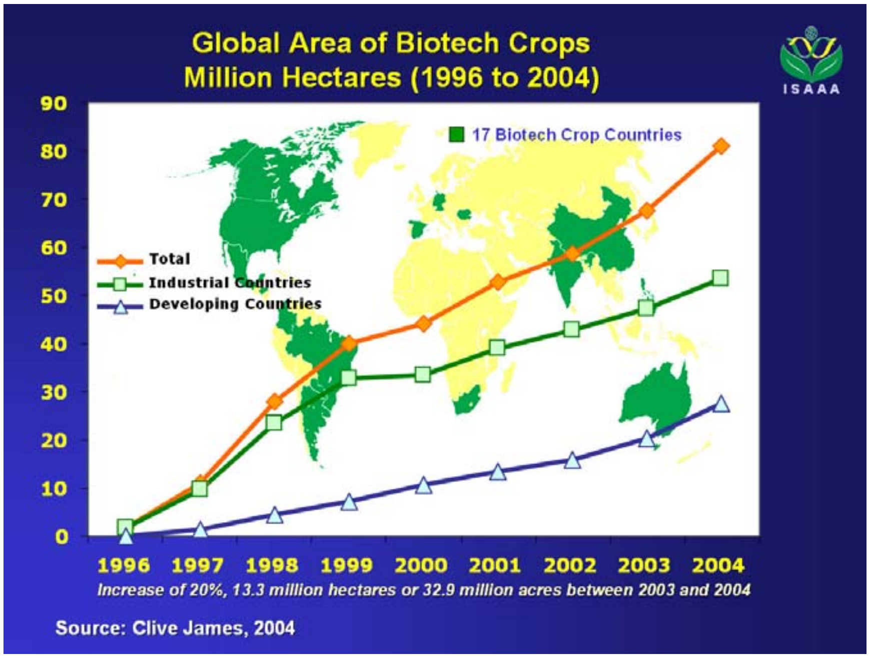
Genetic modification is the use of modern biotechnological techniques to alter the genetic makeup of certain living organisms such as plants, animals, or bacteria in a way that does not occur naturally by mating and/or natural recombination (EEC, 1990). In the past, crop breeders were limited to transferring genes from one crop variety to another. With GM technology, the genes transferred into the organism can be from the same organism, a related organism or from a totally unrelated organism such as a bacterium, a virus or an animal. Moreover, it is possible to incorporate genes for the desired trait without incorporating undesirable traits.

With its immense potential of diversification in its products and the existence of abundant potential inputs, GM technology has been introduced as a solution to world hunger and ecological crisis created by industrial agriculture. Specifically, the technology has four broad goals, namely enhancing the product characteristics, improving plant resistance to pests and pathogens, increasing output and improving nutritional value (Kendall, *et al.,* 1997). Table 2.1 summarizes categories of characteristics for which GM crops have either been developed or for which research is ongoing.

The first GM crops were commercialized by China in the early 1990s; yet the acceleration in the use of the technology corresponds to late 1990s after its introduction to US in 1994 (Nelson, 2001). Between 1996 and 2004, GM crop area increased more than 47 fold, from 1.7 million hectares to 81 million hectares (James 2004). The number of countries growing biotechnology tripled during the same period (James 2004). While a considerable amount of GM crop area is in industrial countries (66%), the proportion of GM crops grown in developing countries has increased consistently every year. Most GM crops are grown in North America, followed by Argentina, Brazil and China (James, 2004). Figure 2.1 displays the increasing adoption of GM technology both in industrial and developing countries.

Table 1.1. Improved characteristics of transgenic crops

GM Characteristics	***Rationale***	***Examples***
Herbicide Tolerance	More efficient herbicide use and/or safer herbicide use	Glyphosate-tolerant soybeans, canola
Disease/insect tolerance	Reduction in pesticide use and/or more efficient pest control	Bt-cotton, corn, potatoes
Quality Improvements	Development of new foods or sources of new products	Ripening-delayed tomato
Tolerance to biological stresses	Improved resistance to droughts, easier nitrogen fixation	Research on drought-tolerant corn
Productivity enhancements	Higher output per unit land	High yielding rice and corn



Increase of 20%, 13.3 million hectares or 32.9 million acres between 2003 and 2004

Source: Clive James, 2004

Figure 1.1. Global area of GM crops from 1996 to 2004

The most widely adopted GM products are herbicide tolerant (HT) and insect resistant (IR) crops. The insertion of a herbicide tolerance gene enables farmers to spray wide-spectrum herbicides on their fields killing all the plants but the GM herbicide tolerant crop; whereas the insertion of a special protein which is harmful for the target pest prevents the IR crop from being eaten by the pest, aiming to decrease or eliminate pesticide use. Table 2.2 summarizes the dominant GM crop varieties by their commercial planting area.

Table 1.2. Dominant GM crops

GM Crop	***Grown Area in Million Hectares***
Herbicide tolerant soybean	48.4
Bt Corn (IR)	11.2
Bt Cotton (IR)	4.5
Herbicide tolerant corn	4.3
Herbicide tolerant canola	4.3
Bt/Herbicide Tolerant Corn	3.8
Bt/Herbicide Tolerant Cotton	3.0
Herbicide Tolerant Cotton	1.5
Total	**81.0**

These applications are in essence a continuation of the current agriculture regime which relies on herbicide and pesticide use to fight against weeds and pests. Advocates of this technology claim that pest and weed management will be enhanced via genetically acquired herbicide tolerance/pest resistance and that yields will increase accordingly (See, for example, Nap *et al.*, 2003; Bennett *et al.*, 2004; Carpenter *et al.*, 2001).

Currently all commercialized insect resistant GM crops contain genes expressing *Bt* toxins obtained from *Bacillus thuringiensis*, which resulted in the convention of calling these crops *Bt-crops* rather than IR crops[1]. *Bacillus thuringiensis* is a naturally occurring soil bacterium, which produces "Cry" proteins that are toxic to insects (Teulon, *et al.*, 2002). A unique feature of *Bt* as a pesticide (either synthetic or biological) is that a specific Cry protein is toxic only to specific groups of insects and has no known effects on mammals. These characteristics make *Bt* pesticides very popular in general and very crucial specifically for the organic food industry (Nelson *et al.*, 2001). However one problem with synthetic *Bt* pesticides is that the toxic effect breaks down quickly after exposure to ultraviolet radiation, heat or dry conditions, which possibly occurs before functioning. Modifying a certain crop to produce its own *Bt* protein overcomes this problem since *Bt*-plants generally express the toxin throughout the plant and for the duration of the growing cycle.

As to the herbicide tolerant crops, the most common application is tolerance to glyphosate, which is a broad-spectrum, non-selective herbicide whose commercial name is "Roundup". Prior to the introduction of herbicide tolerant soybeans and sugar beet,

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[1] From this point on, the name *Bt*-crop will stand for *insect resistant crops* in accordance with this convention.

growers chose from many herbicides, often applying three or more active ingredients, some of which would cause severe damage to the growing plants, or cause harm to crops that commonly follow. Genetically modified herbicide tolerant crops can overcome this problem by allowing growers to rely on one herbicide to control a broad spectrum of weeds without harming the crops.

1.2. Debates on Ecological Impacts of Genetically Modified Crops

The next decades may see an exponential growth in GM product development as researchers gain increasing access to genomic resources that are applicable to organisms. However, there is an ongoing risk debate and a widespread opposition to GM technology due to an unresolved host of questions about food safety, environmental effects, economic impacts, and ethical issues. According to proponents, GM crops are a natural extension of traditional crop breeding and they offer a solution to feed the growing world population by increasing agricultural efficiency. However, opponents claim that GM crops possess unknown ecological risks, promote the further industrialization of agriculture, reduce biodiversity, favor monocultures and direct R&D according to commercial criteria rather than public benefit. Among these issues the potential environmental impacts of GM crops are critical for this study, since we aim to evaluate the sustainability of pest management via GM technology by focusing on the fundamental feedback mechanisms in the environment.

Introduction of exotic genes into crops can have unpredictable ecological impacts. While some possible problems are unique to this technology, some of the ecological problems associated with GM are no different than the problems already raised by agricultural intensification. Yet, GM technology may result in a uniquely rapid increase in agricultural intensification, further complicating such problematic issues. Indeed, many problems that were faced with increasing intensification of agriculture must be addressed once again to better assess the risks associated with GM technology. Some frequently stated arguments include the following phenomena:

- Transgenes can spread to wild and weedy relatives, creating ‘superweeds’ if the transgene increases the fitness of the weedy species (Hails, 2005; Halfill, 2002 etc.). Hybridization between compatible plant species is a natural phenomenon, so not an issue triggered by GM crops. However, the risk with the GM crops is that fitness enhancing transgenes will move to wild relatives resulting in weed shifts, which may trigger unpredictable ecological consequences. For example, weeds containing a transgene that provides resistance to draught have an increased fitness in nature and may compete more with beneficial plant species leading to the latter’s suppression. On the other hand, herbicide tolerant superweeds will increase the complexity of the farming management problem due to their resistance to the selected herbicide(s). Current reliance on just a few broad-spectrum herbicides already makes it likely that herbicide tolerance will develop fast. Herbicide tolerant superweeds will further speed up this phenomenon.

- Insect resistant GM crops will speed up the spread of resistance in major insect pests. If a pest population already has a high background level of resistance genes, then resistance may be spread in the population within three to four years even when the refuge strategy enforced by EPA (Environmental Protection Agency) is applied (Levidow, 1999). When pest species become resistant to major toxins, farmers will switch to more toxic pesticides and the use of pesticides will be boosted contrary to reduced use of pesticides claim. Moreover when a population becomes resistant to a specific toxin, it is quite likely that it evolves resistance to other toxins with the same mode of action. Tabashnik has reported a case where one resistance gene conferred resistance to four different toxins (Tabashnik et al., 1997). Hence it may be impossible to generate new effective pesticides in the long run, especially when the various constraints imposed by environment, human health, and production and R&D costs are considered (Laxminarayan, 2000).

- A plant that germinates from a seed left behind in the field from a previous crop is called *volunteer*. Volunteer GM crops may be problematic weeds for the other crops in the rotation since they may have fitness increasing properties (Kwon, *et* al., 2001). Volunteerism is a concern for GM herbicide tolerant crops especially if the same herbicide is used throughout the rotation, because these plants are not controllable by the herbicide just like the resistant weeds.

- GM crops may have deleterious effects on non-target organisms, such as beneficial insects and birds. It has already been demonstrated that a *Bt*-corn variety can kill the larvae of monarch butterflies (Losey, *et al.,* 1998). Moreover, *Bt* is argued to increase the mortality rate of green lacewing which is a beneficial insect (Hilbeck, 1998).

- A loss in biodiversity may be observed due to severe weed control and due to toxins released from GM crops. Biodiversity is an essential resource for agriculture along many fronts; from incorporating traits for resistance to diseases and pests, to improved nutritional qualities, more effective soil nutrient uptake, and more environmentally friendly methods to control pests (Srivastava, *et. al*, 1996). Moreover, biodiversity generally brings stability in the sense that diverse systems can display more resistance towards changes in response to factors imposed from outside (Norton, 1987).

All of these possible problems challenge the claimed benefits of GM insect resistant and herbicide tolerant crops and raise concerns about the sustainability of a pest management practice that relies on these products. Considering the huge and continuous investment in agricultural biotechnology, such concerns become further notable. Hence, it is not surprising that these issues have been the subject of many theoretical studies apart from the ongoing intensive empirical work.

Much of the modeling work focuses on the pest resistance problem in *Bt*-crops (Linacre *et al*., 2003; Laxminaryan *et al.,* 2000; Hurley, 2005, etc). Most of these studies investigate the effectiveness of EPA’s refuge strategy to slow down resistance evolution and try to optimize the refuge proportion with respect to different objectives. However there is no comprehensive model which focuses on feedback processes in the environment to come up with a generic sustainability analysis of *Bt*-crops. None of the models consider the pest's interactions with its predator. In their generic model for a dynamic analysis of food-prey-predator system in agriculture, Mediavilla *et al* show that the prey-predator dynamics is an important factor to consider in the investigation of the effects of agricultural practices (Mediavilla *et al*., 2004). Especially, the effect of *Bt* toxin on the predator species may be a determining factor for the long-term success of the pest control.

Not much effort has been devoted to modeling the effects of herbicide tolerant crops on farming management. There are studies that present micro-level models of gene flow phenomenon and some of them analyze its realization in HT crops (see for example Richter *et al*., 2004). However, the focus of these models is not the long term effects of HT crops but the gene flow process itself. In one macro-level model, Watkinson *et al* concentrates on the effects of HT crops on farm bird population (Watkinson *et* al., 2000). However, the study does not cover superweed formation, herbicide resistance development and certain feedback relations existing in the population dynamics, as criticized by Firbank *et al* (Firbank *et al*., 2000). Two highly specific and detailed simulation models are built by Danish EPA which account for the aforementioned phenomena (Danish EPA, 1999). However since their model purpose is to come up with a comprehensive analysis of selected HT crops on Danish agriculture, they do not constitute a generic framework for the sustainability analysis of agriculture with HT crops.

In this study we intend to assess the sustainability of agriculture with GM insect resistant and herbicide tolerant crops by modeling the interactions of these crops with farming management, the focus being on the potential environmental and ecological problems discussed in the literature.

2. PROBLEM DESCRIPTION AND OBJECTIVE

Bt-crops are presented as powerful solutions to enhance pest management with the ultimate goal of higher yields. Yet, sustainability of their merits is debatable due to certain feedback processes in the environment, the major one being pest resistance evolution via natural selection. If a plant produces a compound toxic to an insect species, any individual of that species not susceptible to that toxin has reproductive advantage. Because insects have a short lifespan and high reproductive capability, resistance can spread quickly throughout the population. A pest population which is resistant to the specific *Bt*-toxin can no longer be controlled by that toxin and most probably by other toxins with the same mode of action. In these cases, the possible comparative benefits of *Bt*-plants would only hold for a relatively short period and agriculture with *Bt*-crops will be far from being sustainable. Hence, pest resistance development raises a point of concern for preserving chemical treatments as a tool for pest management for future use.

Another feedback process influential on the success of a pest control program is target pest resurgence. Target pest resurgence occurs when a pest control treatment damages the predators of the pest species as well. If that is the case, a sharp decrease in the predator population will be observed for two reasons: first due to the toxin used in the treatment; second due to the initial drop in pest density caused by the treatment, i.e. due to food scarcity. Then, any pest individuals that survive due to resistance or luck, or that migrate to the area find themselves with a plentiful food resource but few if any natural enemies (predators). In this case, a pest population boom is very likely to happen. (Moreover, if the treatment damages a non-target species which is the predator of some secondary pest, that secondary pest may grow and become a major pest, further increasing the complexity of the pest management problem.)

The problems of target pest resistance, resurgence and secondary pest outbreaks have been frequently reported in chemical pest management. These problems have resulted in the application of more and other pesticides leading to further resistance, further resurgence and further secondary pests, and so to more pesticide, more problems and more expense… (See Cowan, 1996 for a detailed analysis) This has been called the *pesticide treadmill*, implying that once farmers get on, they find it difficult to get off (Wilson, 2001). Being more effective pesticides, *Bt*-crops are very likely to cause the same problems probably more quickly and with higher intensity.

Likewise, alleged benefits of HT crops, namely increase in yields, need to be justified via a long-term dynamical analysis since these claims underrate the possible effects of gene flow and herbicide resistance evolution phenomena. With GM herbicide tolerance technology, continuous use of a single herbicide is encouraged and such consistent selection pressure has been most responsible for herbicide resistance evolution. Hence, introduction of a GM crop tolerant to a specific herbicide could result in accelerated evolution of herbicide resistance. Moreover if herbicide tolerance genes escape to closely related weedy species, superweeds may evolve. Superweeds are essentially herbicide resistant weeds; hence they increase the weed management burden since their control necessitates a different herbicide or other additional techniques. If they are not suppressed they may speed up the spread of resistance in the weed population. In addition, HT crops can become problematic agricultural weeds themselves in the next rotation or in an adjacent field by gene flow. Since they are herbicide tolerant just like superweeds, this would increase the difficulty of farming management. All these issues may further increase herbicide usage in the long run since farmers will again need to spray additional herbicides getting on the aforementioned pesticide treadmill. Indeed, herbicide use is already reported to have increased in certain regions due to such reasons (Benbrook, 2001) Significant increase in yields would not be observed either, unless HT crop is modified to be tolerant to the pack of herbicides applied.

Many of these possible consequences of adopting GM technology are not evident in the short run. There are some experimental field studies to come up with a farm-scale evaluation of GM crops such as DEFRA (Department of Environment and Rural Affairs) projects in UK (Squire *et al.,* 2003; Firbank *et al.*, 1999) and projects of Danish Environmental Protection Agency (Danish EPA, 1999); however such studies are costly and have to be pursued for sufficiently long time in order to grasp the slow occurring effects. Hence, a dynamic modeling and simulation of interactions of GM herbicide tolerant and insect resistant crops with farming management that takes into account the long-term environmental feedback structures will be helpful in the evaluation of sustainability of this technology.

In this study, two system dynamics models are built to analyze GM crops in terms of agricultural sustainability. Instead of assuming a single insect resistant and herbicide tolerant crop, these two varieties are investigated in two separate models, called IR and HT models respectively. This approach decreases model complexity without sacrificing fundamental feedbacks and interactions so as to bring about a clearer demonstration of possible ecological phenomena. It is also in accordance with the current situation in the biotechnology market, where dominant GM crops are either insect resistant or herbicide tolerant, but not both.

A previous model developed by Dogan and Karanfil (Dogan, et.al, 2002) provides a generic framework that comprises the gene flow phenomenon and the formation of superweeds and resistant pests through mutation. Certain causal structures and assumptions of this model are taken as a starting point. However, the core of this study is modeling the spread of resistance in the population through natural selection, which has been the biggest source of failure in chemical pest control. Considering GM technology as one step forward in agricultural intensification, both models search the limits of this intensification on a comparative basis with the technology’s conventional counterparts. Hence, the models are intended to provide an experimentation platform to study the impacts of agriculture with GM crops on farming practices under various policies, scenarios and conditions. This way, a generic sustainability analysis of GM-based agriculture will be possible.

3. METHODOLOGY

This study aims to build a model for the dynamic analysis of agriculture with GM crops, with particular emphasis on the environmental feedback mechanisms. However, the non-linear feedback nature of the system under concern makes it hard, if not impossible, to analyze it through analytical methods. With its power in handling complex feedback structures in large-scale natural or socio-economic systems, System Dynamics approach turns out to be appropriate for this analysis regarding the purpose of the study.

System dynamics is a computer based approach for modeling complex dynamic systems with particular focus on the inherent feedback structure, and experimenting with these models to design policies for improved performance. The simulation-based experimentation procedure of system dynamics helps to understand the dynamic complexity of the studied system, to identify the important policy entrance points, and to test long-term system-wide effects of policies (Sterman, 2000). This is an important advantage of the methodology over other quantitative approaches considering the purpose of this study.

System dynamics methodology focuses on predicting the *dynamic patterns* that would result from adopting a given set of policies, rather than predicting the values of the system variables point by point. This aspect makes system dynamics particularly applicable to long-term strategic policy analysis, which is another reason why it is evaluated to be appropriate for the problem under concern.

System dynamics modeling utilizes two basic variable types (Sterman, 2000). *Stocks* (or *level* variables) represent accumulations in the system such as population, inventory, capital stock etc. They are manipulated via instantaneous inflows and outflows, which are referred as *flow* (or *rate*) variables such as births, shipments and depletion. Models built with this methodology correspond to a set of differential or difference equations, in which stocks represent state variables and flows represent rates of change.

Stock variables are represented by boxes in model diagrams and rates are represented as valves on thick arrows that flow in or out of level variables. Arrowheads indicate the directions of these flows. An example of a stock-flow diagram for a simple Population model can be seen in Figure 4.1. The stock variable of the system is *Population*. *Population* is filled by the *births* flow and depleted by the *deaths* flow. Remaining variables *birth fraction* and *death fraction* are called *auxiliary* (or converter) variables and they are used for calculation of flows and defining the links between components of the system. Finally, curved thin arrows indicate causal relation between two variables in the system.

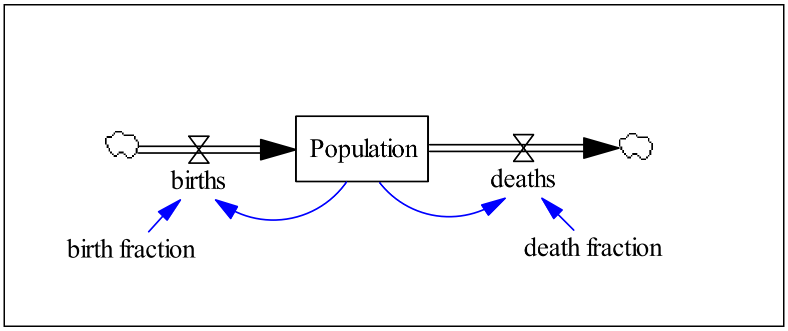

Figure 3.1. Representation of stock and flow variables in model diagrams

System dynamics methodology, which is composed of an iterative modeling process and a following experimentation, is mainly composed of five stages (Sterman, 2000).

The first step is problem articulation where the dynamic problem under concern is identified. In this step, system boundary and the time horizon of the study are set and key variables are determined.

In the second stage of the methodology, a dynamic hypothesis about the dynamic problem being studied is built. This process requires characterization of the feedback relationships between system variables with respect to the observed past or potential future behaviors of the system.

The third step consists of building the simulation model based on the hypothesis developed regarding the causal structure. After building the stock-flow structure of the model, parameters, equations and initial conditions are estimated based on the observed past data and system behavior.

The fourth step of the methodology is testing the validity of the built model with respect to the problem of concern. Validation process is actually performed almost concurrently with model building, rather than being a separate stage. The core of validation is testing the validity of the model structure against the real system relations regarding the dynamic hypothesis. Behavioral validation, which is comparing the output of the model with observed real dynamic behavior, is performed only if the model structure is validated. The plausibility of the model under extreme conditions and robustness of model behavior to uncertainties in initial conditions and parameters are important criteria in validity testing (Barlas, 1996).

The final step of the methodology is the use of the constructed and validated model in experiments for the purpose of policy analysis and design. This experimentation step enables to study behavior of the system under various policies, scenarios and conditions. Results obtained throughout the experimentation procedure provide effective information that helps to identify policy entrance points, and evaluate and design useful policies.

4. THE INSECT RESISTANT CROP (IR) MODEL

The model developed in this study is intended to consider the feedback processes in the environment in the analysis of long-term impacts of GM insect resistant crops (or in accordance with the convention, *Bt*-crops) on farming management. The model is built on farm scale, where *Bt*-corn is planted in an isolated land. As proposed in the objective statement, the purpose of the model is to bring forth insights for the evaluation of GM technology in terms of sustainability. To evaluate whether a new agricultural practice is in accordance with the sustainability principle, a systems approach is essential to explore the interconnections between farming and other aspects of the environment. The model tries to capture this perspective by incorporating the dynamics of the pest population into the analysis. The key aspect in modeling the pest population is keeping track of the spread of resistance in the population. Susceptible pests actually constitute a stock to be managed, if there is a concern for preserving chemical treatments as a tool for pest management for future use. Hence, dynamics of susceptible pest subpopulation is critical to monitor. In order to better account for the fundamental feedback mechanisms which will be determining for the behavior of the system, pest's interaction with its predator is also taken into consideration. This way, the model will be able to mimic a realistic range of ecological phenomena.

The basic relations in the system are summarized in the causal loop diagram below. The fundamental dynamics of the system is generated by the intervention of the agricultural practices with the positive feedback loops that determine the genotypic ratios in the population. Resistance is usually characterized by a single allele of a gene and all natural populations regardless of the application of any pesticide probably contain individuals that show resistance to one or more pesticides. When the number of resistant individuals increases, so does the frequency of resistance alleles in the population, all other things being equal. This frequency is the determinant of the proportion of pest births which are resistant, where high frequencies imply high proportions. Hence the subpopulation of resistant individuals increases with increasing resistance allele frequency, which will further increase this frequency in the next generation. The analogous of this positive feedback loop which is depicted on the northwest corner of Figure 4.1. exists for susceptible individuals. Yet, without any constraint in nature favoring one of the subpopulations, the increase rate in a subpopulation is proportionate to its genotypic ratio. That is, the increase in one subpopulation is accompanied by an increase in the other subpopulation such that the frequency of resistant individuals remains constant.

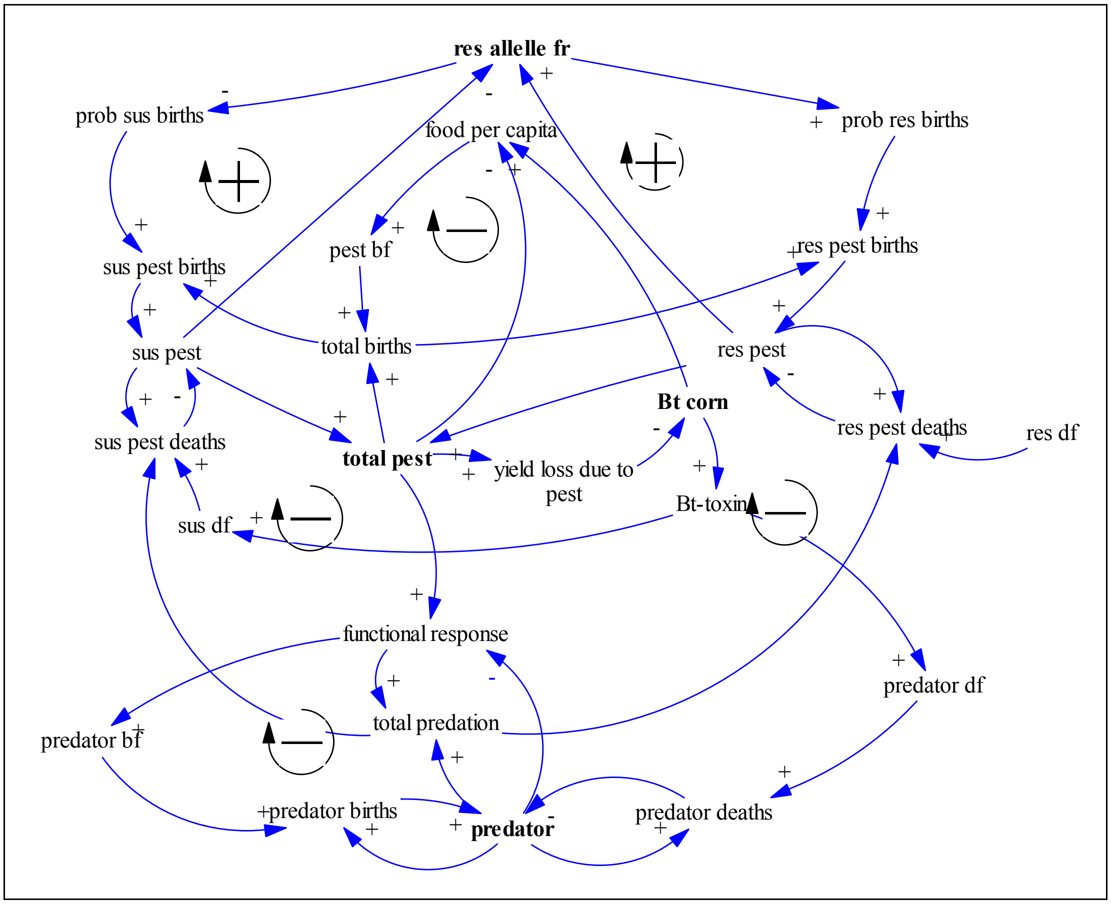

Figure 4.1. Causal loop diagram for the IR-Model

What characterizes the spread of resistance in the population is the introduction of a selection pressure in the environment, which in this case is planting *Bt*-corn. Planting *Bt*-corn will increase the amount of *Bt*-toxin the pest population is exposed to. The toxin will increase the death fraction of the susceptible individuals in the population, which will reduce the population of susceptibles. However, it will not have an effect on the resistant subpopulation. Therefore the frequency of resistance allele in the population will increase, which results in an increasing probability of having resistant births. The next generation will have a higher number of resistant individuals and if that population is also exposed to *Bt* toxin, with further lessened susceptible pests, resistance allele fraction will further increase. These positive feedback loops together with the consistent selection pressure may eventually result in the extinction of susceptible individuals. Such a pest population can no longer be controlled by this toxin (and most probably by other toxins with the same mode of action). Therefore, consistent application of a pesticide would fail to control the pest population in the long run.

The level of a pest population is naturally kept within certain limits by food availability and by interactions with predators. For the former, food per capita is the determining factor. Increasing pest population decreases food per capita. Under the pressure of food scarcity, birth rate is reduced, which decreases the level of population in the next generation. Hence the level of pest population is balanced by this negative feedback loop depicted between the two positive feedback loops. Since the crop is planted each year and the loss of crop biomass caused by pests has a relatively small effect on the food availability for pests, food scarcity turns out to be only a seasonal issue and this structure is not the determining feedback relation for the problem of concern.

On the other hand, pest's interactions with its predator may determine the success of a pest control strategy. As the pest population increases, food per capita for the predator population increases. Hence, the number of pests captured per predator per unit time, namely the *functional response*, increases, which means increased total predation. Increasing total predation implies a reduction in the pest population over time, through increased pest deaths. This closes the negative feedback loops (highlighted in the middle of the diagram in Figure 4.1) balancing the level of the pest population. On the other hand, with increasing food availability, predators reproduce more, attaining a higher level of population. However, increasing predator population implies a decrease in food per capita, which decreases the number of pests that can be captured per predator, completing the negative feedback loop in the southwest corner of the diagram. These counteractive feedback loops keep both pest and predator populations within limits in their natural ecology.

Yet, when a severe pest control program is applied, like in the case of planting a *Bt*-crop, it may be the case that the toxin also harms the predator of the pest species. If that is the case, a sharp decrease in the predator population will be observed for two reasons: first due to the toxin; second due to the initial drop in pest density caused by the treatment, i.e. due to food scarcity. When the number of predators decline, death rate of the pest population decreases. Moreover, since the number of pests diminish due to the toxin, food per capita for pests is increased. Then, any pest individuals that survive find themselves with a plentiful food resource but few if any natural enemies. In this case, a population outbreak is very likely to happen. This phenomenon, examples of which have been observed many times in the short history of chemical treatments, is called *target pest resurgence*. It may be observed even if the toxin does not directly affect the predator species, as long as the pest population suffers from a dramatic decline due to the treatment. Target pest resurgence very much devalues the merits of a pest control program.

4.1. Main Assumptions of the IR-Model

- There is a single pest species in the field
- There is no migration for pest and predator species
- Crop and its residue is the only food source for the pest
- The pest is the only food source of the predator
- Pest and predator species exhibit logistic growth when left alone
- Mutations are negligible

- The species mate randomly
- Resistance is expressed by two alleles of a single gene
- Heterozygote pests are entirely susceptible to toxin
- The effect of abiotic factors such as rain or wind on yield is constant, hence omitted

4.2. Overview of the Model Sectors

The model consists of primarily 3 sectors, being *Pests*, *Crop* and *Predator*. The fundamental interactions between these sectors are prey-predator type interactions. Pests feed solely on the crop and its residue, whereas predators feed solely on the pests. These interactions are the key determinants of the size of each population. Another critical interaction is the GM crop's functioning as a pesticide, potentially suppressing the pest population.

Though the aim of the study is to come up with a general assessment of Bt-crops, in order to be able to calibrate the model and build a platform for further in-depth research, it is wise to concentrate on a specific *Bt*-crop. A wide spread *Bt*-crop, *Bt-corn*, and its major pest *European corn borer* (ECB) are chosen to study.

4.2.1. Pest Sector

In this sector, the pest population is modeled. It is assumed that there is only one pest species of concern in the field.

Since the major concern of the study is to model the spread of resistance to *Bt*-toxin, the pest population is disaggregated to three sub population stocks with respect to the three genotypes RR, RS and SS (R denoting resistant, S denoting susceptible). As can be seen from the stock-flow diagram in Figure 4.2, these stocks are named as *pestRR, pestRS* and *pestSS* respectively, and have the same flow structures. The proportion of total births allocated to each genotypic group is determined through the resistance allele frequency in the population. Pest birth fraction is a function of food availability; therefore it is determined by the feedback from *crop* sector. Mortality has two components, being death due to abiotic factors (including the pesticide applications) which is denoted by *deaths* and death due to predation which is denoted by *killed*. For the former, RR genotype is resistant to the toxin and has a constant death fraction, whereas the death fraction of susceptible individuals is a function of *Bt*-toxin in land. For the latter, total predation is allocated between the genotypic groups in proportion to the frequency of each group in the population.

4.2.2. Crop Sector

In this sector, the artificial process of planting the crop is modeled; hence the structure is responsible for simulating the seeds being sown, plants being grown and being harvested at a predetermined time. The corresponding flows can be seen from the stock-flow diagram in Figure 4.2. Since pests feed on crop residue in between the consecutive planting seasons, the amount of residue is a significant parameter. Corn residue is modeled as a stock variable whose level is directly proportional to the yield and diminishes through decaying. It is assumed that the residue is cleaned at the beginning of a new planting season.

In this model, for the sake of simplicity it is assumed that there is no weed problem for the crop; hence it is only the pests that cause the yield losses.

4.2.3. Predator Sector

In this sector, the population of a predator of the pest is modeled. It is assumed that there is a single predator species and that this predator feeds solely on the pest. Predation rate of an individual predator depends on the pest density with a relation called functional response. This rate is an indicator of food availability and food availability is a determinant of predator birth fraction, which closely ties the pest and its predator producing the well known prey-predator cycles to be seen later.

In order to examine the potential for target pest resurgence, in one version of the model, predators are also assumed to be damaged by the toxin, via an interaction with the crop sector. In Figure 4.2, this case is depicted.

4.3. Description of Pest Sector

4.3.1. Background Information: *Natural Selection in Action*

A key aspect of the model is accounting for the increasing pesticide resistance problem, which in return may devalue the merits of genetic modification. The spread of pesticide resistance is due to natural selection occurring more rapidly than usual. To understand and model this phenomenon, the Hardy-Weinberg law will be reviewed and utilized.

4.3.1.1. <u>Population Genetics and the Hardy-Weinberg Law.</u> Genes contain information about a specific trait or characteristics like eye color or blood type. The alternative forms of a gene, which are called alleles, lead to alternative forms of a trait. For example, blue eyes or brown eyes are represented by different alleles of the eye color gene. Organisms have two alleles for each trait and this pair of alleles determines the genotype of the individual for that trait. If a given trait is characterized by the two alleles, R and S, the three possible genotypes are RR, RS and SS. These genotypes are observed with certain frequencies in a population.

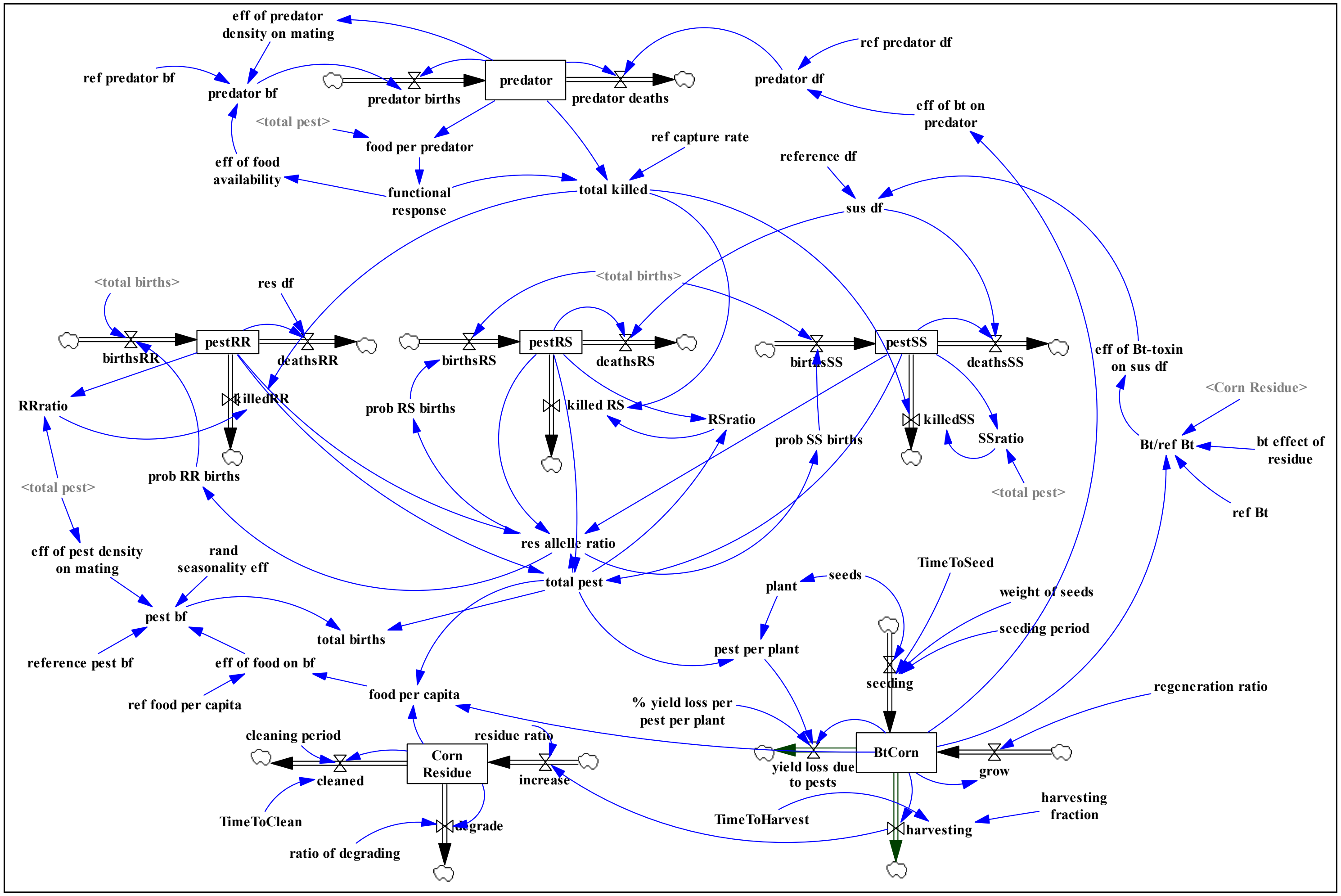

Figure 4.2. Stock-flow diagram for the IR-Mode

In order to have an idea of the genetic make up of a population in the following generations, keeping track of the changes in allele and genotype frequencies is essential. Actually, the basis of evolution is the changes in the frequencies of the alleles in a population.

Population genetics study the changes in allele and genotype frequencies in a population over time. The most fundamental model of population genetics is the *Hardy-Weinberg model*, which describes and predicts genotype and allele frequencies in a non-evolving population. The model has five basic assumptions:

- The population is large
- There is no gene flow between populations, i.e. no migration
- Mutations are negligible
- Individuals are mating randomly
- Natural selection is not operating on the population

Under these assumptions, the model depicts the genotype and allele frequencies for the next generation in the following manner. If a given trait is characterized by two alleles, R and S, the population consists of R alleles with frequency p and S alleles with frequency q where q=1-p. Given random mating, the probability of drawing two R alleles is p^2; one R and one S alleles in either order is $pq + qp = 2pq$, and two S alleles is q^2. These probabilities represent the chances of forming RR, RS and SS individuals, respectively. If the next generation consists of n individuals, the number of RR, RS and SS individuals are p^2n, $2pqn$ and q^2n respectively. Since RR individuals carry two alleles of R, RS individuals carry one and SS carry none, the total number of R alleles in this generation becomes:

$$2(p^2n) + 2pqn = 2pn \tag{4.1}$$

Similarly, the number of S genes is 2qn. The proportion of the gene pool of this generation consisting of R allele is then:

$$\frac{\text{2pn}}{\text{2pn+2qn}} = p \tag{4.2}$$

which is identical to the proportion in the parental generation. Thus allele frequencies p and q, and genotypic frequencies p^2, 2pq and q^2 do not change from one generation to another; which is known as the *Hardy-Weinberg Law*. Such a population whose genotype and allele frequencies are unchanged over successive generations is said to be in Hardy-Weinberg equilibrium. However, these conditions of Hardy-Weinberg equilibrium are hardly met, so allele frequencies do change from generation to generation.

Assumptions (2), (3) and (4) are already stated in the section Main Assumptions of the Model. The first assumption is also valid because a species is called a pest and is exposed to treatment only if it attains a considerably high population level. However, for the agricultural system of concern, the last assumption is invalid; therefore one cannot expect a genotypic equilibrium in an environment where selection pressure is consistent in the form of consistent herbicide/pesticide applications.

Yet, the mechanics of computing the proportions of each genotype for the next generation is valid as long as random mating is assured. Hence, the following formulas will be used in determining the genotype frequencies:

$$proportion\_RR = p \times p \tag{4.3}$$

$$proportion\_SS = q \times q \tag{4.4}$$

$$proportion\_RS = 2 \times p \times q \tag{4.5}$$

It is easy to show that

$$p^2 + 2pq + q^2 = 1, \quad \text{where} \quad q = 1 - p \tag{4.6}$$

4.3.1.2. Pesticide Resistance. Pesticide resistance is assumed to be characterized by a single allele of a single gene. There are two types of alleles R and S, being resistant and susceptible respectively. Each pest has two alleles, one coming from the mother and one from the father. If an individual has two R alleles, its genotype is RR, so called resistant homozygote. If it has one S and one R allele, its genotype is RS, so called susceptible heterozygote. Finally if it has two S alleles, its genotype is SS, so called susceptible homozygote.

The prevalence of evidence suggests that resistance is a recessive trait (Nelson *et al*., 2001) and current resistant management strategies depend on this fact.[2] Hence, only RR individuals are considered to be resistant to the specific *Bt* toxin. Heterozygote individuals are also not affected by the toxin if it is applied at a low dose, however most common *Bt*-crops are already inserted a high dose. Therefore, in the model, only RR individuals are assumed to be resistant.

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[2] Yet this may change from species to species and from toxin to toxin. Moreover, there are studies, which present contradictory findings. (e.g. Huang *et al.*, 1999)

It is argued that resistance typically comes with a fitness cost. Fitness cost can be defined as the possible drawbacks of increased fitness to the environment. If resistance comes with an evolutionary fitness cost, this implies that in the absence of pesticide, mortality is higher among resistant individuals than among susceptibles. One example is that an insect with a thicker shell may be less affected by a toxin than one with a thinner shell. Yet, heavier shell could make its bearer an easier prey for predators.

In the model, a fitness cost is assigned by setting the death fraction of resistant individuals to a higher value than that of the death fraction of susceptibles in the absence of *Bt*-toxin.

4.3.2. Description of the Structure

In the model, there are three stock variables associated with three genotypes of the pest population, being *pestRR*, *pestRS and pestSS*. These stocks actually represent pest densities, with the unit being pest per decar. As seen from the stock-flow diagram in 4.2, pest stocks are filled by *births* inflow and drained by *deaths* and *killed* outflows. *killed* outflow represents deaths due to predation, while *deaths* accounts for deaths due to abiotic reasons.

In this sector, if a variable (flow, auxiliary or constant) common to the three stocks is defined, genotypic information is put either as a suffix or a prefix to the variable name, such as *birthsRR* or *SSratio*. In providing the variable definitions and equations in this chapter, only those related to the *pestRR* stock will be explained when the structure is identical for the three genotypic subpopulations. Hence, unless otherwise stated, the definitions provided for *pestRR* stock will be valid for *pestRS* and *pestSS* stocks as well.

As explained in the model overview, pest population follows a logistic growth in the model. Normally, ECBs have two generations in a year with certain egg laying, larval hatch and pupating periods (OMAFRA, 1998). However for the purposes of this study, assuming that they follow a logistic growth will not affect the general behavior of the model.

Births inflow is determined by the total number of births, *total births* and the genotypic proportion of these births, *prob RR births*. That is, each pest stock receives a proportion of the total births and this proportion is determined by the associated genotypic frequencies.

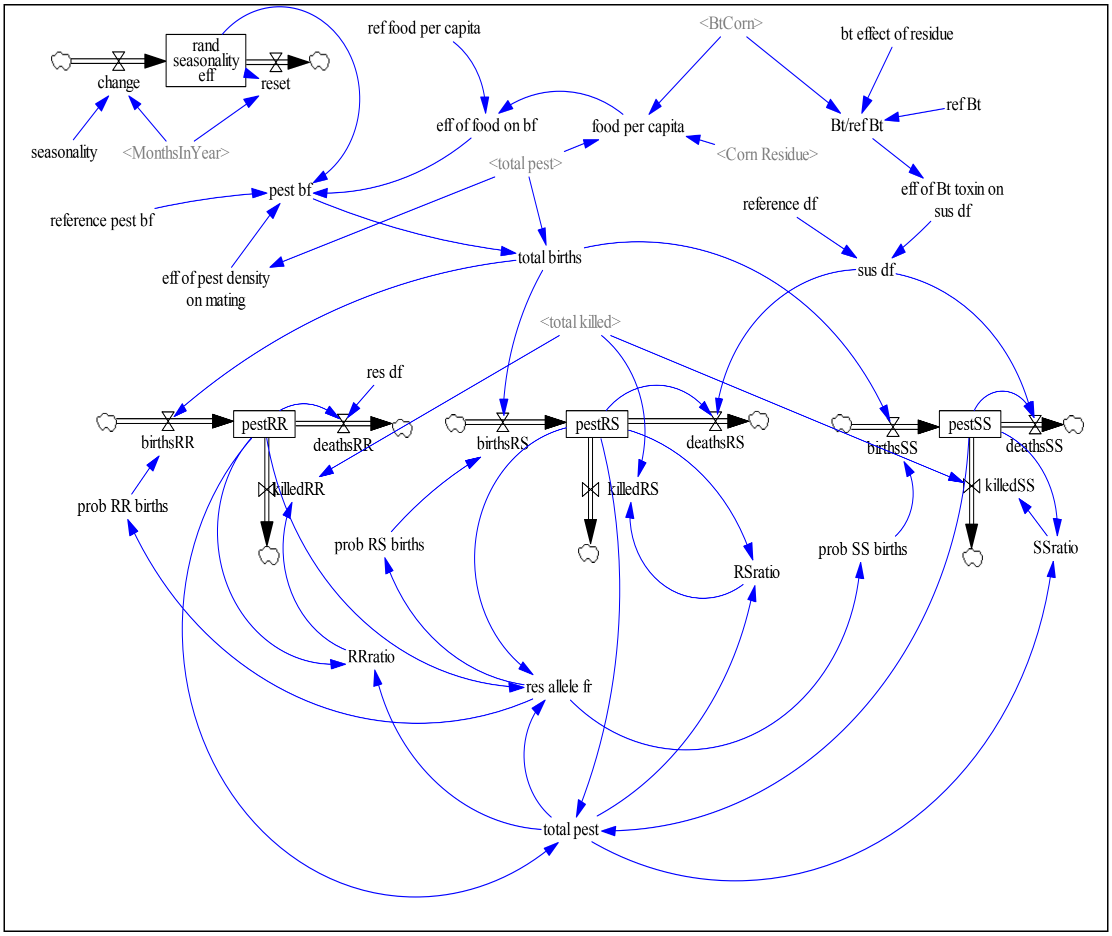


Figure 4.3. Stock-flow diagram of the Pest sector

$$birthsRR = total\ births \times prob\ RR\ births \tag{4.7}$$

Total number of births is determined by pest birth fraction, *pest bf* and the current pest population size, *total pest.*

$$total\ births = total\ pest \times pest\ bf \tag{4.8}$$

where

$$total\ pest = pestRR + pestRS + pestSS \tag{4.9}$$

The key variable in determining the genotypic frequencies is the resistance allele fraction, *res allele fr*. Each *pestRR* individual contributes two R alleles to the gene pool, whereas *pestRS* individuals only one. Given that each individual has two alleles for this trait:

$$res\ allele\ fr = \frac{2 \times pestRR + pestRS}{2 \times total\ pest} \tag{4.10}$$

As explained in the Hardy-Weinberg model, the proportion of *total births* that belong to *pestRR*, *pestRS* and *pestSS* are p^2, 2pq and q^2 respectively, where p is the *res allele fr*. In the model, these proportions are called *prob RR births*, *prob RS births* and *prob SS births* respectively.

Pest birth fraction is mainly determined by the availability of food on the land. Corn is the primary food source of ECB. It attacks many other cultivated crops such as potatoes and soybeans but since in our model the agricultural practice is monoculture and migration is not allowed, the only food source of ECB is taken to be corn. ECB overwinters on corn residue between the harvest and the following growing period. Therefore, the effect of food availability on pest birth fraction is determined by the amount of corn and corn residue on the land. Apart from the effect of food availability, *eff of pest density on mating* also affects birth fraction at very low values of pest density. Finally, to somewhat account for the fluctuations in the pest population level due to ignored factors such as climatic changes, stochasticity is added to *pest bf* through the variable *rand seasonality eff.* Hence

pest bf=

$$eff\ of\ food\ on\, bf \times reference\ pest\ bf \times eff\ of\ pest\ density\ on\, mating \times rand\ seasonality\ eff \tag{4.11}$$

The variable *eff of food on bf* is a function of relative food per capita,

$$eff\ of\ food\ on\, bf = \mathrm{f}(\frac{food\ per\ capita}{ref\ food\ per\ capita}) \tag{4.12}$$

Reference pest bf is defined to be the maximum possible birth fraction, attained when there is no scarcity of food. Therefore, the highest value of *eff of food on bf* is 1.

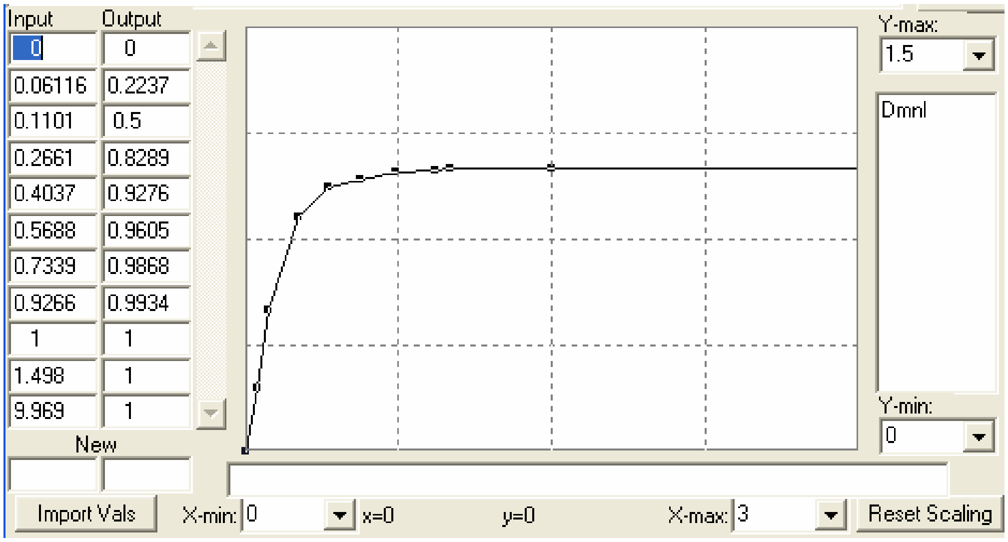
Food per capita is the amount of food per pest, which is computed by:

$$food\ per\ capita = \frac{BtCorn + Corn\ Residue}{total\ pest} \tag{4.13}$$

When there is no food, we expect that the individuals do not reproduce. Therefore when *food per capita* is 0, *eff of food on bf* is also 0. *Ref food per capita* is again a hypothetical reference value to represent the sufficiency food level. When *food per capita* reaches *ref food per capita*, the population does not suffer from food scarcity and attains its maximum birth fraction. Therefore

$$eff\ \ of\ \ food\ on\, bf\,(1)=1 \tag{4.14}$$

We expect that increasing food availability increases the birth fraction with decreasing rate, since as the food availability approaches sufficiency level, an extra unit will no longer have a significant contribution. The resulting function is depicted in Figure 4.4.



Input	Output
0	0
0.06116	0.2237
0.1101	0.5
0.2661	0.8289
0.4037	0.9276
0.5688	0.9605
0.7339	0.9868
0.9266	0.9934
1	1
1.498	1
9.969	1

Y-max: 1.5

Dmnl

Y-min: 0

X-min: 0

x=0

y=0

X-max: 3

Figure 4.4. Effect of food availability on pest birth fraction as a function of relative food per capita

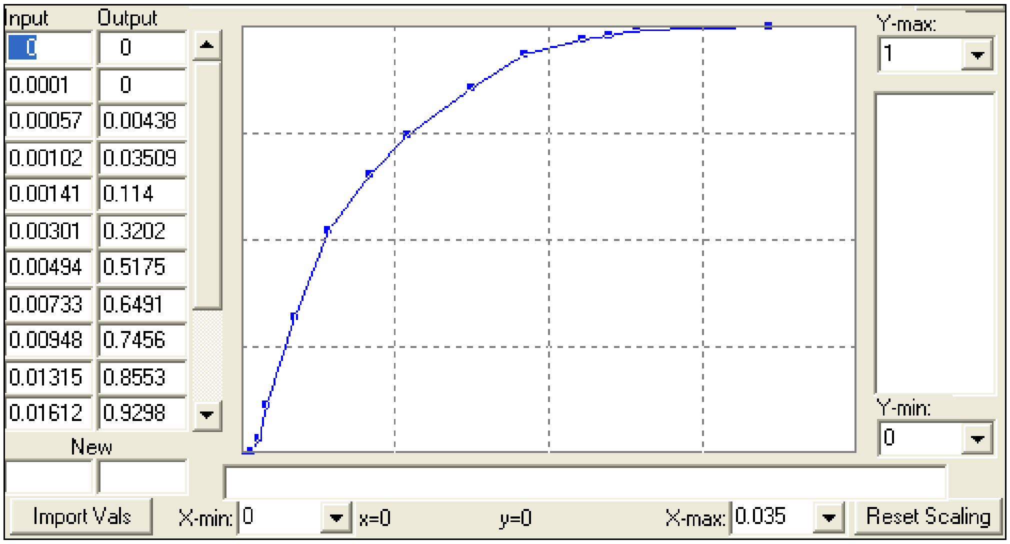
Eff of pest density on mating is a function of pest population.

$$eff\ \ of\ \ pest\ density\ on\ mating = f\,(total\ pest) \tag{4.15}$$

When pest population is asymptotically approaching zero, it is hard, if not impossible, for a pest individual to find a mate within the distance it can seek. Birth fraction is reduced under such circumstances and it is zero at the limit.

$$\mathit{eff\ \ of\ \ pest\ density\ on\ mating}(0)=0 \tag{4.16}$$

Yet, beyond a threshold predator, mating burden is surpassed and this function is ineffective on determining birth fraction.



Input	Output
0	0
0.0001	0
0.00057	0.00438
0.00102	0.03509
0.00141	0.114
0.00301	0.3202
0.00494	0.5175
0.00733	0.6491
0.00948	0.7456
0.01315	0.8553
0.01612	0.9298

Y-max: 1
Y-min: 0
X-min: 0
x=0
y=0
X-max: 0.035

Figure 4.5 Effect of pest density on mating as a function of *total pest*

Finally, *rand seasonality eff* is obtained via a pink noise named as *seasonality*. The variable takes the value of the pink noise *seasonality* at the beginning of each season and remains at the same level throughout the season. It is incorporated into the model in order to account for the possible effects of unaccounted factors such as climatic changes on the dynamics of the pest population.

Reference birth fraction can be estimated from existing data on ECB that can be found in the literature (See for example IPM at the University of Illinois, 2004). In one breeding period, an ECB female lays approximately 400 eggs. If we assume that population has equal number of females and males, and considering only the births, population in the next generation would be 200 times the population of the current generation. Since ECB has 2 generations in a year,

$$P_{t+1} = P_t \times 40000 \tag{4.17}$$

where P_t is the current population and P_{t+1} is the population in the next year.

Considering the fact that the time unit is month in our simulations, we can rewrite it as:

$$P_{t+12} = P_t \times 40000 \tag{4.18}$$

In the model,

$$P(t) = P_0 \times e^{bf \times t} \tag{4.19}$$

The following approximation can be made for bf:

$$P_0 \times e^{\mathrm{bf} \times 12} = P_0 \times 40000 \tag{4.20}$$

Therefore

$$bf \times 12 = \ln(40000) \tag{4.21}$$

$$bf = 0.88 \tag{4.22}$$

This value is used as the reference birth fraction value.

Killed outflow is determined by the total number of pests killed by predators, *total killed,* and the genotypic frequencies. The assumption is that the predators do not distinguish between the genotypes and therefore total number of killed individuals is distributed among the three stocks proportionate to the genotypic frequencies.

$$killedRR = total\ killed \times RRratio \tag{4.23}$$

Death outflow is determined by the size of the pest stock and per month death fraction associated with each stock as can be seen from Figure 4.3. *sus df* is the per month death fraction of *pestSS* and *pestRS*; whereas *res df* is the per month death fraction of *pestRR.*

$$deathsRR = pestRR \times res\ df \tag{4.24}$$

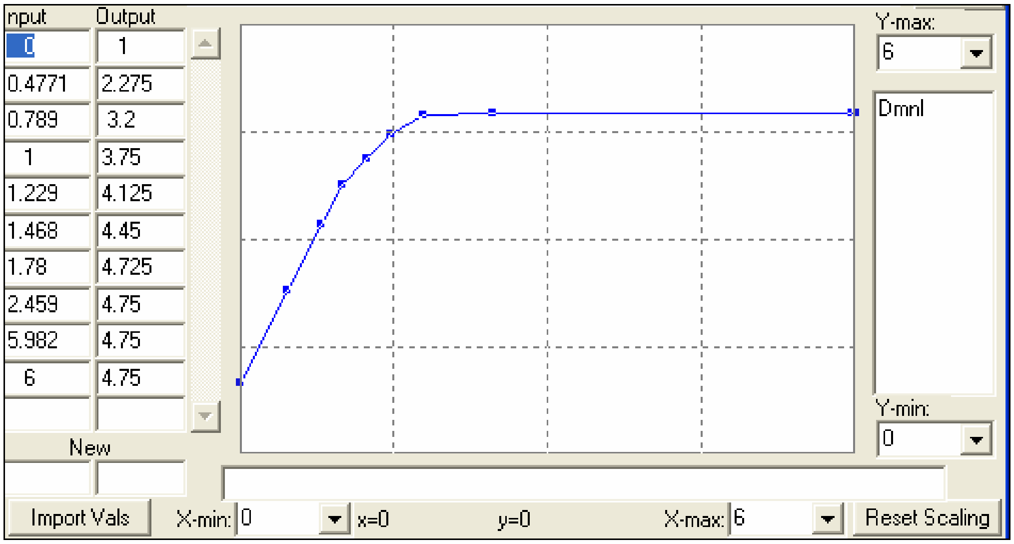
In the model, *Bt*-toxin affects the mortality fraction *sus df* of susceptible homozygote pests, *pestSS,* and susceptible heterozygote pests, *pestRS,* which is accounted for by the variable *eff of Bt toxin on sus df.*

$$sus\ df = reference\, df \times eff\ of\ Bt\ toxin \tag{4.25}$$

where sus ref df represents the per month death ratio of pests in the absence of pesticide.

The efficiency of the most effective *Bt*-crops is on average 95%, whereas conventional pesticides result in an average of 75% pest control. Using these facts, *eff of Bt toxin* is formulated as a function of toxin density *Bt/ref Bt*. The effect of increasing toxin density increases the effect on susceptible death fraction but with less and less response.

ref Bt is a hypothetical measure which represents the level of *Bt*-toxin achieved via conventional pesticides. Hence, *Bt/ref Bt* accounts for the relative power of the *Bt*-crop compared to that of conventional pesticides'. When *Bt/ref Bt*=1, the resulting mortality fraction has to be 0.75. Since *sus ref df* is estimated to be 0.2, at *Bt/ref Bt* =1, *eff of Bt-toxin on sus df* is approximately 3.75. To achieve a maximum mortality ratio of 0.95, the function values approach the asymptote, 4.75.



nput	Output
0	1
0.4771	2.275
0.789	3.2
1	3.75
1.229	4.125
1.468	4.45
1.78	4.725
2.459	4.75
5.982	4.75
6	4.75

Y-max: 6

Dmnl

Y-min: 0

X-min: 0

x=0

y=0

X-max: 6

Figure 4.6. Effect of *Bt*-toxin on susceptible death fraction as a function of Bt/ref Bt

Whether corn residue also expresses *Bt*-toxin depends on the product type. Some will be expressing the toxin in the stalks as well, while some express it throughout the harvested parts. Even if the residue expresses *Bt*-toxin, it is a lower dose than that of the harvested parts. To account for this fact, the variable *bt effect of residue* is used. The value of this hypothetical variable is

$$0< bt\ effect\ of\ residue <1 \tag{4.26}$$

Therefore *Bt/ref Bt* is formulated as

$$Bt/ref\ Bt = \frac{BtCorn + bt\ effect\ of\ residue \times Corn\ Residue}{ref\ Bt} \qquad (4.27)$$

To estimate the value for reference death fraction, we have to consider mortality due to abiotic reasons, which is reported to be on average 90%. This per year value is converted to a per month estimation by a similar approximation done for estimating *reference pest bf* and 0.2 is set to be the reference death fraction of susceptible pests, *sus ref df*.

In the literature, it is assumed that resistant pests suffer from a fitness cost of resistance. To account for that, death fraction of resistant subpopulation *res df* is set to a value higher than *sus ref df*.

4.4. Description of Crop Sector

4.4.1. Background Information: *Bt-corn*

Bt-corn varieties are developed to fight against European corn borers, the major pest of corn. Presence of the *Bt* gene in a corn hybrid does not increase yield, it only aids in preventing yield loss due to corn borers. The first *Bt*-corn hybrids were introduced by Novartis Seeds and Mycogen Seeds in 1996 and up to now several other seed companies have also incorporated this technology into their product lines. These *Bt*-corn hybrids marketed by different brands each have a gene from the bacteria *Bacillus thuringiensis* which produces a protein that kills European corn borer larvae. However, different brands have different promoter genes. The promoter gene allows the *Bt* gene to be turned on and different promoter genes may allow the *Bt*-toxin to be expressed at different times of the year or different parts of the plant. For example, one brand (YieldGard) expresses the toxin throughout the plant, being equally effective for the two generations of the pest; another (the 176 event of Knockout and Nature Gard) expresses only at certain parts and being more effective for the first generation of ECB (Bessin, 2004). Therefore, the brands do not have identical control performances. The most effective brand is reported to provide 95% average control of ECB, where insecticides provide on average 75% control. For the scope of this study, it is convenient to consider a high-dose effectiveness and perform sensitivity analysis on control effectiveness of the *Bt*-corn.

4.4.2. Description of the Structure

Life cycle of a crop is roughly composed of three stages: sowing, growing, harvesting. Seeds are sown at a predetermined amount which guarantees the intended plant population to a degree. Sowing occurs at a predetermined period, which may change from region to region. Growth of a crop is limited by the carrying capacity of the area for that crop and the yield is also affected by other stress factors such as pests and unfavorable weather conditions. The crop is harvested at a predetermined harvest period as the crop reaches maturity. The model tries to mimic these stages in corn's life cycle, assuming that the effects of climatic factors are constant. Corn biomass is represented by the *BtCorn* stock. As can be seen from the stock-flow diagram in Figure 4.7, *BtCorn* is filled with *seeding* and *growing* inflows, and drained by *harvesting* and *yield loss due to pests* outflows. The accumulation is in terms of biomass and has the unit kg/da. Therefore the related flows have the unit kg/(da*Month).

Seeding rate is determined by *seeds,* which is a constant denoting the required number of seeds per area to achieve the intended plant population, and weight of these seeds *weight of seeds*. This inflow is only activated at the predetermined dates of seeding, which is achieved by the binary variable *TimeToSeed*. Finally, *seeding period* represents the necessary time for the seeding process to be completed. Therefore:

$$seeding = \frac{seeds \times weight\ of\ seeds \times TimeToSeed}{seeding\ period} \tag{4.28}$$

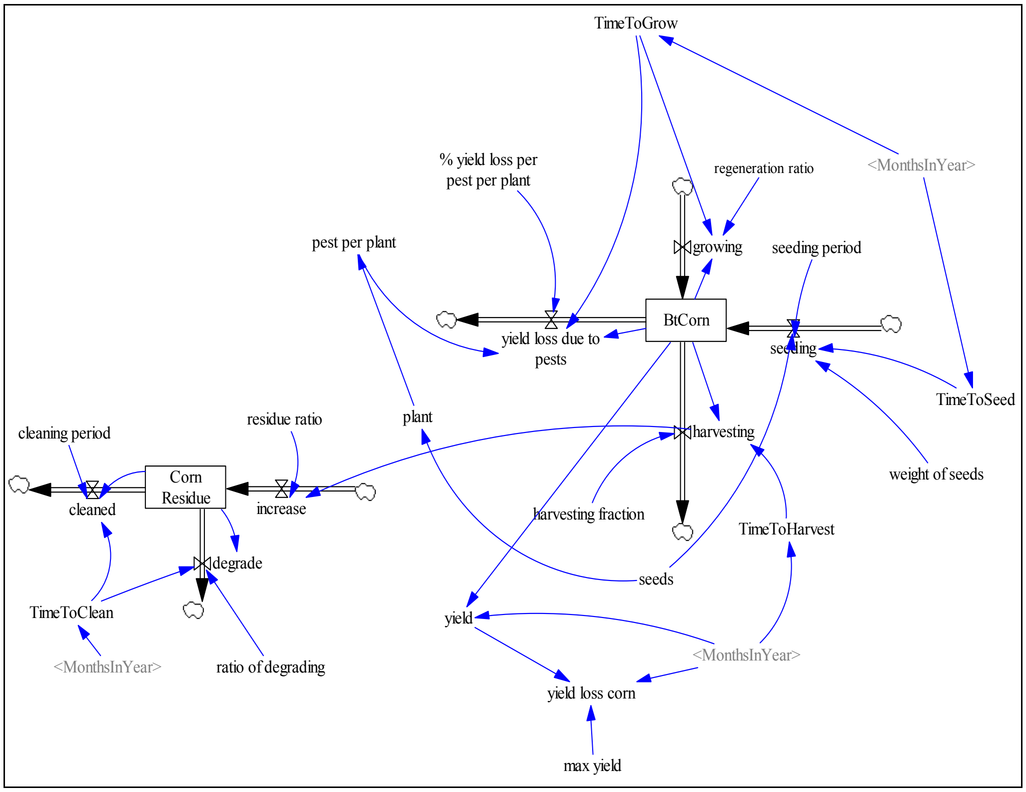


Figure 4.7. Stock-flow diagram of the Crop sector

Sowing and harvesting dates are approximated from the report of USDA Agricultural Statistics Board on Usual Planting and Harvest Dates for US Corn (USDA, 1997). Parameter values for *seeds*, *weight of seeds* and *plant* are estimated from Iowa State University Corn Planting Guide (Iowa State University Department of Agronomy, 2001).

Growing rate is determined by *regeneration ratio*, which is per month growth ratio of the plant, and the biomass of the plant, *BtCorn* and lasts throughout the period determined by *TimeToGrow*.

$$growing = BtCorn \times regeneration\ ratio \times TimeToGrow \qquad (4.29)$$

In the model, regeneration ratio is experimentally determined and set to a value which guarantees the expected corn yield in the absence of pests.

Harvest is the complete draining of the *BtCorn* stock. It occurs at a predetermined time period, which is modeled by the binary variable *TimeToHarvest* and with per month draining fraction of *harvesting fraction.*

$$harvesting = BtCorn \times TimeToHarvest \times harvesting\ fraction \tag{4.30}$$

Yield loss due to pests is determined by *BtCorn* biomass, *% yield loss per pest per plant* and *pest per plant*.

$$yield\ loss\ due\ to\ pests = BtCorn \times \%yield\ loss\ per\ pest\ per\ plant \times pest\ per\ plant \tag{4.31}$$

Pest per plant is computed as:

$$pest\ per\ plant = \frac{total\ pest}{plant} \tag{4.32}$$

% yield loss per pest per plant is reported to be around 5% in several sources (Such as Sloderberg, 1997; OMAFRA, 1998). A monthly estimate for it is 0.014, which is used as the parameter value for *% yield loss per pest per plant.*

At harvest corn stalks and cobs are left out in the farm land. This is the primary habitat and food source for ECB between the two growing cycles of corn. As can be seen from the stock-flow diagram in Figure 4.7, the amount of residue left is represented by the stock variable *Corn Residue.* When the harvest is completed, this stock is filled immediately by the *increase* inflow. The residue remains in land till the following sowing period, at which time it will be cleaned out totally. However in the meantime, much of the residue already degrades. These phenomena are modeled via *clean* and *degrade* outflows respectively.

Increase inflow is determined by the *harvest* outflow of *BtCorn* and the *residue ratio* which represents the proportion of the plant not harvested. In the literature harvest indices for crops are reported, which represent the quantity of harvestable biomass per unit total biomass produced. *residue ratio* can be thought as (1 - Harvest index). For corn, harvest index is reported to be on average 0.5, which makes *residue ratio* 0.5 as well. The equation becomes:

$$increase = harvesting \times residue\ ratio \tag{4.33}$$

Degrade outflow is determined by the size of the residue stock *Corn Residue*, and the fraction of degrading residue per time, *ratio of degrading*.

$$degrade = Corn\ Residue \times ratio\ of\ \ degrading \tag{4.34}$$

Cleaned outflow is determined by the size of the residue stock *Corn Residue*, and the *cleaning period.* Since cleaning occurs right before the seeding period of the following year, the flow is activated by the variable *TimeToClean*.

$$cleaned = \frac{Corn\ Residue \times TimeToClean}{cleaning\ \ period} \tag{4.35}$$

4.5. Description of Predator Sector

4.5.1. Background Information: *Functional Response*

Functional response of predators to prey availability is defined as all the predator's behaviors that determine how many prey it will capture per unit time. It is measured in number of prey captured/predator/time.

The predation rate of predators is in general influenced by prey availability per predator. Typically, as prey population grows, predation rate increases but with less and less response and finally approaches a limit asymptotically as seen in Figure 4.8.

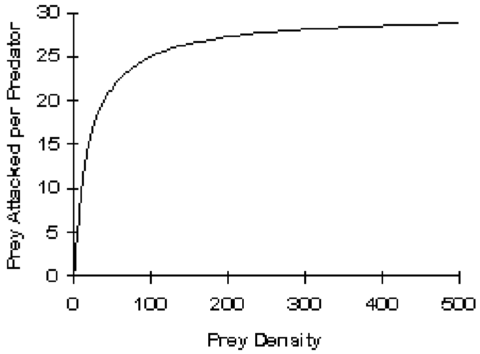


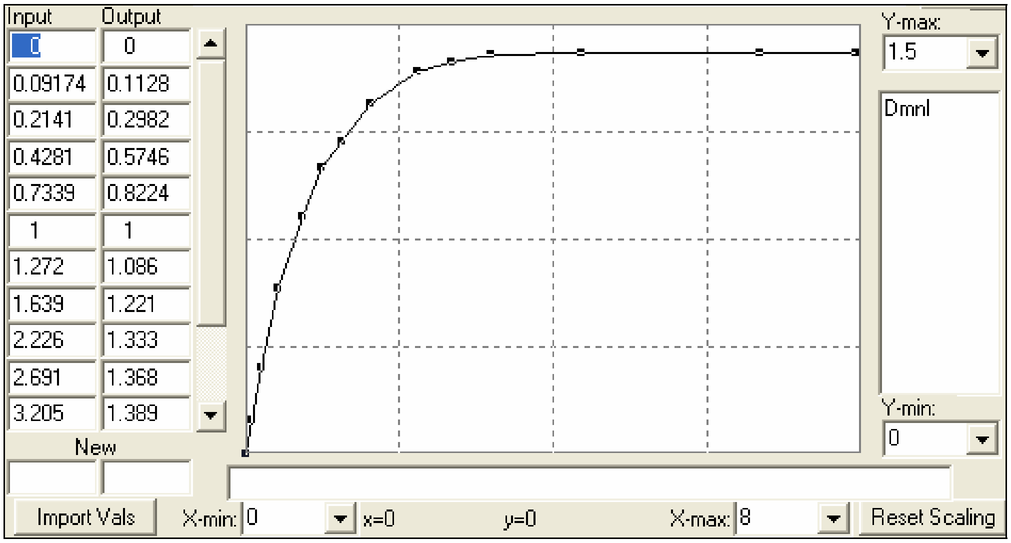
Figure 4.8. An example functional response curve

In the model, *functional response* is modeled slightly different in order to obtain a more realistic formula. First, rather than directly giving the number of preys attacked by the predator, the function relates the values to a reference number of preys attacked, i.e. states what percent of the reference value, *ref capture rate*, is attacked per predator per time. Second, it is a function of pest per predator, rather than the pest population.

$$\textit{functional response} = \mathrm{f}\left(\frac{\textit{pest per predator}}{\textit{ref pest per predator}}\right) \qquad (4.36)$$

where *ref pest per predator* represents the normal level of per predator food availability.

When there is no pest, *pest per predator* becomes null. Hence, no pests can be attacked and *functional response* returns 0. When *pest per predator* is equal to *ref pest per predator*, number of pests captured per predator per time in unit land, must be at its reference value, *ref capture rate.* Hence *functional response* returns 1.



Input	Output
0	0
0.09174	0.1128
0.2141	0.2982
0.4281	0.5746
0.7339	0.8224
1	1
1.272	1.086
1.639	1.221
2.226	1.333
2.691	1.368
3.205	1.389

Y-max: 1.5

Dmnl

Y-min: 0

X-min: 0

x=0

y=0

X-max: 8

Figure 4.9. Functional response as a function of normalized pest per predator

Therefore total number of killed pests per unit time per unit land is calculated as:

$$\mathit{total\ killed} = \mathit{functional\ response} \times \mathit{ref\ \ capture\ rate} \times \mathit{predator} \tag{4.37}$$

4.5.2. Description of the Structure

In the model, it is assumed that predators exhibit logistic growth. As can be seen from the stock-flow diagram of this sector in Figure 4.10, the population of predators is represented by the *Predator* stock. *Predator* is filled by *predator births* and drained by *predator deaths*.

Predator births inflow is determined by the size of the predator population *Predator,* and predator birth fraction, *predator bf.*

$$\mathit{predator\ births} = \mathit{Predator\ \ density} \times \mathit{predator\ bf} \tag{4.38}$$

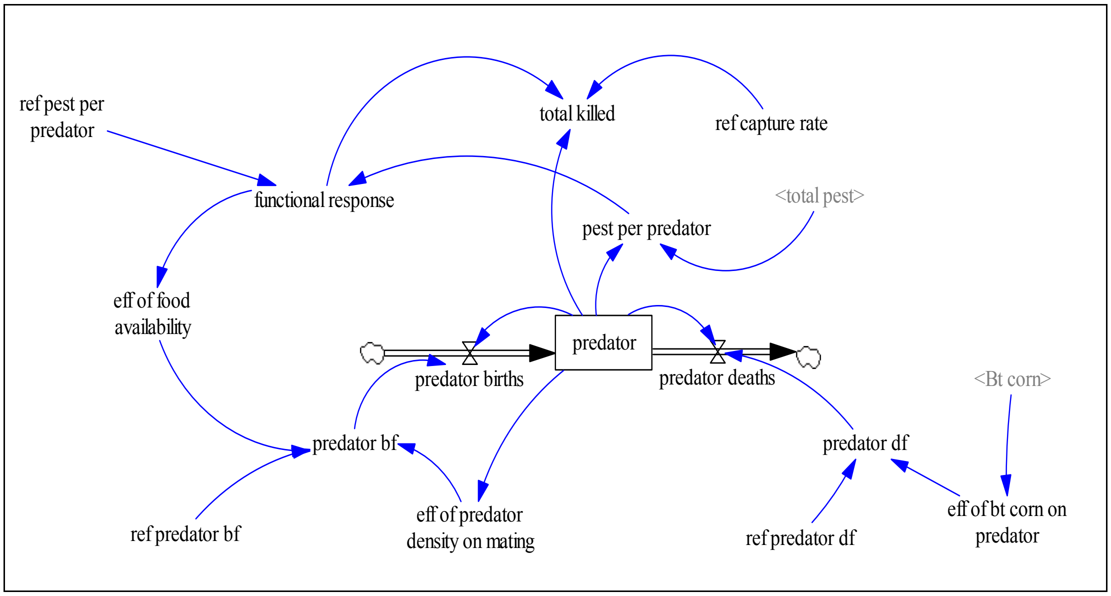


Figure 4.10. Stock-flow diagram of the Predator sector

Predator bf formulation is density dependent and it constitutes another component of the tie between the predator and the pest population. *predator bf* is determined by food availability per predator and the effect of current predator on mating.

$$predator\ bf\ =$$

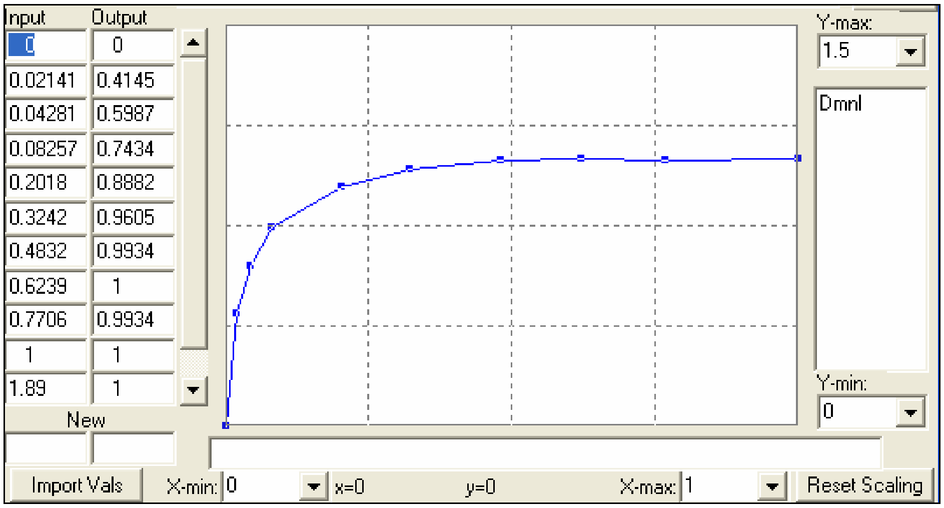
$$ref\ \ predator\ bf \times eff\ \ of\ \ food\ availability \times eff\ \ of\ \ predator\ \ density\ \ on\ \ mating \tag{4.39}$$

Eff of food availability is a function of *functional response*.

$$eff\ \ of\ \ food\ \ availability = f\,(\,functional\ \ response) \tag{4.40}$$

By considering food per capita instead of aggregate food availability, competition between predator individuals is also taken into consideration. When food per capita is lower than the sufficiency level *ref food per capita*, population tends to limit reproduction.

When there is no food, i.e. when the number of pests asymptotically approaches zero, *eff of food availability* returns 0. When food per capita increases, birth rate tends to increase. When food per capita is equal to its reference level, making *functional response* 1, *eff of food availability* returns 1, so that the reference birth fraction is attained. With further increasing functional response, birth fraction continues to increase and then asymptotically reaches the maximum possible birth fraction.



Input	Output
0	0
0.02141	0.4145
0.04281	0.5987
0.08257	0.7434
0.2018	0.8882
0.3242	0.9605
0.4832	0.9934
0.6239	1
0.7706	0.9934
1	1
1.89	1

Y-max: 1.5

Dmnl

Y-min: 0

X-min: 0

x=0

y=0

X-max: 1

Figure 4.11. Effect of food availability on predator birth fraction as a function of functional response

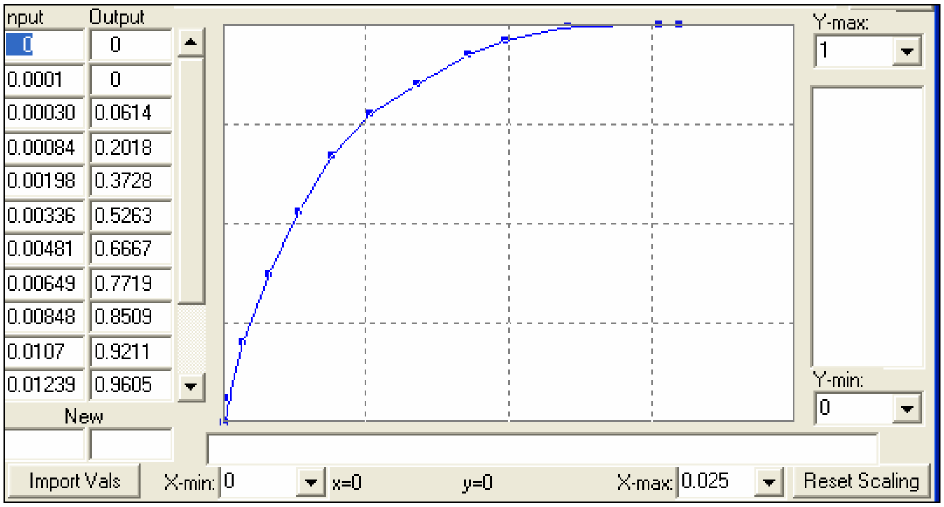
Eff of predator on mating is a function of predator population.

$$eff\ \ of\ \ predator\ density\ on\ mating = f\,(Predator) \tag{4.41}$$

When predator is asymptotically approaching zero, it is hard, if not impossible, for a predator individual to find a mate within the distance it can seek. Birth fraction is reduced under such circumstances and it is zero at the limit.

$$eff\ \ of\ \ predator\ density\ on\ mating(0) = 0 \tag{4.42}$$

Yet, beyond a threshold predator, mating burden is surpassed and this function is ineffective on determining birth fraction.



nput	Output
0	0
0.0001	0
0.00030	0.0614
0.00084	0.2018
0.00198	0.3728
0.00336	0.5263
0.00481	0.6667
0.00649	0.7719
0.00848	0.8509
0.0107	0.9211
0.01239	0.9605

Y-max: 1
Y-min: 0
X-min: 0
x=0
y=0
X-max: 0.025

Figure 4.12 Effect of predator on mating as a function of *Predator*

Predator deaths outflow is determined by the size of the predator population, *Predator*, and the per month predator death fraction, *predator df*.

$$Predator\ deaths = Predator \times predator\ df \tag{4.43}$$

There are two scenarios related to the determination of *predator df*: Predators may or may not be affected by the *Bt*-toxin in land. In the model diagram, the first case is shown. If *Bt* toxin does not affect the predator species, then predator death fraction is equal to the reference death fraction, *ref predator df*. If it does, it is assumed that *Bt*-corn increases *predator df* by the factor *eff of bt on predator*.

$$predator\ df = eff\ \ of\ \ bt\ on\ predator \times ref\ \ predator\ df \tag{4.44}$$

where *ref predator df* is the normal death fraction in the absence of the effect of toxin.

Since the mechanics of this type of a detrimental effect has not been revealed, the increase in the death fraction is linked directly to the presence of *Bt*-corn in land as the only food source for the pest species. The following formula guarantees that when *Bt*-corn is planted, *predator df* is increased by 30% and when it is not, *predator df* stays the same.

$$Eff\ \ of\ \ bt\ corn\ on\ predator = IFTHENELSE(BtCorn > 0{,}1.30{,}1) \qquad (4.45)$$

5. ANALYSIS AND VALIDATION OF THE IR-MODEL

In order to analyze the system behavior, the model described in the previous chapter is simulated via Vensim DSS software. Simulation time unit is chosen to be months and a sufficiently small value of time step (1/32) is used. Since some of the impacts are evident only in the long run, time horizon of the study is set to 500 months. In this chapter, base behavior of the IR-model will be analyzed and the results of the experiments conducted in order to test the validity of the IR-model are summarized.

5.1. Analysis of the Base Behavior

The base behavior of the model depicts the long term behavior of the system in case the conditions provided in the model description persist. These dynamics constitute a reference in interpreting the consequences of the sensitivity and policy runs. In this section base behavior of the IR-model is demonstrated through dynamic behaviors of selected variables. In order to provide a better understanding of the system, a comparison to the behavior obtained from planting conventional corn is also provided (see Appendix C.2 for the modified model description) Finally, the assumption of predators' not being adversely affected by *Bt*-corn is challenged in the last subsection and the obtained results are compared to the base behavior as well as the dynamics of the conventional corn system.

5.1.1. Base Behavior of the Model

The fundamental behavior of the model is the spread of resistance in the pest population, which is depicted by the graph of *res allele ratio*. Under the consistent selection pressure selecting out resistant individuals, this ratio exhibits an S-shaped growth. When resistance allele ratio exceeds 0.5, the complete development of resistance comes about almost immediately. Therefore this level is accepted to be a critical level.

When Figure 5.1 is investigated, it can be seen that this level is reached at t=123 and it only takes approximately 10 more months till the population is entirely resistant.

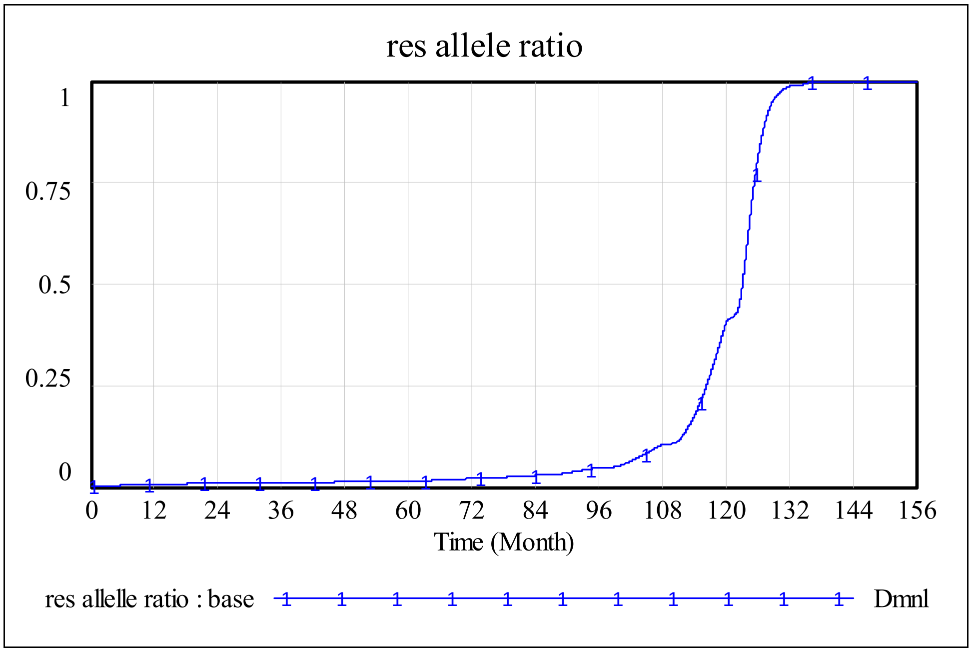


Figure 5.1. Dynamics of pest resistance allele frequency in the base run

When the graph of *total pest* is analyzed (Figure 5.2), it is seen that due to the high control efficiency of *Bt*-corn, the pest population suffers from a sharp decline, which is followed by a decline in the predator population (Figure 5.3). Decrease in its natural enemies results in a rise in the pest population and it starts to fluctuate within acceptable levels (below one pest per plant). Until the 125th period, level of the population is away from being critical. However, after resistance is spread, *Bt*-corn loses its effectiveness as a pest control means and a pest infestation occurs, which is slightly alleviated by the resulting increase in the predator population. One important point is that transition to this infestation state is sharp, i.e. the farmer may spend an almost pest-free year and then the next year may face with an infestation.

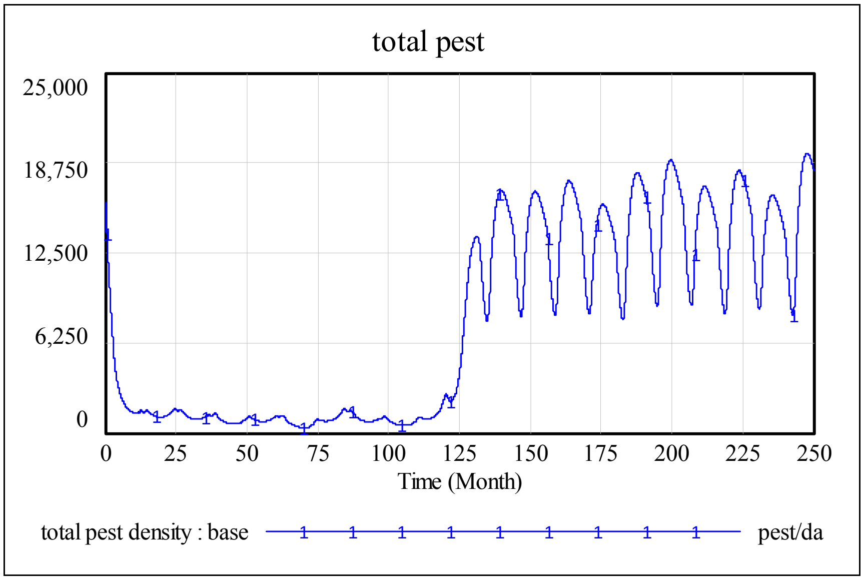


Figure 5.2. Dynamics of pest population in the base run

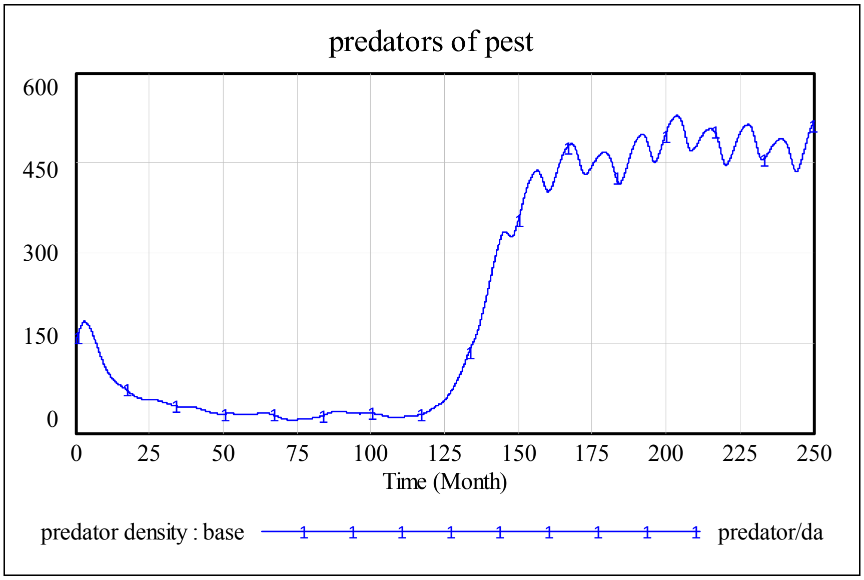


Figure 5.3. Dynamics of predator population in the base run

Homozygote susceptible subpopulation (*pestSS)* gradually diminishes first due directly to *Bt*-toxin, and then due to decreasing frequency of susceptibility allele. On the other hand, *pestRS* whose initial level is very low first increases due to increasing resistance allele frequency, then decreases due to decreasing susceptible allele frequency. When resistance allele approaches unity, both susceptible subpopulations go extinct.

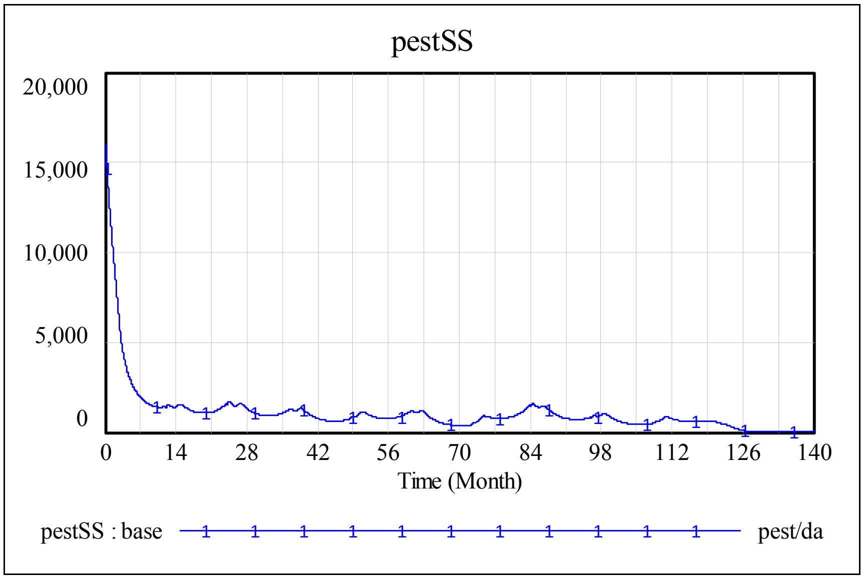


Figure 5.4. Dynamics of homozygote susceptible (SS) pest population in the base run

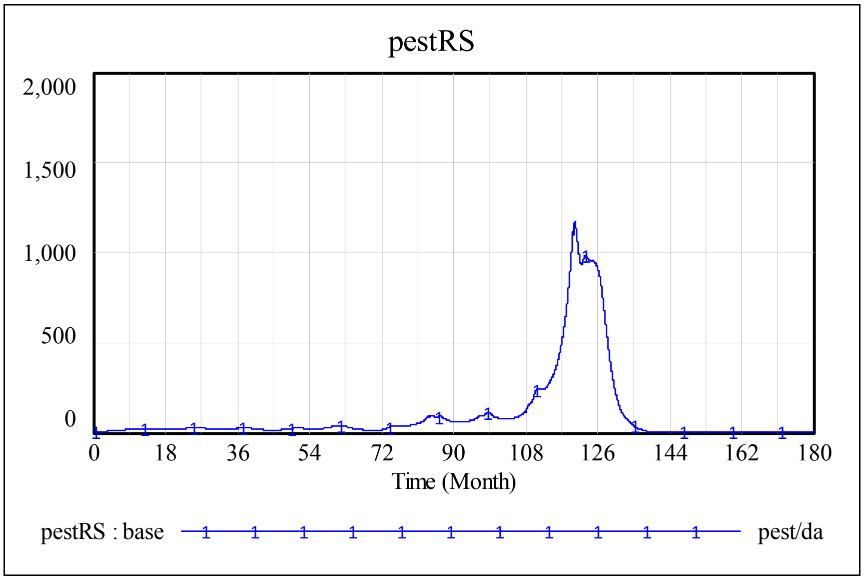


Figure 5.5. Dynamics of heterozygote susceptible (RS) pest population in the base run

What is faced meanwhile by the producer is revealed by the yield loss graph (Figure 5.6). When pest population is controlled successfully by *Bt*-corn, yield losses are small. Yet, after the spread of resistance, they reach very critical levels.

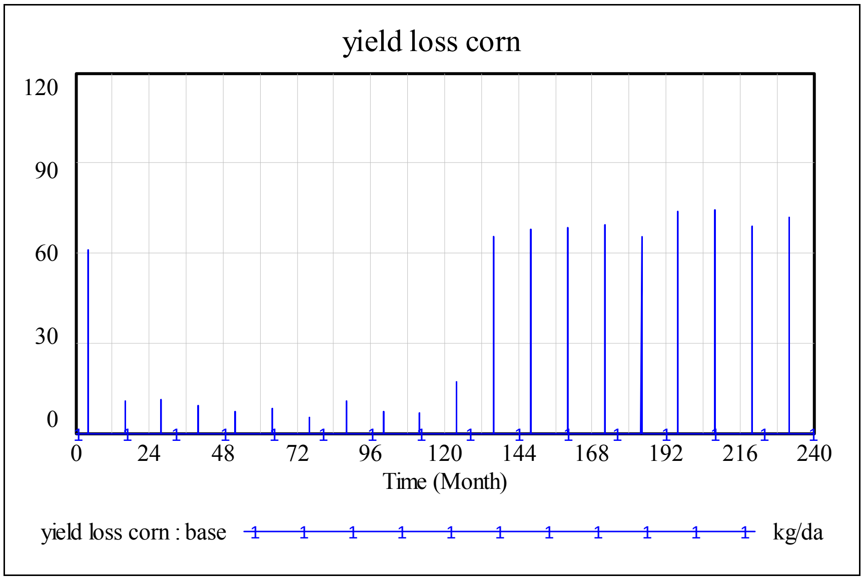


Figure 5.6. Corn yield loss due to pests in the base run

5.1.2. Comparison with Planting Conventional Corn

In order to comprehend the above results, one needs to assess what would happen if conventional corn is planted instead of a *Bt*-corn variety (see Appendix C.2 for modified equations). If the conventional corn is not treated with conventional spray pesticides, pest population is seen to fluctuate around an equilibrium level (Figure 5.7). The fluctuation is due to the random effect of seasonal variations, such as climatic changes, accounted by the variable *rand seasonality eff*. Note that the equilibrium level of the pest population is the same equilibrium level attained after the spread of resistance in the base run. Hence, this level represents the level of pest population where no effective pest control strategy is applied.

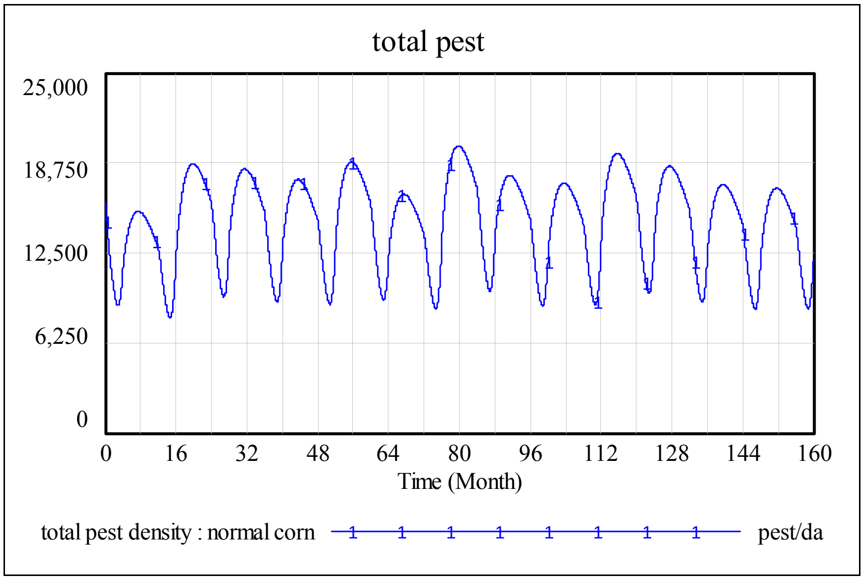


Figure 5.7. Dynamics of pest population when conventional corn is planted and not treated with pesticide

Corn is usually treated with pesticides[3]. It can be seen from Figure 5.8 that using a *Bt*-pesticide that kills on average 75% of the susceptible pests twice a season regardless of the pest density is not a treatment as efficient as *Bt*-corn. However, since in this case spread of resistance is very slow compared to *Bt*-corn treatment (Figure 5.9), this relatively low efficiency is sustained for a longer period. Hence, as seen in Figure 5.10 long run cumulative yield losses are lower than those obtained when *Bt*-corn is planted, if the structure is preserved for both cases. Assuming that a pesticide or Bt-plant to which pests gained resistance will stay in use is unrealistic. However, the point that is tried to be made via this analysis is that comparative benefits of using *Bt*-corn decreases and the need for an alternative product rises in relatively short periods.

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[3] Though not always, due to the poor effectiveness of spray pesticides on ECB. (Benbrook, 2001)

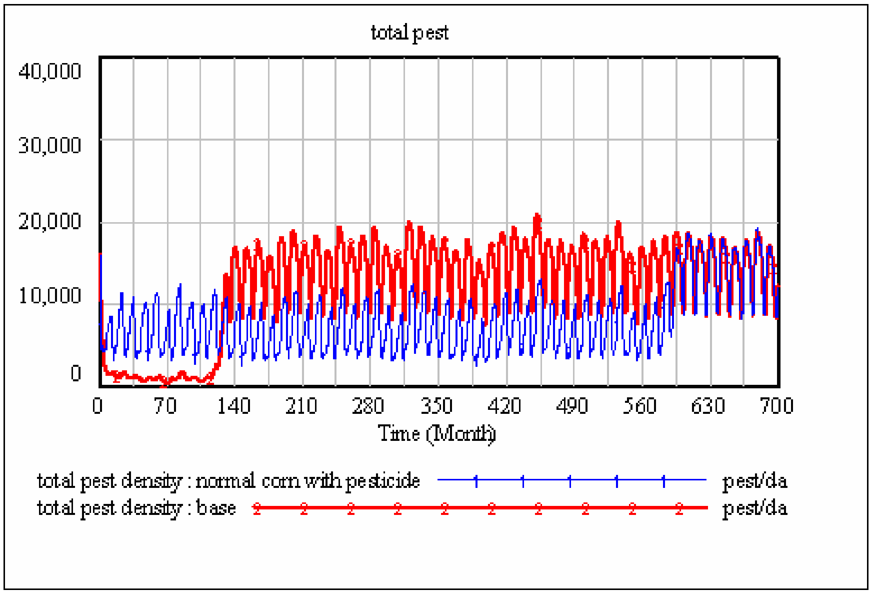


Figure 5.8. Comparison of pest population dynamics corresponding to *Bt*-corn and *Bt*-pesticide treatments

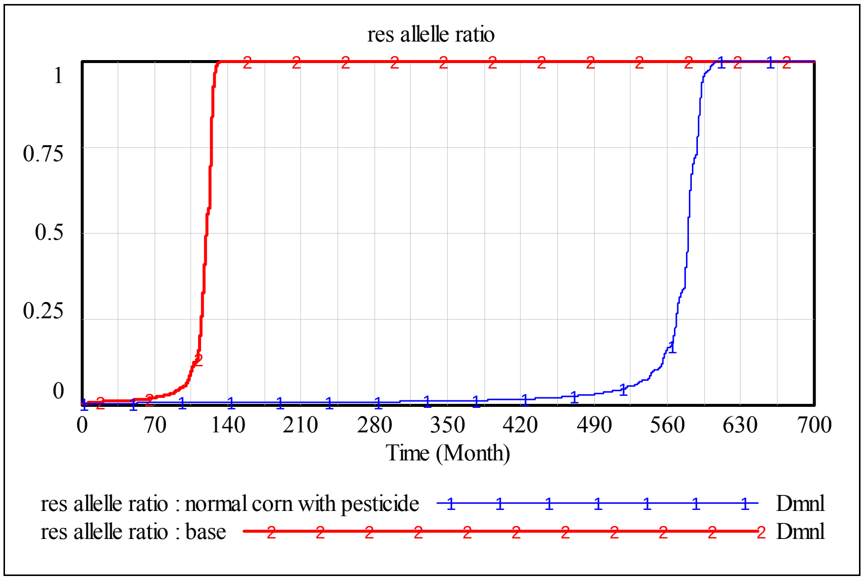


Figure 5.9. Comparison of resistance allele ratio dynamics corresponding to *Bt*-corn and *Bt*-pesticide treatments

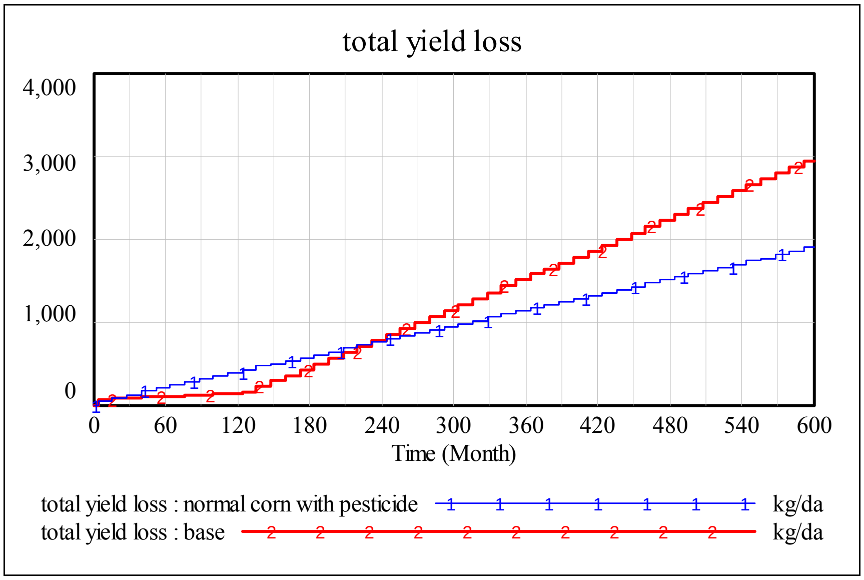


Figure 5.10. Comparison of cumulative yield losses corresponding to *Bt*-corn and *Bt*-pesticide treatments

5.1.3. Base Behavior When Predators are Also Harmed by Bt-corn

Mortality among predators may increase with either directly or indirectly exposure to *Bt*-toxin, the latter being through feeding on pests which were exposed to the toxin. Hilbeck *et al* showed that green lacewings, which are beneficial predatory insects, had a higher death rate and delayed development when fed European corn borers which had eaten *Bt*-corn compared with lacewings fed borers given non-*Bt* corn (Hilbeck *et al*., 1998). However, there are some other studies which claim that there is no significant difference in the mortality fractions (See for example Pilcher *et al.*, 1997). If the death rate of the predator increases due to *Bt*-corn, then the predator species may obtain a lower equilibrium level and may even go extinct on the farm scale. These result in intensified pest infestations as well as loss of farm land biodiversity in the latter case. Since the effect of *Bt*-corn on these predators is unclear but critical, base dynamics will be analyzed considering both possibilities.

In this setting, planting *Bt*-corn causes a considerable increase (35%) in the predator mortality fraction. Like in the first case, pest population suffers a dramatic decline due to the effective control by *Bt*-corn. Having been deprived of its unique food source and being also damaged by the toxin, predator population goes to farm-scale extinction (Figure 5.11).

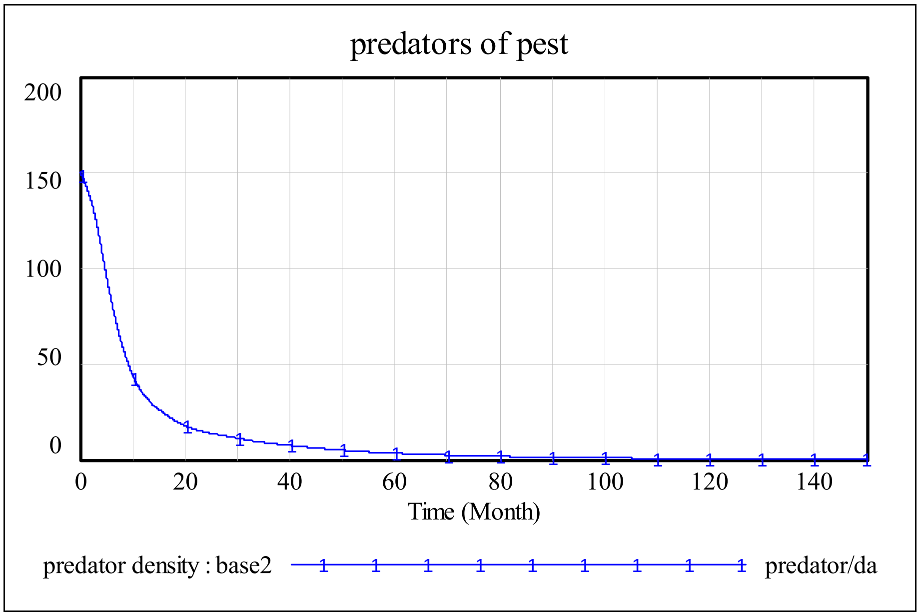


Figure 5.11. Dynamics of predator if predators are assumed to be harmed by *Bt*-toxin

Loss of its natural enemies results in a rise in the pest population and it starts to fluctuate within acceptable levels (below one pest per plant). However, when resistance is spread throughout the population, a sudden pest outbreak is observed.

Since the population eliminated two major causes of mortality, being *Bt*-toxin and predators, the infestation is more severe as seen in Figure 5.12. In this setting *Bt*-corn loses effectiveness in less than 11 years if used consistently in spite of the low initial resistance allele ratio. Corresponding yield losses can be seen in Figure 5.13.

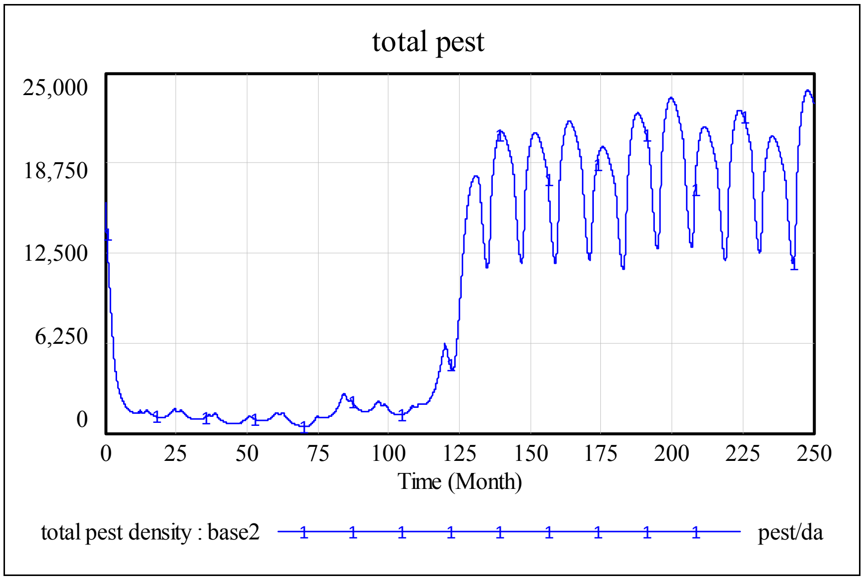


Figure 5.12. Dynamics of pest population if predators are assumed to be harmed by *Bt*-toxin

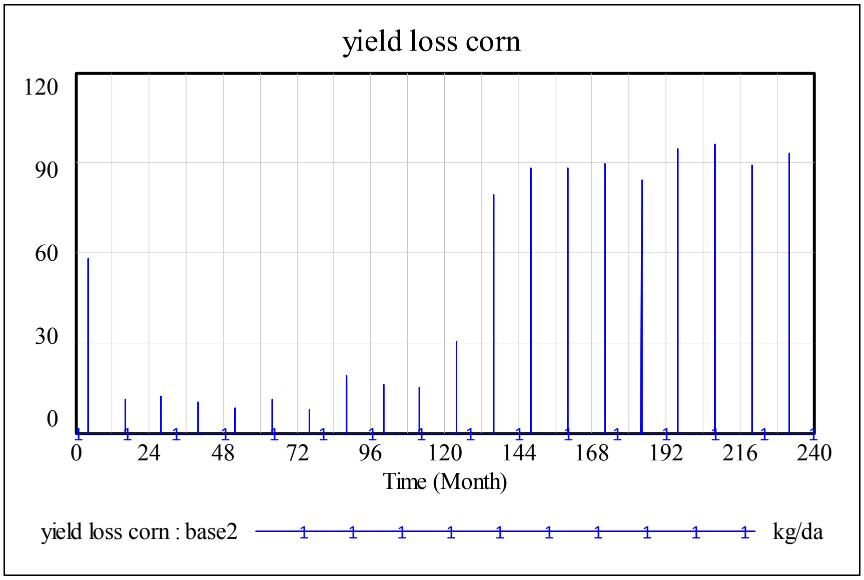


Figure 5.13. Corn yield loss if predators are assumed to be harmed by *Bt*-toxin

In the figure below, dynamics of the pest population in the two cases are compared. The unfavorable impact of target pest resurgence due to farm scale extinction of predators can easily be seen. Pest infestation in the second case is much more severe than that in the base run, which implies higher yield losses. This result is also depicted in the cumulative yield loss graph in Figure 5.15.

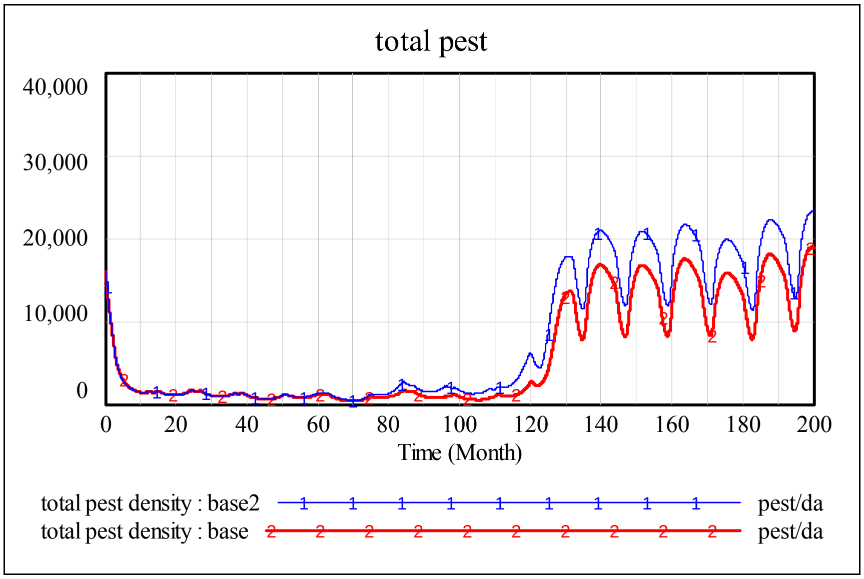


Figure 5.14. Comparison of pest population dynamics when predators are assumed to be harmed by *Bt*-corn and the base run

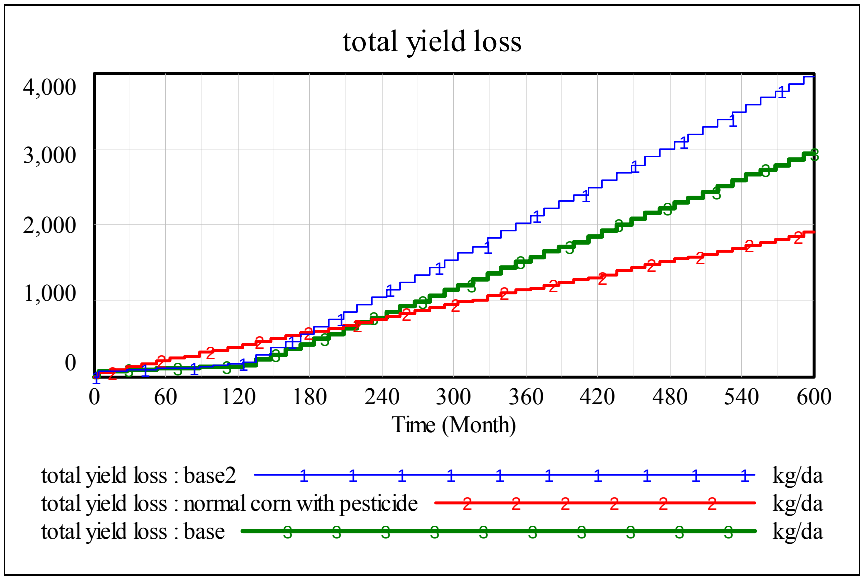


Figure 5.15. Comparison of cumulative yield losses in the two cases of the *Bt*-corn treatments and in Bt-pesticide treatment

Though not modeled in this study, it is easy to show that if *Bt*-crop damages the predator of a potential pest species, whose level is kept in control mainly by this predator, the species may emerge as a pest species due to the very same mechanisms. Hence, before releasing *Bt*-crops into the market, such non-target effects have to be tested thoroughly to avoid these undesirable effects. The incident with Monarch butterflies is a good example of how loose the control mechanisms have been.

5.2. Validation of the Model

In this section, the results of the experiments conducted in order to test the validity of the IR-model are summarized. Since system dynamics models are essentially built to design and test different policy alternatives in order to improve system behavior, the internal structure of the model has to represent the real system adequately with respect to the purpose of the model. Hence validation of a system dynamics model primarily concentrates on the internal structure of the model with the ultimate goal of "obtaining the right behavior for the right reasons" (Barlas, 1996). A formal validation process has to be performed in order to detect structural flaws in the model. Once the analyst is convinced that the structure is valid, tests for behavioral accuracy can then be performed.

As mentioned in the methodology section, validation process is very much integrated to model building process, rather than being a separate stage after the model is built. Especially, direct structure tests, which are basically direct comparisons of the model structure with the knowledge about the real system of concern, are performed concurrently with model building. Due to the lack of data for the dynamics of pest and predator populations, it will not be possible to perform rigorous behavioral validation; hence model validation is primarily demonstrated on the basis of structure tests invoking extreme condition and behavior sensitivity tests.

5.2.1. Extreme Condition Tests

In extreme condition tests, certain variables are assigned extreme values in order to check if the model exhibits the anticipated dynamic behavior that would be exhibited by the real system under that extreme condition. The reason behind testing the model at its extremes is that it is much easier to anticipate the behavior of large-scale complex systems under extreme conditions. By comparing the anticipated behavior with the results generated by the model, the modeler can catch the structural errors in the model.

5.2.1.1. No Corn is Planted. When corn is not planted, pests are expected to go extinct on the farm scale, since corn is assumed to be their unique food source. Likewise, when pests go extinct, predators will go extinct as well. These anticipated behaviors are generated by the model as can be seen from Figure 5.16 and Figure 5.17.

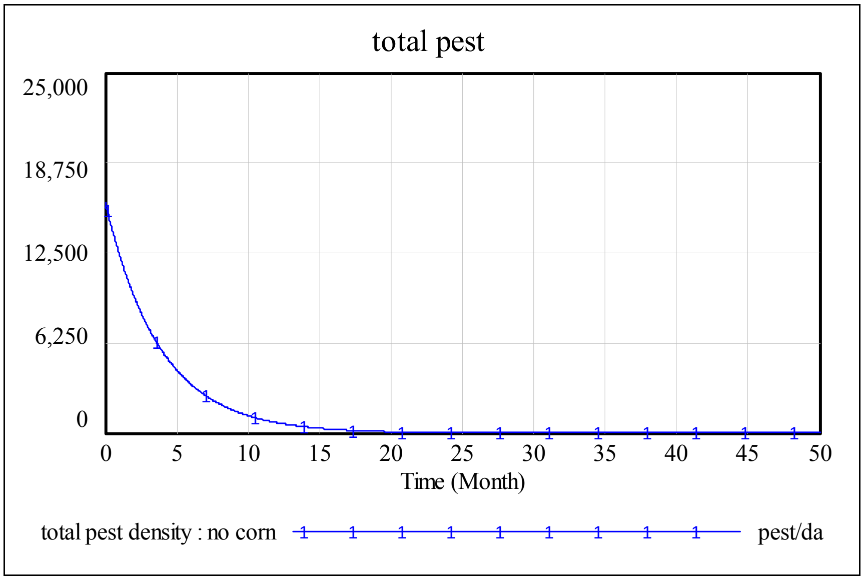


Figure 5.16. Dynamics of the pest population when corn is not planted

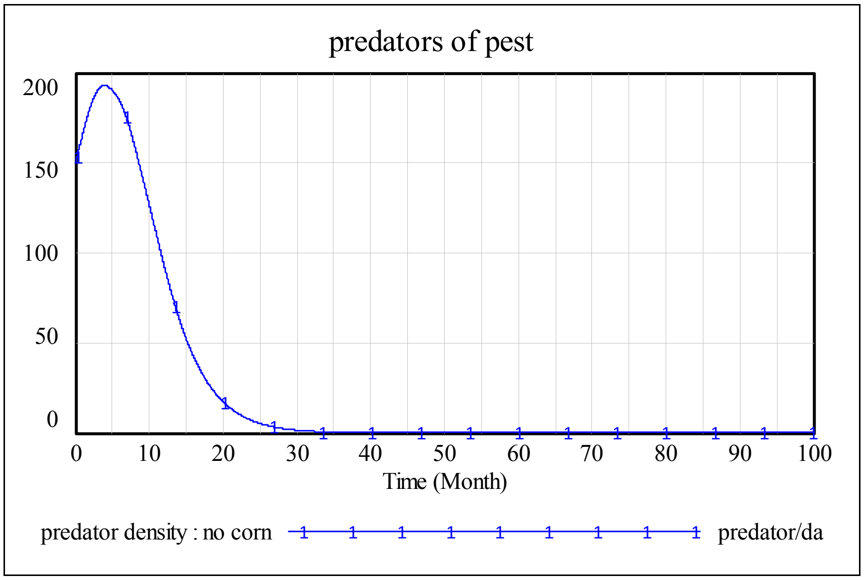


Figure 5.17. Dynamics of the predator population when corn is not planted

5.2.1.2. No Selection Pressure. When a constraint is exerted on an ecosystem, it will put pressure on all species within the system to adapt to the constraint in order to survive. Species with higher ability of adaptation to these new conditions will be selected out from those not able to adapt, the latter eventually going extinct. This is called selective or selection pressure.

In this context, this constraint is the ability to live in the presence of a pesticide and the pressure is exerted by planting *Bt*-corn or spraying a pesticide. When these actions are not taken, there is no selection pressure on pests. However, if it is assumed that resistance comes with a fitness cost, then it is also a form of selection pressure selecting out susceptibles in the absence of pesticide.

From the Hardy-Weinberg law, we expect in our model that when there is no selection pressure, resistance allele frequency remain the same, which is validated by the figure below:

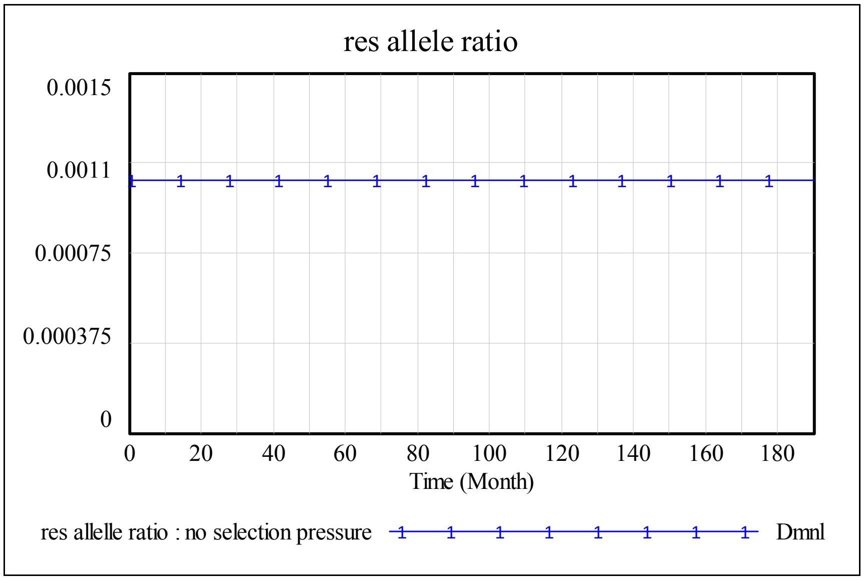


Figure 5.18. Resistance allele ratio when no selection pressure is exerted

If we assume that resistance comes with a fitness cost, this will select out susceptible individuals in the absence of Bt-toxin resulting in a decrease in the resistance allele ratio (Figure 5.19).

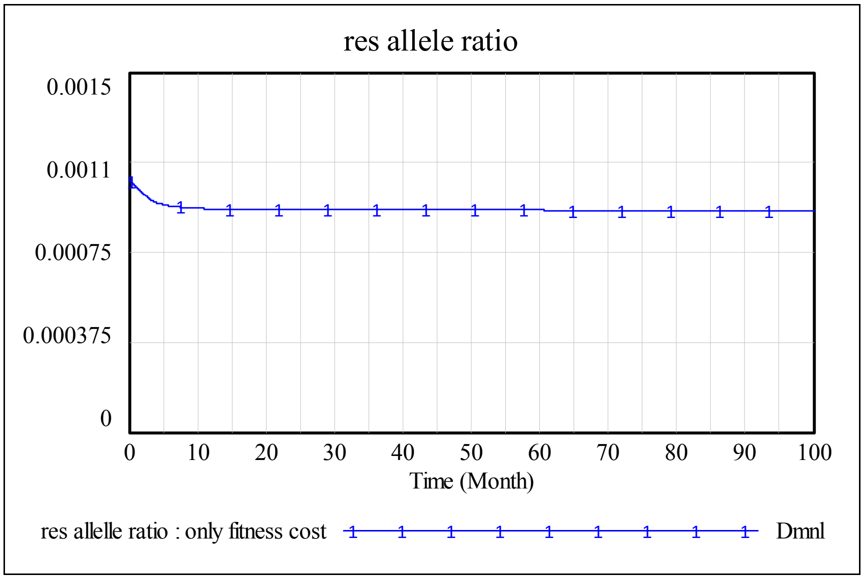


Figure 5.19. Resistance allele ratio when the only selection pressure is fitness cost of resistance

5.2.1.3. <u>No resistance allele in the population</u>. Since we assumed that mutation is negligible, there cannot be any resistance evolution when the initial resistance allele frequency is zero. As can be seen from the figure below, this is depicted by the model behavior.

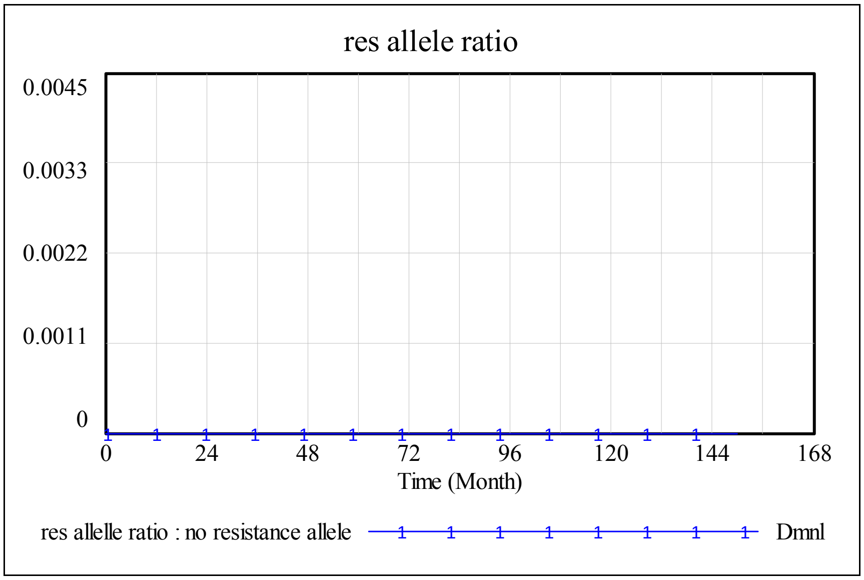


Figure 5.20. Resistance allele ratio when there is no resistance allele initially

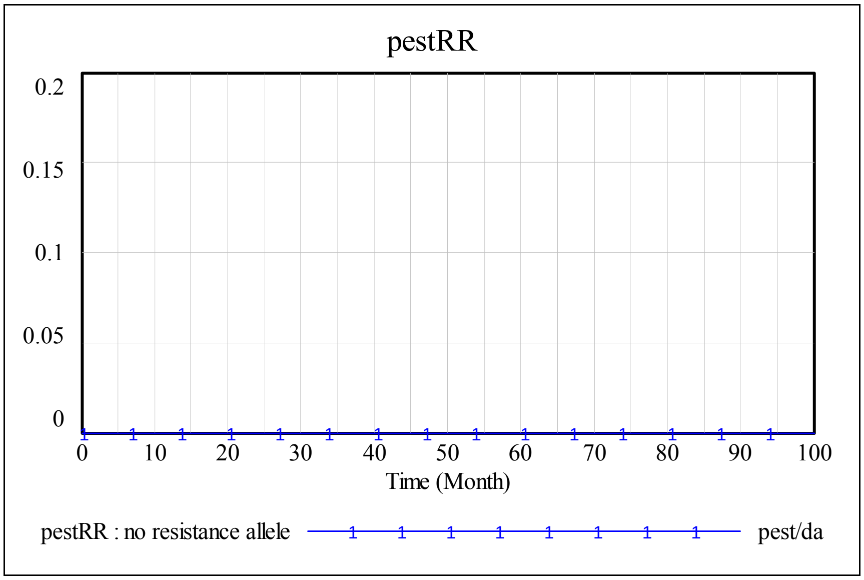


Figure 5.21. Resistant (RR) pest population when there is no resistance allele initially

5.2.1.4. No Resistant Individual. When there is no resistant individual in the population, it does not necessarily imply that there is no resistant allele in the field. Resistance gene may be present if there are heterozygote individuals; hence spread of resistance is still an issue. When the model is simulated by setting the initial number of resistant individuals to zero and heterozygote individuals to a level above zero, it is seen that resistance is spread in the population in accordance with the anticipation.

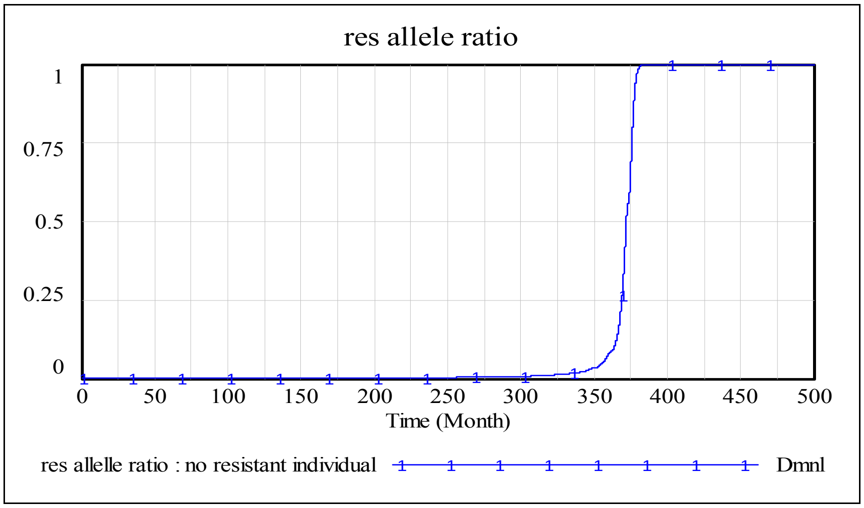


Figure 5.22. Dynamics of resistance allele ratio when there is no resistant individual initially

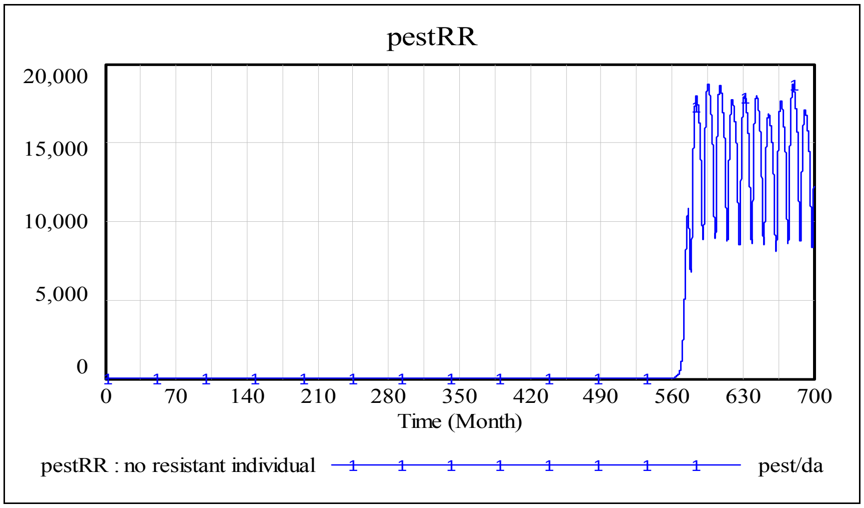


Figure 5.23. Dynamics of resistant pest subpopulation (RR) when there is no resistant individual initially

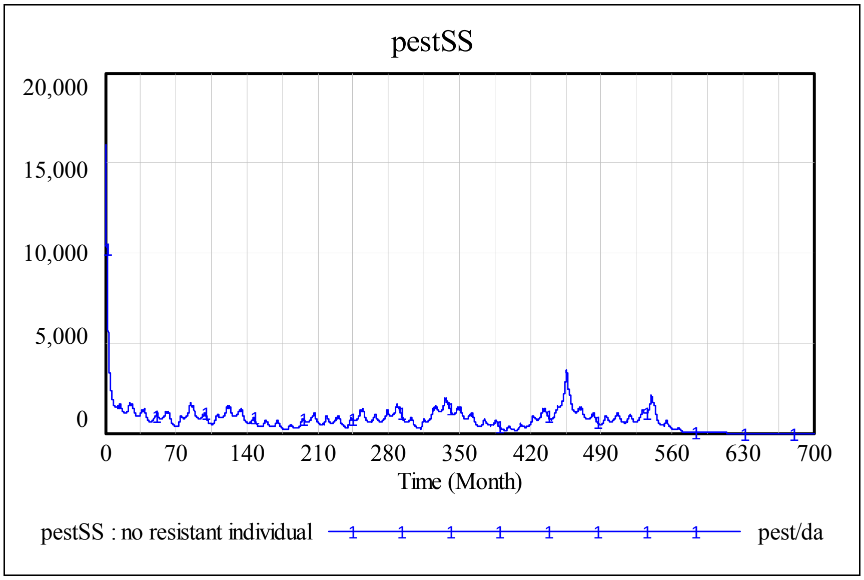


Figure 5.24. Dynamics of susceptible pest population (SS) when there is no resistant individual initially

5.2.2. Sensitivity Analysis

5.2.2.1. Fitness Cost. Fitness cost is incorporated into the model by assigning resistant pest subpopulation a higher reference mortality fraction. When fitness cost is high, spread of resistance is delayed and the population level attained thereafter is lower.

In the sensitivity test below, *res df* is allowed to take random values from a Uniform distribution between 0.2 and 0.3, the former implying no fitness cost (since it is equivalent to the reference death fraction of susceptible pests), and the latter implying an extremely high fitness cost. It is confirmed that rate of resistance development is sensitive to this parameter (Figure 5.25).

To see the effect of this parameter on the size of the pest population, two values for *res df* are tested, being 0.2 and 0.3. Though the fundamental behavior does not change, population size is significantly reduced with increasing fitness cost. Moreover the rate of resistance development is lower (Figure 5.26).

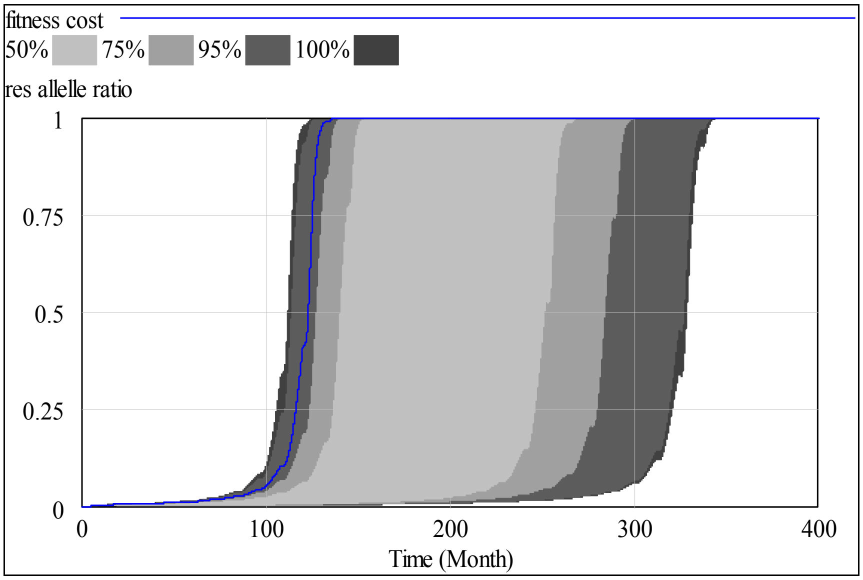


Figure 5.25. Sensitivity of resistance allele ratio to fitness cost of resistance

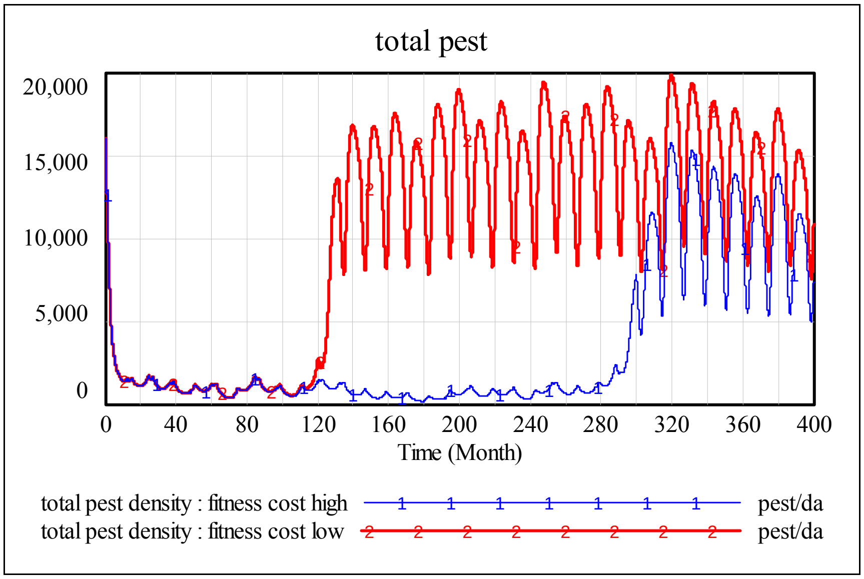


Figure 5.26. Comparison of pest population dynamics corresponding to different values of fitness cost of resistance

5.2.2.2. Initial Resistance Allele Ratio. Initial resistance allele frequency is expected to differ considerably from region to region and in general it depends upon insect species, target toxin and past exposure to *Bt*. Many studies reported resistance allele frequencies ranging from 0 to 0.16 for different Bt crops, generally being lower than 0.01 (e.g. Tabashnik *et al.*, Gould *et al*). In the base run, 0.001 is used in accordance with Gould *et al.* The initial resistance allele frequency is one of the key determinants for the rate of resistance development. The figure below displays the dynamics of resistance allele ratio with respect to different values of the initial resistance allele frequency and in a population where there is no fitness cost of resistance and a high dose *Bt* is used consistently. The table displays the initial resistance allele frequency at each run.

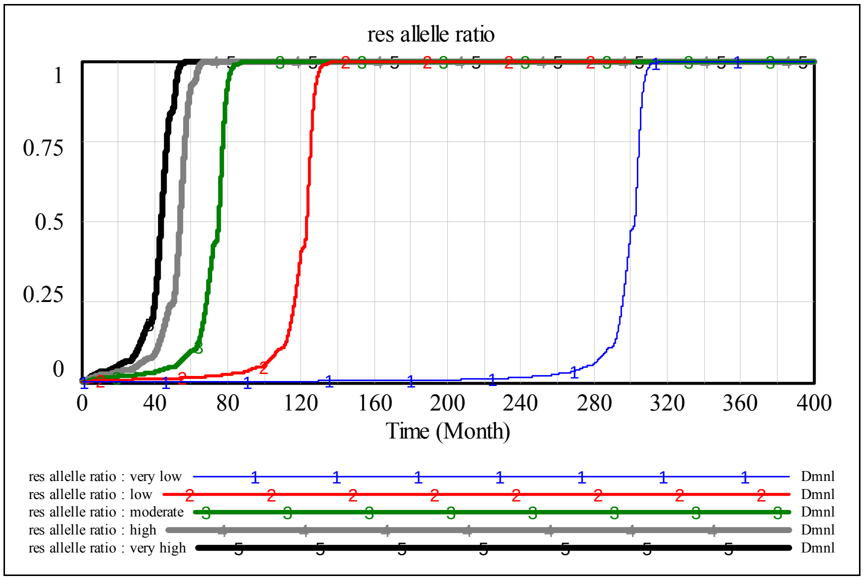


Figure 5.27. Comparison of resistance allele ratio dynamics corresponding to different initial resistance allele ratios

Table 5.1. Initial resistance allele ratios in each run of the sensitivity analysis

Run	**Resistance Allele Ratio**
Very high	0.005
High	0.004
Moderate	0.003
Low	0.001
Very low	0.0005

It is clear from the figure that initial value of resistance allele frequency in the population is an important determinant of the rate of resistance development.

5.2.2.3. Initial Size of the Pest Population. In these two runs, two extreme conditions for the initial size of the pest population are tested. For *few pests* this size is 4000, whereas for *many pests* it is 32000. To avoid a confounding effect, initial resistance allele frequency is kept the same in these runs. It is seen that fundamental dynamic behavior of the model does not depend on the size of the initial pest population. Resistance is spread in the pest population and then the population fluctuates between levels, which are critical in terms of yield loss in both of the runs. The level of population attained after the spread of resistance is also insensitive to the initial size of the population. However, the rate of resistance development is higher when the pest population is bigger due to higher number of resistant pests passing on resistance genes to the offspring (Figure 5.29).

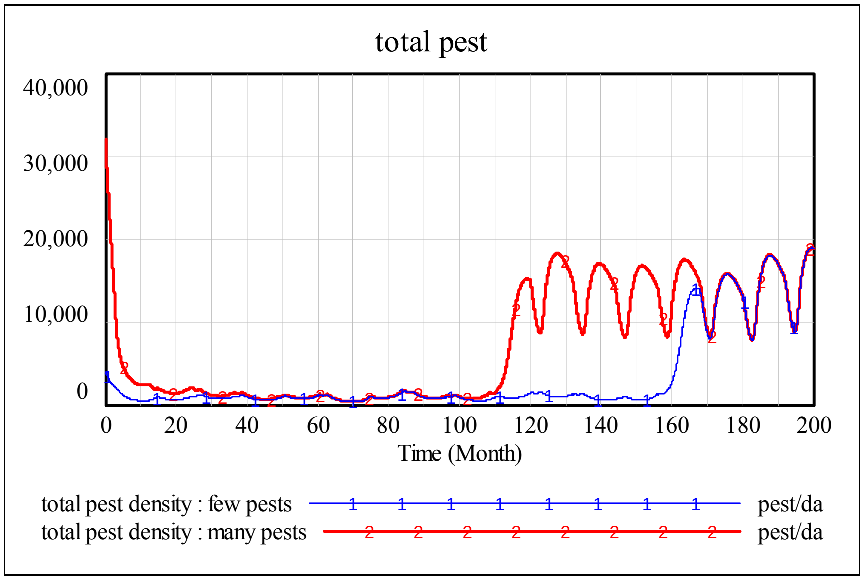


Figure 5.28. Comparison of pest population dynamics corresponding to different initial population sizes

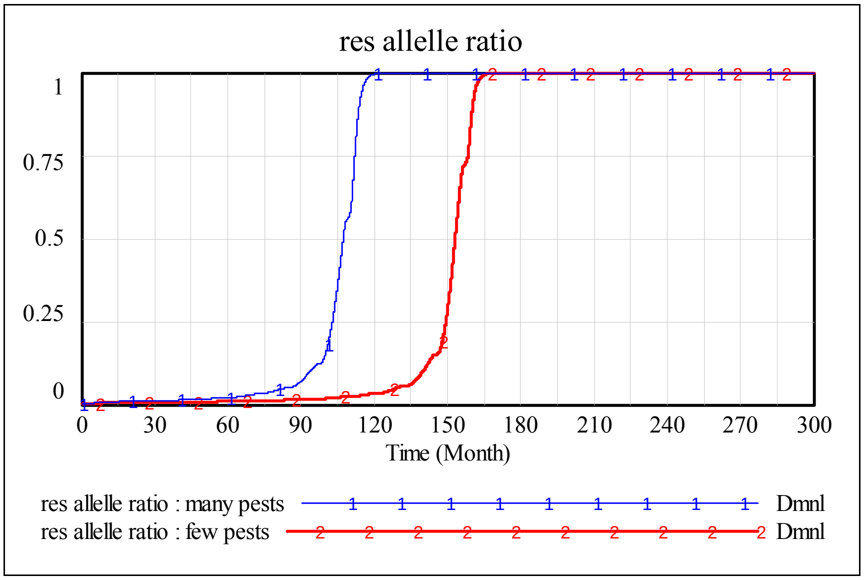


Figure 5.29. Comparison of resistance allele ratio dynamics corresponding to different initial population sizes

6. SCENARIO AND POLICY ANALYSIS OF THE IR-MODEL

Sensitivity analyses in the previous section depicted that on the contrary to the robustness of the fundamental dynamic behavior (i.e. resistance evolution followed by a pest outbreak), *time* to resistance evolution which is an important parameter determining the success of the method is quite sensitive to various parameter values. Analyses in the literature focus on specific cases and generally do not challenge their parameter settings. Since the objective of this study is to portray the long term impacts of GM crops under various scenarios and conditions, in this section IR-Model will be tested under different scenarios. Then, the refuge strategy which is the panacea solution proposed by EPA will be taken into consideration under different scenarios. Finally, a strategy that takes the population dynamics into account, hence which is in accordance with the Integrated Pest Management principles will be tested.

6.1. Scenario Analysis

6.1.1. A Best-Case Scenario

If for a specific pest species and a specific toxin, there is a considerable fitness cost of resistance and for a certain region the initial resistance allele ratio is very low, then the rate of resistance evolution is low. In addition, if the selected toxin does not harm predators, then planting a *B*t-crop is less problematic. In this best case scenario, initial resistance allele ratio is set to 0.0006 and a severe fitness cost is provided by setting *res df* to 0.25 where *sus ref df* is 0.20. Besides, predators are not damaged directly by *Bt*-toxin. It is seen from Figure 6.1 that resistance evolution is pretty much delayed under these favorable conditions.

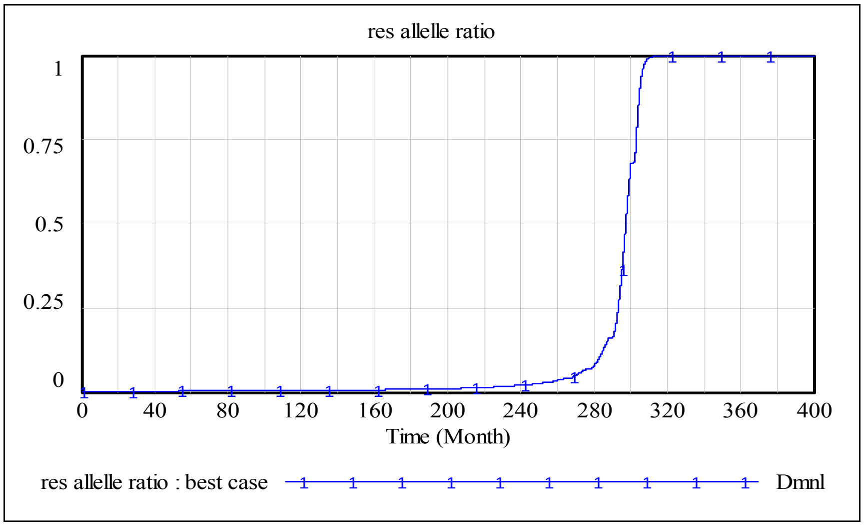


Figure 6.1. Dynamics of resistance allele ratio in the best case scenario

As seen from Figure 6.2, until t=315 pest population is suppressed very successfully, however pest outbreak is inevitable after the spread of resistance in the pest population. Since control efficiency is preserved for a considerably long period in spite of the consistent application of the same treatment, *Bt*-corn is advantageous compared to conventional pesticide treatment for a longer time in this optimistic scenario (Figure 6.3 and Figure 6.4).

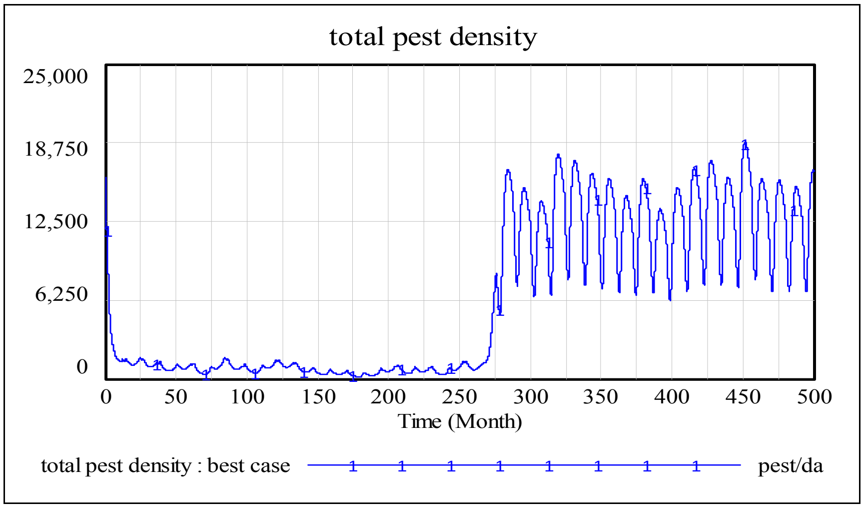


Figure 6.2. Dynamics of the pest population in the best case scenario

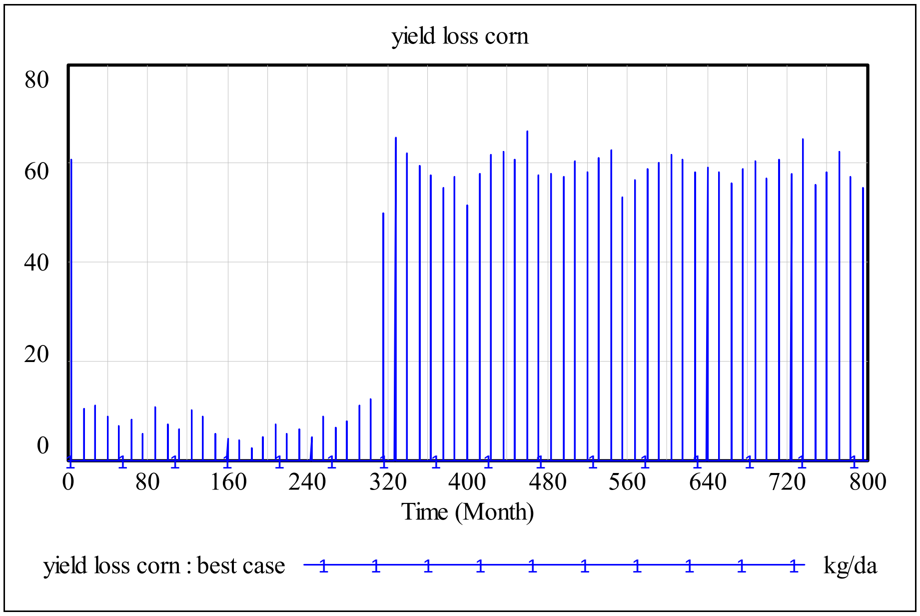


Figure 6.3. Bt-corn yield loss in the best case scenario

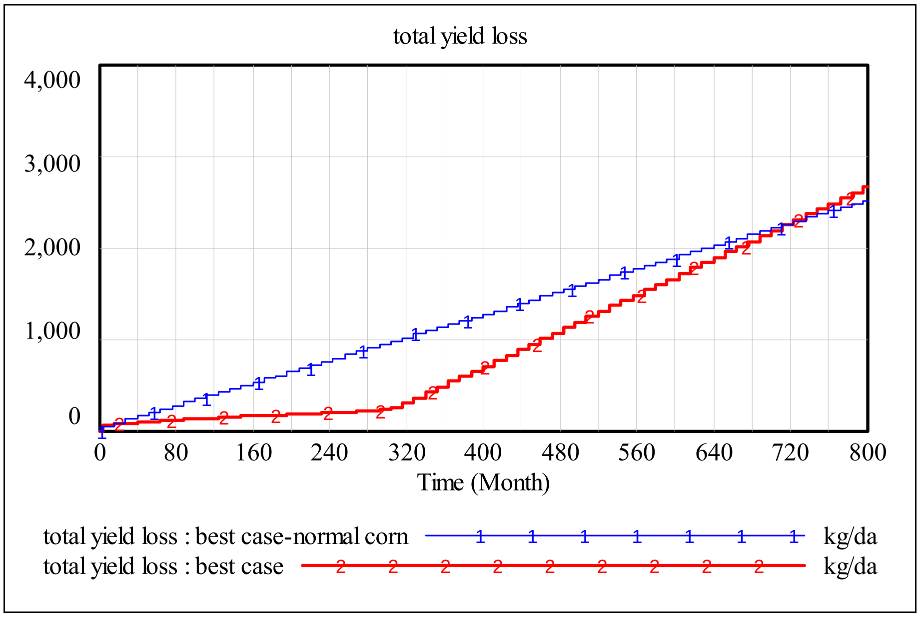


Figure 6.4. Comparison of cumulative yield losses corresponding to planting *Bt* and conventional corn under the best case scenario

6.1.2. A Worst-Case Scenario

Just like the conditions favoring the best case scenario, conditions shaping the other extreme are also possible. Resistance need not come with a fitness cost and resistance allele ratio may be initially high probably due to having been exposed to the toxin before. To demonstrate these conditions, initial resistance allele ratio is set to 0.005 and death fraction of resistant pests is taken to be equal to the normal death fraction of susceptibles. Besides, predators are assumed to be damaged by the toxin.

It is seen from Figure 6.5 that under these conditions the rate of resistance evolution is very high. In five years, the pest population on the farm-land becomes thoroughly resistant to the specific toxin.

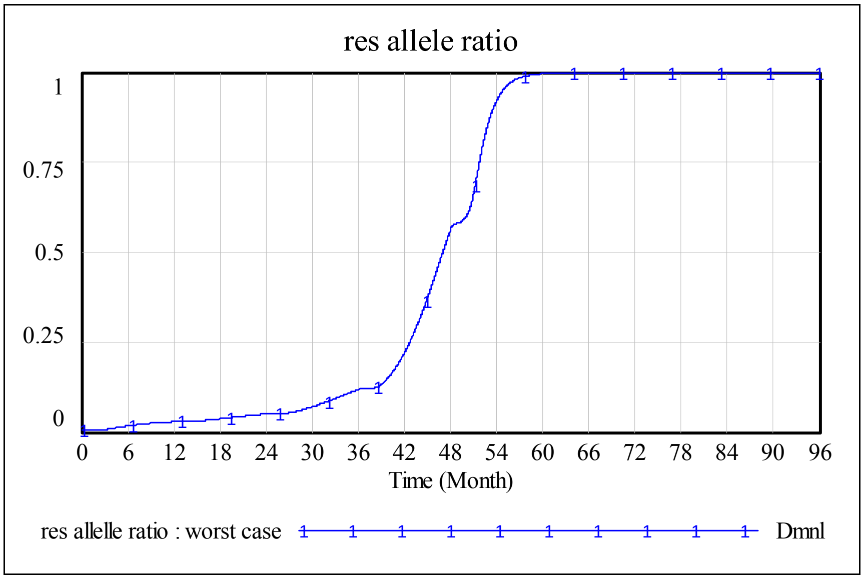


Figure 6.5. Dynamics of resistance allele ratio in the worst case scenario

Since resistance evolution is fast, pest outbreak is faced earlier than the base run as can be seen from Figure 6.6. Moreover predator species face with farm scale extinction, which makes the pest species more problematic.

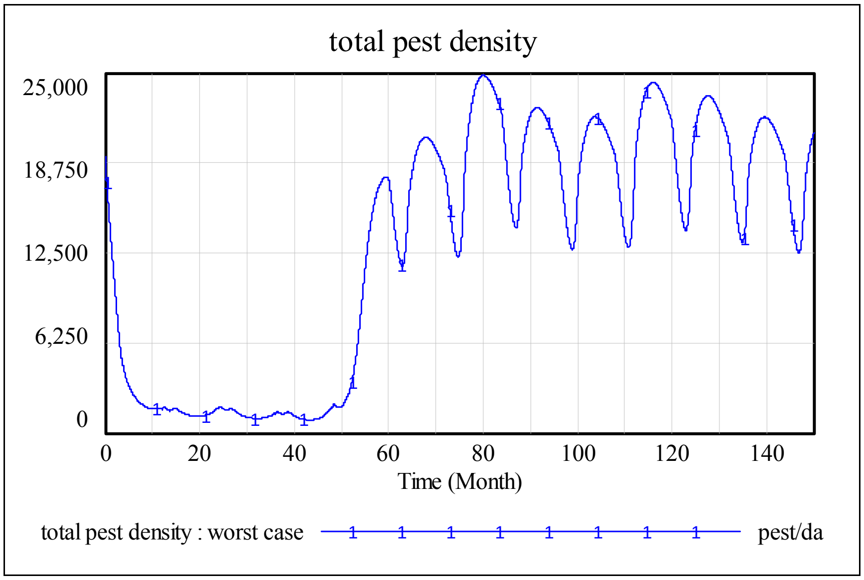


Figure 6.6. Dynamics of the pest population in the worst case scenario

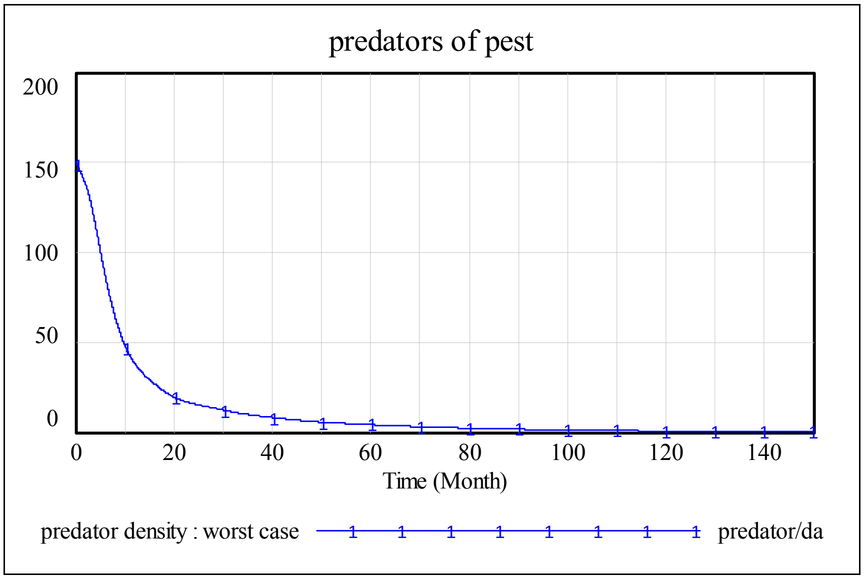


Figure 6.7. Dynamics of the predator population in the worst case scenario

The corresponding yield losses can be seen in Figure 6.8. When these losses are compared to those obtained from planting conventional corn (Figure 6.9), it is seen that *Bt*-corn's advantage is limited to the first 5 years.

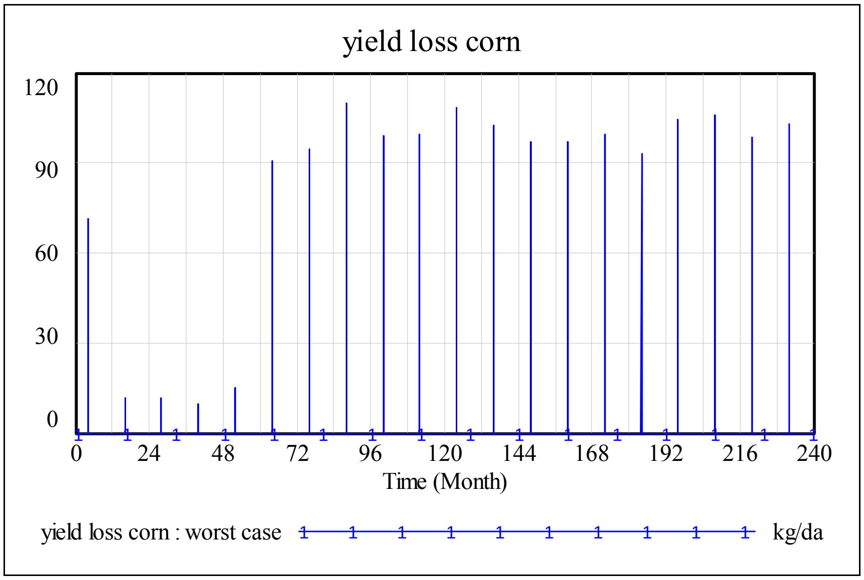


Figure 6.8. Corn yield loss in the worst case scenario

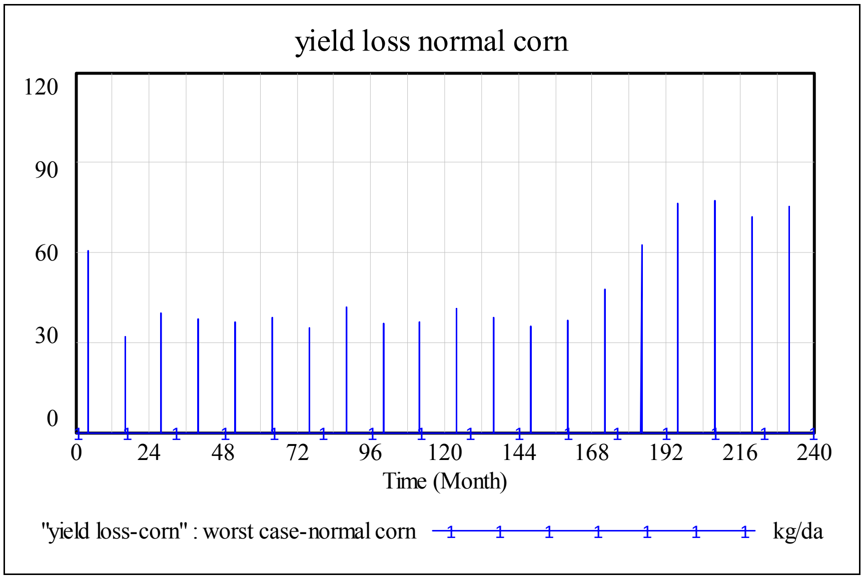


Figure 6.9. Conventional corn yield loss in the worst case scenario

Assuming that *Bt*-pesticide which is used to treat the conventional corn does not damage the predator species, it is seen that the equilibrium level of the pest population after the spread of resistance is higher when *Bt* is planted implying an increased burden of pest management compared to planting conventional corn (Figure 6.10). This drawback of planting *Bt*-corn is also depicted in the cumulative yield loss graph in Figure 6.11.

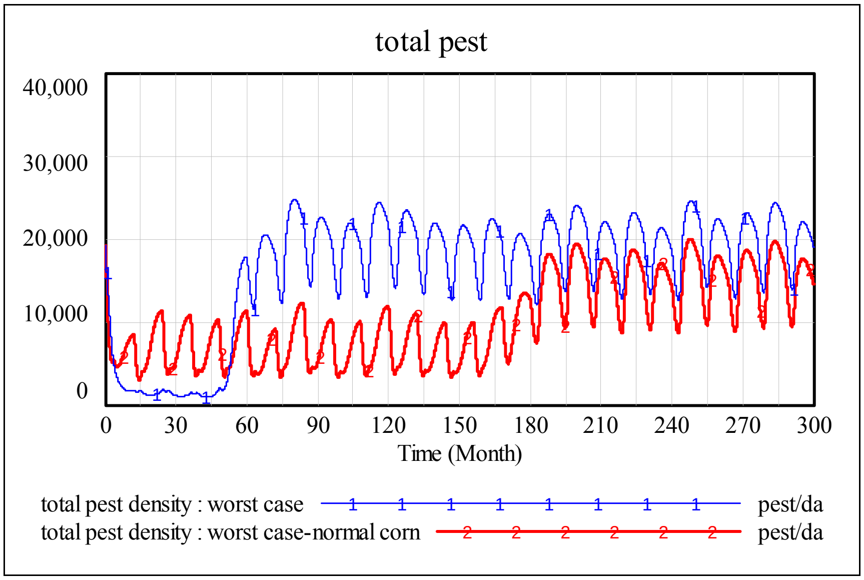


Figure 6.10. Comparison of pest population dynamics corresponding to planting *Bt* and conventional corn, under the worst case scenario

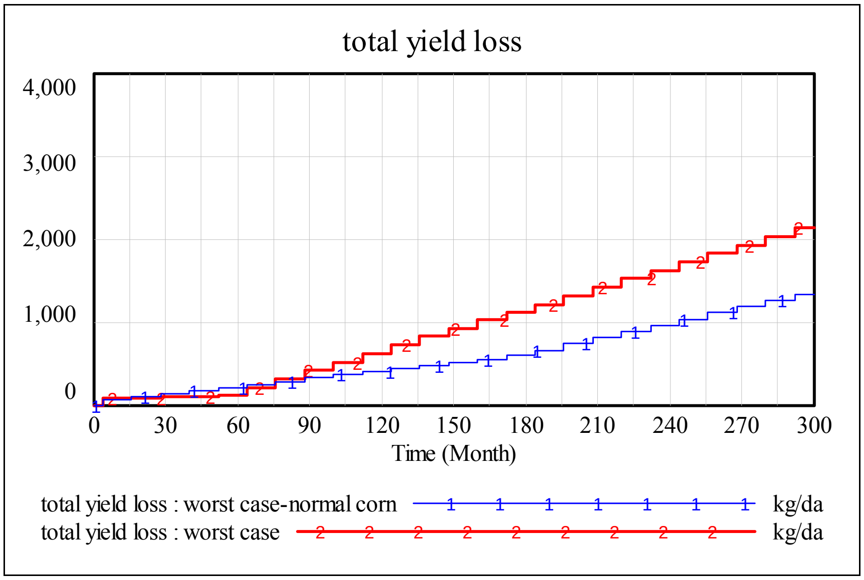


Figure 6.11. Comparison of cumulative yield losses corresponding to planting *Bt* and conventional corn under the worst case scenario

6.1.3. Farmers Switch to Pesticide

Assuming a pesticide or Bt-plant to which pests gained resistance will stay in use is not realistic. In this section, the farmer is modeled to switch to planting conventional corn and treating with a non-*Bt* pesticide two seasons after the spread of resistance. It is assumed that resistant pests are not resistant to the new pesticide (i.e. there is no cross-resistance) and those pests that are resistant to this pesticide is very few in number. If the farmers switch to a less effective pesticide after the spread of resistance (due to the fact that more effective pesticides are generally prohibited because of their environmental risks) treating conventional corn with pesticides turns out to be as efficient as *Bt*-corn in the long run when cumulative yield losses are taken into account (Figure 6.12). It is even a better treatment if *Bt*-corn harms predators since in this case farmers will face with a more severe pest infestation due to loss of predators. As seen from Figure 6.13, the equilibrium level of the pest population in this case is higher than the level attained by treating conventional corn with pesticide.

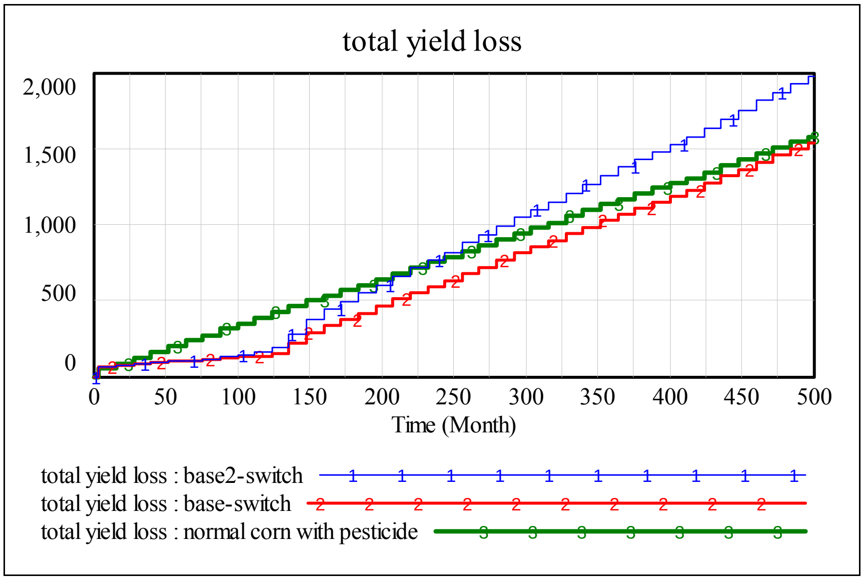


Figure 6.12. Comparison of cumulative yield losses occurring in both cases of the base run and in conventional corn when farmers switch from *Bt*-corn to conventional corn after resistance is spread

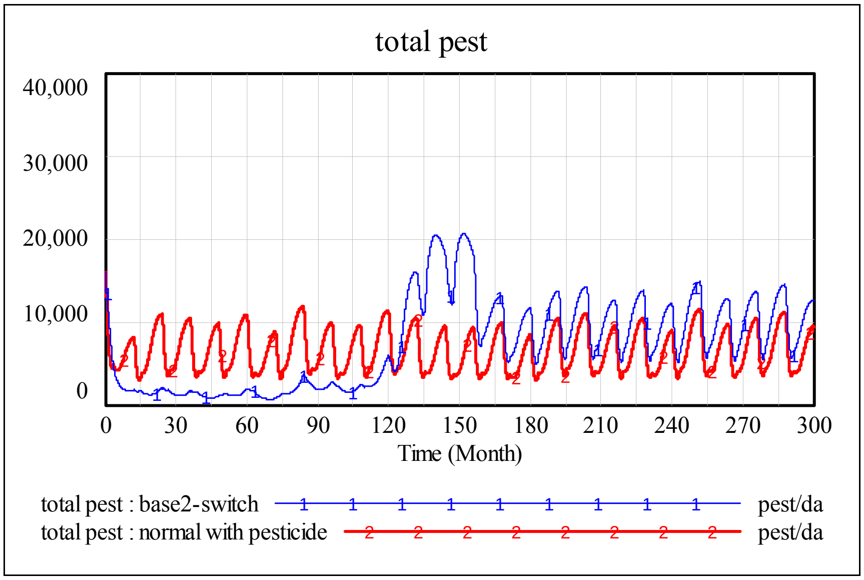


Figure 6.13. Comparison of pest population dynamics corresponding to *Bt*-corn and *Bt*-pesticide treatments if *Bt*-corn also harms predators and when farmers switch from *Bt*-corn to conventional corn after resistance is spread

6.1.4. Partially Resistant Heterozygote Pests

Though initial resistance allele ratio seem to be the most effective parameter determining the rate of resistance development, it is argued that phenotype of heterozygote pests is the most influential issue (see for example Tuli *et al.,* 2000). In the IR-Model, heterozygote pests are assumed to be completely susceptible since "the prevalence of evidence suggests that resistance is a recessive trait" (Nelson *et al*., 2001) and since current resistant management strategies rely on this assumption. However, susceptibility/resistance is generally considered to be a matter of degree, rather than a binary distinction and the survival rate of heterozygote pests may change from species to species. Furthermore, the validity of the susceptibility assumption has not been proved for many pest species.

In this scenario, heterozygote pests have a certain resistance to the toxin, which is accomplished by lowering the effect of *Bt*-toxin on heterozygote death fraction, *pestRS df*.

Hence heterozygote survival rate is now between resistant and susceptible homozygote survival rates.

As seen from Figure 6.14, resistance may be spread throughout the population in as little as four years when heterozygote pests are not completely susceptible. Hence, phenotype of heterozygote pests is clearly more determining for the rate of resistance development than the initial resistance allele ratio. This shows that without confirming that heterozygote pests are completely susceptible to a specific toxin, it is inadvisable to adopt the technology for consistent application.

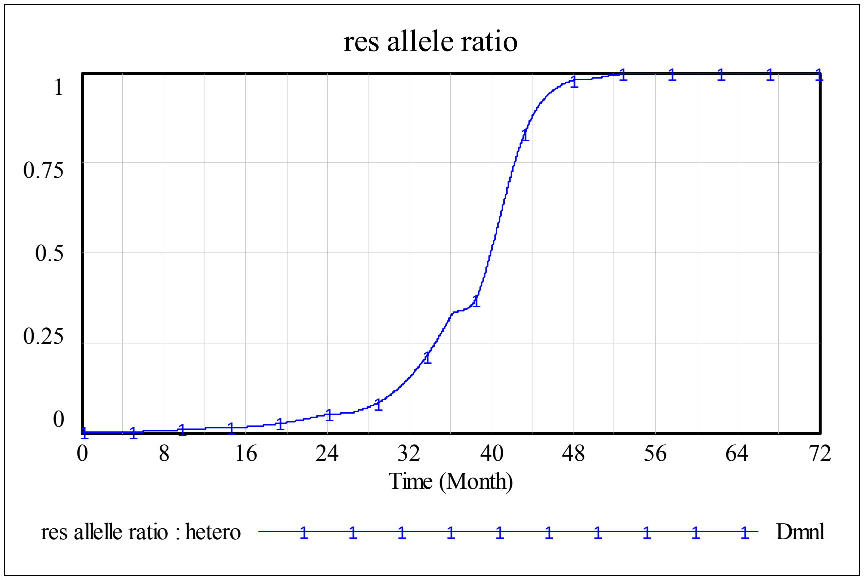


Figure 6.14. Dynamics of resistance allele ratio when heterozygote pests are semi-resistant

If conventional corn were planted in this scenario, resistance evolution is delayed by almost 8 years compared to planting *Bt*-corn (Figure 6.15 and 6.16). However, losing the usability of a less harmful (more environmentally friendly) toxin in 12 years shall not be considered as an achievement either.

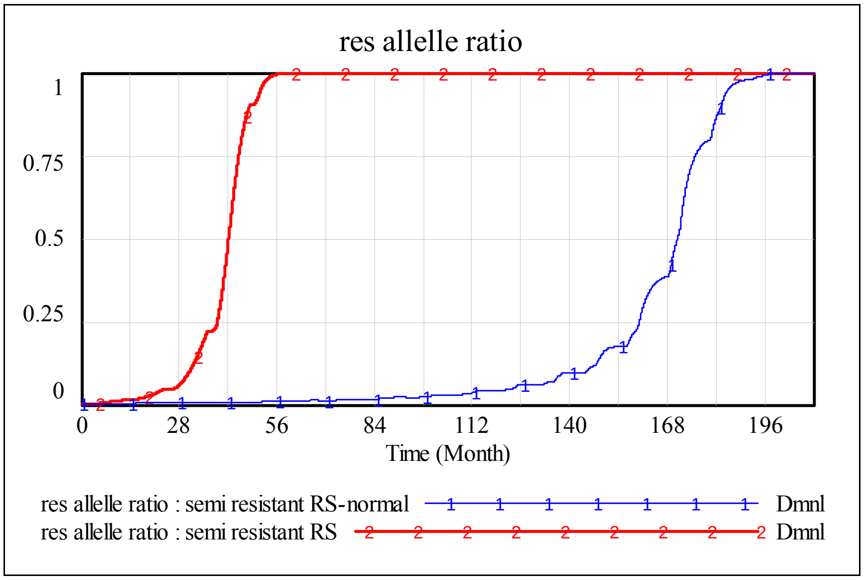


Figure 6.15. Comparison of resistance allele ratio dynamics corresponding to planting *Bt* or conventional corn when heterozygote pests are semi-resistant

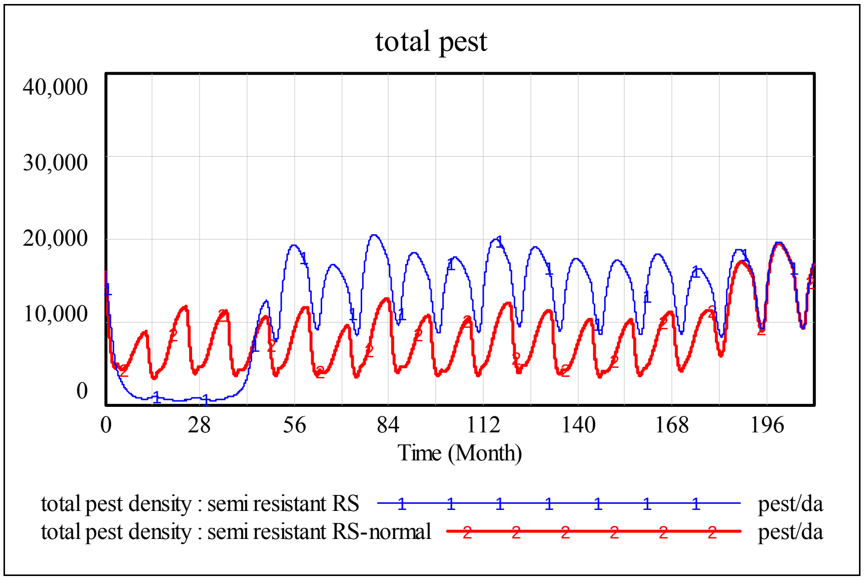


Figure 6.16. Comparison of pest population dynamics corresponding to planting *Bt* or conventional corn when heterozygote pests are semi-resistant

6.2. Policy Analysis

6.2.1. Refuge Strategy

EPA asks all farmers who use *Bt*-crops to spare a portion of their land as a refuge for susceptible pests, though noncompliance to this requirement is not subject to any legal action. An example of a refuge is a block of non-*Bt* corn planted near a *Bt*-corn field. Refuge supplies a source of susceptible pests that can mate with resistant pests. Assuming that resistance is a rare occurring and recessive trait, with the supply of susceptible individuals the probability that a resistant insect from a *Bt* field choose another resistant insect as a mate is considerably reduced. Hence, by preventing the pairing of resistant genes, these refuges try to ensure that susceptibility is passed on to offspring (EPA, 2002).

Determining the size and the design of refuges is an issue because it must be guaranteed that susceptible pests in the refuge are within mating distance with resistant pests. As the distance between the refuge and the *Bt*-corn area increases, it is less likely that such mating between susceptible and resistant individuals occurs. Hence the design of the refuge area determines the success of the strategy and no design guarantees that susceptible pests are evenly distributed in the area ensuring perfect randomness in mating. However, in the model for the sake of simplicity it is assumed that mating is random throughout the land. Hence the results obtained will be optimistic in estimating the resulting delay in resistance development.

In the model, conventional corn is planted in the refuge area and it is assumed that pests are distributed evenly throughout the land. Refuge ratio is taken to be 20% in accordance with EPA requirements (see Appendix C.3 for related model equations). This area is treated with a non-*Bt* pesticide and it is assumed that there is no cross-resistance. Hence pests that are resistant to Bt-corn are susceptible to this pesticide. Resistance to the non-Bt pesticide is ignored due to low rate of resistance development when pesticides are used. Under these conditions, it is seen from Figure 6.17 that resistance development is delayed by almost 15 years. Hence pest population remains in reasonable levels in the first 26 years (Figure 6.18).

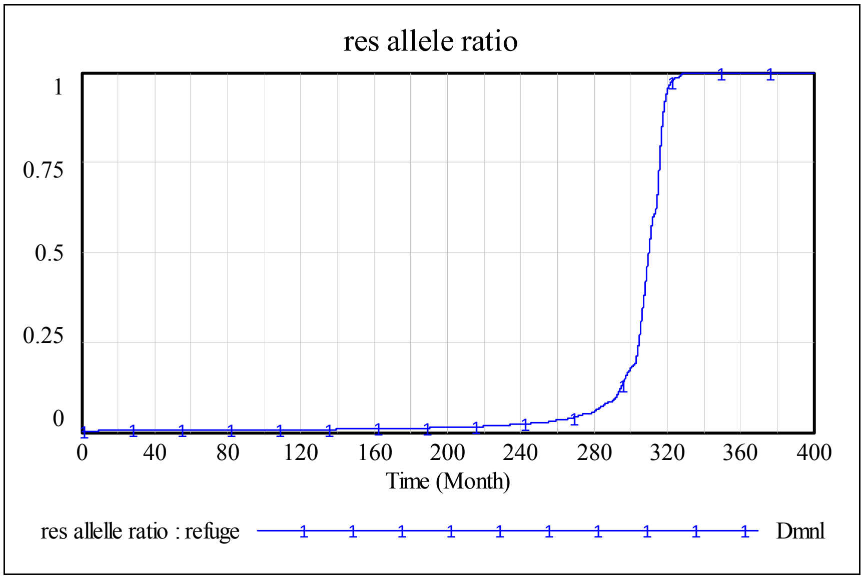


Figure 6.17. Dynamics of resistance allele ratio under the refuge strategy

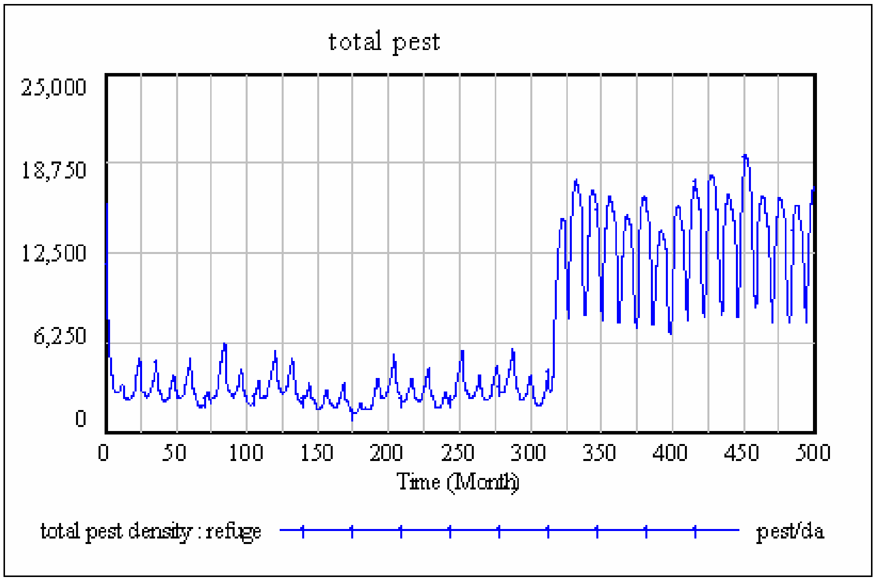


Figure 6.18. Dynamics of the pest population under the refuge strategy

Yield losses before the spread of resistance are higher than those in the base run, however this control efficiency can be preserved for a longer period.

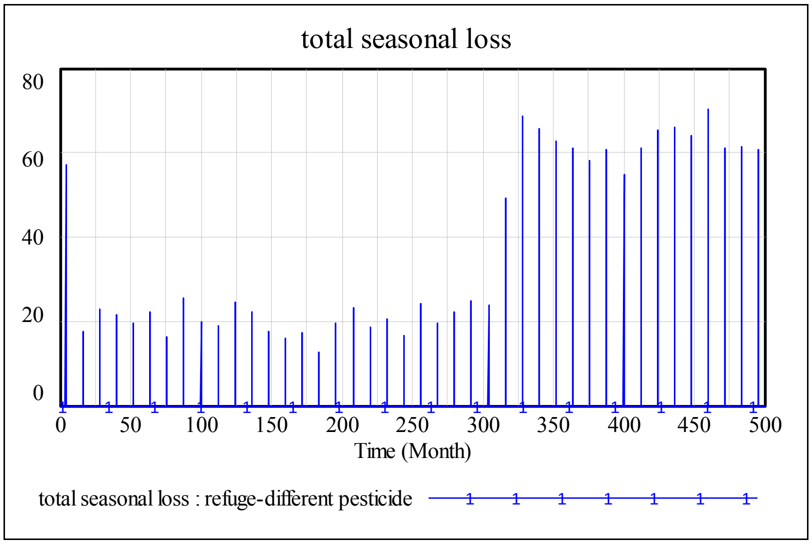


Figure 6.19. Yield losses under the refuge strategy

On the other hand, resistant pests may be cross resistant to the pesticide that is used to treat the refuge. This happens especially when the two pesticides have a common mode of action. A pest which is resistant to *Bt*-corn may very well be resistant to another pesticide which acts similarly to *Bt*-corn. In this case, resistance to the pesticide that is used to treat the refuge evolves just exactly the same way as resistance to *Bt*-corn develops.

To demonstrate this phenomenon, initial population of resistant pests is assumed to be also resistant to the pesticide. Hence, as resistance dynamically develops, *Bt*-resistant pests also gain pesticide resistance. As seen in Figure 6.20, in this case resistance development is delayed only by six years. Figure 6.21 shows that the advantage of planting refuge is limited to these six years. Hence, under the case of cross-resistance benefits gained from planting refuges decline considerably.

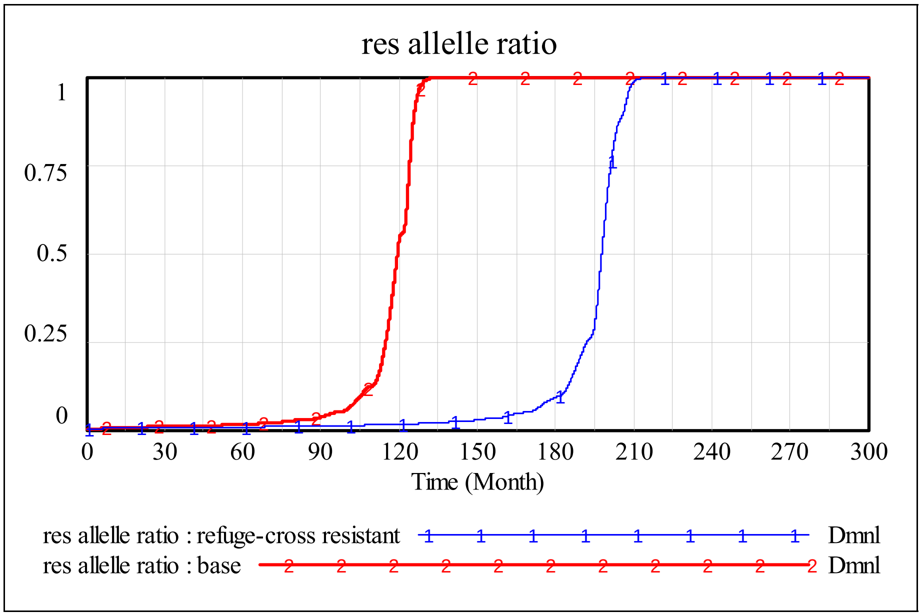


Figure 6.20. Resistance allele ratio dynamics in the base run versus under the refuge strategy when refuge is treated with a pesticide to which Bt-resistant pests are resistant

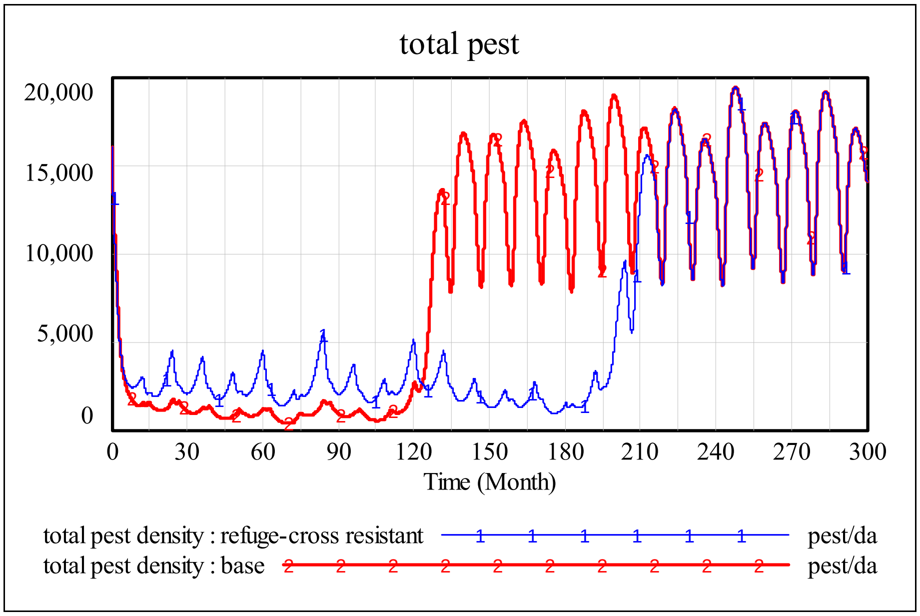


Figure 6.21. Pest population dynamics in the base run versus under the refuge strategy when refuge is treated with a pesticide to which Bt-resistant pests are resistant

Another refuge strategy proposed by EPA has been sparing only 5% of the land as refuge but not treating that part of the field (EPA, 2002). As seen in Figure 6.22, this strategy is not successful enough in delaying resistance development when compared to the first strategy.

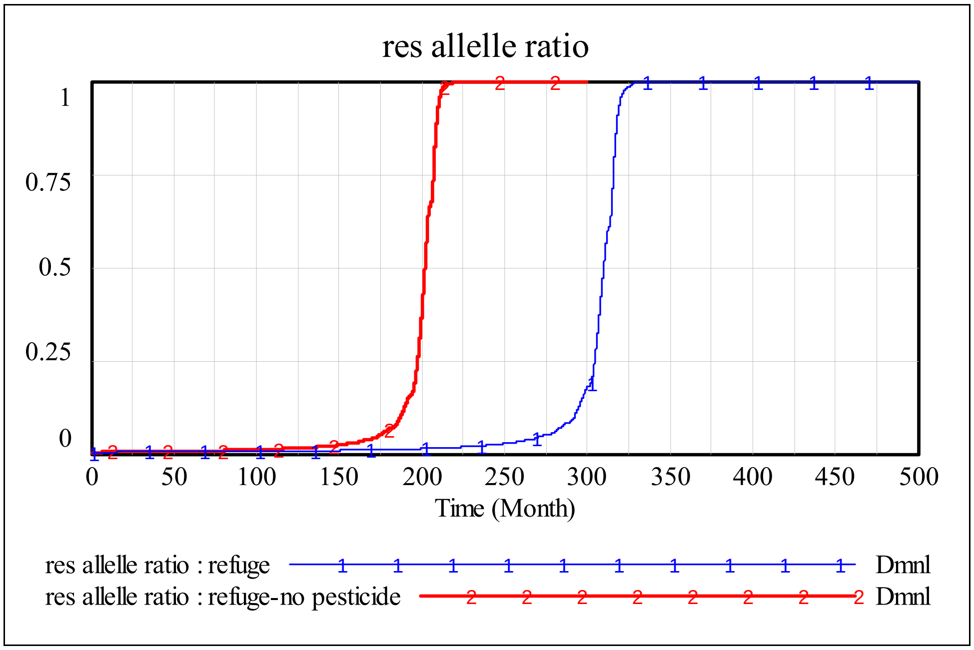


Figure 6.22. Comparison of resistance allele ratio dynamics corresponding to the two different refuge strategies

In the analyses above, refuge strategy is tested assuming that the setting of the base behavior holds. However, if refuge strategy is applied assuming that the conditions shaping the worst-case scenario (which are high level of initial resistance allele ratio and no fitness cost of resistance) hold, it can be seen from Figure 6.23 that resistance evolution is not delayed sufficiently. Pest outbreak emerges at the ninth year in spite of the refuge (Figure 6.24).

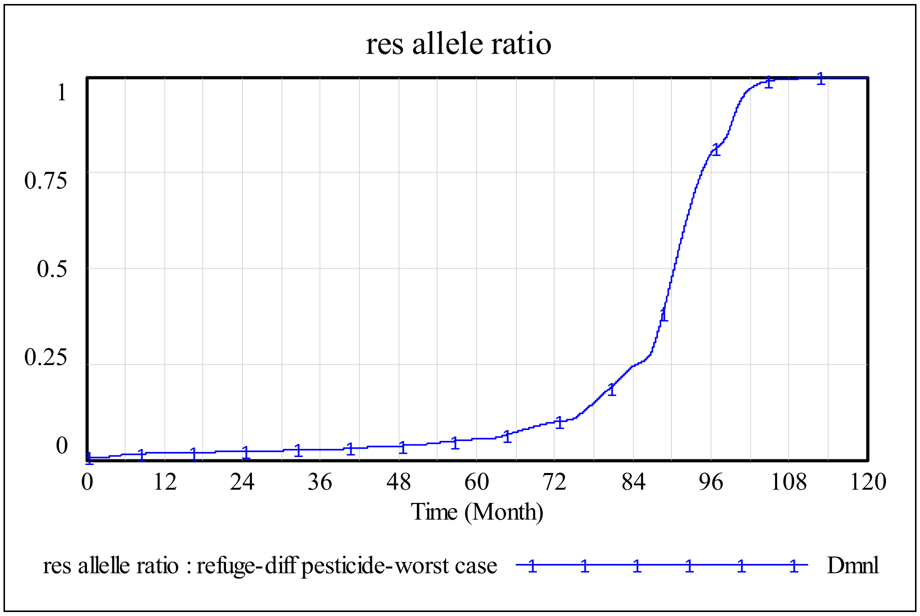


Figure 6.23. Dynamics of resistance allele ratio when refuge strategy is applied under worst case conditions

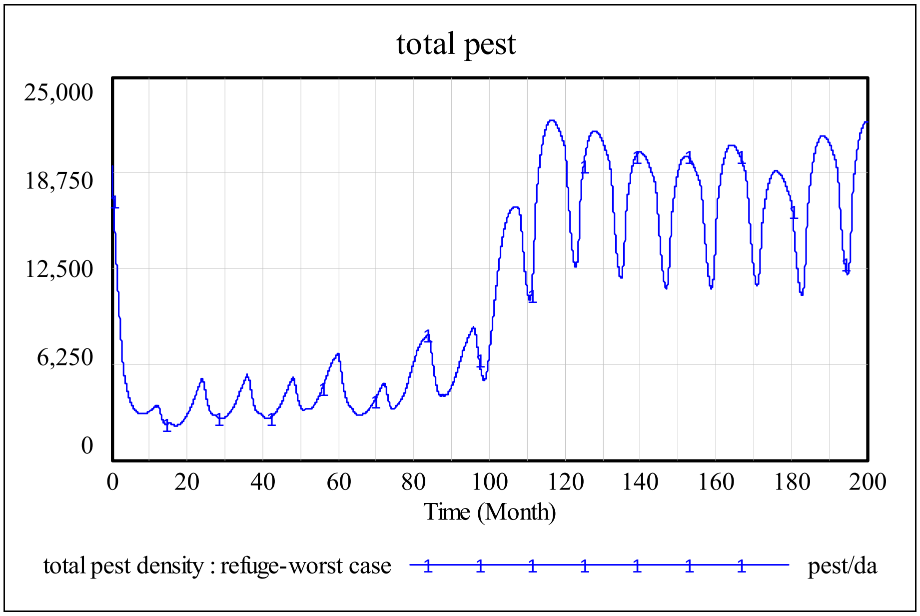


Figure 6.24. Dynamics of pest population when refuge strategy is applied under the worst case conditions

On the other hand, if the susceptibility assumption of heterozygote pests is challenged, it seen that refuge strategy is no longer effective in delaying resistance evolution.

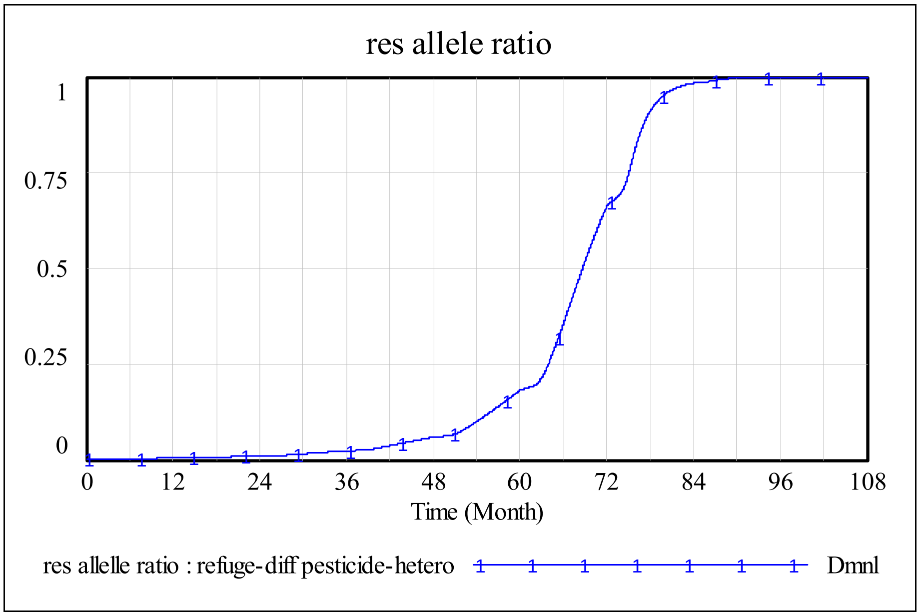


Figure 6.25. Resistance allele ratio dynamics when refuge strategy is applied under the scenario of semi-resistant heterozygote pests

The last two cases show that the refuge strategy is not necessarily a panacea. Depending on the feedback from the environment, trying to design and maintain refuges may turn out to be a futile attempt.

6.2.2. An Integrated Pest Management (IPM) Strategy

Integrated pest management (IPM) is a knowledge intensive approach to pest control that utilizes regular monitoring to determine if and when treatments are needed. An IPM program might contain biological controls such as building predator traps, cultural controls such as crop rotation and as a last resort chemical control with least toxic chemicals to keep pest number low (Cowan *et al*., 1996).

To implement an IPM strategy, a farmer typically monitors his crop and the pest populations, estimates the potential economic damage and if the damage threshold is reached, applies the most appropriate measures. IPM strategies try to enhance natural interactions rather than overriding them by eliminating some species (Cowan *et al*., 1996). Hence IPM is potentially a sustainable alternative to chemical control. Moreover, in the presence of a pest infestation controls are applied only if the cost of application is less than the economic value of pest damage, i.e. when pest population reaches a certain economic threshold level. Hence, IPM tries to minimize economic damage rather than physical damage. For these two reasons, many argue that it is a superior means of pest control.

Building and testing a thorough IPM model for the crop of concern is not the objective of this study, hence will be left as a future work. However, a hypothetical strategy will be tested in this section to mimic the fundamental idea behind IPM so as to demonstrate a means of integrating *Bt*-crops with IPM techniques. The model uses a given economic threshold, which is used for the decision "plant or do not plant *Bt*-corn". Hence *Bt*-corn is planted only those years when there is an expectation for a more severe pest outbreak, otherwise conventional crop is planted and a different pesticide is applied.

As seen in Figure 6.26, under this strategy *Bt*-corn becomes a tool to be evoked when there is good reason to believe that pest outbreak is likely to occur. Hence, it is planted less frequently than the conventional corn.

When conventional corn hybrid is planted, pesticide is sprayed twice a season (Figure 6.27). However, since pesticides are not as effective and durable as *Bt*-crops, pest population cannot always be suppressed as efficiently (Figure 6.28).

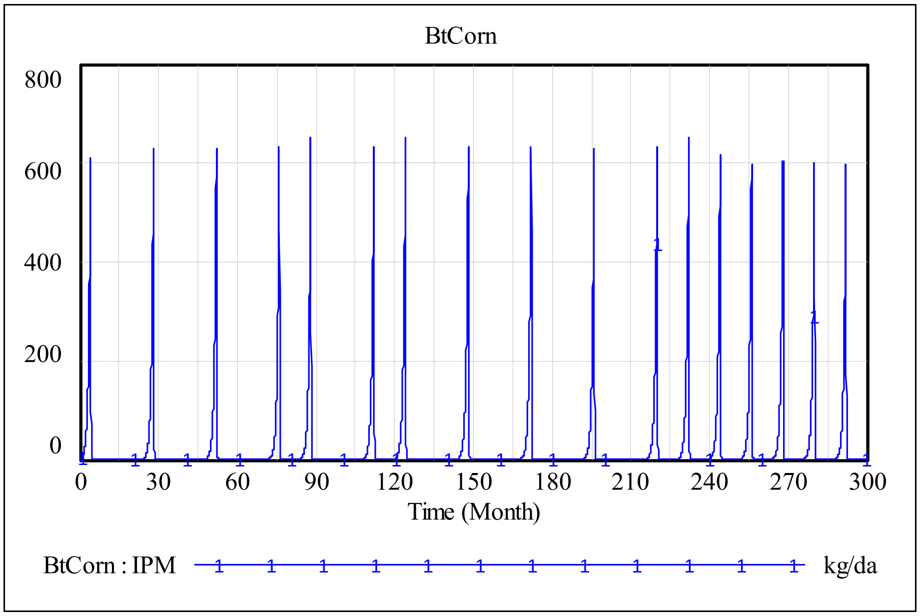


Figure 6.26. Years when Bt-corn is planted

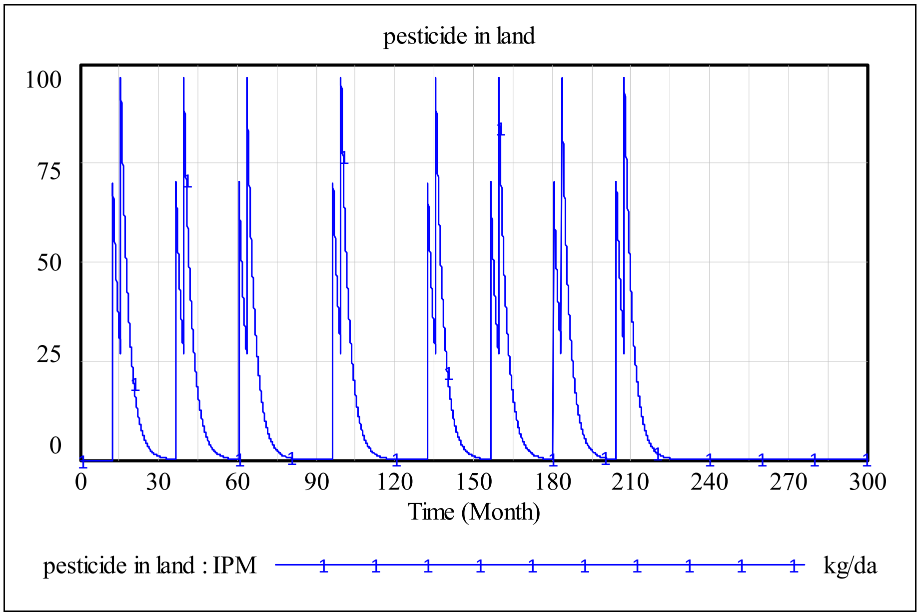


Figure 6.27. Pesticide usage under the IPM scenario

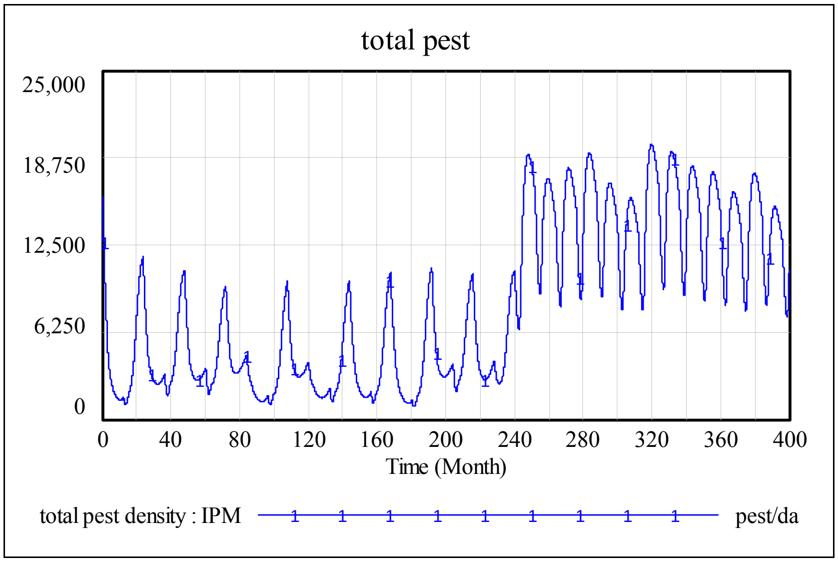


Figure 6.28. Dynamics of the pest population under the IPM scenario

Yet, by limiting the usage of *Bt*-crop, the strategy turns out to provide a smaller rate of resistance development compared to the base run (Figure 6.29). Hence, though at certain years high yield losses occur, the strategy is superior in terms of sustainability.

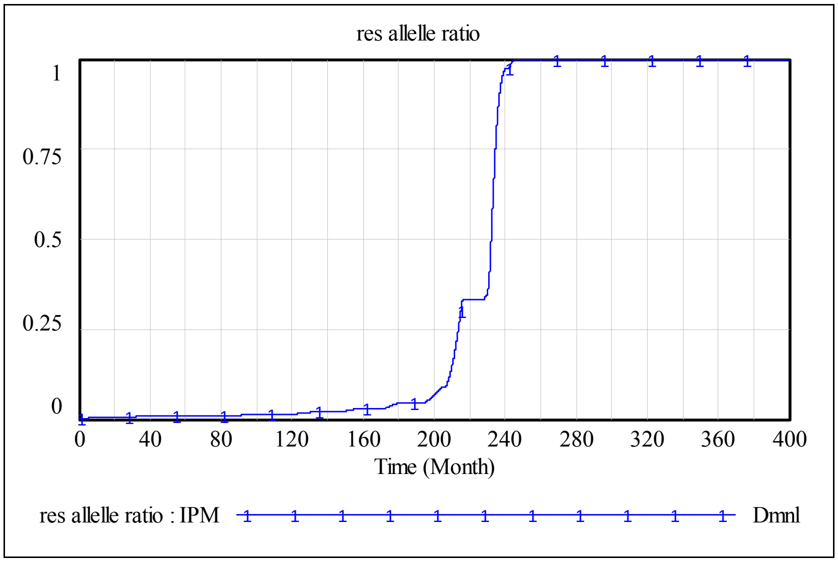


Figure 6.29. Dynamics of resistance allele ratio under the IPM scenario

However, the strategy is not as effective as planting 20% refuge and treating it with a different pesticide in terms of delaying resistance formation (Figure 6.30) and cumulative yield loss (Figure 6.31).

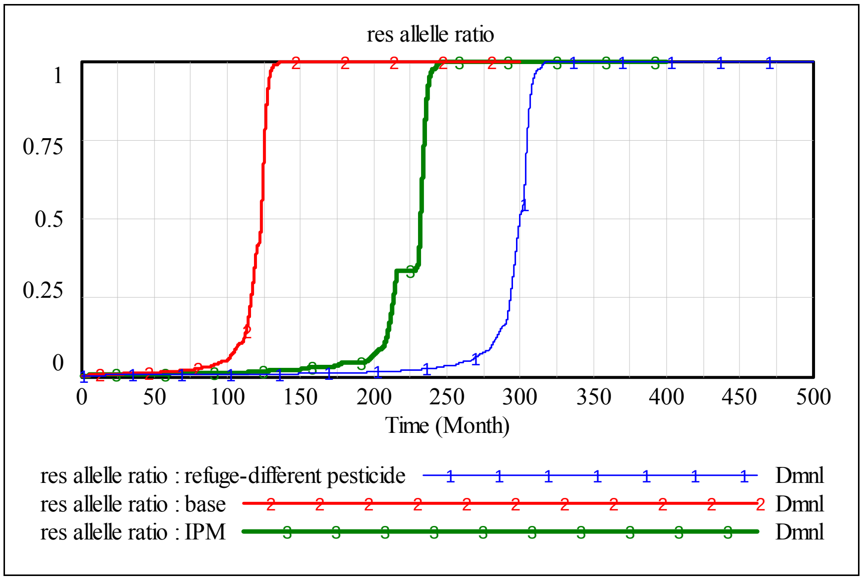


Figure 6.30. Comparison of resistance allele ratio dynamics occurring in the base run, IPM and refuge strategies

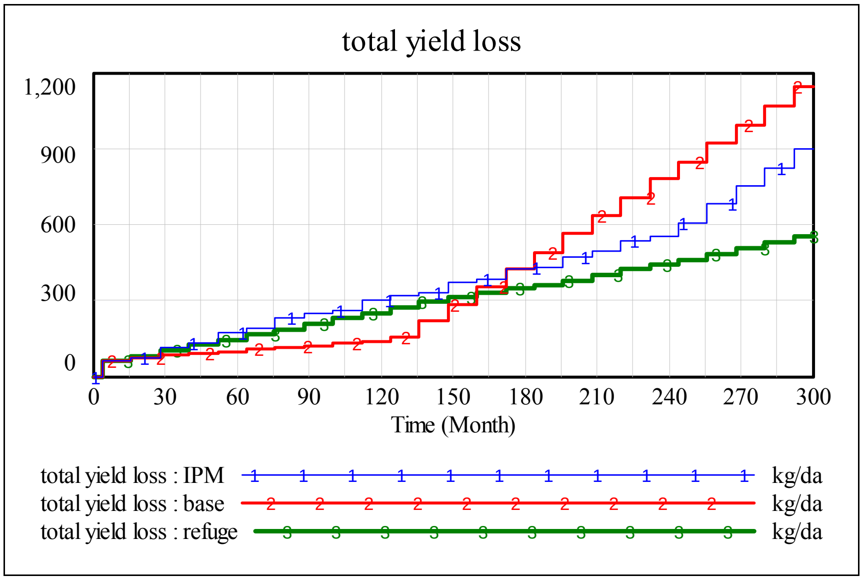


Figure 6.31. Comparison of cumulative yield losses occurring in the base run, IPM and refuge strategies

6.2.3. Further Discussion on the Model Behavior

The main concentration in the analysis of the model behavior has been resistance evolution due to consistent selection pressure, particularly when it occurs slowly, when it occurs faster and how it can be delayed. Certainly, the *Bt*-crop is not the only product on the shelves for the control of the pest of concern and biotechnology industry may supply new varieties into the market when pests gain resistance to one specific *Bt*-crop. However, susceptibility to relatively less toxic products such as *Bt*-toxins is a resource to be protected. It is generally concluded that *Bt* pesticides have low dietary, worker, and ecological risks compared to the alternatives that might replace it if resistance develops (EPA, 2002). For most farmers, the development of resistance to specific *Bt* toxins would mean a return to synthetic insecticides, thereby offsetting the claimed benefits of reducing pesticide use. For the organic food industry, which heavily relies on *Bt* as bio-pesticides, migration of resistant pests will bring about the necessity to find substitute treatment techniques. Moreover, experience has shown that simply changing pesticides is not enough to overcome resistance problem, mostly due to the fact that resistance to a pesticide comes with cross resistance to a range of pesticides. Hence, planting *Bt*-crops consistently as a monoculture is far from being a sustainable alternative to conventional techniques in spite of its attractive short-term benefits. However, this discussion is not meant to promote the "conventional" way of agriculture. Conventional seeds are usually treated with pesticides and the adverse effects of spray pesticides on environment have been demonstrated many times. Hence, the issue is finding a more sustainable means of pest management as an alternative to both strategies.

The summary of the obtained results can be seen in Table 6.1.

Table 6.1. Summary of the results from the IR-Model

Scenarios:	**GM Crop**	**Conventional Crop**
Base Run	• Pest resistance develops in 10 years • Short run cumulative yield losses are lower	• Pest resistance develops in 50 years • Long run cumulative yield losses are lower
If Predators Are Also Harmed by Bt-crop	• Pest resistance develops in 10 years • A more severe pest infestation at equilibrium	N/A
If Heterozygote Pests Are Partially Resistant	• Pest resistance develops in 5 years • Refuge policy is futile	• Pest resistance develops in 17 years
Very low initial resistance allele frequency; very high fitness cost of resistance	• Pest resistance develops in 25 years • Cumulative yield losses are lower even in the long run	• Pest resistance develops in 130 years
Very high initial resistance allele frequency; no fitness cost of resistance	• Pest resistance develops in 5 years • Refuge policy is futile	• Pest resistance develops in 15 years • Cumulative yield losses are lower in the medium term
Refuge Policy	• Delays resistance evolution by 15 years	N/A
Refuge Policy: If resistant pests are cross resistant to pesticide	• Delays resistance evolution by only 6 years	N/A
Integrated Pest Management	• Delays resistance evolution by 10 years	N/A

7. THE HERBICIDE TOLERANT CROP (HT) MODEL

The second model developed in this study aims to introduce the feedback processes in the environment to the analysis of long-term impacts of HT crops on farming management. As is the case with the first model, the purpose of this model is to bring forth insights for the evaluation of this new technology in terms of sustainability. The model is built on farm scale considering an isolated land where HT crop is planted. The core aspect is increased herbicide tolerance through natural selection due to overuse of herbicides. This phenomenon, which constitutes the common grounds for the two models, is not specific to HT crops; however it is very likely that it will be intensified via this technology. Hence, to better illustrate the effects of HT crops, the analyses will be performed by simultaneous comparison with its conventional counterparts. The second phenomenon investigated in the model is "superweeds" formation via hybridization of the HT crop with its wild relatives. When hybridization occurs between a HT crop and its weedy relative, the herbicide tolerance transgene can spread to this species making the species resistant to the herbicide which is sprayed to eliminate it. This further complicates weed management, speeding up the spread of resistance in the weed species.

The basic relations in the system are summarized in the causal loop diagram below. The fundamental dynamics of the system is the spread of resistance throughout the population and is generated by the intervention of the agricultural practices with the positive feedback loops at the bottom of the diagram that determine the genotypic ratios in the population. The relations defining the genotypic ratios and resistance allele frequency are identical to those described in the first model; therefore will not be discussed again in this section. The rest of the process is also pretty much the same. When herbicides are used to control the weed population, mortality rate of susceptible weeds increases, which will reduce the subpopulation of susceptible weeds. However, herbicides will not have an effect on the resistant weeds subpopulation. Therefore the frequency of resistance allele in the population will increase, which results in an increasing probability of having an increased number of resistant seeds. The next generation will have a higher number of resistant individuals and if that population is also exposed to the same herbicide, with further lessened susceptible weeds, resistance allele fraction will further increase. These positive feedback loops highlighted at the bottom of the diagram together with the consistent selection pressure may eventually result in the extinction of susceptible individuals. Such a weed population can no longer be controlled by this toxin (and most probably by other toxins with the same mode of action). Therefore, consistent application of a herbicide fails to control the weed population in the long run.

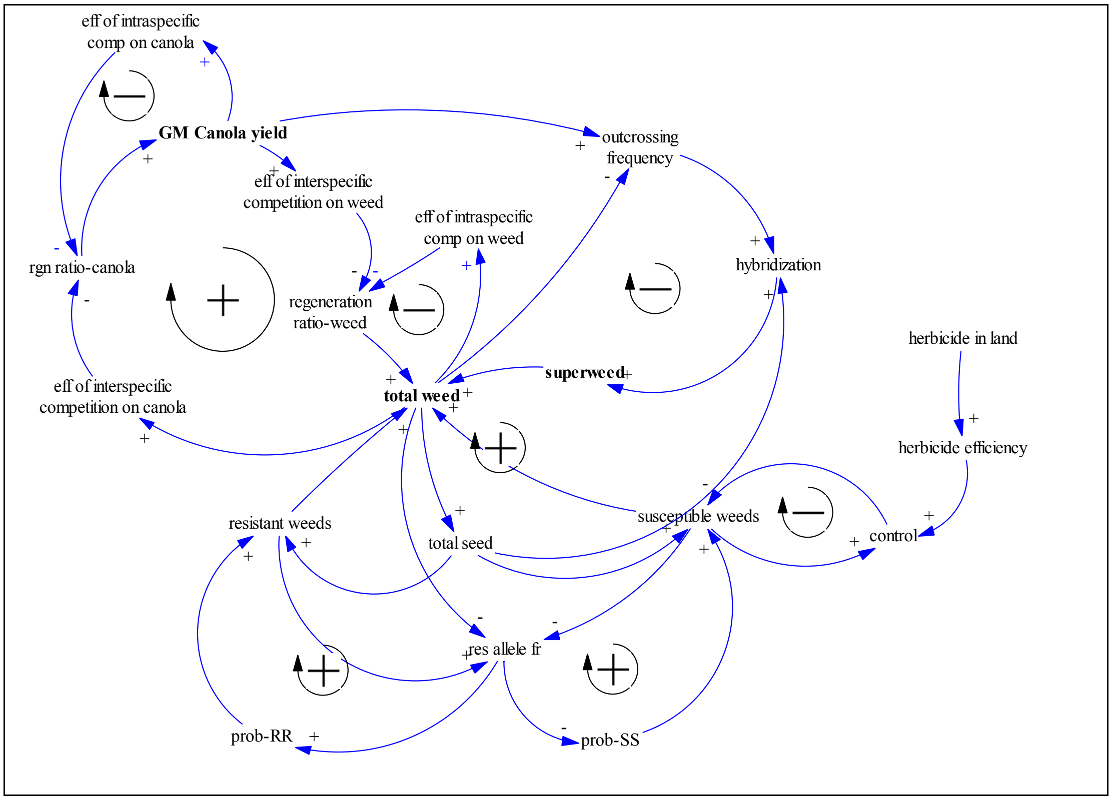


Figure 7.1. Simplified causal loop diagram of the HT-Model

The backbone of the model structure is the competition among plants. Weeds are called pests since they compete with the crop putting a constraint on their growth. When there are more weeds in the land, the effect of competition on the crop increases, which reduces the regeneration rate of the crop. This in return decreases the yield. When the biomass of the crop is reduced, the effect of competition on weed population is less. Hence regeneration rate of weeds increases. Consequently, other things being equal the weed biomass increases, which completes a major reinforcing feedback loop highlighted at the northwest corner of the diagram in Figure 7.1. On the other hand, as weed biomass increases, so does the effect of intraspecific competition on weed, which reduces the regeneration rate of the weed species. This in return decreases the weed biomass, completing the negative feedback loop in the middle of the diagram. This negative feedback loop and its analogous for the crop depicted in the northwest corner of the diagram regulate the plant populations and prevent the plants from growing infinitely.

Another important phenomenon occurs due to the interactions of the weed population with the crop. As the weed biomass increases, so does the amount of seed produced. When there are more seeds, the number of hybrid seeds is higher; hence increasing the total amount of seeds increases the hybridization rate. A higher rate of hybridization implies forming more superweeds, which are essentially weeds that are herbicide resistant. Hence total biomass of weeds is further increased, closing the reinforcing feedback loop in the middle of the diagram. On the other hand, the fraction of hybrid seeds within total seed production is determined by the outcrossing frequency. When a weed plant is surrounded by the crop plants, it is more likely that it be pollinated by a crop plant, which implies that outcrossing frequency increases with increasing crop biomass and decreases with increasing weed biomass. Hence, when the weed biomass is increased, hybridization rate is reduced due to decreasing outcrossing frequency. This results in a reduction in the superweed population, which in return decreases the total weed population, completing the balancing feedback loop at the northeast corner of the diagram in Figure 7.1.

7.1. Main Assumptions of the HT-Model

- There is a single weed species in the field
- There are no pests
- The effect of abiotic factors such as rain or wind on plant populations is constant, hence omitted

- Mutations are negligible
- Resistance is expressed by two alleles of a single gene
- Heterozygote weeds are entirely resistant to herbicide
- All the resistant species, i.e. resistant weeds, GM canola and superweeds are 100% resistant to the herbicide
- Superweeds are morphologically identical to the weeds, except for their reproduction capacity. Hence their seeds can be regarded as weed seeds, treating the second-generation hybrids as weeds.
- A single herbicide is used to treat the weeds.

7.2. Overview of the Model Sectors

The model consists of primarily 3 sectors, being *Crop*, *Weed* and *Superweeds*. The fundamental interaction between these sectors is the competition among these plants. Weeds (including superweeds) compete with canola. This competition determines the size of each plant population and basically the amount of yield from an agro-economic perspective. In order to suppress the weed populations, herbicide is applied once or more throughout the season, which is modeled under *Weed* sector.

7.2.1. Crop Sector

In this sector, the artificial process of planting a crop is modeled; i.e. the structure is responsible for simulating the seeds being sown, plants being grown and being harvested at a predetermined time. Dynamics of the population size (i.e. yield) in this sector is influenced by the feedbacks from *Weeds* and *Superweeds* sectors in the form of competition.

7.2.2. Weeds Sector

In this sector, the weed population is modeled. It is assumed that there is only one weed species of concern in the field. Unlike the crop, seeding rate for weeds is not exogenously set by farmers but determined by their own deposited seeds in soil, which are called seedbanks. Therefore, this sector has two subsectors, one to model the current population of weeds, the *Weed Biomass* and one to model the seedbanks, the *Weed SeedBank.*

Since one concern of the study is the evolution of resistance to herbicides, weed population is disaggregated to three subpopulation stocks with respect to the three genotypes RR, RS and SS to be able to analyze this phenomenon. Each weed stock has its own *SeedBank*, leading to a 6-stock system. As can be seen from the stock-flow diagram in Figure 7.2, the two subsectors are closely interrelated since the size of one is determined by the size of the other. Dynamics of the weed population size is also influenced by the feedback from *crop* sector in the form of competition, and by the herbicide applications both as a mortality factor and a selection pressure mechanism.

Herbicide application is also modeled within this section. The amount of herbicide sprayed is given with respect to the weed biomass. Herbicide usage is a critical factor that affects the sizes of susceptible populations. The effect of herbicide on the weed population as well as the conventional crop is a function of the amount of herbicide in land. Herbicides do not lose effectiveness immediately but have certain persistence in land, which is determined by their half lives. In the model, herbicide in land is a stock filled by the sprayed amount and drained proportionately to its half life.

7.2.3. Superweeds Sector

In GM debates, hybrid weeds containing a transgene that provides resistance to a herbicide, or any other property which increases their fitness to the environments are called superweeds. In this sector, the hybridization process between GM crop and its weed is modeled, which will end up with herbicide resistant weeds. This process is somewhat simplified in the model, as modeling the entire hybridization process is out of the scope of this study.

As can be seen from the stock-flow diagram in Figure 7.2, superweeds are modeled similarly to the weed populations. One stock represents the superweed population; another stock represents the inventory of hybrid seeds. Again, the population is disaggregated with respect to its genotypes. Dynamics of the superweed population size is also influenced by the feedbacks from HT crop in the form of hybridization and competition.

7.3. Background Information: Modeling Competition

Competition may be defined as " the active seeking after, or utilization of a given resource by two or more individuals of the same or different species, the resource itself being actually or potentially limiting" (Putmann, 1984). It is called *intraspecific* if it occurs between individuals of the same species, and *interspecific* if it occurs between the individuals of different species. Competition among plants can be for soil nitrogen, light and other required minerals obtainable from soil. Weeds are plants which successfully compete with the food crop, resulting in a decreased yield. Therefore, modeling competition among the plant species is a key aspect of HT-Model.

In the model, the formulation for the effect of competition is based on the *carrying capacity* concept. Carrying capacity represents the limit exerted by the environment on the population size of a species on a given area. It is assumed that beyond that limit the species can no longer obtain sufficient nutrition from soil and therefore growth is stopped. Hence, as the total biomass on the specific area approaches the carrying capacity of the species of concern, the net growth rate of the species decreases. In the model, competition affects the net growth rate of the species by changing its regeneration ratio.

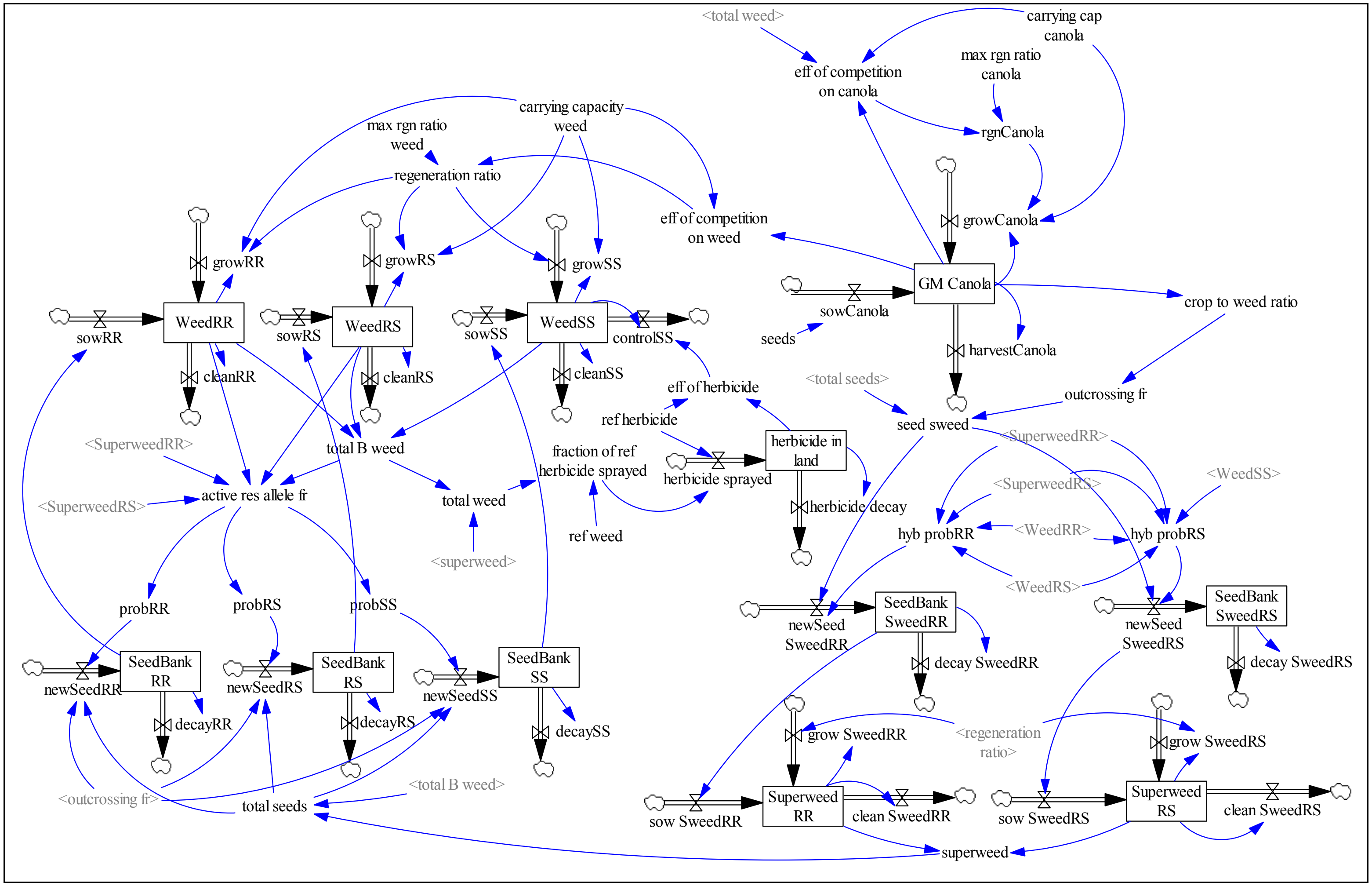


Figure 7.2. Stock-flow diagram of the HT-Model

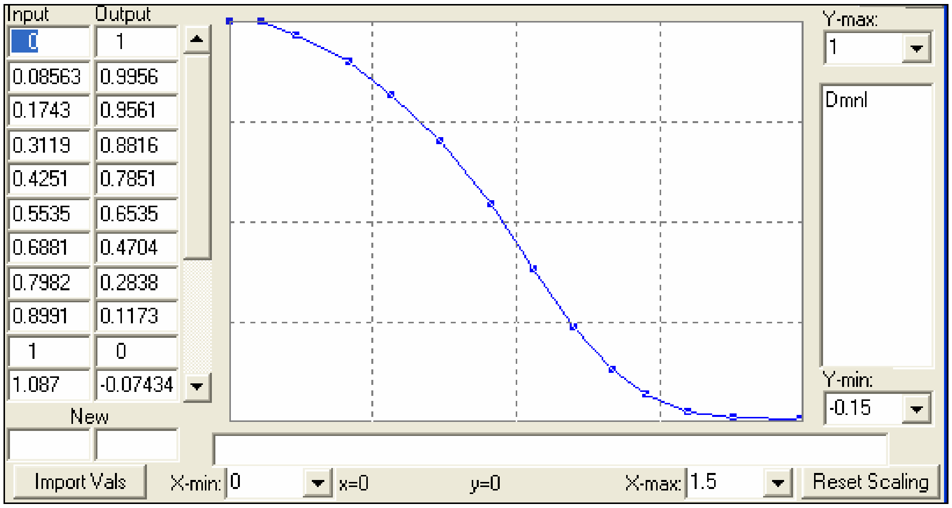
$$\mathit{Net\ Growth\ Rate} = \mathit{Biomass} \times \mathit{Regeneration\ Ratio} \qquad (6.1)$$

$$\mathit{Regeneration\ Ratio} = \mathit{Maximum\ Regeneration\ Ratio} \times \mathit{Effect\ of\ \ Competition} \,(6.2)$$

The effect of competition on species *i* is a function of the ratio of total biomass to the carrying capacity of the species.

$$\mathit{Effect\ of\ \ Competition\ on\ Species\ i} = f\left(\frac{\mathit{Biomass}_i + \alpha \times \sum \mathit{Biomass}_j}{\mathit{carrying\ capacity}_i}\right) \qquad (6.3)$$

In the equation, α represents the relative impact of the competing species on resource usage. α=1 when the competing species exploits the resource just in exactly the way as the species under concern does. *Effect of Competition on Species i* approaches zero when this scaled total biomass approaches the carrying capacity of the species of concern. If the carrying capacity is overshoot, the function takes negative values implying a negative net growth rate. Hence the shape of the function is as follows:



Input	Output
0	1
0.08563	0.9956
0.1743	0.9561
0.3119	0.8816
0.4251	0.7851
0.5535	0.6535
0.6881	0.4704
0.7982	0.2838
0.8991	0.1173
1	0
1.087	-0.07434

Y-max: 1

Dmnl

Y-min: -0.15

X-min: 0

x=0

y=0

X-max: 1.5

Figure 7.3. Effect of competition as a function of the ratio of total biomass to the carrying capacity of the species of concern

7.4. Description of the Crop Sector

7.4.1. Background Information: *GM canola*

In order to be able to calibrate the model and build a platform for further in-depth research, it is wise to concentrate on a specific GM crop for this study. *Canola* (Oilseed rape or *Brassica Napus*) is appropriate for demonstration purposes since it hybridizes with wild relatives relatively more frequently which makes it more probable to have superweeds as a problem. In the literature, high hybridization rates between canola and birdseed rape (field mustard, wild turnip or *Brassica rapa*) have been reported and birdseed rape is a common weed in many places where canola is grown which makes the situation problematic (Jorgensen 1994, Halfhill *et al.*, 2002, etc). This study will focus on canola and birdseed rape as the crop-weed pair in order to better demonstrate the potential drawbacks of superweed formation.

Canola seeds tolerant to various herbicide classes have been grown commercially for more than a decade. The reason behind modifying crops for herbicide tolerance is that while application of any chemical to a crop has the potential to cause an unfavorable response, herbicides typically have a greater potential to cause damage than other chemicals that are applied since they have the ability to kill plants. These yield losses may be as high as 25% depending on the type and timing of the herbicide. (ISU Weed Science Online, 1996)

The genetic modification enables glyphosate to be sprayed on weeds without destroying the canola plants. Major herbicide tolerant varieties in the market are resistant to glyphosate, which is an effective, non-selective herbicide. Hence, in this study glyphosate tolerant canola will be studied.

7.4.2. Description of the Structure

In the model, canola biomass is represented by the *GM Canola* stock. This stock is filled with *sowCanola* and *growCanola* inflows, and drained by *harvestCanola* outflow. The accumulation is in terms of biomass and has the unit kg/da.

Canola seeds are sown at a predetermined amount and date, and the crop is harvested completely at a predetermined harvest date as the plants reach maturity. Growth of the crop is limited by the carrying capacity of the area for that crop and competition with other plants as explained under Section 7.3.

Sowing rate is determined by the predetermined amount *seeds*, which denotes the required number of seeds per area to achieve the intended plant population, and weight of these seeds *seedweight* (Figure 7.6). This inflow is activated at the predetermined dates of seeding, which is achieved by the auxiliary (and binary) variable *TimeToSeed*. Finally, *seeding period* represents the necessary time for the seeding process to be completed. Therefore:

$$sowCanola = \frac{seedweight \times seeds \times TimeToSeed}{seeding\ period} \tag{6.4}$$

Growing rate is determined by the biomass of the plant and regeneration ratio, *rgrCanola*, which is per month growth ratio of the plant.

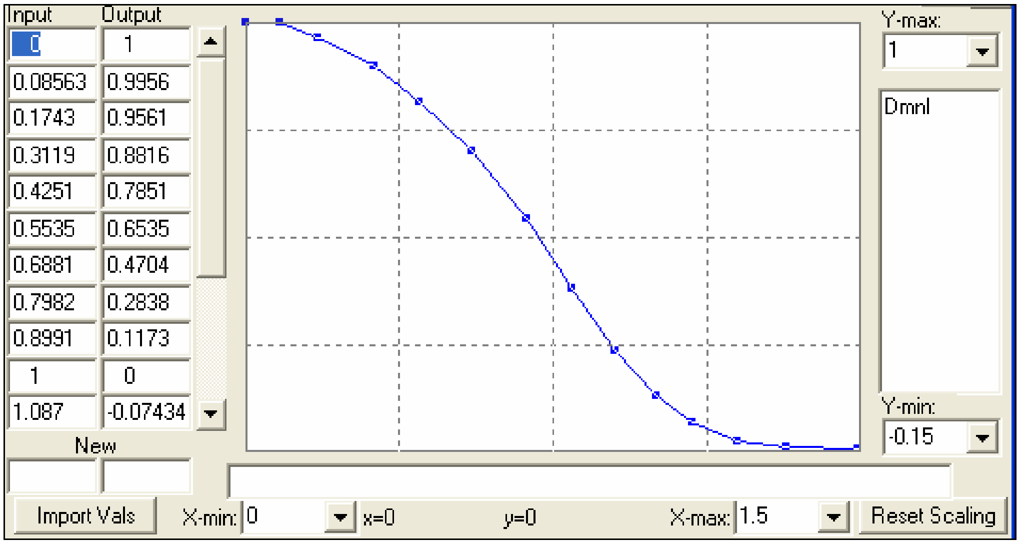
$$growCanola = GM\ \ Canola \times rgrCanola \tag{6.5}$$

However, as explained above, regeneration ratio is not a constant. It decreases with increasing competition. Therefore, in the model there is a maximum attainable ratio of growth *max rgn ratio Canola* and the *eff of competition on canola* which determines the actual regeneration ratio at a given time.

$$rgrCanola = max\ rgn\ ratio\ Canola \times eff\ \ of\ \ competition\ on\ canola \qquad (6.6)$$

Eff of competition on canola is a function of relative biomass on land, where relative biomass represents the ratio of *carrying cap canola* which is already occupied. As explained in Section 7.3, interspecific competition is scaled by a constant *alfa Canola* which represents the relative impact of the competing species on resource usage.

$$eff\ \ of\ \ competition\ on\ canola = f\,(\frac{GM\ Canola + alfa\ Canola \times total\ weed}{carrying\ cap\ canola} \qquad (6.7)$$



Input	Output
0	1
0.08563	0.9956
0.1743	0.9561
0.3119	0.8816
0.4251	0.7851
0.5535	0.6535
0.6881	0.4704
0.7982	0.2838
0.8991	0.1173
1	0
1.087	-0.07434

Y-max: 1

Dmnl

Y-min: -0.15

X-min: 0

x=0

y=0

X-max: 1.5

Figure 7.4. Effect of competition on canola as a function of relative biomass

HarvestCanola is the complete draining of the *GM Canola* stock. It occurs at a predetermined time period, which is modeled by the auxiliary variable *TimeToHarvest.* Harvesting takes a period of time equivalent to *harvesting period.*

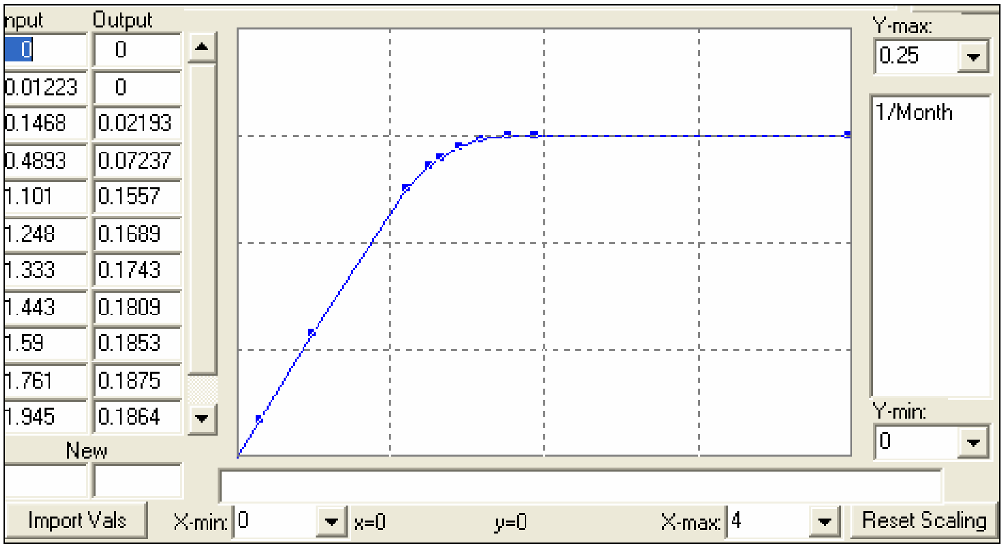
$$harvestCanola = \frac{GM\ \ Canola \times TimeToHarvest}{harvesting\ \ period} \tag{6.8}$$

When conventional canola is planted instead of a GM variety, herbicide application results in a yield loss due to crop response. This loss is modeled via the outflow *yield loss due to herbicide*, which is a function of *eff of herbicide on canola*.

$$yield\ \ loss\ due\ to\ herbicide = GM\ \ Canola \times eff\ \ of\ \ herbicide\ on\ canola \tag{6.9}$$

Eff of herbicide on canola is a function of herbicide density.

$$eff\ \ of\ \ herbicide\ on\ canola = f\,(\frac{herbicide\ in\ land}{ref\ \ herbicide}) \tag{6.10}$$



nput	Output
0	0
0.01223	0
0.1468	0.02193
0.4893	0.07237
1.101	0.1557
1.248	0.1689
1.333	0.1743
1.443	0.1809
1.59	0.1853
1.761	0.1875
1.945	0.1864

Y-max: 0.25

1/Month

Y-min: 0

X-min: 0

x=0

y=0

X-max: 4

Figure 7.5. Effect of herbicide on canola as a function of herbicide density

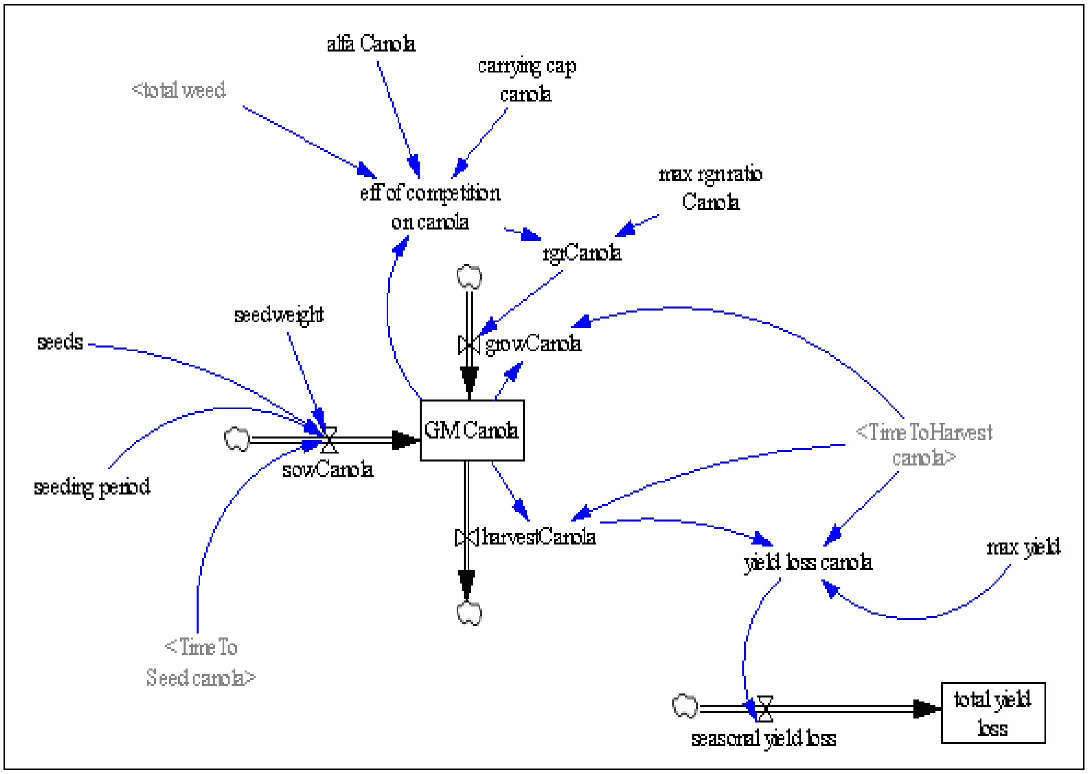


Figure 7.6. Stock-flow diagram of the Crop sector

7.5. Description of the Weed Sector

7.5.1. Description of the Structure for Weed Biomass Subsector

Unlike the crops, seeding rate for weeds is not exogenously set by farmers but determined by their own deposited seeds in soil, which are called seedbanks. Therefore, to model the weed population, two stocks are needed: one for the current weed biomass, one for the deposited seeds in the soil. Since one concern of the model is the evolution of resistance to herbicides, weed population is disaggregated to three sub population stocks with respect to the three genotypes RR, RS and SS as *WeedRR*, *WeedRS* and *WeedSS* respectively. The overall weed population is denoted by *total B weed*.

$$total\ B\ weed = WeedRR + WeedRS + WeedSS \qquad (6.11)$$

Each weed stock has its own *SeedBank*, leading to a 6-stock system as can be seen from the stock-flow diagram in Figure 7.2. Glyphosate resistance is dominant and segregates in accordance with Mendelian principles. (Hall *et al.*, 2000) Therefore, if R represents resistance allele, individuals with genotypes RR and RS are resistant and have the same phenotype, whereas individuals with SS genotype are susceptible. Only susceptible individuals are considerably controllable by herbicide applications and there is no evidence for a fitness cost of resistance. (Danish EPA, 1999)

In order to discriminate the variables belonging to each *Weed* subpopulation, the genotype is used as a suffix while naming the variables used in the model. For example, *sowRR* represents the seeding inflow of the stock *WeedRR*. However, in providing the variable definitions in this chapter, these suffixes are omitted for identical variables used for all subpopulations.

Growth of weeds is similar to that of crops. Weed stocks are filled with *sow* and *grow* inflows, and drained by *clean* outflow. Only *WeedSS* has the additional outflow *control,* which models the death of susceptible weeds due to herbicide applications. *clean* outflow is used due to the practical fact that during the harvest time the farmland is also cleaned from weeds. Since this process would in any case dominate death by natural means, a death outflow is not modeled separately.

Sow inflow of the *Weed* stock is determined by the *SeedBank* size and germination frequency. Germination frequency, *grm*, is the fraction of seeds in the *SeedBank* that begins to grow per time.

$$sow = SeedBank \times grm \tag{6.12}$$

Grow inflow is determined by the size of *Weed* stock and regeneration ratio, which is modeled the same way as for the crop.

$$grow = Weed \times rgr\ weed \tag{6.13}$$

Clean outflow is activated when it is the harvest period on the farmland. Like harvest, it drains its stock completely.

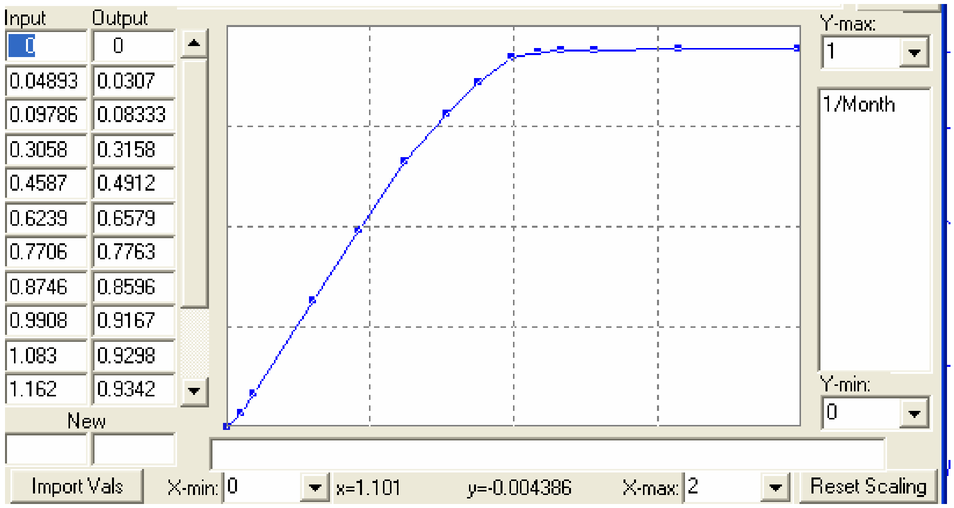
$$clean = \frac{Weed \times TimeToHarvest\ Weed}{harvesting\ period} \tag{6.14}$$

Control outflow models the impact of herbicide on the *Weed* stock. Herbicide kills a fraction of susceptible weed population and this fraction is modeled as *effect of herbicide.*

$$control = Weed \times effect\ of\ herbicide \tag{6.15}$$

Effect of herbicide is a graphical function of the normalized amount of herbicide.

$$effect\ of\ herbicide = f\left(\frac{herbicide\ in\ land}{ref\ herbicide}\right) \tag{6.16}$$



Input	Output
0	0
0.04893	0.0307
0.09786	0.08333
0.3058	0.3158
0.4587	0.4912
0.6239	0.6579
0.7706	0.7763
0.8746	0.8596
0.9908	0.9167
1.083	0.9298
1.162	0.9342

Y-max: 1
1/Month
Y-min: 0
X-min: 0
x=1.101
y=-0.004386
X-max: 2

Figure 7.7. Effect of herbicide on susceptible weeds as a function of herbicide density

Herbicide in land is a stock filled by *herbicide sprayed* and drained by *herbicide decay*. Herbicide sprayed is modeled in two different ways for two different herbicide strategies. In the first strategy, a fraction of *ref herbicide* which is determined by weed density is sprayed when it is *TimeToSpray*. Hence:

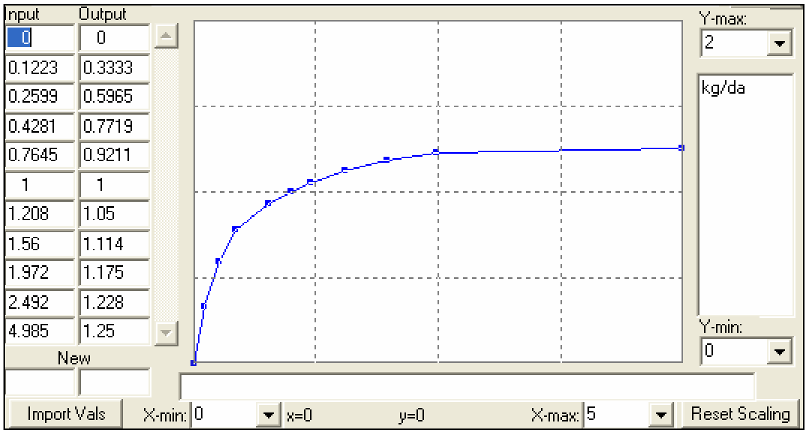
$$herbicide\ sprayed =$$

$$fraction\ of\ \ ref\ \ herbicide\ sprayed \times ref\ \ herbicide \times TimeToSpray \qquad (6.17)$$

Fraction of ref herbicide sprayed is a function of weed density.

$$fraction\ of\ \ ref\ \ herbicide\ sprayed = f\left(\frac{total\ \ weed}{ref\ \ weed}\right) \qquad (6.18)$$

When there is no weed, herbicide is not sprayed. As weed biomass approaches its reference value the prescribed amount of herbicide, *ref herbicide*, is sprayed and the function returns 1. When weed biomass exceeds its reference value, herbicide usage exceeds the prescribed amount, increasing up to a threshold value.



Input	Output
0	0
0.1223	0.3333
0.2599	0.5965
0.4281	0.7719
0.7645	0.9211
1	1
1.208	1.05
1.56	1.114
1.972	1.175
2.492	1.228
4.985	1.25

Y-max: 2

kg/da

Y-min: 0

X-min: 0

x=0

y=0

X-max: 5

Figure 7.8. Fraction of the prescribed amount of herbicide that is sprayed as a function of weed density

In the second strategy, the amount of herbicide to spray is predetermined and is equivalent to *ref herbicide*. Hence *herbicide sprayed* becomes:

$$herbicide\ sprayed = TimeToSpray \times ref\ \ herbicide \tag{6.19}$$

Sprayed herbicide accumulates in the soil; however this accumulated chemical breaks down and loses effectiveness gradually. *herbicide decay* is determined by *herbicide in land* and *decay ratio*. Decay ratio is estimated from the half life of glyphosate.

$$herbicide\ decay = herbicide\ in\ land \times decay\ ratio \tag{6.20}$$

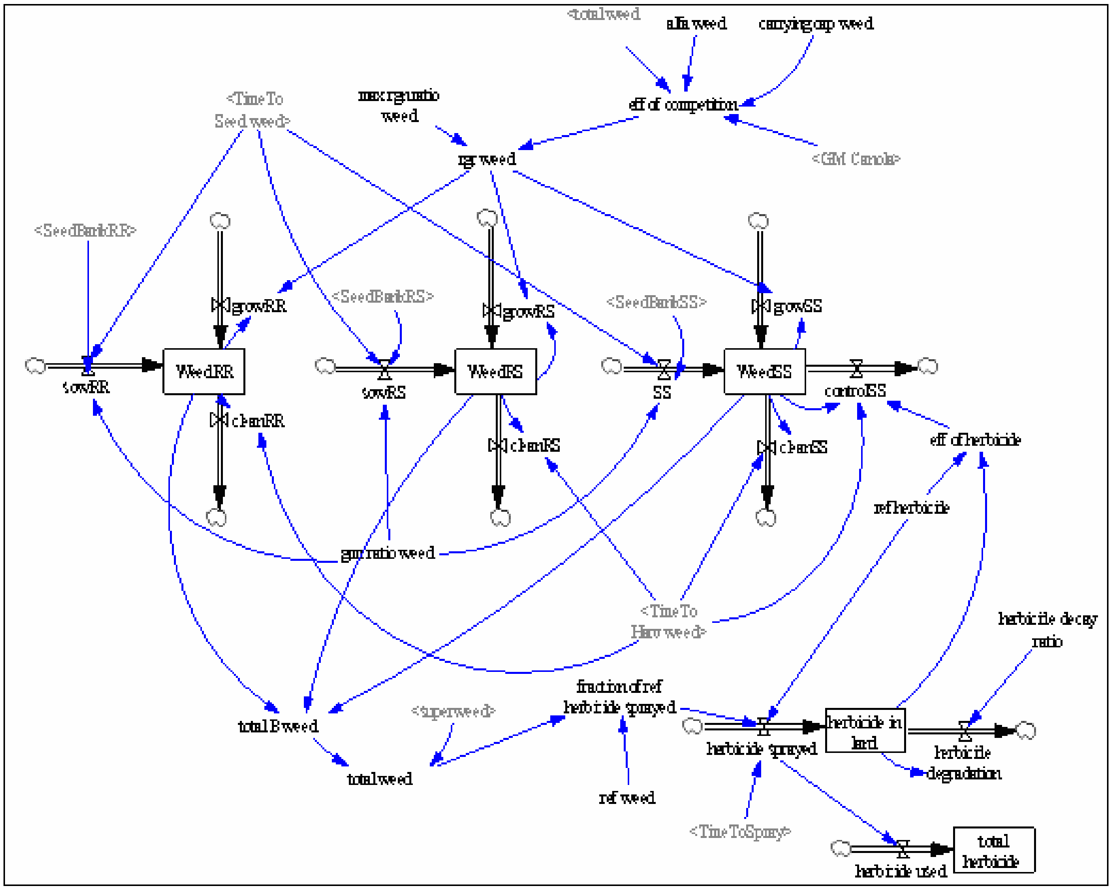


Figure 7.9. Stock-flow diagram of the Weed Biomass subsector

7.5.2. Description of Structure for Weed Seedbanks Subsector

Seedbank can be thought as a bank into which the species deposit their annual seed production. When seeds are sown, this stock of seeds is reduced. Moreover some of the seeds in this stock decay or die due to various reasons throughout the year. Therefore a *SeedBank* stock is filled by *newSeed* inflow and drained by *grm* (germinated seed) and *decay* outflows (Figure 7.10). Its unit is determined as kg/da to be in accordance with other biomass units in the model, though in the literature seeds/da is the preferred unit. There are three *SeedBank*s corresponding to the three genotypes of *Weed*s, being *SeedBankRR, SeedBankRS and SeedBankSS.*

New seeds are formed when plants reach maturity and then they are buried in the soil, i.e. deposited in the seedbank. Seed production is determined by the biomass at harvest time, and the *harvest index*, which indicates the ratio of seeds to overall biomass. However a portion of these seeds are lost mostly due to predation, which is accounted for by the variable *seedloss* in the model. Seed production by superweeds also contributes to *total seed*. Hence, the variable *total seeds* is computed as:

$$\text{total seed} =$$

$$(\text{total B weed} \times \text{harvest index} + \text{superweed} \times \text{HI Sweed}) \times \text{seedloss} \qquad (6.21)$$

A fraction of these seeds (*outcrossing fr*) are formed though hybridization, hence are deposited into the seedbanks of superweeds. The remaining seeds are directed into weed seedbanks with respect to the associated genotypic frequencies. The proportion of these seeds that belong to seedbanks RR, RS and SS are p^2, 2pq and q^2, respectively, where p is the frequency of the resistance allele in the population. In the model, these proportions are called *probRR*, *probRS* and *probSS* respectively.

Therefore

$$newSeedRR = probRR \times total\ seed \times (1 - outcrossing\ fr) \tag{6.22}$$

The frequency of resistance allele, which determines the genotypic frequencies, depends on the current population of *WeedRR*, *WeedRS*, *SuperWeedRR* and *SuperWeedRS*. It is called the *active res allele fr* in the model since it excludes the genotypic distribution of the seedbanks, which also determine the genotypic frequencies of the population. However, to compute the genotypic distribution of new seeds, only the genotypes of individuals who produce these seeds are relevant.

Each RR seed (*Weed* or *Superweed*) contributes two R alleles to the gene pool, whereas RS seeds only one. Each individual has two alleles for resistance trait. Since seed production is proportional to the related biomass and its harvest index, the formulation for *active resistance allele fr* becomes:

$$\frac{(2 \times WeedRR + WeedRS) \times harvest\ index + (2 \times SuperweedRR + SuperweedRS) \times HI\ Sweed}{2 \times (total\ \ weed \times harvest\ index + superweed \times HI\ Sweed)} \tag{6.23}$$

The amount of decaying seeds is determined by the size of the *SeedBank* and the proportion of the seeds that decay per month, i.e. *decay fr*.

$$decay = SeedBank \times decay\ fr \tag{6.24}$$

Finally, germination outflow *grm* is equal to *sowWeed* inflow.

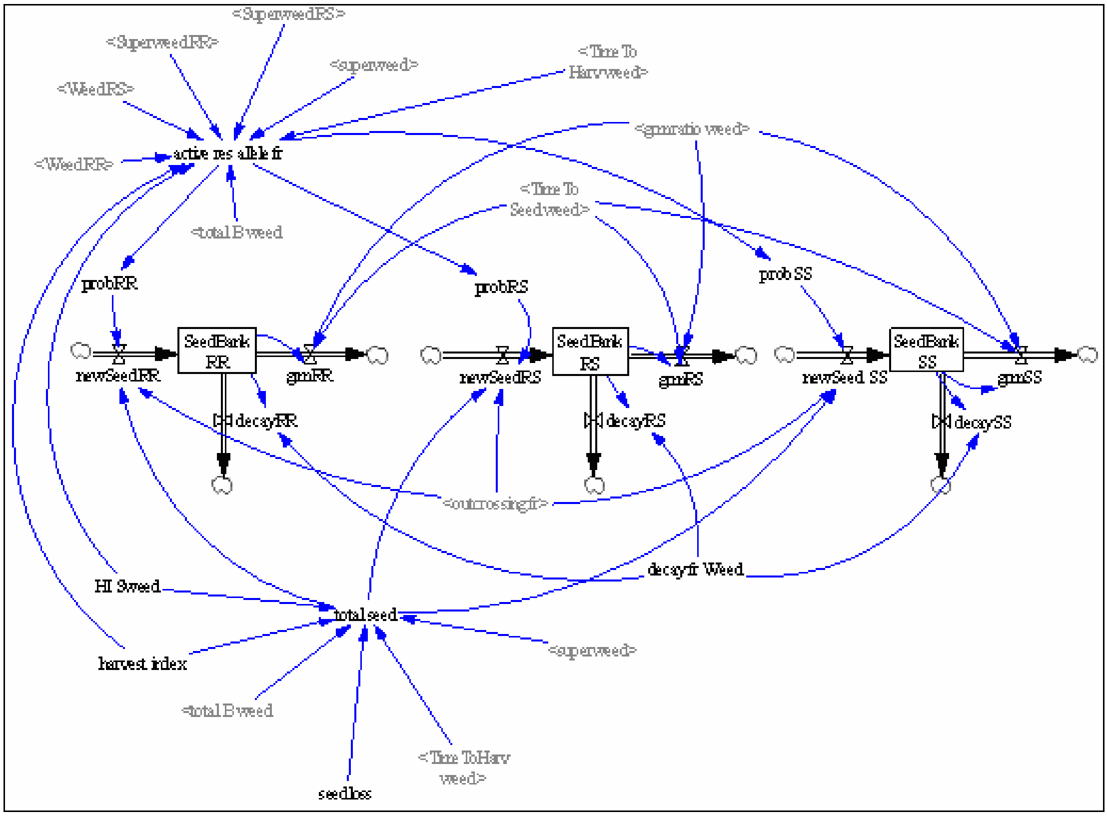


Figure 7.10. Stock-flow diagram of the Weed Seedbanks subsector

7.6. Description of the Superweeds Sector

7.6.1. Background Information: Hybridization

Gene flow is the movement of genes between populations of a species (Kwon *et al.,* 2001). In plants, this occurs via dispersal of seeds or pollens. Gene flow can also occur from a crop to wild plants that are sexually compatible with the crop and hybridization takes place between these plants. Before the risk assessment of GM crops has been an issue, it was thought that successful hybridization between crops and wild relatives was infrequent. With an increasing research interest on this topic, it was discovered that hybridization is the rule rather than the exception. (Halfhill *et al.,* 2002) The risk with the HT crops is that transgenes will move to wild relatives resulting in weed shifts, which may trigger unpredictable ecological consequences. If this gene is expressed in a weed species, it may considerably increase the fitness of the species in nature. For example, weeds containing a transgene that provides resistance to a herbicide would be a problem for the agricultural system. In GM debates, such weeds are called s*uperweed*s, implying their increased fitness.

Yet, not all hybrids are fit to survive and establish new, independent populations. Relative fitness of the first and advanced generations is to be estimated to comment on the hybrid's chance to persist. The first generation of the hybrids between two species is called F1 hybrids. F2 hybrids are second-generation hybrids resulting from crosses between F1 hybrids. On the other hand, there are backcross hybrids BC1, resulting from crossings between F1 and parent species, and BC2 from crossings between BC1 hybrids. In the literature, there are various studies addressing the relative fitness of these generations of various crops, including HT canola (Hails *et al.*, 2005). Unlike many other species, F1 generation of canola and birdseed rape hybrids does not suffer from severe reduced fitness. Only the seed productivity is reduced, which affects the population size of F2 and BC1 generations (Hails *et al.*, 2005). GM canola with the brand name Quest is homozygous in glyphosate resistance (Hall *et al.*, 2000); hence F1 hybrids are either RR or RS, both resistant to glyphosate.

7.6.2. Description of the Structure

In the model, *Superweed*s are modeled similarly to the weed populations: *Superweed* stocks represent the superweed population, whereas *SeedBank Sweed* stocks represent the inventory of hybrid seeds (see the stock-flow diagram in Figure 7.8).

Since modeling the entire hybridization process is out of the scope of this study, it is assumed that after F1 generation, seeds of superweeds are transferred to the weed seedbanks. Hence backcrosses are not modeled. This is justified with the fact that after one generation, many of the progeny are similar to the weeds (Halfhill *et al.,* 2002). Therefore, in the model having two stocks for *Superweed*s is sufficient, which are *SuperweedRS* and

SuperweedRR. These stocks are filled with seeds from their seedbanks *Seedbank SweedRS* and *Seedbank SweedRR* respectively. Seeds produced by superweeds are a part of *total seeds,* which are distributed among weed seedbanks regarding the genotype frequencies.

In formulating the *Superweed* population, the typical weed formulations are used choosing growth parameters identical to those of *Weed*'s. *Seedbank Sweed* is again structurally identical to the previous seedbank formulations. The only difference is in the formulation of *seed Sweed.* These hybrid seeds that will enter the seedbank of superweeds are modeled as a fraction of total seeds produced by the weed population. This fraction represents the hybridization rate between the two species and is denoted by *outcrossing fr*.

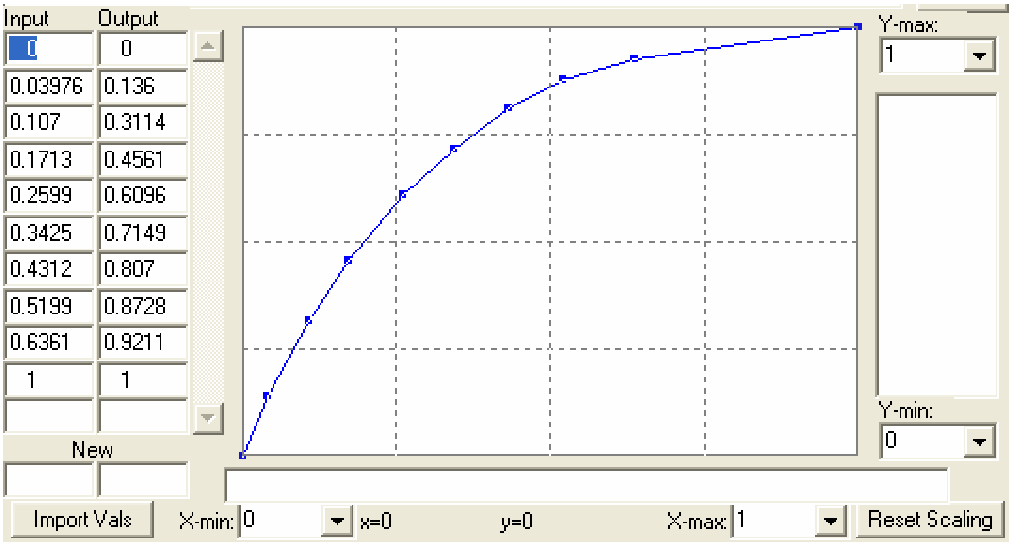
$$seed\ sweed = total\ seed \times outcrossing\ fr \tag{6.25}$$

The more the weed is surrounded by canola plants, the higher the hybridization rate between the two. Different studies point out a quite wide range of hybridization rates, yet all emphasize that as the distance between canola and the weed decreases and as the ratio of crop to wild relative increases, hybridization rate increases (Halfill *et al.,* 2002). Hence in the model, *outcrossing fr* is a function of crop to weed ratio. A reference value which occurs at high crop to weed ratios is provided and the effect of relative abundance of crop is accounted for by *eff of crop to weed ratio on hybridization*, which is a graphical function of *crop to weed ratio*.

$$outcrossing\ fr = ref\ \ frequency \times eff\ \ of\ \ crop\ to\ weed\ \ ratio\ on\ hybridization \tag{6.26}$$

$$eff\ \ of\ \ crop\ to\ weed\ ratio\ on\ hybridization = \mathrm{f}\left(\frac{crop\ to\ weed\ ratio}{ref\ \ ratio}\right) \tag{6.27}$$

$$crop\ to\ weed\ ratio = \frac{GM\ \ Canola}{total\ weed} \tag{6.28}$$



Input	Output
0	0
0.03976	0.136
0.107	0.3114
0.1713	0.4561
0.2599	0.6096
0.3425	0.7149
0.4312	0.807
0.5199	0.8728
0.6361	0.9211
1	1

Y-max: 1

Y-min: 0

X-min: 0

x=0

y=0

X-max: 1

Figure 7.11 Effect of crop to weed ratio on hybridization frequency as a function of crop to weed ratio

Since superweeds are morphologically closer to weeds, many parameters (regeneration ratio, germination ratio, decay ratio, seed loss) are taken equal to those of W*eed*'s. The only difference is in harvest index. Hybrids reproduce less than their parents, forming a fewer number of seeds. Therefore harvest index for superweeds, *HI Sweed,* is taken to be lower than that of canola and the weed.

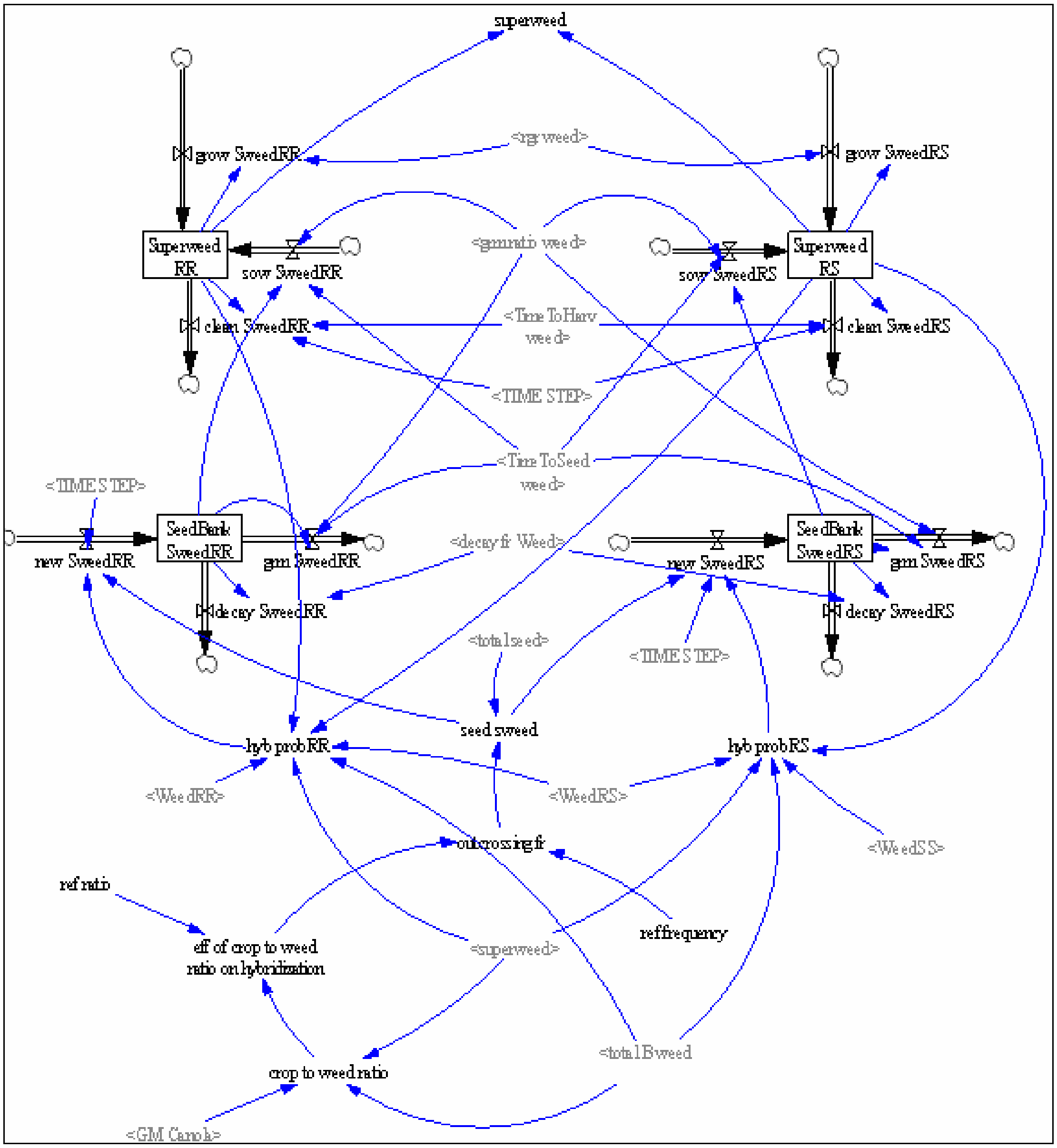


Figure 7.12. Stock-flow diagram of Superweed sector

8. ANALYSIS AND VALIDATION OF THE HT-MODEL

In order to analyze the system behavior, the model described in the previous chapter is simulated via Vensim DSS simulation software. Simulation time unit is chosen to be months and a sufficiently small value of time step (1/32) is used. Since some of the impacts are evident only in the long run, time horizon of the study is set to 500 months. In this chapter, base behavior of the HT-model will be analyzed and the results of the experiments conducted in order to test the validity of the HT-model are summarized.

8.1. Base Behavior of the Model

Base behavior of the model will be analyzed under two herbicide strategies and by simultaneous comparison with conventional canola. In the first strategy, the *variable herbicide strategy*, weed biomass is observed twice a season and the amount of herbicide to spray is determined as a function of this density. In the second strategy, the *constant herbicide strategy*, herbicide is sprayed twice a season at a predetermined amount.

8.1.1. Herbicide Sprayed as a Function of the Weed Biomass

The fundamental behavior of the model is the spread of resistance in the population, which is depicted by the graph of *res allele fr*. Similar to the IR-Model, under the consistent selection pressure due to herbicide, this ratio exhibits an S-shaped growth. However, in this case population in successive seasons includes susceptible individuals coming from the seedbank, which occasionally decreases the frequency of resistant alleles.

As seen in Figure 8.1, spread of resistance in the population is fast in spite of the very low initial resistance allele frequency (0.0000003). This is due to the high efficacy of glyphosate, dominance of the resistance allele and formation of superweeds which further contributes resistance alleles into the seedbanks.

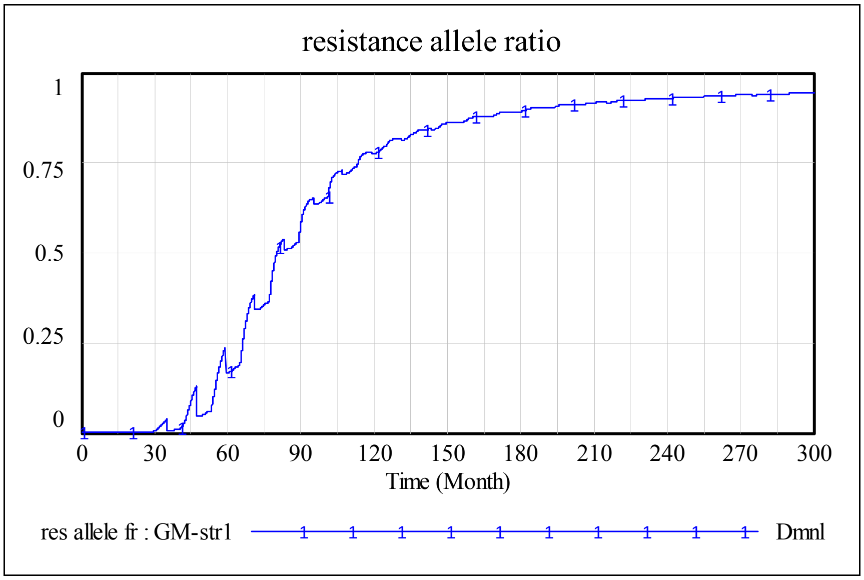


Figure 8.1. Dynamics of resistance allele ratio in the base run under the variable herbicide strategy

When the graph of *total weed* is analyzed (Figure 8.2), it is seen that the weed population can be suppressed by herbicide applications at first. However, with increasing resistance in the population, the herbicide loses effectiveness and the weed population attains infestation levels.

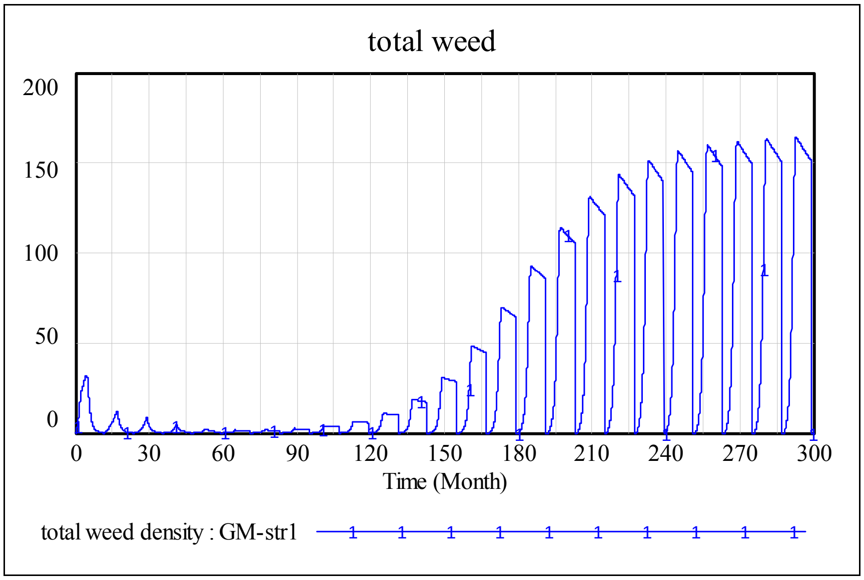


Figure 8.2. Dynamics of the weed population in the base run under the variable herbicide strategy

As can be seen from the graph in Figure 8.3, yield losses are very small initially since the weed population is suppressed. Yet, with increasing weed biomass they increase up to one third of the expected yield.

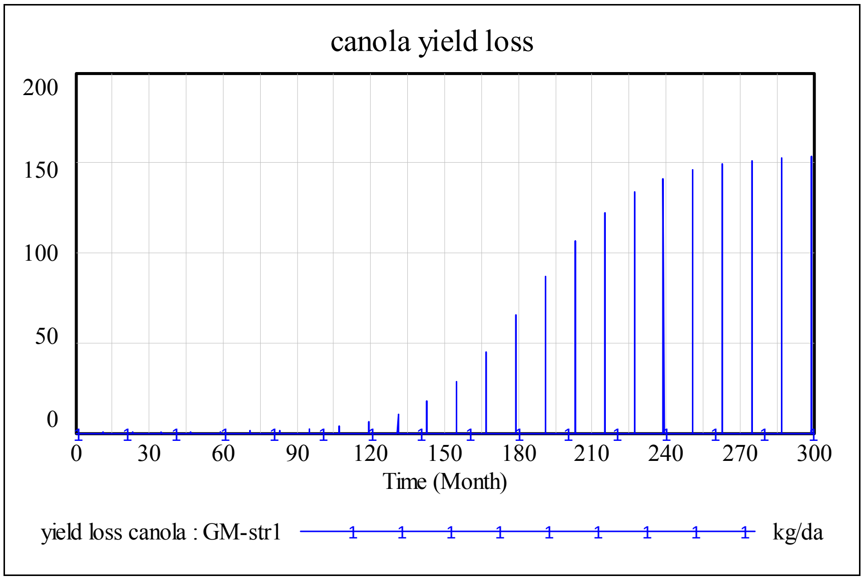


Figure 8.3. Canola yield loss in the base run under the variable herbicide strategy

Though the population of superweeds is relatively small (Figure 8.4), it has a particular role in increasing yield losses. First, it consists solely of resistant individuals, which means that the superweed population cannot be suppressed by the herbicide. Second, it has a considerable contribution of resistance allele into the seedbanks, which increases the probability of having resistant individuals.

If the land is planted with a conventional canola variety instead of GM Canola, the crop also suffers from the effects of the herbicide (see the modified equations for conventional canola in Appendix D.2). In the absence of weeds, the yield loss caused by two times applying glyphosate throughout the season is taken to be 4% (ISU Weed Science Online, 1996). In addition, superweeds are not formed since conventional canola seeds are not resistant to the herbicide.

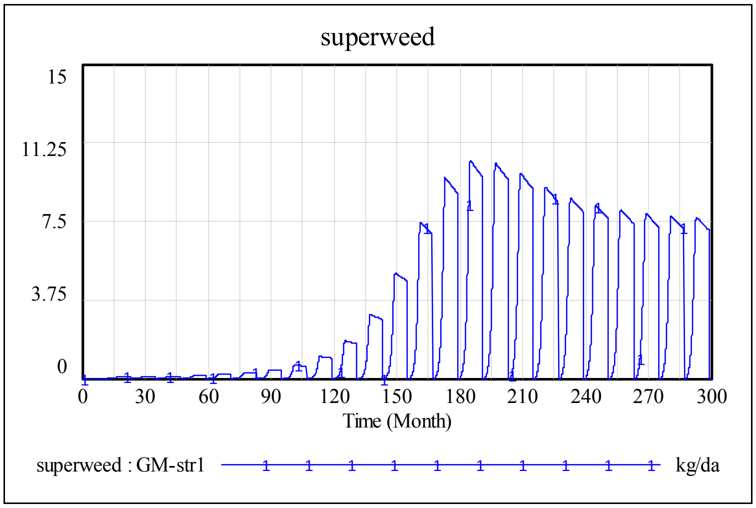


Figure 8.4. Dynamics of superweed population in the base run under the variable herbicide strategy

When the dynamics of the weed population under the cases of GM and conventional canola are compared, it is seen that weed population resurges earlier in the GM case (Figure 8.5). Since superweeds increase the resistant weed population, more weed survives the herbicide, and more herbicide is sprayed. Increased herbicide usage results in an increased rate of resistance development as seen in Figure 8.6.

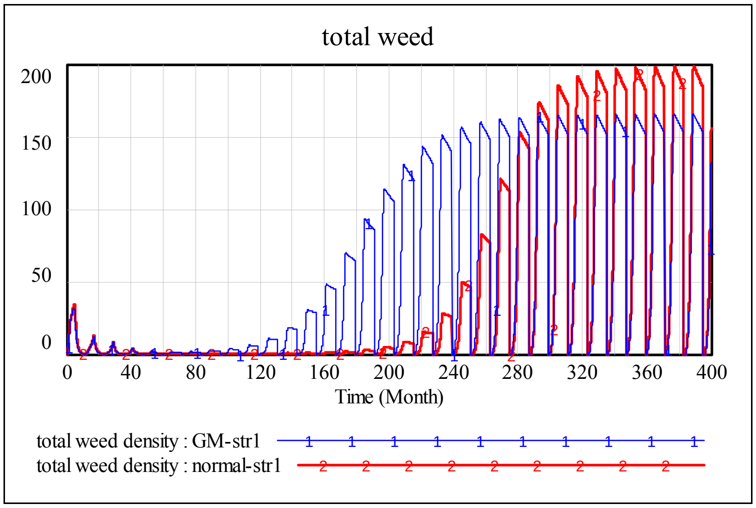


Figure 8.5. Comparison of weed population dynamics in GM canola and in conventional canola fields under the variable herbicide strategy

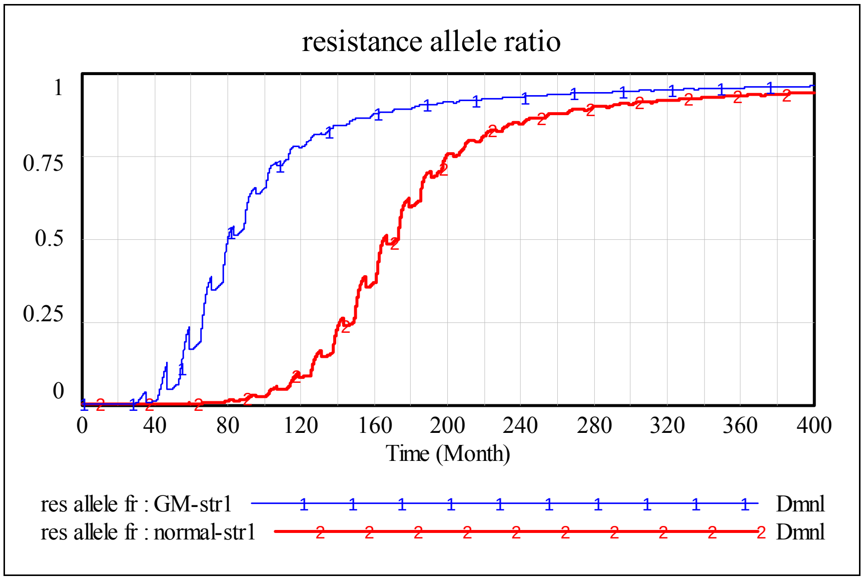


Figure 8.6. Comparison of resistance allele ratio dynamics in GM canola and in conventional canola fields under the variable herbicide strategy

The reason behind the higher equilibrium level of weed biomass in conventional canola is that conventional canola suffers from yield losses due to herbicide (as can be seen from Figure 8.7), which results in a lower canola biomass; hence a higher weed biomass due to decreased competition.

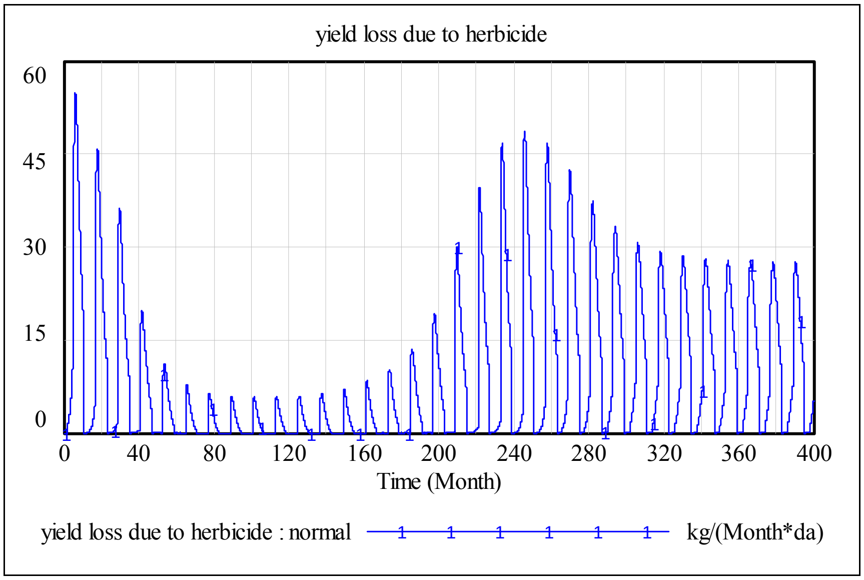


Figure 8.7. Canola yield loss due to herbicide when conventional canola is planted under the variable herbicide strategy

When cumulative yield losses are compared, it can be seen that in the short run planting GM canola provides higher yield efficiency (Figure 8.8). However, this comparative benefit decreases in a relatively short period due to faster evolution of resistance in the weed population in GM canola case compared to that in conventional canola.

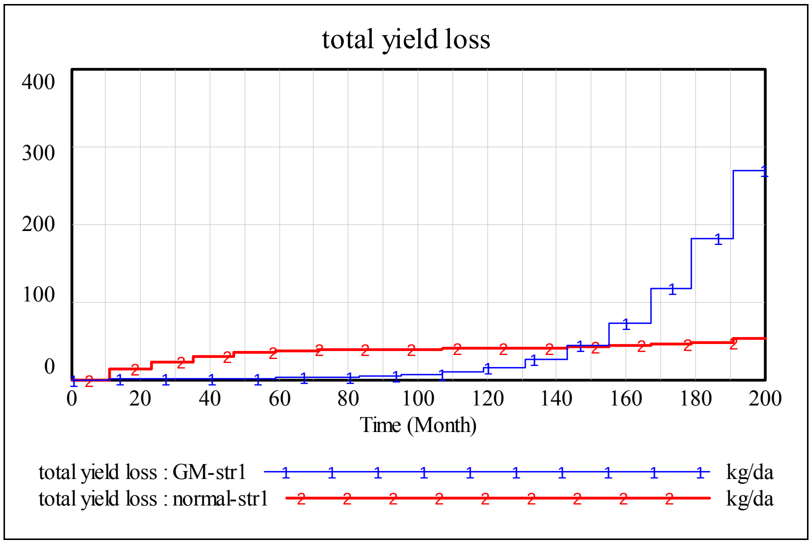


Figure 8.8. Comparison of cumulative yield loss occurring in GM canola and in conventional canola fields in the short run under the variable herbicide strategy

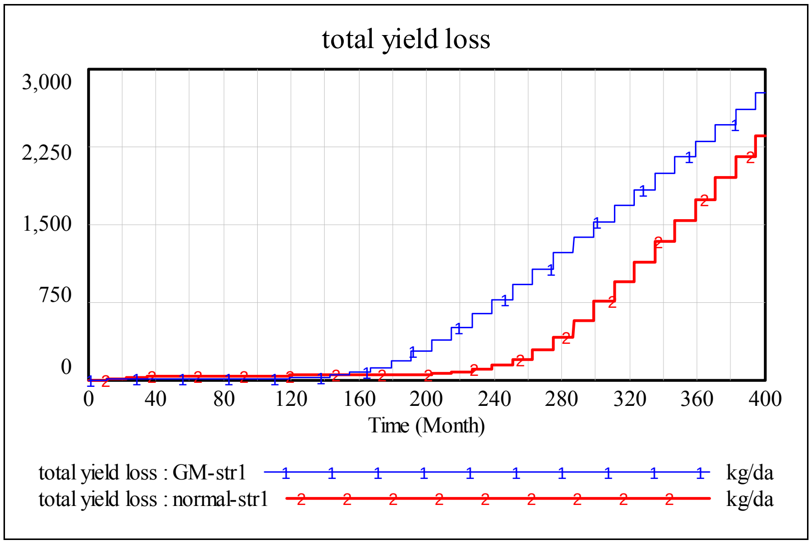


Figure 8.9. Comparison of cumulative yield loss occurring in GM canola and in conventional canola fields under the variable herbicide strategy

Furthermore, cumulative herbicide usage is higher when GM canola is planted (Figure 8.10), which increases the input costs in agriculture and, perhaps more critical, invokes concerns for food safety.

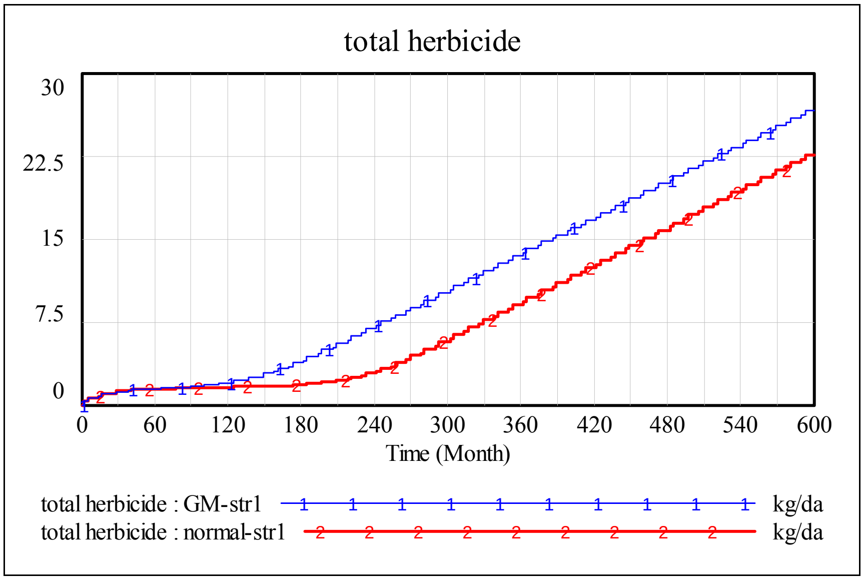


Figure 8.10. Comparison of cumulative herbicide usage in GM canola and in conventional canola fields under the variable herbicide strategy

In these basic results, hybridization rate is kept in an average level regarding the reported values for canola and its weed birdseed rape. A higher/lower rate will increase/decrease the yield loss difference between GM and conventional canola, which will be demonstrated in the scenario analysis.

8.1.2. Herbicide Sprayed Independent of the Weed Biomass

In the constant herbicide strategy, the amount of herbicide sprayed is independent of the weed biomass and the same prescribed amount is used for both GM and conventional canola fields. Since the general dynamic behavior of GM canola in this case is almost identical to the behavior obtained under the variable herbicide strategy, these dynamics will not be reanalyzed and only comparative analyses of GM and conventional canola under this strategy will be provided.

As depicted in the resistance allele ratio graph in Figure 8.11, the gap between the rates of resistance development in GM and conventional canola is reduced compared to the first case. This is mainly because equivalent amount of herbicide is applied in both fields. The other reason is that since weed population is almost eradicated due to high control efficacy, superweeds cannot come out to considerable levels at first. Hence, they do no grow sufficiently to result in a significant difference in the rate of resistance development.

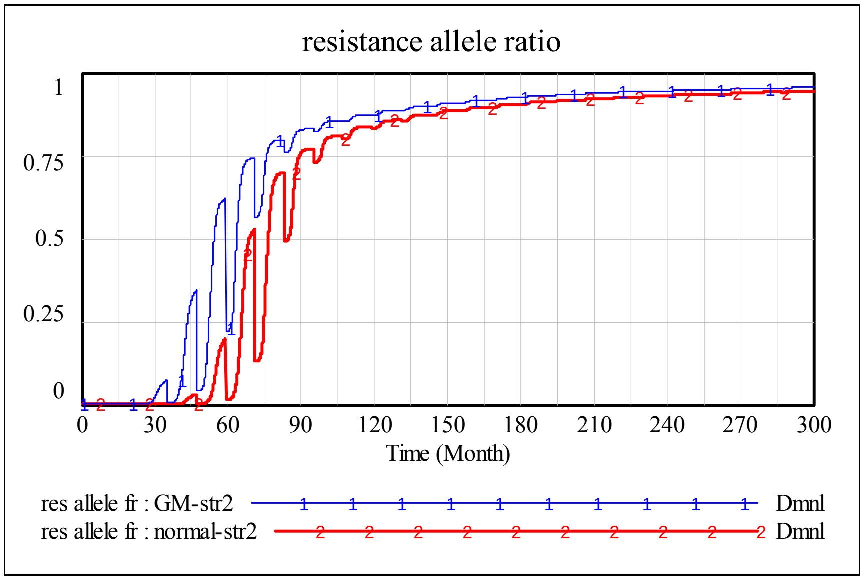


Figure 8.11. Comparison of resistance allele ratio dynamics in GM canola and in conventional canola fields under the constant herbicide strategy

When the comparative graph of weed population is investigated (Figure 8.12), it can be seen that conventional canola is advantageous only for a small period. Since herbicides inhibit the canola population in conventional canola, weeds gain competitive advantage and obtain a higher equilibrium level. The cumulative yield loss graph in Figure 8.13 also shows that under this strategy, GM canola turns out to be a preferable control means.

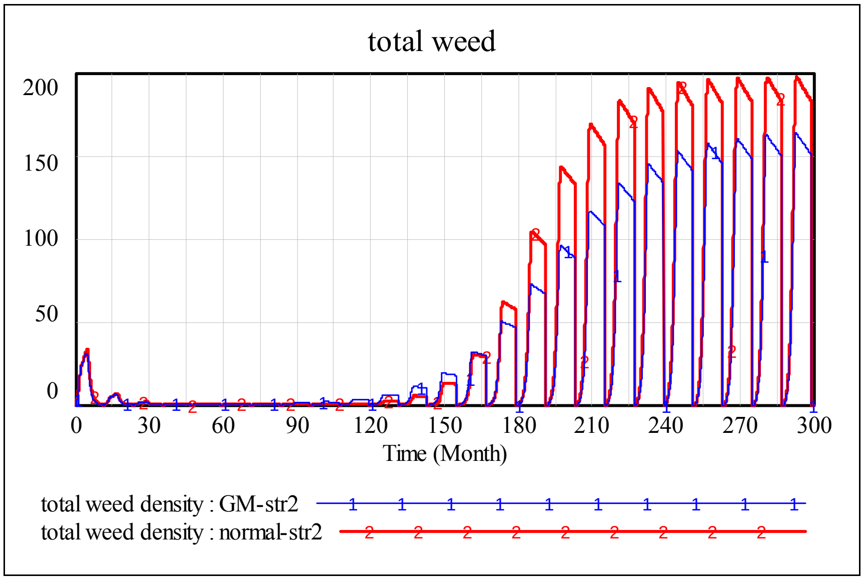


Figure 8.12. Comparison of weed population dynamics in GM canola and in conventional canola fields under the constant herbicide strategy

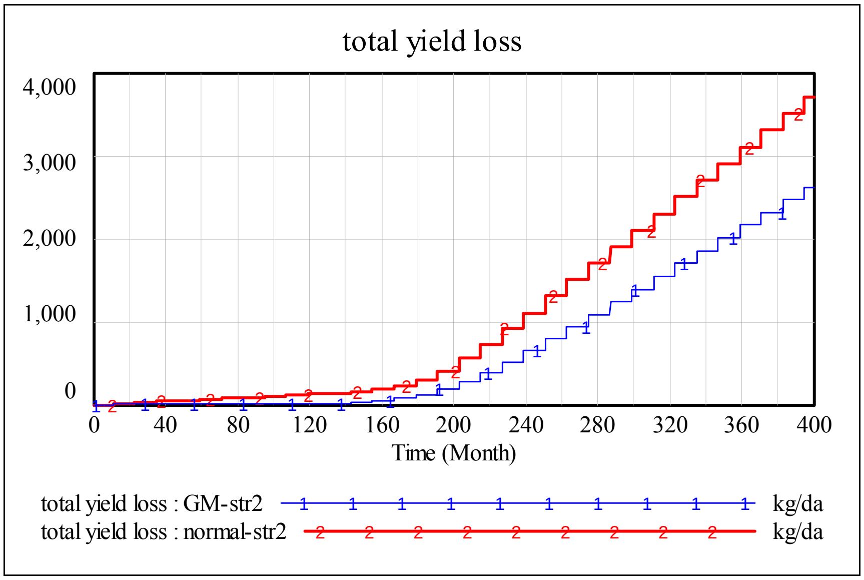


Figure 8.13. Comparison of cumulative yield losses occurring in GM canola and in conventional canola fields under the constant herbicide strategy

Note that in this strategy herbicide usage is promoted which is contrary to the objectives of an environmentally friendly weed management program. In Figure 8.14, cumulative herbicide usages under the two strategies and for the two plant varieties (GM and conventional canola) are compared. It is seen that the best strategy in reducing herbicide use is planting conventional canola and applying herbicide as a function of weed biomass.

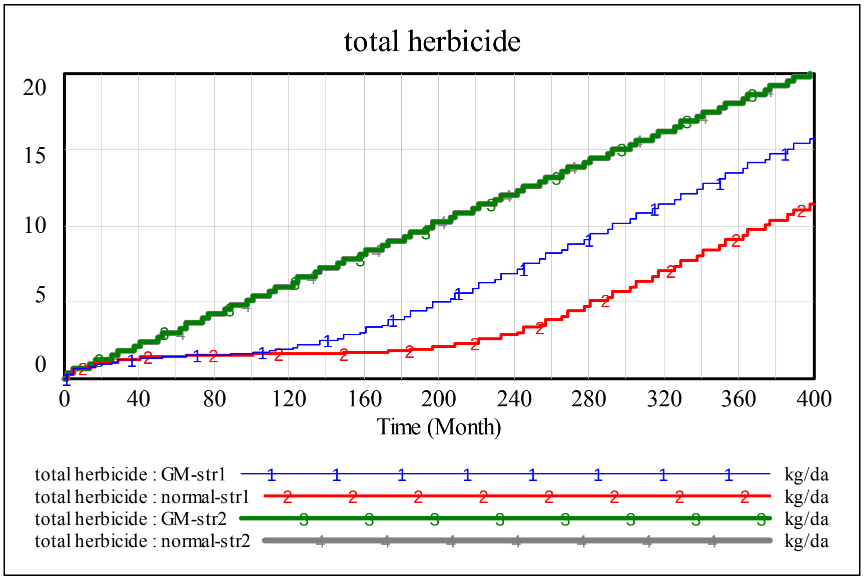


Figure 8.14. Comparison of cumulative herbicide usage under two herbicide strategies and two plant varieties GM and conventional

On the other hand, when cumulative yield losses are compared, for the first 160 months planting GM canola under the second strategy remains superior in yield efficiency (Figure 8.15). However, since resistance evolution is slow when conventional canola is planted under the first strategy, long run cumulative yield losses are lower than those obtained when GM canola is planted, if the structure is preserved (Figure 8.16). Assuming that a herbicide to which weeds gained resistance will stay in use is unrealistic. However, the point that is tried to be made via this analysis is that comparative benefits of using GM canola decreases and the need for an alternative product rises in relatively short periods.

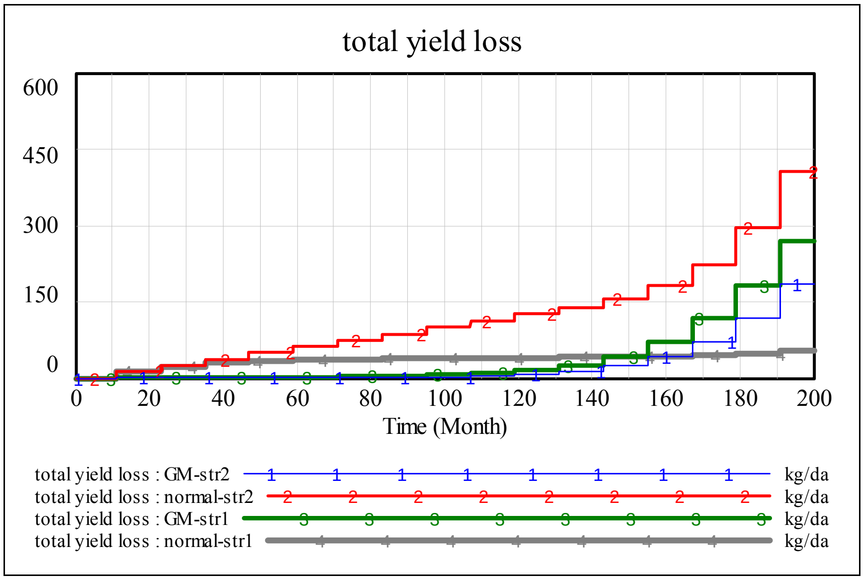


Figure 8.15. Comparison of cumulative yield loss occurring under two herbicide strategies and two plant varieties in the first 200 months

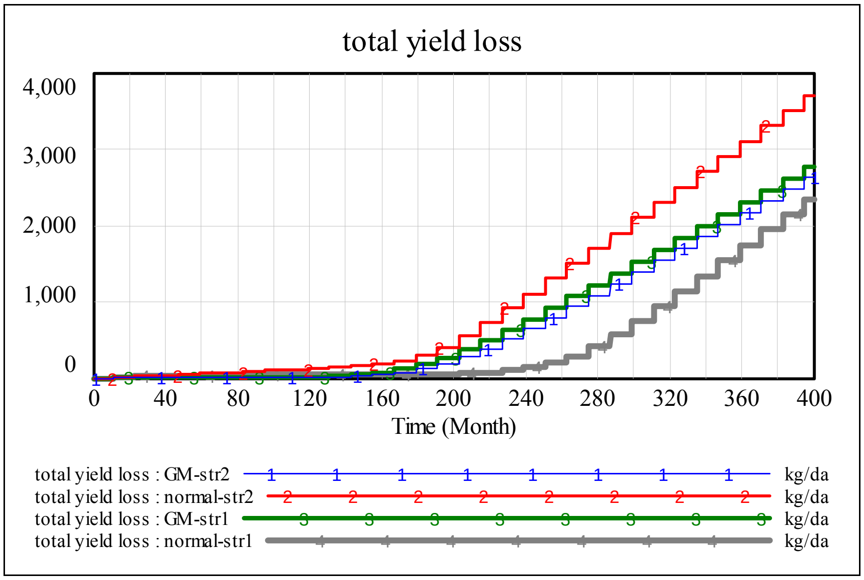


Figure 8.16. Comparison of cumulative yield loss occurring under two herbicide strategies and two plant varieties in the long run

8.2. Validation of the HT-Model

Just like in the validity testing of the IR model, the validation of the model is primarily demonstrated on the basis of extreme condition tests performed on the base model.

8.2.1. Extreme Condition Test: No Canola Seeds Are Sown

When *GM canola* is not planted, *Superweeds* cannot emerge, which is captured in the model as shown in the figure below.

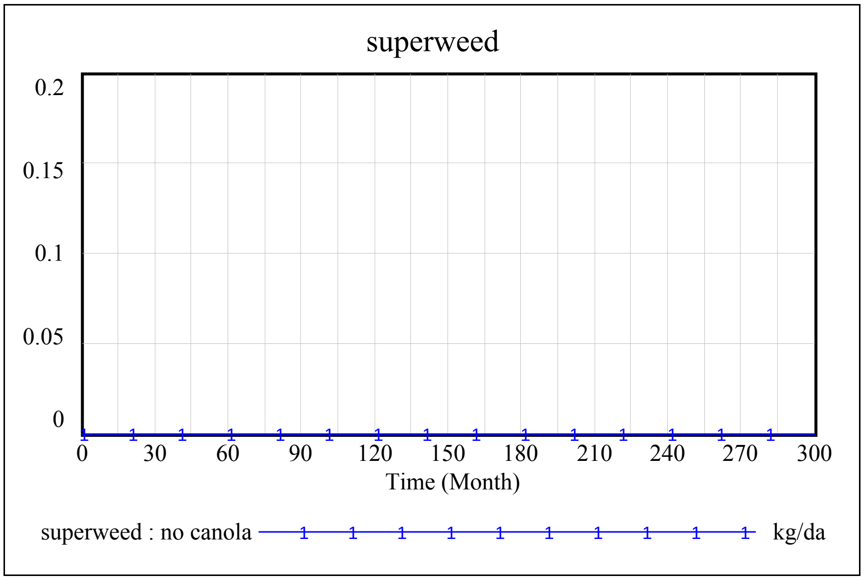


Figure 8.17. Superweed population when canola is not planted

With this extreme condition, there is no interspecific competition for weed, moreover, it is not cleaned since there is no canola harvest. Hence it is expected to behave as if isolated, i.e. reach its carrying capacity and saturate there. This holds for the model output, as shown below.

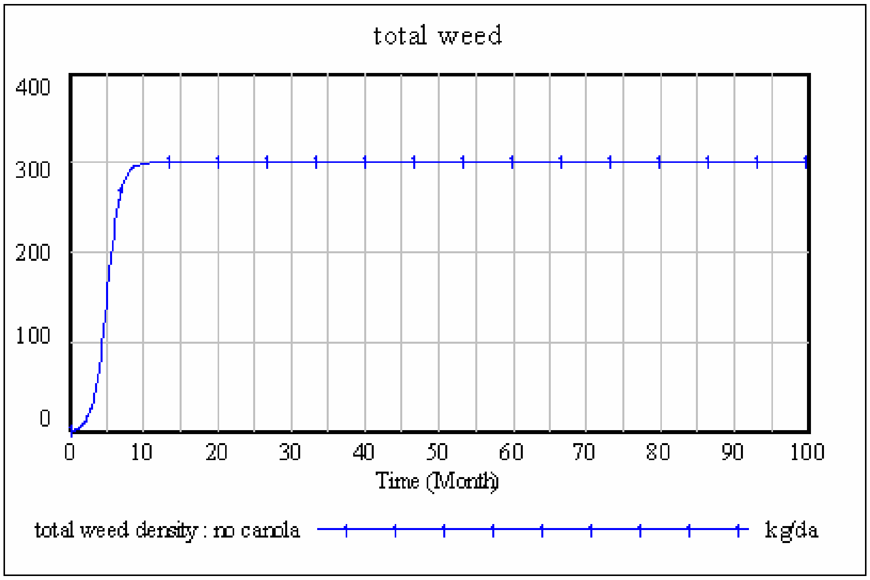


Figure 8.18. Dynamics of weed population when canola is not planted

8.2.2. Extreme Condition Test: No Weed Seedbanks

When weed seedbanks are empty, weeds cannot grow, as depicted in Figure 8.19.

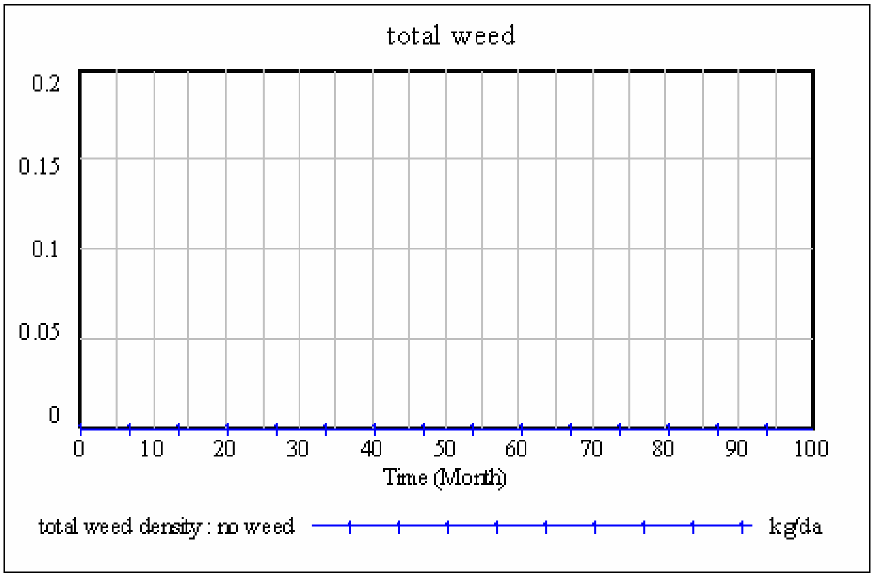


Figure 8.19. Dynamics of weed population when its seedbanks are empty

When there is no weed, there cannot be hybridization, hence superweeds cannot emerge.

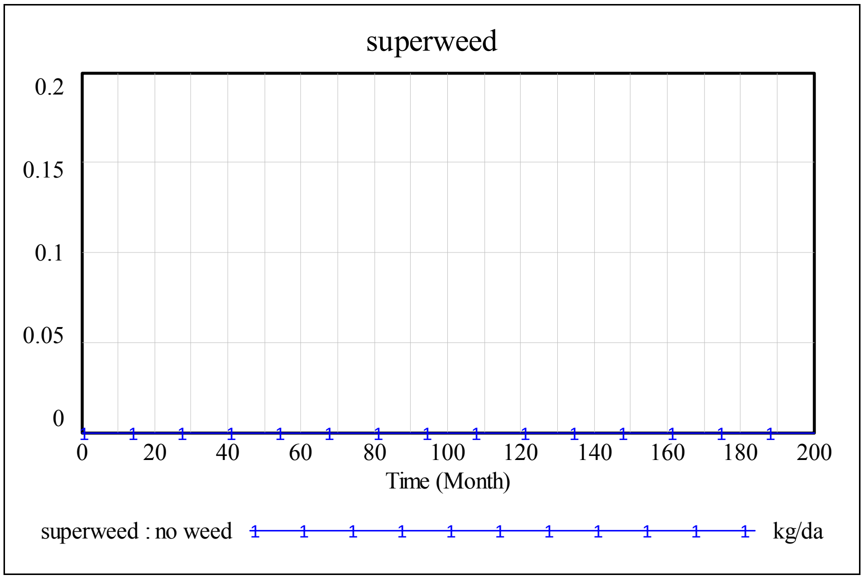


Figure 8.20. Superweed population when weed seedbanks are empty

GM canola behaves as if isolated, without weed pressure.

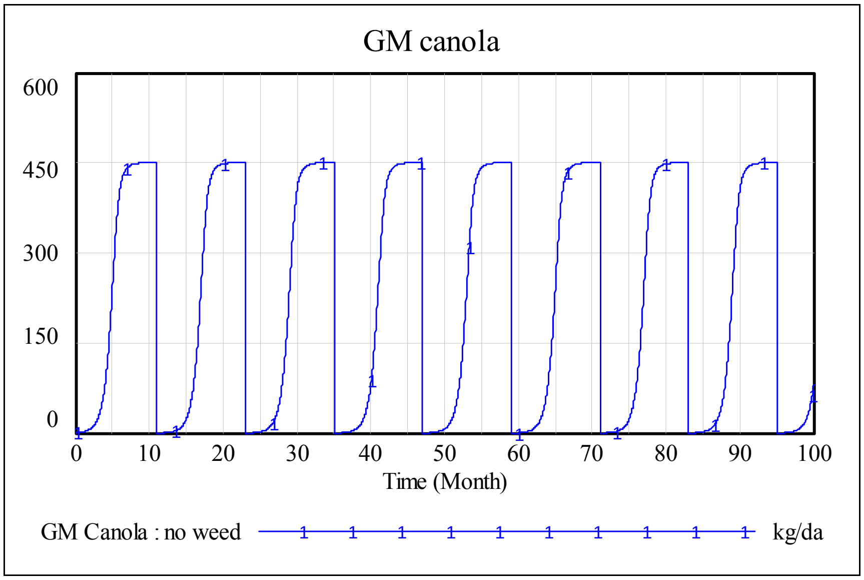


Figure 8.21 GM canola population when weed seedbanks are empty

8.2.3. Extreme Condition Test: No Resistance Allele

When there is no resistance allele in a weed population and mutation is not allowed, it is expected that resistance cannot be spread in the population no matter how much selection pressure is exerted. However, when a HT crop with a considerable hybridization rate with its weedy relative is planted, formation of superweeds introduces resistance alleles into the population, thereby allowing resistance evolution.

It is seen from Figure 8.22 that resistance is spread in the population very quickly. Figure 8.23 shows the resulting dynamics of weed population.

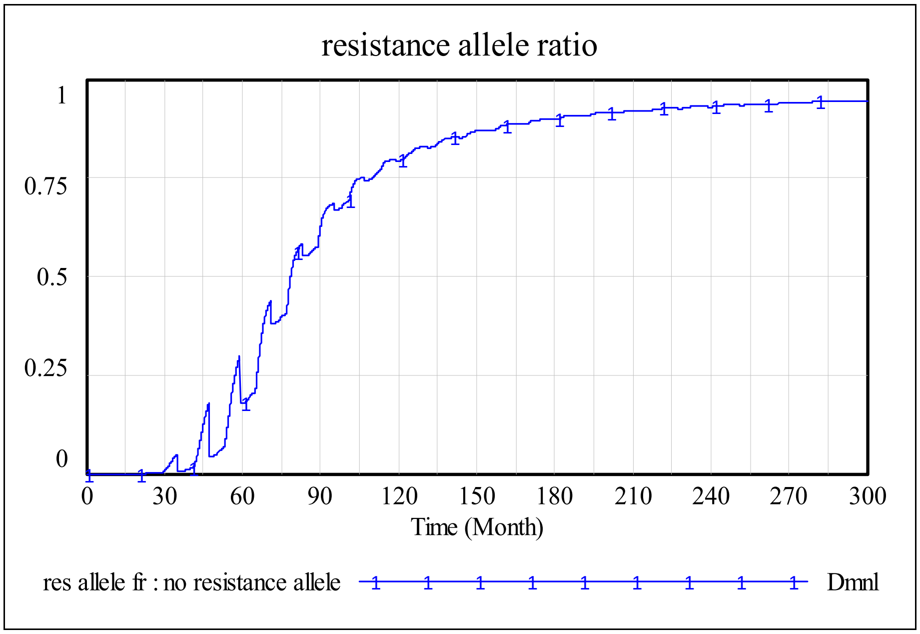


Figure 8.22 Dynamics of resistance allele ratio when initially there is no resistance allele in the population

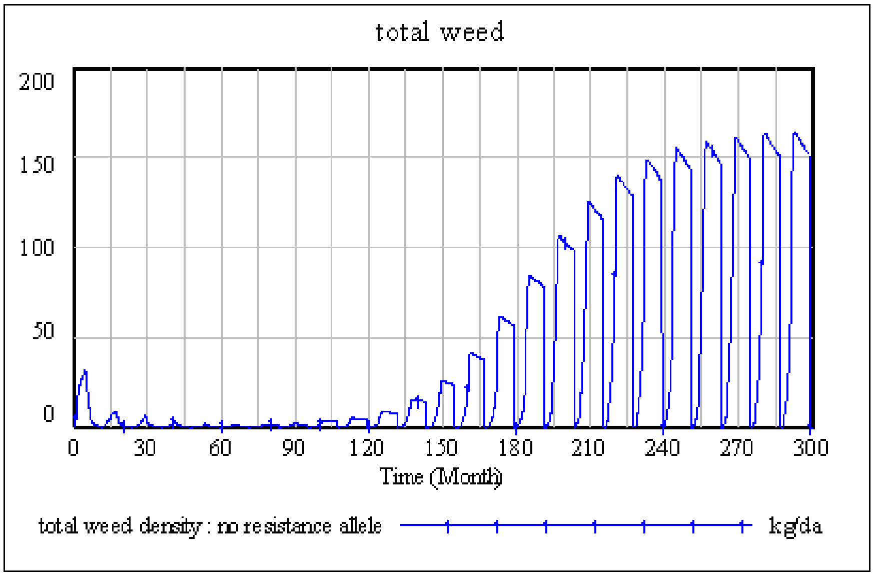


Figure 8.23 Dynamics of weed population when initially there is no resistance allele in the population

If there were no hybridization, resistance allele ratio would stay at zero as seen in Figure 8.24.

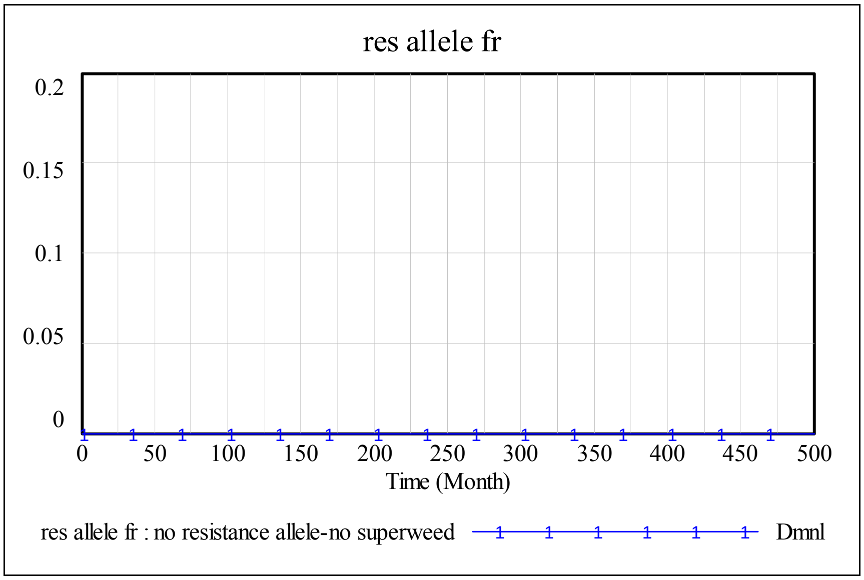


Figure 8.24. Resistance allele ratio when there is no resistance allele initially and hybridization is not allowed

9. SCENARIO ANALYSIS OF THE HT-MODEL

Base behavior of the HT-model is obtained assuming a certain parameter setting. When the parameter values are challenged via sensitivity analyses performed by changing a variable or variables at a time, it is seen that there is a great deal of numeric sensitivity involved in the model. However, most of the results obtained do not alter the inferences made from the base run. Hence, only two scenarios that are shaped via these analyses will be presented. Then, in the last section GM canola will be analyzed as a part of a crop rotation in order to see the effects of volunteer canola on yield efficiency and to initiate a further research avenue.

9.1. A Less Problematic Weed Population

Relative intensity of competition is determined by *alfa* values, *alfa weed* and *alfa Canola*. For instance, higher *alfa weed* implies faster habitat saturation for weed, hence a lower weed biomass at equilibrium. In the base run *alfa weed* is arbitrarily set to 1, which enables a persistent and problematic weed population. In Figure 9.1, weed population dynamics in the base run (*GM*) is compared to the case when *alfa weed* is 1.2 (*high alfa-GM*) and it is confirmed that for higher values of *alfa weed*, weed infestation is less of a burden. Accordingly, yield losses are also lower (Figure 9.2).

Note that, in this case it takes more time for the weeds to resurge compared to the base run. This implies that formation of a considerable superweed population is also delayed (Figure 9.3).

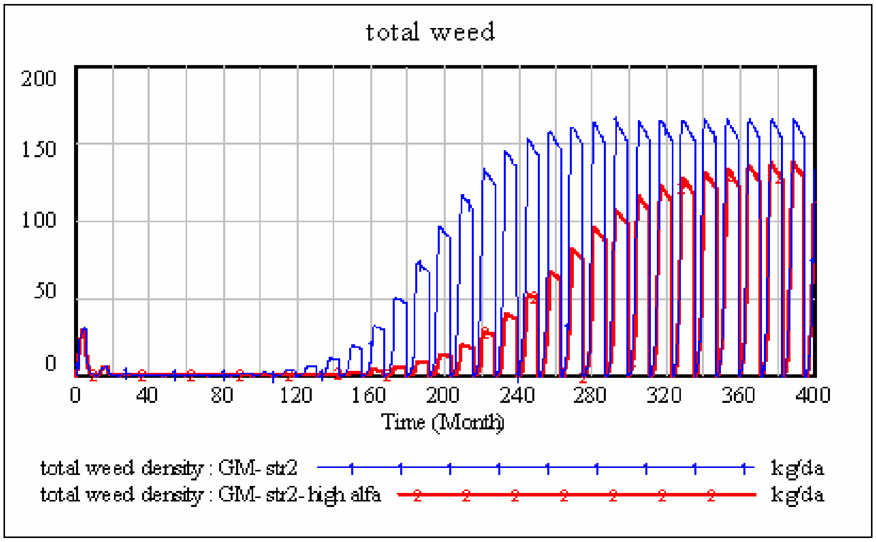


Figure 9.1. Comparison of weed population dynamics corresponding to the base run and the sensitivity run where *alfa weed* is high

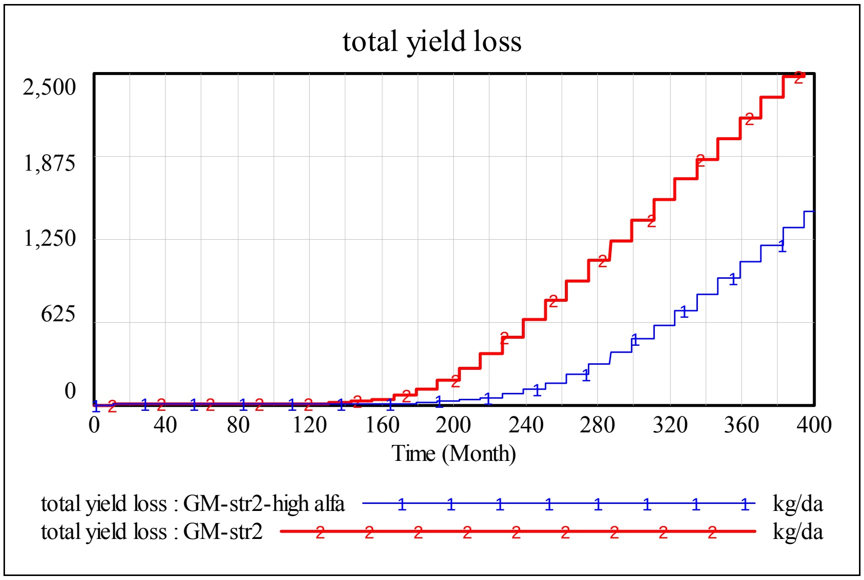


Figure 9.2. Comparison of cumulative yield losses corresponding to the base run and the sensitivity run where *alfa weed* is high

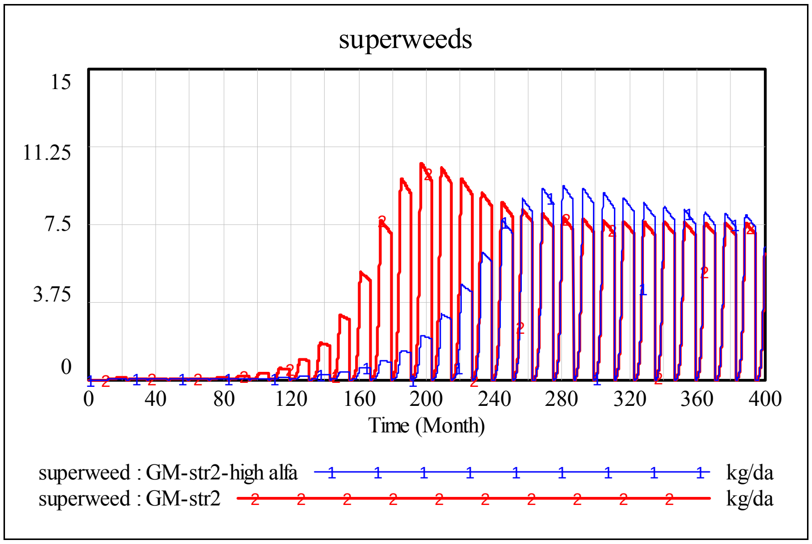


Figure 9.3. Comparison of superweed biomass occurring with different values of *alfa weed*

When GM and conventional canola are compared under this scenario, it is seen that the short-term advantage of planting GM canola becomes more evident and persists for a longer time mostly due to delayed superweed formation and delayed resistance evolution (Figure 9.4).

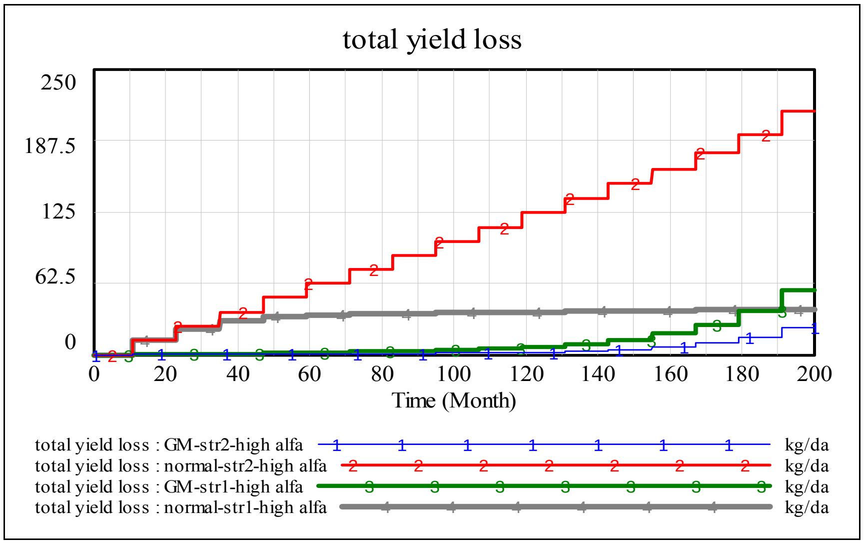


Figure 9.4. Comparison of cumulative yield loss occurring under two herbicide strategies and two plant varieties in the first 200 months when *alfa weed* is high

However, long term advantage of planting conventional canola under the variable herbicide strategy still prevails.

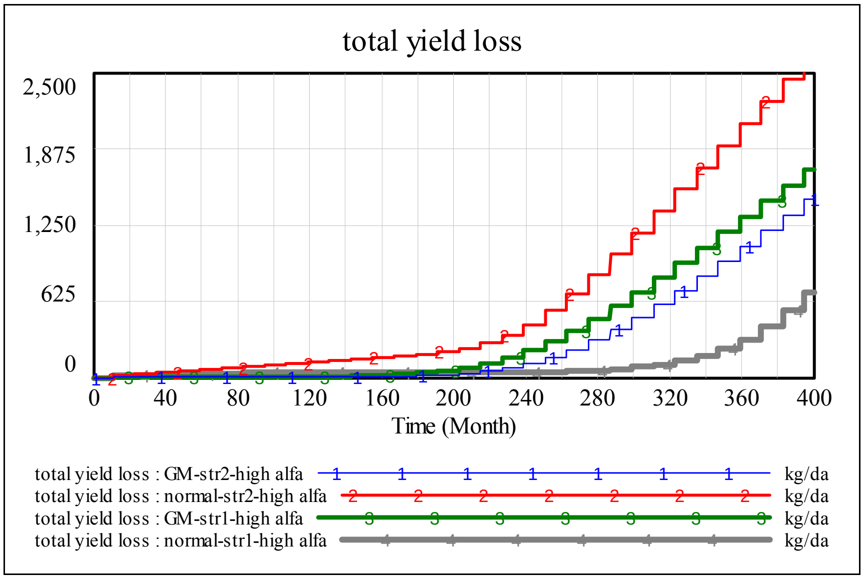


Figure 9.5. Comparison of cumulative yield loss occurring under two herbicide strategies and two plant varieties in the long run when *alfa weed* is high

Hence in less competitive weed populations spread of herbicide resistance is slower, which results in a slower decrease in comparative benefits of planting HT crops.

9.2. Resistant Weeds Showing Susceptibility at High Doses of Herbicide

In the model it is assumed that resistant weeds of both RR and RS genotype are completely immune to the herbicide in use. However, in reality it is often the case that resistance is a matter of degree and especially heterozygote weeds are expected to show some susceptibility at doses higher than the amount sufficient to kill susceptible weeds. In order to see whether treating resistance in this kind of a continuum changes the general behavior, all weed genotypes are modeled to be controlled by the herbicide at different rates such that *WeedRR* are only modestly affected at very high doses, while *WeedRS* shows some susceptibility even at the current dose.

Since in this scenario *WeedRS* and *WeedRR* are also controllable to a degree, it is no surprise that weed biomass attains a lower equilibrium than the base run as seen from Figure 9.6. Also note that resistance evolution is slower since *WeedRR* also suffers from selection pressure and provides less RR seeds to the seedbanks.

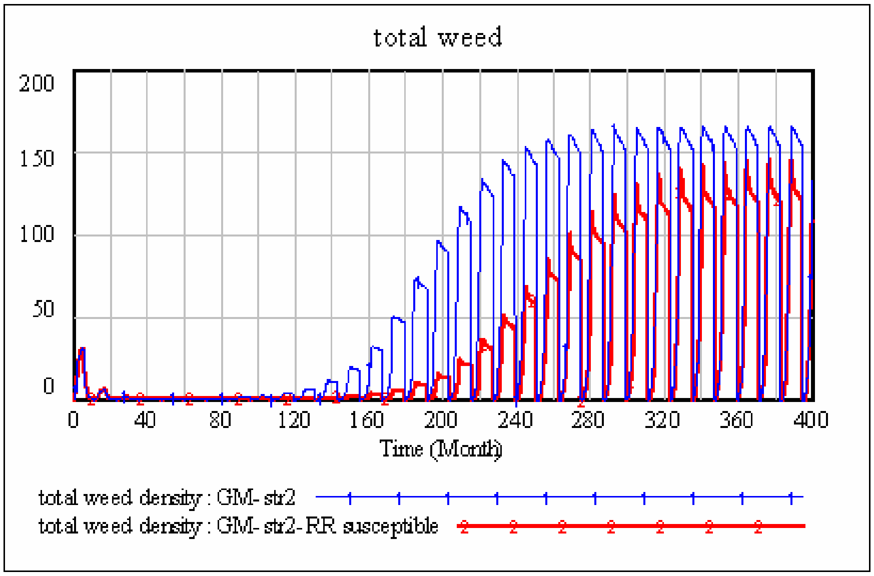


Figure 9.6. Comparison of weed population dynamics corresponding to the base run and the sensitivity run where resistant weeds show susceptibility at high doses

Yet, these changes do not alter the aforementioned results of the comparison between GM and conventional canola. Still, planting GM canola under the second strategy is preferable in the short run and planting conventional canola under the variable herbicide strategy is preferable in the long run (Figure 9.7 and Figure 9.8).

However, a different picture is obtained if herbicide spray rate is increased. Now, increased herbicide usage makes a difference even after the spread of resistance since resistant weeds are also controllable to a degree at high doses. Since HT crops prevent crop damage, it is probable that GM farmers increase their herbicide spray rates in order to fully benefit from the merits of the costly seeds they purchase.

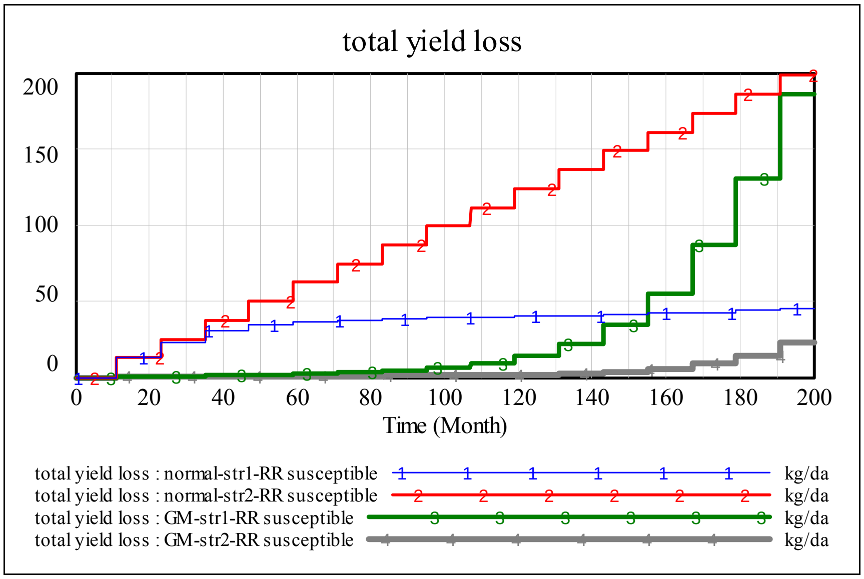


Figure 9.7. Comparison of cumulative yield losses in the first 200 months corresponding to two plant varieties under two herbicide strategies when resistant weeds show susceptibility at high doses

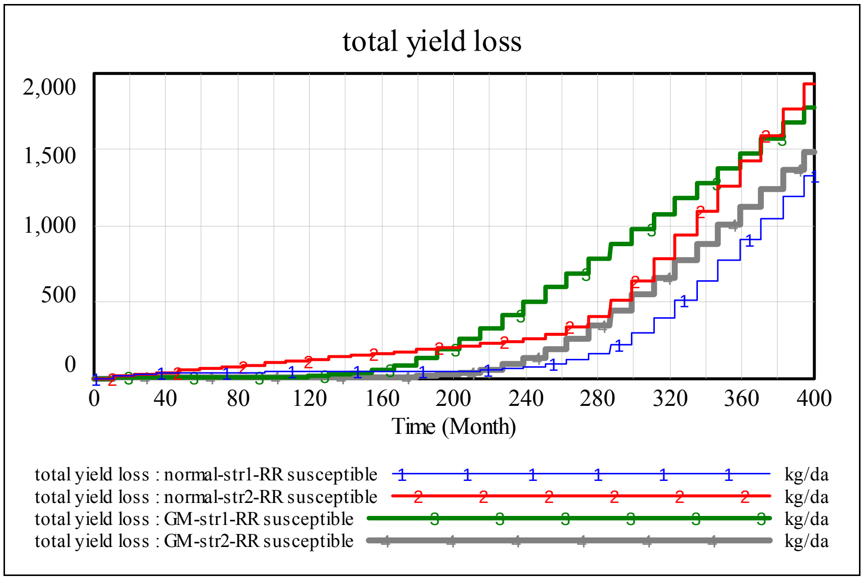


Figure 9.8. Comparison of long run cumulative yield losses corresponding to two plant varieties under two herbicide strategies when resistant weeds show susceptibility at high doses

Indeed, farmers are reported to spray considerably more herbicides on HT soybean (Benbrook, 2003). However, for conventional canola increased herbicide usage implies increased yield losses due to herbicide. Hence, it is likely that farmers planting conventional canola will be reluctant to spray at higher rates. Regarding this possibility, in this scenario GM farmer sprays at a higher rate than the rates used for conventional canola planting. Under the variable herbicide strategy, this is achieved by modifying the function *fraction of ref herbicide sprayed* so that it saturates at a higher value implying a higher rate of herbicide application as weed biomass increases. Under the second strategy, simply the predetermined amount is increased by 20%. When cumulative yield losses of the two plant varieties under the two herbicide strategies are compared, planting GM canola under the constant herbicide strategy and at an increased rate provides the highest yield efficiency throughout the simulation.

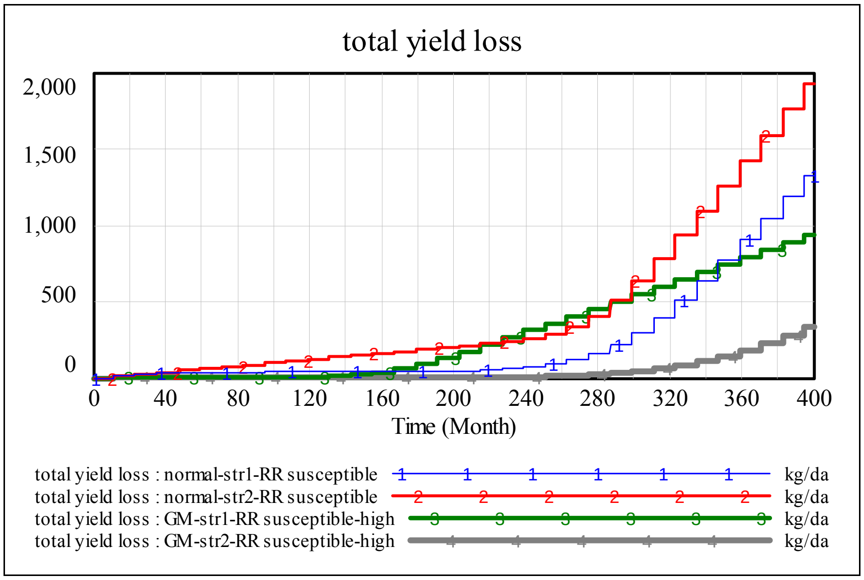


Figure 9.9. Comparison of cumulative yield losses corresponding to two plant varieties under two herbicide strategies when resistant weeds show susceptibility at high doses and when GM farmers spray at higher rates

However, this benefit comes with the cost of increasing herbicide usage dramatically, as seen in Figure 9.10.

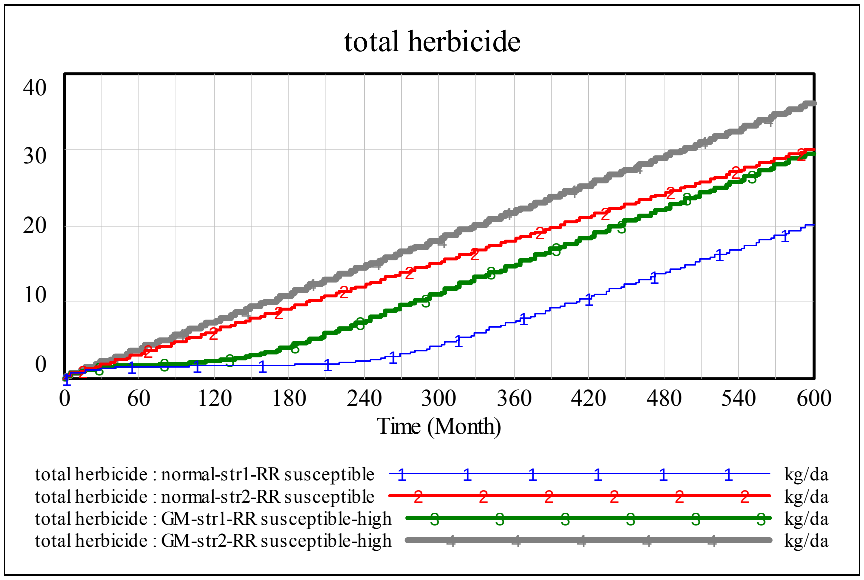


Figure 9.10. Comparison of cumulative herbicide usage corresponding to two plant varieties under two herbicide strategies when resistant weeds show susceptibility at high doses and when GM farmers spray at higher rates

9.3. Canola Planted in Rotation with Wheat

In the model, agriculture with HT crops is investigated assuming that each year the same crop is planted as a monoculture. However, it is highly recommended that crops be planted in rotation for various reasons such as sustaining soil nutrients and providing a natural means of pest control. In this section, a rotation scenario will be investigated under the two herbicide strategies discussed in Chapter 8 and by simultaneous comparison with conventional canola. A four-year rotation cycle of *Canola-Wheat-Wheat-Canola* is studied and it is assumed that the same herbicide is used throughout the rotation. *Wheat* is not herbicide tolerant and model equations for *Wheat* are identical to those of conventional *Canola* except for parameter values (see Appendix D.3 for the related modified equations and the model description for rotation analysis).

When crops are planted in rotation, volunteer crops become an issue. A plant that germinates from a seed left behind in the field from a previous crop is called *volunteer*.

Volunteerism is a concern for genetically modified herbicide tolerant crops especially if the same herbicide is used throughout the rotation, because these plants are not controllable by the herbicide just like the resistant weeds. The problem is more severe for crops whose seeds have higher chance of persisting in the soil, i.e. who have high seed dormancy or longevity. Canola has considerable seed dormancy. It is reported that volunteer canola can persist up to 4 years after planting. (Hall *et al*., 2000) Seed losses range from 3.3-10%, providing ample source of seed to establish a weed seedbank. This amount of seed losses is 9-56 times the normal seeding rates of canola and occurs even if good harvest management practices are followed, since it seems to be a result of the shattering nature of canola. (Shirtliffe, 2003) Consequently, volunteer canola has been reported to evolve into a common weed.

In the model, only canola volunteers are taken into account for the sake of simplicity relying on the fact that modeling wheat volunteers which are essentially weeds that can be suppressed by the herbicide does not quite enrich the analysis. Canola volunteers are modeled similarly to the weed population, having a current population stock *Canola Vol* and a seedbank, *Seedbank Vol*. Size of the volunteer seedbank depends on the amount of canola seeds escaped from harvest, persistency of these seeds in the seedbank and the amount of seeds lost due to predation or other reasons. Size of the current volunteer population depends on the size of the seedbank and intraspecific and interspecific competition. Canola volunteers are problematic only when wheat is planted

When GM canola is planted, *CanolaVol* constitutes a considerable weed population as seen in Figure 9.11. This weed population competes with the rotation crop, *Wheat*, resulting in higher yield losses compared to planting conventional canola where volunteers can be suppressed by the herbicide (Figure 9.12).

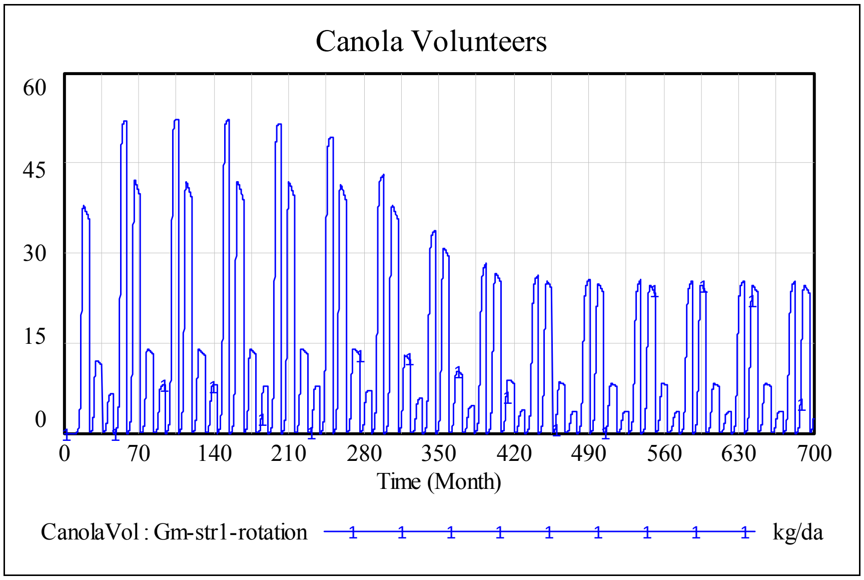


Figure 9.11. Dynamics of canola volunteer population when GM canola is planted in rotation with wheat

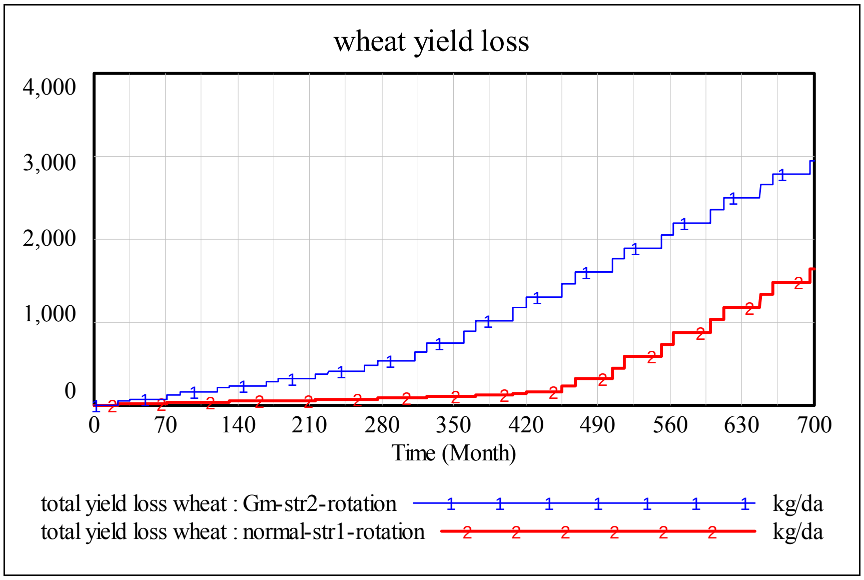


Figure 9.12. Cumulative yield loss when GM canola is planted under the constant herbicide strategy versus when conventional canola is planted under the variable herbicide strategy, both in rotation

GM canola still provides higher yield efficiency in the short run, though with decreased discrepancy (Figure 9.13). However, when the overall yield efficiency of the rotation is considered and it is assumed that only glyphosate is used throughout the rotation, this short term advantage is lost (Figure 9.14). This loss of advantage is due to the fact that canola volunteers cannot be suppressed by glyphosate, which increases weed biomass.

Of course, a different or an additional herbicide can be used for the rotation crop in order to suppress canola volunteers. Indeed, rotating herbicides is a recommended strategy to delay the evolution of herbicide resistance. However, it must be kept in mind that when a species gains resistance to a herbicide, it most probably becomes resistant to a range of herbicides with a similar mode of action. Finding an appropriate herbicide to suppress volunteers will be especially an issue if crops are modified to be tolerant to a pack of herbicides.

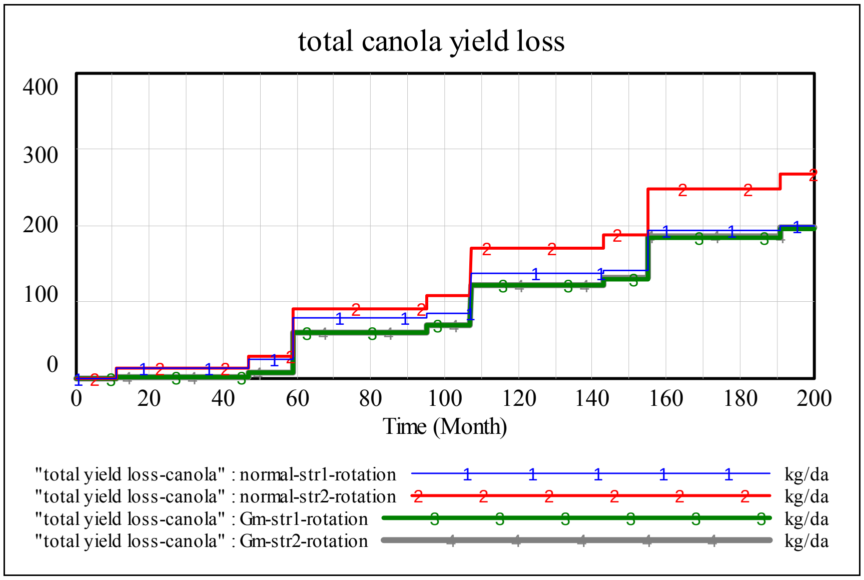


Figure 9.13. Comparison of cumulative canola yield losses in the first 200 months occurring under two herbicide strategies and two plant varieties, all in rotation

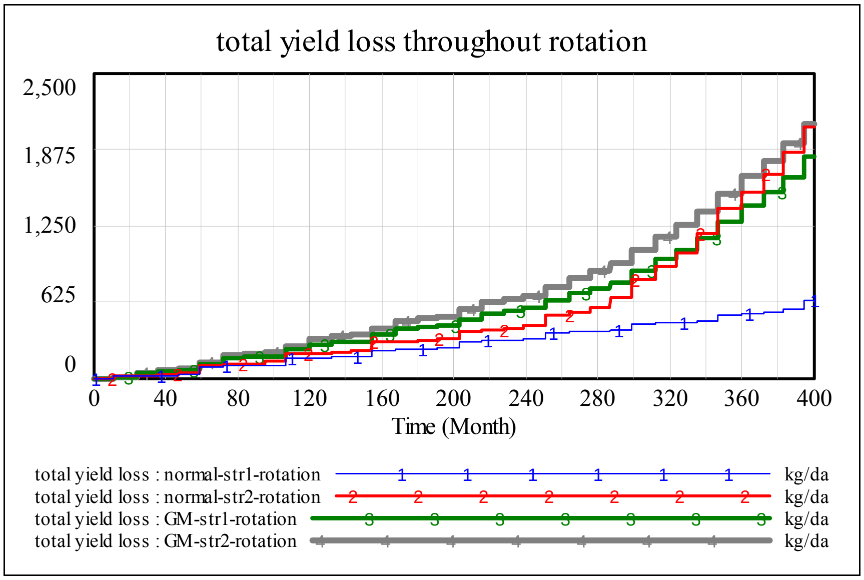


Figure 9.14. Comparison of long run cumulative yield losses throughout the rotation occurring under two herbicide strategies and two plant varieties, all in rotation

Table 9.1. Summary of the results from the HT-model

		GM crop		**Conventional crop**	
		Variable Herb. Strategy	***Constant Herb. Strategy***	***Variable Herb. Strategy***	***Constant Herb. Strategy***
SCENARIOS	*Base Run*		• Lowest yield loss in the short run • worst rate of resistance development	• Lowest yield loss in the long run • lowest herbicide usage • lowest rate of resistance development	• Worst yield loss both in the short and the long run
	Less Problematic Weeds		• Lowest yield loss in the short run • worst rate of resistance development (though slower than the base run)	• Lowest yield loss in the long run • lowest herbicide usage • lowest rate of resistance development (all lower than the base run)	• Worst yield loss both in the short and the long run (though lower than the base run)
	Resistant Weeds Showing Susceptibility at High Doses	• At higher herbicide doses lowest yield loss in the short run	• At higher herbicide doses lowest yield loss both in the short and the long run • highest herbicide usage	• Lowest rate of resistance development • lowest herbicide usage (both lower than the base run)	
	Rotation	• Lowest canola yield loss in the short run	• Lowest canola yield loss in the short run • highest total yield loss in the long run	• Lowest total (wheat and canola) yield loss • lowest herbicide usage • lowest rate of resistance development	

10. CONCLUSIONS AND FURTHER RESEARCH

The purpose of this research is to study the long-term impacts of GM crops on crop yield and pesticide/herbicide use under various scenarios and policies, the focus being on the potential environmental and ecological problems discussed in the literature. Two models are built for this purpose: The first one for insect resistant (IR) GM crops, the second one for herbicide tolerant (HT) GM crops. These systems are compared to their conventional counterparts in order to appreciate the relative benefits and disadvantages.

Under consistent selection pressure exerted by using the same pesticide (whether an insect resistant *Bt*-crop or a pesticide), resistance evolution is inevitable. Although resistance evolution is inevitable, the special role of *Bt*-crops is especially notable. With the high-dose efficacy of *Bt*-crop, resistance evolution is shown to happen in 10 years, while it would take 50 years with conventional pesticides under the same conditions. If pests have already been exposed to the toxin resulting in a higher resistance allele ratio, resistance evolution can be completed in 5 years. When resistance is spread in the population, *Bt*-crop loses its effectiveness as a pest control means and pest infestation becomes inevitable. Hence, delaying this phenomenon is essential for the sustainability of agriculture with these crops. One important point depicted in this study is that transition to this infestation state may be quite sharp, i.e. the farmer may spend an almost pest-free year and then may face an infestation the next year. Hence not observing any resistance at all to a *Bt*-crop this season does not guarantee that the crop will be effective the next season.

The results of policy analysis of the IR-model show that building refuges for susceptible pests can delay resistance development as much as 15 years when 20% of the land is planted with non-*Bt* crop and this refuge is treated with a non-*Bt* pesticide to which *Bt*-resistant pests are susceptible. On the other hand, if the pesticide which is used to treat the refuge is a *Bt* pesticide or one to which *Bt*-resistant pests gained resistance through cross-resistance, this effectiveness of refuge falls down to 6 years. Another refuge strategy proposed by Environment Protection Agency which prescribes that 5% of the land is spared as a non-treated refuge turned out to be less effective, providing a delay of approximately 6 years.

On the other hand, if heterozygote pests show resistance to a degree, the refuge strategy turns out to be futile. Resistance is spread in 7 years in spite of the refuge. Likewise, if the land had already been exposed to *Bt*-pesticide resulting in a higher level of resistance allele ratio initially, the strategy is not sufficient to keep the *Bt*-crop useful more than 9 years.

Another result depicted by the IR-model is that whether *Bt*-crops have a harmful effect on the predators of target pests can be a determining factor on the performance of the pest management system. When predators are harmed by the *Bt*-crop, pest population is shown to attain a higher equilibrium level, thereby constituting a more severe pest management problem.

The situation with genetically modified herbicide tolerant (HT) crops is slightly different in the sense that weed management strategy is not altered with the advent of GM varieties. The same herbicides are normally used whether the crop is GM or conventional. What makes the difference in terms of sustainability and yield efficiency is HT crop's advantage of eliminating yield loss due to herbicide and disadvantage of forming resistant hybrid weeds so called the superweeds. In the HT-model, agriculture with herbicide tolerant crops is analyzed under two herbicide strategies. In the first one which is called the variable herbicide strategy, the amount of herbicide to spray is a function of weed biomass while in the second one, the constant herbicide strategy, herbicide is sprayed at a predetermined amount.

Under the variable herbicide strategy, planting HT crops are shown to be more effective in the short run, but this comparative benefit decreases quickly due to faster evolution of resistance in the weed population in the GM case compared to the conventional crop case. What makes HT crop disadvantageous is the formation of superweeds, which increases both the weed burden and the rate of resistance development. One important phenomenon depicted is that in the GM case, herbicide resistance is developed even if there is no resistance allele in the weed population initially, which is not the case for conventional crops. Superweeds contribute resistance alleles to the gene pool and speed up the spread of resistance; hence the herbicide becomes ineffective more rapidly, which outweighs HT crop's advantage of increased crop safety. Moreover, since superweeds are resistant to the herbicide and cannot be suppressed, weed biomass is increased in their presence, pushing up the need for spraying herbicide. Hence, in the GM case cumulative herbicide usage is higher, which also increases the rate of resistance development.

If the amount of herbicide sprayed is fixed and high enough, weed population is so suppressed that hybridization is almost ruled out. In this case superweeds cannot emerge until the resistant weed population attains a considerable level. Hence, conventional crop loses its advantage of lower weed biomass and decreased herbicide use. Furthermore, it suffers from a higher yield loss due to the adverse effects of the herbicide. Hence, when herbicide is sprayed at a predetermined rate, HT crop provides superior yield efficiency compared to conventional crop under the same strategy. When the four possible cases are compared, it is seen that this is the best option in terms of short-term yield efficiency. Yet, this herbicide strategy increases the cumulative herbicide usage, which increases input costs to agriculture and more critically, invokes food safety concerns. Furthermore planting conventional crop under the variable herbicide strategy results in a lower cumulative yield loss in the long run, due to slower resistance evolution.

On the other hand, if HT crop is planted within a rotation where the same herbicide is used consistently, volunteers of the GM crop become a severe weed burden since it is resistant to the herbicide. In this case, when the overall yield loss throughout the rotation is considered, HT crop is inferior in terms of yield efficiency under both herbicide strategies.

In general, when weed biomass is used to determine the amount of herbicide sprayed, cumulative herbicide usage is reduced, which makes it a better practice in terms of environmental impacts. Moreover, since in this case weed biomass does not go down to extremely low levels, the strategy does not threat other organisms that feed on weed seeds. However, one must note that in any case, for HT crops or conventional ones, consistent application of a single herbicide is not a sustainable means of weed management since resistance development is fast.

To sum up, as depicted in the long term yield loss behaviors of *Bt* and HT crops, relative benefits of using these crops decrease and the need for alternative crops rises in relatively short periods. To overcome the resistance barrier, a sustainable and integrated system needs to be developed regarding the specific conditions on the land of concern. However, one rightful concern is that the short-term success of insect resistant and herbicide tolerant crops will delay the intensive search for novel non-pesticide based pest management technologies and methods.

There are several avenues in which this research can be expanded. One first step is incorporating both pest and weed problems in a single model. These issues generally coexist in practice, resulting in further complicated interactions. Hence challenging the assumptions of *no weeds* for the IR-model and *no pests* for the HT-model may bring to light interesting results. Moreover, biotechnology industry has developed GM crops which provide both insect resistance and herbicide tolerance. An analysis of long term impacts of these crops on agriculture may reveal a broader range of possible environmental phenomena.

Since, the focus of this study is the sustainability of agriculture with *Bt* and HT crops, economic returns have not been analyzed. The models can be extended to incorporate a long term profitability analysis. However, considering the fact that the market for GM products involves somewhat a higher uncertainty due to unresolved risk issues associated with the technology, the results of such a study should be interpreted with caution.

This research does not consider market's bringing forth new products or technologies when *Bt*-crop loses its effectiveness. Farmers' shifting to new products can be incorporated into the models as a further research topic. Such an effort is initiated in IR-Model's policy analysis by letting farmers switch to a new pesticide after the resistance is spread. However, that switch is exogenous to the model structure rather than being an output of farmer's decision making. In a regional agriculture model, it would be interesting to incorporate farmer behavior (about shifting to new products, compliance to refuge requirement, adopting or leaving GM technology, etc) into the analysis of long term impacts of GM crops.

Finally, as a step in the analysis of effects of GM crops on biodiversity, HT-Model can be further extended to incorporate population dynamics of farm birds and similar animals that feed on weed seeds. Whether birds can survive the effects of intensified weed management is occasionally raised as a concern. A comparison of conventional and HT crop under the aforementioned strategies can reveal interesting results in this biodiversity loss perspective.

APPENDIX A: LIST OF VARIABLES, DEFINITIONS AND UNITS: IR-MODEL

A.1. Pest Sector

Variables used in Pest sector are listed below. In providing the variable definitions and units, only those related to the *pestRR* stock will be explained when the structure is identical for the three genotypic subpopulations. Hence, the definitions provided for *RR* are valid for *RS* and *SS* stocks as well.

birthsRR: Pest births of RR genotype (pest/da/Month)

bt effect of residue: Fraction denoting the toxic effect of corn residue (unitless)

Bt/ref Bt: Ratio of *Bt* activity in land to the reference *Bt* level (unitless)

deathsRR: Pest deaths of RR genotype (pests/da/Month)

eff of Bt toxin on sus df: Effect of Bt-corn on death fraction of susceptible pests (unitless)

eff of food on bf: Effect of food availability on pest birth fraction (unitless)

eff of pest density on mating: Effect of pest density on predator birth fraction (unitless)

food per capita: The amount of corn and corn residue available per pest (kg/pest)

killedRR: Pests of RR genotype killed by the predator (pests/da/Month)

pest bf: Fraction of pests giving births (1/Month)

pestRR: Resistant (RR) pest population per unit land (pest/da) *pestRS:* Heterozygote susceptible (RS) pest population per unit land (pest/da)

pestSS : Homozygote susceptible (SS) pest population per unit land (pest/da)

prob RR births: Proportion of RR birth in the total births (unitless)

ref Bt: Reference level of Bt-toxin (kg/da)

sus ref df: Reference death fraction for susceptible pests attained when there is no Bt-toxin in land (1/Month)

reference pest bf: Maximum birth fraction attained when food is sufficient (1/Month)

ref food per capita: The amount of food per pest necessary to attain the maximum birth fraction (kg/pest)

res allele ratio : Ratio of resistance allele in the gene pool to the total number of alleles in the pest population (unitless)

res df: Fraction of resistant pests dying (1/Month)

RRratio: Ratio of RR pests in the pest population (unitless)

sus df: Fraction of susceptible pests dying per unit time (1/Month)

total births: Pest births (pests/da/Month)

total killed: Number of pests killed by the predator per unit land (pests/da/Month)

total pest: Aggregate pest population per unit land (pest/da)

A.2. Crop Sector

BtCorn: Biomass of GM corn (kg/da)

cleaned: Rate of depletion of corn residue through cleaning (kg/da/Month)

cleaning rate: Fraction of corn residue cleaned (1/Month)

Corn Residue: Biomass of corn residue left in the field after the harvest (kg/da)

degrade: Rate of corn residue degradation (kg/da/Month)

grow: Rate of Bt-corn growth from seeds to maturity (kg/da/Month)

harvesting: Rate of Bt-corn harvesting (kg/da/Month)

harvesting fr: Fraction of Bt-corn harvested (1/Month)

increase: Rate of increase in the corn residue (kg/da/Month)

plant: Number of individual corn plant in the field (plant/da)

pest per plant: Average number of pests per each Bt-corn plant (pest/plant)

ratio of degrading: Fraction of corn residue degraded (1/Month)

residue ratio: Ratio of corn residue amount to corn biomass at harvest (unitless)

seeding: Rate of sowing corn seeds (kg/da/Month)

seeding period: Period through which corn seeding is performed (Month)

seeds: Number of plant seeds sown per unit land (plant/da) *regeneration ratio:* Corn biomass growing rate(1/Month)

TimeToClean: Time of the year when the field is cleaned from residue (unitless)

TimeToHarvest: Time of the year when corn is harvested (unitless)

TimeToGrow: Time of the year when corn is growing (unitless)

TimeToSeed: Time of the year when corn seeds are sown (unitless)

weight of seeds: weight of each corn seed (kg/plant)

yield loss due to pests: Rate of decrease in corn biomass due to pests (kg/da/Month)

% yield loss per pest per plant: % decrease in corn biomass per each pest per plant (plant/pest/Month)

A.3. Predator Sector

eff of bt on predator: Effect of Bt-corn on predator death fraction (unitless)

eff of food availability: Effect of pest per predator on predator birth fraction (unitless)

eff of predator on mating: Effect of predator on predator birth fraction (unitless)

functional response: Fraction of reference capture rate that can be achieved in the current pest density (unitless)

pest per predator: Average number of pests per predator (pest/predator)

predator: Predator population per unit land (predator/da)

predator bf: Fraction of predators giving birth (1/Month) *predator births:* Number of predator births (predator/da/Month)

predator deaths: Number of predator births (predator/da/Month)

ref capture rate: Reference number of pests captured per predator when pest density is at its sufficiency (reference) level (pest/predator/Month)

ref predator bf: Reference value of predator birth fraction when food availability is at its sufficiency (reference) value (1/Month)

ref predator df: Reference value of predator death fraction when Bt-toxin or Bt-corn does not have any adverse effect on predators (1/Month)

A.4. Pesticide Sector

evacuation ratio: Fraction of pesticide in land that decays per unit time (1/month)

pesticide degradation: The amount of herbicide degrading per unit time (g/da/Month)

pesticide in land: The amount of pesticide effective in land (g/da)

prescribed amount: The amount of pesticide recommended to spray at a time (g/da)

ref pesticide: The reference amount of pesticide in land that provides the maximum control efficiency (g/da)

TimeToSpray: Time of the year when pesticide is sprayed (unitless)

A.5. Refuge Sector

birthsRR refuge: Pest births of RR genotype (pest/da/Month) *deathsRR refuge:* Pest deaths in refuge belonging to RR genotype (pests/da/Month)

pest per corn: Average number of pests per each conventional corn plant (pest/plant)

pestRR refuge: Resistant (RR) pest population per unit refuge area (pest/da)

plant-ref: Number of individual corn plants in refuge (plant/da)

refuge ratio: The ratio of refuge area to the overall land (unitless)

RRratio refuge: Ratio of RR pests in refuge to the pest population (unitless)

sus refuge df: Fraction of susceptible pests in refuge dying per unit time (1/Month)

total seasonal loss: Total yield loss in a season (kg/da)

yield loss corn: Conventional corn yield loss in a season (kg/da)

yield loss conventional corn: Bt-corn yield loss in a season (kg/da)

APPENDIX B: LIST OF VARIABLES, DEFINITIONS AND UNITS: HT-MODEL

B.1. Crop Sector

alfa Canola: Relative impact of the competing species on the regeneration rate of the crop GM canola (unitless)

carrying cap canola: The maximum biomass of GM-canola that the specific habitat can support (kg/da)

eff of competition on Canola: Effect of inter and intraspecific competition on the regeneration ratio of canola (unitless)

GM canola: Biomass of GM canola (kg/da)

grow-Canola: Rate of GM canola growth from seeds to maturity (kg/da/Month)

harvest-Canola: Rate of GM canola harvesting (kg/da/Month)

HI-Canola: Ratio of canola seeds produced to the overall biomass of canola (unitless)

Loss-Canola: Fraction of canola seeds that are lost due to predation etc. (unitless)

max rgn ratio-Canola: Maximum attainable regeneration ratio of canola (1/Month)

max yield: Maximum attainable canola yield in the absence of competing species (kg/da)

rgr Canola: Canola biomass growing rate(1/Month)

seeding period: Period through which corn seeding is performed (Month) *seeds:* Number of plant seeds sown per unit land (plant/da)

seedweight: weight of each canola seed (kg/plant)

sow-Canola: Rate of sowing canola seeds (kg/da/Month)

TimeToSeed-Canola: Time of the year when canola seeds are sown (unitless)

TimeToHarvest-Canola: Time of the year when canola is harvested (unitless)

total yield loss: Cumulative yield loss throughout the simulation (kg/da)

yield loss-canola: The discrepancy between the maximum attainable yield and the current yield (kg/da)

yield loss due to herbicide: Rate of yield loss in conventional canola due to the detrimental effects of the herbicide

B.2. Weed Sector

In providing the variable definitions and units, only those related to the *WeedRR* stock will be explained when the structure is identical for the three genotypic subpopulations. Hence, the definitions provided for *RR* are valid for *RS* and *SS* stocks as well.

active res allele fr: Ratio of resistance allele to the total number of alleles in the gene pool of the current weed population (unitless)

alfa weed: Relative impact of the competing species on the regeneration rate of the weed population (unitless) *carrying cap weed:* The maximum biomass of weed that the specific habitat of concern can support (kg/da)

cleanRR: Rate of cleaning weeds during harvest (kg/da/Month)

decay fr weed: Fraction of weed seeds in the seedbank that decay per unit time (1/Month)

decayRR: Rate of seed decaying out of the weed seedbank (kg/da/month)

eff of competition on weed: Effect of interspecific and intraspecific competition on the regeneration ratio of weed (unitless)

eff of herbicide on weed: Fraction of susceptible weeds that are killed via the herbicide per unit time (1/Month)

fraction of ref herbicide sprayed: Fraction of the prescribed amount of herbicide that is sprayed at a given season (unitless)

grm RR: Rate of seeds germinating out of the weed seedbank (kg/da/Month)

grm ratio weed: The fraction of weed seeds in the weed seedbank that germinates per unit time (1/Month)

growRR: Rate of weed growth from seeds to maturity (kg/da/Month)

harvest index: The ratio of harvested part to the overall biomass (unitless)

herbicide decay ratio: Fraction of herbicide in land that decays per unit time (1/month)

herbicide degradation: The amount of herbicide degrading per unit time (kg/da/Month)

herbicide in land: The amount of herbicide effective in land (kg/da)

herbicide sprayed: The amount of herbicide sprayed per unit time (kg/da/month) *max rgn ratio weed:* Maximum attainable regeneration ratio of canola (1/Month)

newSeed-RR: Rate of addition of new seeds with RR genotype to the weed seedbank of RR genotype (kg/da/month)

prescribed amount: The amount of herbicide recommended to spray at a time (kg/da)

probRR: Proportion of weed seeds that are of RR genotype (unitless)

ref herbicide: The amount of herbicide necessary to attain the maximum efficacy of weed control (kg/da)

res allele fr: Ratio of resistance allele to the total number of alleles in the in the gene pool of the weed population (unitless)

rgr weed: Weed biomass growing rate(1/Month)

SeedBankRR: The deposited seeds of WeedRR (kg/da)

seedloss: Fraction of seeds that are lost due to reasons such as predation (unitless)

seedweed: Total seed production of the weed species in a given season (kg/da)

sowRR: Rate of sowing weed seeds of RR genotype (kg/da/Month)

spray ratio: Ratio of the prescribed amount of herbicide sprayed per unit time (1/Month)

TimeToSpray: Time of the year when herbicide is sprayed if it is to be sprayed (unitless)

total B weed: Total weed biomass excluding the first generation superweeds (kg/da)

total herbicide: Cumulative herbicide usage throughout the simulation (kg/da) *total seed:* Total seed production of all weeds (including those formed via hybridization) in a given season (kg/da)

total seedbank: Total amount of seeds deposited in weed seedbanks (kg/da)

total weed: Total weed biomass (kg/da)

WeedRR: Weed biomass of RR-genotype (kg/da)

B.3. Superweeds Sector

clean SweedRR: Rate of cleaning superweeds during harvest (kg/da/Month)

crop to weed ratio: Ratio of canola biomass to the total weed biomass (unitless)

decay SweedRR: The rate with which seeds decay out of the superweed seedbank of RR genotype (kg/da/month)

eff of crop to weed ratio on hybridization: Effect of relative biomass of canola biomass on hybridization rate (unitless)

grm SweedRR: Rate of superweed seeds germinating out of the superweed seedbank of RR genotype (kg/da/Month)

grow SweedRR: Rate of superweed growth from seeds to maturity (kg/da/Month)

hyb probRR: Proportion of hybrid seeds that are of RR genotype (unitless)

new SweedRR: Rate of addition of new RR seeds to the seedbank of superweeds with RR genotype (kg/da/month)

outcrossing fr: The proportion of weed seeds that are pollinated by canola (unitless) *ref frequency:* Maximum attainable value of outcrossing frequency (1/Month)

ref ratio: Reference value of crop to weed ratio at which the reference outcrossing frequency is attained (unitless)

SeedBank SweedRR: The deposited seeds of superweeds of RR genotype (kg/da)

seed Sweed: The amount of hybrid seeds to be deposited in the seedbanks of the superweeds (kg/da)

superweed: Total biomass of superweeds (kg/da)

SuperWeedRR: The biomass of superweeds of RR genotype (kg/da)

sow SweedRR: Rate of sowing superweed seeds of RR genotype (kg/da/Month)

B.4. Volunteer Sector

CanolaVol. Biomass of volunteer canola (kg/da)

decayVol. Rate of seed decaying out of the canola seedbank (kg/da/month)

escaped seed fr: Fraction of canola seeds that escape harvest (unitless)

grmSeedVol. Rate of canola seeds that germinates per unit time (kg/da/Month)

grm ratioVol. Fraction of canola seeds that germinates per unit time (1/Month)

growVol. Rate of volunteer canola growth from seeds to maturity (kg/da/Month)

newSeedVol. Rate of addition of new canola seeds to the canola seedbank (kg/da/month)

SeedBankVol. The deposited seeds of canola (kg/da) *seedsVol.* The amount of canola seeds that escape harvest and are deposited in the seedbank for volunteer canola (kg/da)

sowVol. Rate of sowing volunteer canola seeds (kg/da/Month)

cleanVol. Rate of cleaning volunteer canola during harvest (kg/da/Month)

APPENDIX C: EQUATIONS OF THE IR-MODEL

C.1. Base Model

"% yield loss per pest per plant"=0.014

births RR=prob RR births*total births

births RS=prob RS births*total births

births SS=total births*prob SS births

BtCorn= INTEG (+seeding+growing-harvesting-yield loss due to pests,0)

bt effect of residue=0.5

"Bt/ref Bt"=(BtCorn+Corn Residue*bt effect of residue)/ref Bt

change=IF THEN ELSE(MonthsInYear=0, seasonality/TIME STEP, 0)

cleaned=Corn Residue*cleaning period*TimeToClean

cleaning period=4

Corn Residue= INTEG (+increase-cleaned-degrade,0)

deaths RR=pestRR*res df

deaths RS=sus df*pestRS

deaths SS=pestSS*sus df

degrade=Corn Residue*ratio of degrading* (1-TimeToClean)

eff of bt corn on predator=IF THEN ELSE(BtCorn>0, 1.3, 1)

"eff of Bt-toxin on sus df"= WITH LOOKUP ("Bt/ref Bt",([(0,0)-(6,6)],(0,1),(0.477064,2.27525),(0.788991,3.1996),(1,3.7525),(1.22936,4.1249),(1.468,4.4498),(1.78,4.7253),(2.45872,4.75),(5.98165,4.75),(6,4.75)))

eff of food availability= WITH LOOKUP (functional response,([(0,0)-(2,1.5)],(0,0),(0.0214067,0.414474),(0.0428135,0.598684),(0.0825688,0.743421),(0.201835,0.888158),(0.324159,0.960526),(0.495413,0.986842),(0.617737,0.986842),(0.807339,0.980263),(1,1),(1.88991,1),(2,1)))

eff of food on bf= WITH LOOKUP (food per capita/ref food per capita,([(0,0)-(3,1.5)],(0,0),(0.0917431,0.25),(0.192661,0.546053),(0.302752,0.769737),(0.458716,0.907

895),(0.642202,0.960526),(0.770642,0.973684),(0.862385,0.986842),(1,1),(1.49847,1),(9.96942,1)))

eff of pest density on mating= WITH LOOKUP (total pest,([(0,0)-(1000,1)],(0,0),(0.0001,0),(0.000577982,0.00438596),(0.00102752,0.035087),(0.00141284,0.114035),(0.00301835,0.320175),(0.00494495,0.517544),(0.00733945,0.649123),(0.00948012,0.745614),(0.0131498,0.855263),(0.0161193,0.929825),(0.0193945,0.969298),(0.0208716,0.97807),(0.0225688,0.991228),(0.03,1),(1,1),(1000,1)))

eff of predator on mating= WITH LOOKUP (predator,([(0,0)-(1000,1)],(0,0),(0.0001,0),(0.000577982,0.00438596),(0.00102752,0.0350877),(0.00141284,0.114035),(0.00301835,0.320175),(0.00494495,0.517544),(0.00733945,0.649123),(0.00948012,0.745614),(0.0131498,0.855263),(0.0161193,0.929825),(0.0193945,0.969298),(0.0208716,0.97807),(0.0225688,0.991228),(0.03,1),(1,1),(1000,1)))

food per capita=IF THEN ELSE(total pest>0, (BtCorn+Corn Residue)/total pest, 6000)

functional response= WITH LOOKUP (pest per predator/ref pest per predator,([(0,0)-(8,1.5)],(0,0),(0.0917431,0.1228),(0.214067,0.2982),(0.428135,0.5746),(0.733945,0.8224),(1,1),(1.27217,1.086),(1.639,1.221),(2.226,1.333),(2.691,1.368),(3.205,1.389),(3.86544,1.39474),(4.50153,1.39474),(6.92355,1.39474),(7.92661,1.39474)))

growing=BtCorn*regeneration ratio*TimeToGrow

harvesting=BtCorn*TimeToHarvest*harvesting fraction

harvesting fraction=8.5

increase=harvesting*residue ratio

initial RR=16

initial RS=4

initial SS=16000

killedRR=RRratio*total killed

killedRS=RSratio*total killed

killedSS=SSratio*total killed

max yield=672.5

MonthsInYear=MODULO(Time, 12)

pest bf=eff of food on bf*reference pest bf*eff of pest density on mating*rand seasonality eff

pest per plant=IF THEN ELSE(plant>0, total pest/plant, 0) pest per predator=ZIDZ(total pest,predator)

pestRR= INTEG (births RR-deaths RR-killed RR,initial RR)

pestRS= INTEG (births RS-deaths RS-killed RS,initial RS)

pestSS= INTEG (births SS-deaths SS-killed SS,initial SS)

plant=seeds*0.85

predator bf=eff of food availability*ref predator bf*eff of predator on mating

predator births=predator*predator bf

predator deaths=predator*predator df

predator= INTEG (predator births-predator deaths,150)

predator df=ref predator df

prob RR births=res allelle ratio*res allelle ratio

prob RS births=2*res allelle ratio*(1-res allelle ratio)

prob SS births=(1-res allelle ratio)*(1-res allelle ratio)

ratio of degrading=0.1

ref Bt=100

ref capture rate=15

ref food per capita=0.1

ref pest per predator=300

ref predator bf=0.62

ref predator df=0.5

sus ref df=0.2

reference pest bf=0.88

regeneration ratio=1.5

res allelle ratio=ZIDZ(2*pestRR+pestRS, 2*total pest)

res df=0.21

reset=IF THEN ELSE(MonthsInYear=11.9375, rand seasonality eff/TIME STEP, 0)

residue ratio=0.5

RRratio=ZIDZ(pestRR, total pest)

RSratio=ZIDZ(pestRS, total pest)

seasonal loss=yield loss corn/TIME STEP

seasonality=SMOOTHI(white noise, 0.8,0.8)

rand seasonality eff= INTEG (change-reset,0)

seeding=seeds*weight of seeds*TimeToSeed*(1/seeding period)
seeding period=TIME STEP
seeds=5900
SSratio=ZIDZ(pestSS, total pest)
sus df=sus ref df*"eff of Bt-toxin on sus df"
TimeToClean=IF THEN ELSE(MonthsInYear>=0 :AND: MonthsInYear<1, 1, 0)
TimeToGrow=IF THEN ELSE(MonthsInYear>0:AND:MonthsInYear<4, 1, 0)
TimeToHarvest=IF THEN ELSE (MonthsInYear>4:AND:MonthsInYear<6 , 1,0)
TimeToSeed=IF THEN ELSE(MonthsInYear=0, 1, 0)
total births=pest bf*total pest
total killed=functional response*predator*ref capture rate
total pest=pestRR+pestSS+pestRS
total yield loss= INTEG (seasonal loss,0)
weight of seeds=0.000339
white noise=RANDOM NORMAL(0, 1.9, 1, 2000, 17)
yield=IF THEN ELSE(MonthsInYear=4, BtCorn, 0)
yield loss corn=IF THEN ELSE(MonthsInYear=4, (max yield-yield), 0)
yield loss due to pests=BtCorn*"% yield loss per pest per plant"*pest per plant*TimeToGrow

C.2. Modifications and New Equations for Conventional Corn:

corn= INTEG (growing+seeding-harvesting-yield loss due to pests, 0)
eff of Bt toxin on sus df= WITH LOOKUP (pesticide in land/ref pesticide,([(0,0)-(6,6)],(0,1),(0.477064,2.27525),(0.788991,3.1996),(1,3.75),(1.22936,4.1249),(1.468,4.4498),(1.78,4.7253),(2.45872,4.75),(5.98165,4.75),(6,4.75)))
evacuation ratio=0.3
pesticide degradation=evacuation ratio*pesticide in land
pesticide in land= INTEG (+pesticide used-pesticide degradation,0)
pesticide used=prescribed amount*TimeToSpray/TIME STEP
prescribed amount= 90

ref pesticide=100

TimeToSpray=IF THEN ELSE(MonthsInYear=0.5:OR:MonthsInYear=3.5, 1, 0)

C.3. Modifications and New Equations for Refuge Policy

birthsRR refuge=prob RR births*total birth*refuge ratio

corn= INTEG ("growing-normal"+"seeding-normal"-"harvesting-normal"-"yield loss due to pests-normal",0)

deathsRR refuge=pestRR refuge*sus refuge df

eff of bt toxin on sus ref df= WITH LOOKUP (pesticide in land/ref pesticide,([(0,0)-(6,6)],(0,1),(0.477064,2.27525),(0.788991,3.1996),(1,3.7525),(1.22936,4.1249),(1.468,4.4498),(1.78,4.7253),(2.45872,4.75),(5.98165,4.75),(6,4.75)))

killedRR refuge=total killed*RRratio refuge

pestRR refuge= INTEG (birthsRR refuge-deathsRR refuge-killedRR refuge, initial RR refuge)

ref pesticide=100

refuge ratio=0.2

res allelle ratio=ZIDZ(2*(pest RR+pestRR refuge)+pest RS+pestRS refuge, 2*total pest)

RRratio refuge=ZIDZ(pestRR refuge, total pest)

seeding=seeds*(1-refuge ratio)*weight of seeds*TimeToSeed*(1/seeding period)

"seeding-normal"=seeds*refuge ratio*weight of seeds*TimeToSeed/seeding period

sus refuge df=sus ref df*eff of bt toxin on sus ref df

total seasonal loss=yield loss conventional corn+yield loss corn

yield loss conventional corn=IF THEN ELSE(MonthsInYear=4, max yield*refuge ratio-corn yield, 0)

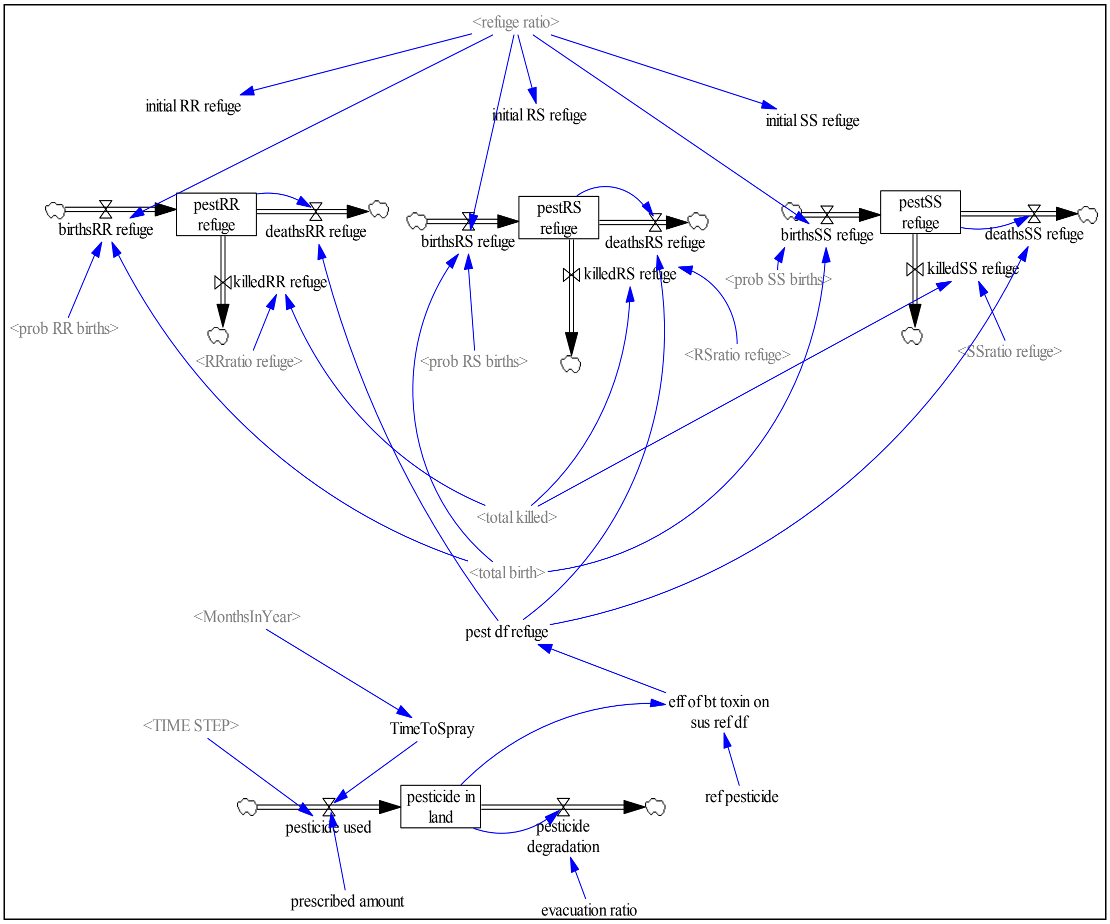


Figure C. 1. Stock-flow diagram of Refuge Sector

APPENDIX D: EQUATIONS OF THE HT-MODEL

D.1. Base Model-GM Canola

active res allele fr= ZIDZ (2*(WeedRR+SuperWeedRR)*TimeToHarv weed+(WeedRS+SuperWeedRS)*TimeToHarv weed, 2*(total B weed+superweed)*TimeToHarv weed)

alfa Canola=1

alfa weed=1

carrying cap canola=450

carrying cap weed=300

cleanRR=TimeToHarv weed*WeedRR/TIME STEP

cleanRS=WeedRS*TimeToHarv weed/TIME STEP

cleanSS=TimeToHarv weed*WeedSS/TIME STEP

clean SweedRR=SuperWeedRR*TimeToHarv weed/TIME STEP

clean SweedRS=SuperWeedRS*TimeToHarv weed/TIME STEP

control SS=eff of herbicide*WeedSS*(1-TimeToHarv weed)

crop to weed ratio=ZIDZ(GM Canola, total B weed+superweed)

decay fr Weed=0.042

decay RR=decay fr Weed*SeedBank RR

decay RS=decay fr Weed*SeedBank RS

decay SS=decay fr Weed*SeedBank SS

decay SweedRR=SeedBank SweedRR*decay fr Weed

decay SweedRS=SeedBank SweedRS*decay fr Weed

eff of competition= WITH LOOKUP ((total weed+alfa weed*GMCanola)/carrying cap weed, ([(0,-0.08)-(1.5,1)],(0,1),(0.0856269,0.995614),(0.174312,0.95614),(0.311927,0.881579),(0.425076,0.785088),(0.553517,0.653509),(0.697248,0.473684),(0.825688,0.285088),(0.905199,0.144737),(0.95107,0.0614035),(1,0),(1.05199,-0.01),(1.20489,-0.012),(1.5,-0.0125))) eff of competition on canola= WITH LOOKUP ((GM Canola+alfa Canola*total weed)/carrying cap canola,([(0,-0.02)(1.5,1)],(0,1),(0.0856269,0.995614),(0.174312,0.95614),(0.311927,0.881579),(0.425076,0.785088),(0.553517,0.653509),(0.697248,0.473684),(0.825688,0.285088),(0.905199,0.144737),(0.95107,0.0614035),(1,0),(1.05199,-0.01),(1.20489,-0.012),(1.5,-0.015)))

eff of crop to weed ratio on hybridization= WITH LOOKUP (crop to weed ratio/ref ratio,([(0,0)-(10,1)],(0,0),(0.0397554,0.135965),(0.107034,0.311404),(0.171254,0.45614),(0.259939,0.609649),(0.342508,0.714912),(0.431193,0.807018),(0.519878,0.872807),(0.636086,0.921053),(1,1),(10,1)))

eff of herbicide= WITH LOOKUP (herbicide in land/ref herbicide,([(0,0)-(2,1)],(0,0),(0.030581,0.0394737),(0.12844,0.166667),(0.262997,0.355263),(0.366972,0.495614),(0.495413,0.644737),(0.642202,0.763158),(0.770642,0.864035),(0.874618,0.907895),(1,0.94),(1.54128,0.956),(1.99388,0.96)))

fraction of ref herbicide sprayed= WITH LOOKUP (total weed/ref weed,([(0,0)-(5,2)],(0,0),(0.122324,0.333333),(0.259939,0.596491),(0.428135,0.77193),(0.764526,0.921053),(1,1),(1.20795,1.05),(1.55963,1.11404),(1.97248,1.17544),(2.49235,1.22807),(4.98471,1.25)))

GM Canola= INTEG (grow Canola+sow Canola-harvest Canola,0)

grm ratio weed=0.4

grm RR=grm ratio weed*SeedBank RR*TimeToSeed weed

grm RS=grm ratio weed*SeedBank RS*TimeToSeed weed

grm SS=grm ratio weed*SeedBank SS*TimeToSeed weed

grm SweedRR=SeedBank SweedRR*grm ratio weed*TimeToSeed weed

grm SweedRS=SeedBank SweedRS*grm ratio weed*TimeToSeed weed

grow Canola=GM Canola*rgr Canola*(1-TimeToHarvest canola)

growRR=rgr weed*WeedRR

growRS=rgr weed*WeedRS

growSS=rgr weed*WeedSS

grow SweedRR=SuperWeedRR*rgr weed

grow SweedRS=SuperWeedRS*rgr weed

harvest Canola=GM Canola*TimeToHarvest canola/TIME STEP

harvest index=0.25

herbicide decay ratio=0.35

herbicide degradation=herbicide decay ratio*herbicide in land

herbicide in land= INTEG (+herbicide sprayed-herbicide degradation,0)

herbicide sprayed= fraction of ref herbicide sprayed*spray ratio*TimeToSpray*ref herbicide

herbicide used=herbicide sprayed

HI Sweed=0.15

hyb prob RR=ZIDZ(0.5*(WeedRS+SuperWeedRS)+WeedRR+SuperWeedRR, total B weed+superweed)

hyb prob RS=ZIDZ(0.5*(WeedRS+SuperWeedRS)+WeedSS,total B weed+superweed)

"initial-RR"=7e-007

"initial-RS"=5e-005

"initial-SS"=5.2

max rgn ratio Canola=1.3

max rgn ratio weed=1.1

max yield=450

MonthsInYear=MODULO(Time, 12)

new SweedRR=hyb prob RR*seed Sweed/TIME STEP

new SweedRS=hyb prob RS*seed Sweed/TIME STEP

newSeed RR=probRR*total seed*(1-outcrossing fr)/TIME STEP

newSeed RS=probRS*total seed*(1-outcrossing fr)/TIME STEP

newSeed SS=prob SS*total seed*(1-outcrossing fr)/TIME STEP

outcrossing fr=ref frequency*eff of crop to weed ratio on hybridization

prescribed amount=150

probRR=active res allele fr*active res allele fr

probRS=2*active res allele fr*(1-active res allele fr)

probSS=(1-active res allele fr)*(1-active res allele fr)

ref frequency=0.3

ref herbicide=150

ref ratio=20

ref weed=25

res allele fr=ZIDZ(2*(SeedBank RR+WeedRR)+(SeedBank RS+WeedRS), 2*(total B weed+total seedbank))
rgr Canola=max rgn ratio Canola*eff of competition on canola
rgr weed=max rgn ratio weed*eff of competition
seasonal yield loss=yield loss canola/TIME STEP
seed Superweed=superweed*HI Sweed*TimeToHarv weed*seedloss
seed Sweed=total seed*outcrossing fr
seed weed=harvest index*seedloss*total B weed*TimeToHarv weed
SeedBank RR= INTEG (+newSeed RR-decay RR-grm RR,"initial-RR")
SeedBank RS= INTEG (+newSeed RS-decay RS-grm RS,"initial-RS")
SeedBank SS= INTEG (+newSeed SS-decay SS-grm SS,"initial-SS")
SeedBank SweedRR= INTEG (+new SweedRR-decay SweedRR-grm SweedRR,0)
SeedBank SweedRS= INTEG (+new SweedRS-decay SweedRS-grm SweedRS,0)
seeding period=TIME STEP
seedloss=0.2
seeds=130000
seedweight=4e-006
sow Canola=seedweight*seeds*TimeToSeed canola/seeding period
sowRR=SeedBank RR*grm ratio weed*TimeToSeed weed
sow RS=grm ratio weed*SeedBank RS*TimeToSeed weed
sow SS=grm ratio weed*SeedBank SS*TimeToSeed weed
sow SweedRR=grm ratio weed*SeedBank SweedRR*TimeToSeed weed
sow SweedRS=grm ratio weed*SeedBank SweedRS*TimeToSeed weed
spray ratio=1/ TIME STEP
superweed=SuperWeedRR+SuperWeedRS
SuperWeedRR= INTEG (+grow SweedRR+sow SweedRR-clean SweedRR,0)
SuperWeedRS= INTEG (+grow SweedRS+sow SweedRS-clean SweedRS,0)
TimeToHarv weed=IF THEN ELSE(TimeToHarvest canola=1, 1, 0)
TimeToHarvest canola=IF THEN ELSE(GM Canola>0:AND:MonthsInYear>11, 1, 0)
TimeToSeed canola= IF THEN ELSE(MonthsInYear=0, 1, 0)
TimeToSeed weed=IF THEN ELSE(MonthsInYear>=0:AND:MonthsInYear<2, 1, 0)
TimeToSpray=IF THEN ELSE(MonthsInYear=2:OR:MonthsInYear=5, 1, 0)

total B weed=WeedRR+WeedRS+WeedSS
total herbicide= INTEG (herbicide used,0)
total seed=seed weed+seed Superweed
total seedbank=SeedBank RR+SeedBank RS+SeedBank SS
total weed=total B weed+superweed
total yield loss= INTEG (seasonal yield loss,0)
WeedRR= INTEG (+growRR+sowRR-cleanRR,0)
WeedRS= INTEG (+growRS+sow RS-cleanRS,0)
WeedSS= INTEG (+growSS+sow SS-cleanSS-control SS,0)
yield loss canola=(max yield-harvest Canola*TIME STEP)*TimeToHarvest canola

D.2. Modifications and New Equations for Conventional Canola

Canola= INTEG (growCanola+sowCanola-harvestCanola-yield loss due to herbicide,0)
newSeedRR=probRR*seed weed/TIME STEP
eff of herbicide on canola= WITH LOOKUP (herbicide in land/ref herbicide, ([(0,0),(4,0.25)],(0,0),(0.0122324,0),(0.0978593,0.0230263),(0.379205,0.0767544), (0.66055,0.15,(0.966361,0.16886),(1.18654,0.1875),(1.34557,0.195175),(1.57798,0.200658),(4.02446,0.199561)))
seed weed=seedloss*total B weed*TimeToHarv weed*((1-outcrossing fr)*harvest index+HI Sweed*outcrossing fr)
yield loss due to herbicide= Canola*eff of herbicide on canola*(1-TimeToHarvest canola)

D.3. Modifications and New Equations for Rotation Analysis

CanolaVol= INTEG (+growVol+sowVol-cleanVol,0)
cleanVol=CanolaVol*TimeToHarv weed/TIME STEP
decayVol=SeedbankVol*decay ratio
eff of comp on vol= WITH LOOKUP ((

GM canola+CanolaVol+alfaCanola*(superweed+Wheat+total B weed))/carrying cap canola,([(0,-0.02)-(1.5,1)],(0,1),(0.0856269,0.995614),(0.174312,0.95614),(0.311927,0.881579),(0.425076,0.785088),(0.553517,0.653509),(0.697248,0.473684),(0.825688,0.285088),(0.905199,0.144737),(0.95107,0.0614035),(1,0),(1.05199,-0.01),(1.20489,-0.012),(1.5,-0.015)))

eff of competition on wheat= WITH LOOKUP ((Wheat+"alfa-wheat"*total weed)/"carrying cap-wheat",([(0,-0.02)(1.5,1)],(0,1),(0.0856269,0.995614),(0.174312,0.95614),(0.311927,0.881579),(0.425076,0.785088),(0.553517,0.653509),(0.697248,0.473684),(0.825688,0.285088),(0.905199,0.144737),(0.95107,0.0614035),(1,0),(1.05199,-0.01),(1.20489,-0.012),(1.5,-0.015)))

eff of herbicide on wheat= WITH LOOKUP (herbicide in land/ref herbicide,([(0,0)(4,0.25)],(0,0),(0.0122324,0),(0.146789,0.0219298),(0.452599,0.066886),(0.892966,0.130482),(1.23547,0.177632),(1.48012,0.201754),(1.61468,0.211623),(1.7003,0.217105),(2.00612,0.218202),(4.02446,0.221491)))

grm ratioVol=0.1

grmSeedVol=SeedbankVol*grm ratioVol*TimeToSeed weed

increase rate=IF THEN ELSE(MonthsInYear=11.75, 1/TIME STEP, 0)

newSeedVol=seeds Vol/TIME STEP

reset=IF THEN ELSE(RotationYear=3:AND:MonthsInYear=11.75, (RotationYear+1)/TIME STEP, 0)

RotationYear= INTEG (increase rate-reset,0)

SeedbankVol= INTEG (+newSeedVol-decayVol-grmSeedVol,0)

seeds Vol=(CanolaVol*TimeToHarv weed+GM Canola*TimeToHarvest canola)*Loss Canola*HI Canola*escaped seed fr

seeds wheat=277647

seedweight wheat=3.4e-005

sowVol=SeedbankVol*TimeToSeed weed*grm ratioVol

Wheat= INTEG (growWheat+sowWheat-harvestWheat-"yield loss due to herbicide-Wheat",0)

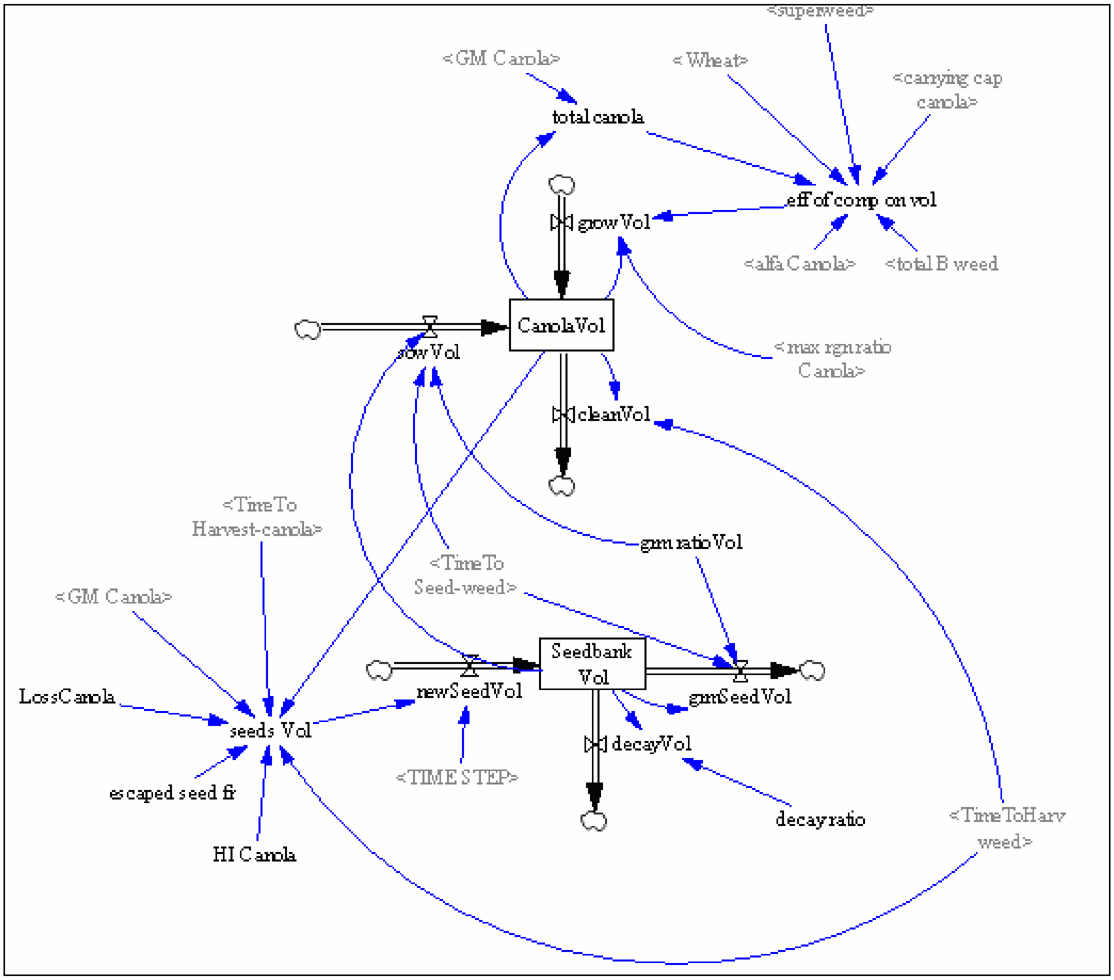


Figure D. 1. Stock-flow diagram of Volunteer sector

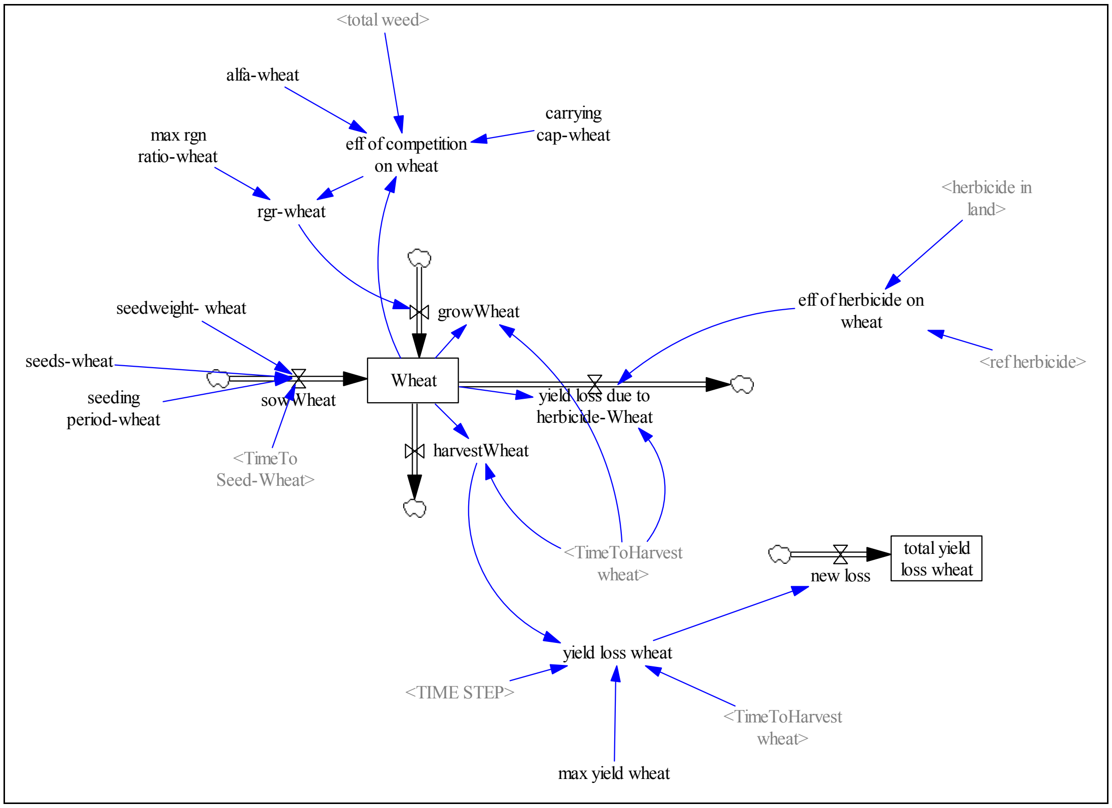


Figure D. 2. Stock-flow diagram of Wheat subsector

APPENDIX E: GLOSSARY

Allele: One of the different forms of a gene that can exist at a single locus

Biotechnology: The science and art of genetically modifying an organism's DNA, such that the transformed individuals can express new traits that enhance survival (e.g., insect or disease resistance, herbicide resistance) or modify quality (e.g., oil, amino acids).

Bt: Bacillus thuringiensis; a bacterium that kills insects; a major component of the microbial pesticide industry

Bt-corn: GM corn, which contains genetic material from one of several strains of the bacterium Bacillus thuringiensis, thereby which has the insect resistance or insecticidal property

Dominant allele: An allele that expresses its phenotypic effect even when heterozygous with a recessive allele; thus if A is dominant over a, then AA and Aa have the same phenotype

ECB: European corn borer; a pest of corn

Fitness cost: The possible drawbacks of increased fitness to the environment

Functional response: All the predator's behaviors that determine how many prey it will capture per unit time

Gene: The basic unit of inheritance and diversity; a section of DNA that codes for a specific product (e.g., protein) or trait

Genotype: The genetic makeup, as distinguished from the physical appearance, of an organism or a group of organisms

Glyphosate: A nonselective (broad spectrum) herbicide effective on a wide spectrum of weeds

Herbicide: Any substance that is toxic to plants; usually used to kill specific unwanted plants

Herbicide Resistance: The inherited ability of a plant to survive and reproduce following exposure to a dose of herbicide normally lethal to the wild type

Herbicide Tolerance: The inherent ability of a species to survive and reproduce after herbicide treatment. This implies that there was no selection or genetic manipulation to make the plant tolerant; it is naturally tolerant.

HI: Harvest Index; an index which denotes the harvested part of the crop relative to the total above ground biomass production

Hybridization: The mating of individuals from different species or sub-species

Integrated Pest Management (IPM): A management approach that integrates multiple, complementary control tactics (e.g., biological control, crop rotation, host plant resistance, insecticides) to manage pests in a profitable, yet environmentally sound manner

Mode of action: The way a pesticide works to kill pests; for example, a poison that works on contact or as a stomach poison

Outcrossing: Cross-pollination, or fertilization of flowers on a plant by pollen from another plant

Pest: A general term for organisms (rats, insects, weeds etc.) which may cause illness or damage or consume food crops and other materials important to humans

Pesticide: A substance that kills harmful organisms (for example, an insecticide or fungicide)

Phenotype: The expression of a specific trait, such as stature or blood type, based on genetic and environmental influences

Recessive allele: An allele that is not expressed in the heterozygous condition

Refuge: An area planted to non-transgenic plants, e.g., non-Bt corn or alternative hosts for European corn borer, where susceptible pests can survive and produce a local population capable of inter-mating with any possible resistant survivors from Bt-corn

Resistance: The capacity of an organism to survive exposure to a toxin

RR: An individual whose both alleles for a specific trait are R (who is homozygous in R)

RS: An individual whose alleles for a specific trait are R and S (heterozygous)

SS: An individual whose both alleles for a specific trait are S (who is homozygous in S)

Transgene: A gene introduced into the genome of an organism by genetic manipulation in order to alter its genotype

Volunteer: A plant that germinates from a seed left behind in the field from a previous crop.

Weed: Any plant that is growing in a place where a human wants a different kind of plant or no plants at all.

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