Dos and Don'ts in Augmentative Biological Control: Insights from Mathematical Modelling

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présentée et soutenue par

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Abstract

This thesis presents the results of the mathematical analysis of models in augmentative biological control. Biological contro in agriculturel involves the use of natural predators to suppress pest invertebrates that attack crops. It is an essential component in efforts to reduce pesticide usage. In this thesis, we model augmentative biological control programs, in which a fixed number of predators are periodically released. Such predators are usually not able to establish in an ecosystem in the absence of the pest. We introduce a general class of impulsive models that describe the intrinsic predator-prey dynamics by a pair of ordinary differential equations and the periodic releases by a discrete equation. We study the variants of this class of models that may arise in a biological control set-up and highlight the consequences on the strategy of releases. In particular, we analyse the effect of intra-predatory interference occurring when the predator preys on the pest, the impact of cannibalism among the predators, and finally the outcome of partial harvests of crops on the biological control program. We also interpret the results of our mathematical analysis as a set of practical guidelines. We report experiments on an agronomic predator-prey system, in which the predator species exhibits interfering behaviour. The experimental results validate our mathematical predictions.

Keywords: impulsive systems, zero-pest solution, local stability, global stability.

Abstract

Résumé

Les travaux présentés dans cette thèse portent sur des problématiques de modélisation mathématique de lutte biologique augmentative et des recommandations pratiques qui en sont dériées. La lutte biologique est une méthode de phytoprotection visant à combattre les ravageurs des cultures à l'aide de leurs ennemis naturels ; son développement est crucial en vue de diminuer l'utilisation de pesticides qui représente un risque notamment pour la santé des agriculteurs et des consommateurs, mais aussi pour l'environnement. Dans cette thèse, nous nous intéressons plus particulièrement à la lutte biologique augmentative, qui consiste à lâcher périodiquement des ennemis naturels qui n'ont pas la capacité de s'établir dans l'environnement en l'absence des ravageurs cibles. Nous introduisons une famille générique de modèles représentant d'une part la relation proie/prédateur à la base de la lutte biologique sous forme d'équations différentielles ordinaires, mais également les lâchers périodiques sous forme d'événements discrets. Nous précisons ensuite ce modèle pour diverses situations rencontrées dans le cadre de la lutte biologique et indiquons quelles en sont les conséquences sur les stratégies de déploiement des ennemis naturels : nous étudions notamment l'effet d'interférences entre prédateurs pour l'accès aux proies, l'existence de relations de cannibalisme entre prédateurs et les conséquences que peuvent avoir des récoltes partielles des plantes sur l'efficacité de la lutte biologique. Enfin, nous résumons tous nos résultats sous forme de recommandations pratiques pour la lutte biologique et en présentons une validation expérimentale sur un exemple agronomique d'intérêt, dans lequel les prédateurs entretiennent des relations d'interférence.

Mots-clefs : systèmes impulsionnels, solution zéro proie, stabilité locale, stabilité globale.

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To my Dadi, Parvati Kumari Nundloll.

1 Introduction

(...) Du plus gros au moindre dans la série animale, le ventre est le souverain du monde ; les données fournies par le manger dominent les autres documents de la vie.
– J-Henri Fabre (1823–1915)

1.1 Understanding biological control

1.1.1 Who kills who?

At the heart of the biological control problem lies a very simple question: "Who kills who?" It is the answer to this question which determines the implementation of a biological control program. In most cases, the motive for killing is, simply, food: one organism kills another and feeds on it. Killing and feeding on one resource may also have indirect effects on another; for instance, the exploitation of a common food source by one organism also leaves another less competitive organism to die out. Not all killing is food-based however - we discuss this in more details shortly.

The idea of an organism as the food of another has been formalised in ecology several decades ago by the concept of *food webs*. This formalism represents the interdependence of species one or more species being the food resource of another or others (Odum, 1968). The network of connections within the food web organise species across different *trophic* or food levels, between which biomass (or energy) flows from a lower level species (a lower trophic level) to a higher level one (a higher trophic level) which feeds on it. Species on one trophic level can also be grouped within *functional groups*, which determines their relationship with respect to a particular organism. Examples of functional groups that are typically located at one trophic level above the organism are predators, parasitoids and pathogens. These attack the organism in different ways and may or may not feed on it. Competitors are another functional group that are at the same trophic level as the organism and compete for similar

[0] "*(...) From the least to the greatest in the zoological progression, the stomach sways the world; the data supplied by food are the chief of all the documents of life.*" Taken from *Souvenirs Entomologiques, Série X, Note 1*, translated by Alexander Teixeira de Mattos (1865–1921).

resources. The different species on which the organism in question feeds on form part of the functional group consisting of its prey.

Another more general formalism is that of the *ecological niche*. This formalism – as proposed by Cohen (1978) – considers, in addition to the trophic relations of a target organism with its immediate food web community, the influence of environmental factors on it. These environmental factors refer to as much to the ambient conditions (such as temperature, light, humidity, as well as climate) as other "non-living resources' it uses such as space (in particular to habitat structure or shelter). All these factors combined with the target organism's behaviour or strategy in optimising its survival define the ecological niche[1].

A systemic definition of biological control fits neatly in this framework as follows:

The *biological control* of a *population* consists of manipulating the *functional groups* and/or *energy pathways* that determine its survival (or disappearance) within its *ecological niche*.

Figure 1.1 represents the ecological niche of a target organism (red box). The **functional groups** (blue boxes, and the autocide) are those that are exploited in biological control programs. The resources (green box) can be living as well as non-living (e.g. space, water, ...) are also used in other forms of control programs. Arrows **i-iv** represent **food pathways** through which there is an actual mass or energy transfer following the direction of the arrows. The arrow **i** corresponding to a host-parasitoid interaction is particular since the killing is performed by an individual whose progeny feeds on the killed organism; however at the population scale it still remains a mass/energy transfer. Broken arrows **v** and **vi** indicate that group at the source of the arrow has a **disruptive effect** on the target: there is no actual mass transfer. Pathogens cause mortality through disease; autocides are sterile conspecifics of the target organism whose progeny is also sterile and cause a population collapse.

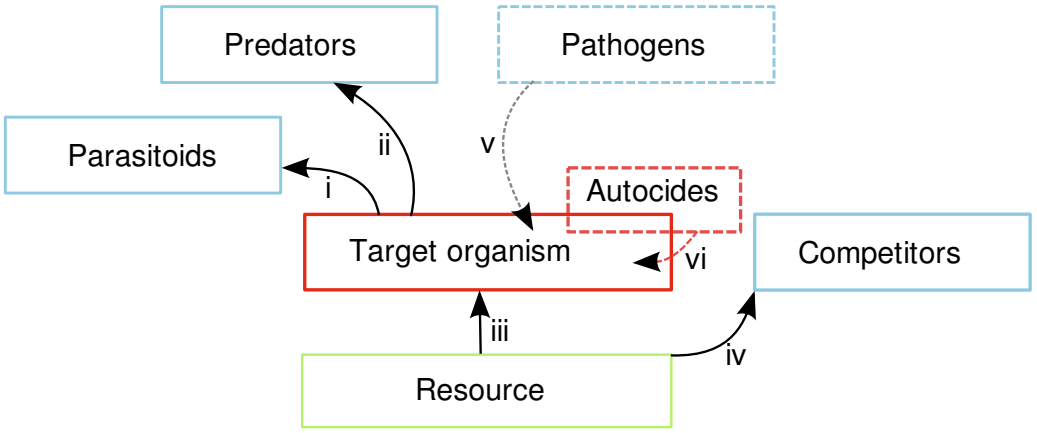

Figure 1.1: An organism (red box) belongs to an ecological niche, which consists of its immediate natural enemies (blue boxes) and its resources - living or nonliving, such as space, water - it consumes (green box) (Hutchinson, 1967). This definition is extended to include autocides. Arrows **i-iv** refer to mass transfer pathways across groups. Arrows **v** and **vi** represent disruptive effects which do not involve mass transfer.

The transfer through the pathways or the intensity of one functional group on another (such as the target organism) is determined by the *size* of the functional groups, the *attack*

[1]This holistic definition proposed by Cohen (1978) has evolved from Elton (1927), through Hutchinson (1967) (as cited by Whittaker et al. (1973)).

mechanisms and the *defence mechanisms* of the various species. These three features are influenced by abiotic factors. In addition, attack and defence mechanisms are usually related to specific phenotypic traits (behavioural and physical characteristics) of the respective organisms (Hill, 1975), some of which can be enhanced or repressed through genetic manipulation.

Attack mechanisms of a natural enemy are determined by intraspecific interactions. Intraspecific competition makes food searching or attacking more difficult. Cannibalism suppresses the need to consume other organisms for survival. Conversely, cooperative behaviours improve foraging abilities. Some species or strains are just also more effective (or virulent in the case of viruses) than others.

Defence mechanisms among animal species include antipredator behaviour such as nest constructions which hinder predator attacks and cooperation where individuals fight back predators. Among plants, defence is more complex: many plants contain chemicals that get released when herbivories attack it; these chemicals may then attract the herbivory organism's enemies (Turlings and Wäckers, 2004) or act directly against the pests ('antibiosis' phenomenon, see e.g. (Gassman et al., 2009)).

Remark: *Organisms in each of the functional groups belong to their respective ecological niches. The predator, for instance, is prey to another predator; considering it as the new 'target organism' shifts its prey (previously considered as the target organism) to the resources level.* ■

1.1.2 Kill the pest!

Crop protection in agriculture

More often than not, in a plantation system, the target is an undesirable (thus defined from the human point of view) organism that destroys a crop. Such an organism is called a *pest*. it needs to be suppressed, if not completely eliminated in order to protect the crop and ensure satisfactory yields.

One of the most common ways to do so has been by applying chemical pesticides. Pesticides modify directly the environment of the organism within its niche. Usually inhaled or absorbed through the exoskeleton, they operate by disrupting nervous or other systemic processes within the target organism. DDT for instance acts on sodium channels in the nerve cells of insects. Organophosphates also attack the nervous system of insects and kill them. Other pesticides are non-lethal to an organism, but will however damage its reproductive system and thus bring about a collapse in the pest population one generation later.

Since the post World War period, chemical pesticides have been easily mass produced and their effectiveness when used against pests explains to a large extent their ubiquity (Metcalf, 1994); so does perhaps the lobbying power of pesticide producers (Van den Bosch, 1978). As one of the pillars of intensive plantation farming programs – which is the most common form of mass agriculture produced today –, the pesticide industry took off during the post World War period and diversified. In 2000, Van Lenteren (2000) reported that the industry was worth $30 billion in the US alone.

Reducing pesticide usage

There are several problems related to pesticide usage. In *higher doses* than that required for insects or other pest control programs, pesticides can also be toxic to more complex organisms such as mammals (including humans). The *persistence* of some pesticides can pose serious risks to human health due to accumulation effects.

The frenzy over pesticides, given their success in pest control programs, has unfortunately led to their abuse in the 1970s. An immediate and visible consequence was that many treated areas in the USA saw their biodiversity collapse. In fact, at that time, most pesticides were broad-spectrum, that is they killed a wide range of species: the use of DDT, for instance, to control pests mass-killed pollinating bees Metcalf (1994). Some regions also suffered from secondary pest outbreaks: herbivory insects that were previously maintained at low level by local enemy populations and were not considered as pests underwent a population explosion causing huge crop destruction (see e.g. King and Powell (1992), as well as references in Metcalf (1994), and Ch.1 in DeBach and Rosen (1991)). They acquired in the process their status as pests. Many pesticides were consequently banned or their use, such as DDT, restricted (Berenbaum et al., 2000). This measure mitigated greatly health and environmental risks.

The mode of action of pesticides (targetting specific physiological processes in a living organism) has however another serious drawback: that of *pest resistance*. Survivors of the first pesticide application reproduce into another generation of resistant individuals who require a higher dose of the same pesticide or the application of a more potent one to be eliminated. This adaptation of the pest population to the pesticide versus the development of new pesticides is referred to as the pesticide treadmill or the pesticide trap.

This issue was reported back in RCEP (1979). This problem remains an important environmental and economic issue to this day. Compiling available data, Metcalf (1994) pointed that more than 500 species in 1989 had developed resistance to at least one class of pesticides (see Figure 1.2). As Hoy (1998) stated on resistance, "Even aside from environmental and health issues [which fuel the pesticide debate], the use of pesticides is not *sustainable*".

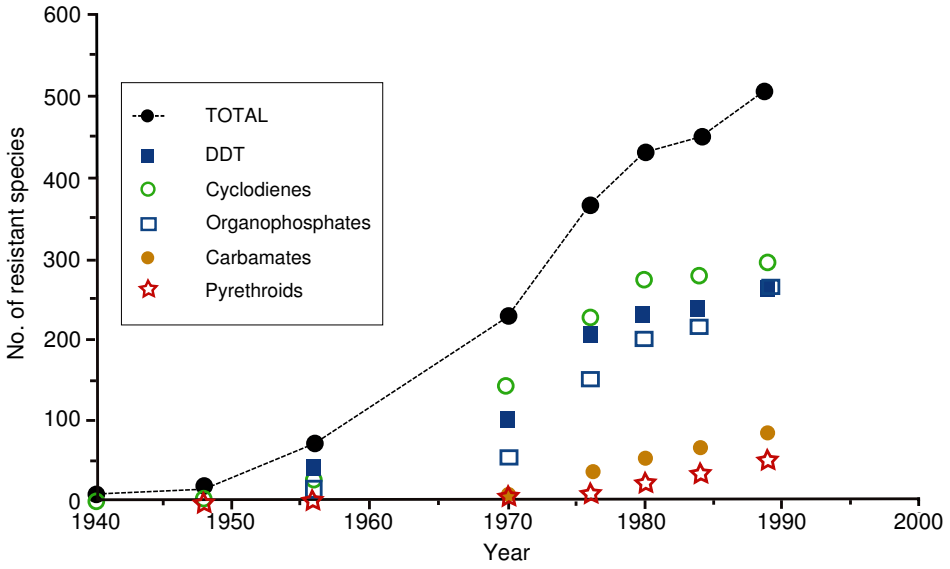

Figure 1.2: Increasing insecticide resistance is major problem in pest control. The data shows the increase for five classes of insecticides over 1940-1989 (from P.251, in Metcalf (1994); see references therein for data sources).

Due to these issues (persistence, resistance and risks to the environment and human health), pesticide usage needs to be reduced. In order to provide adequate pest control, exploiting other dimensions of the ecological niche, namely through biological control, becomes essential.

Remark: *Health and environmental issues motivate a reduction in pesticide usage indirectly, in that they are considered to be the main factors that galvanise public opinion and sway market movements. In fact, while contamination of the environment has been reported widely and in much detail (see e.g.Berenbaum et al. (2000); Voltz et al. (2005); RCEP (2005); Moss (2008)), studies that measure the impact on the environment of this contamination are often too fragmentary and are unable to offer precise indices to be conclusive (see the discussion by Moss (2008), and DEFRA (2006)). Likewise for the impact of pesticides on human health (see e.g. Baldi and Lebailly (2007)). We summarise the results of a small bibliographical research on the matter in Appendix A.* ■

1.1.3 Use predators

In crop protection, one of the ways to exploit the ecological niche of various pests in their control involves the use of their natural enemies. Figure 1.3 gives an example of the food web surrounding a crop.

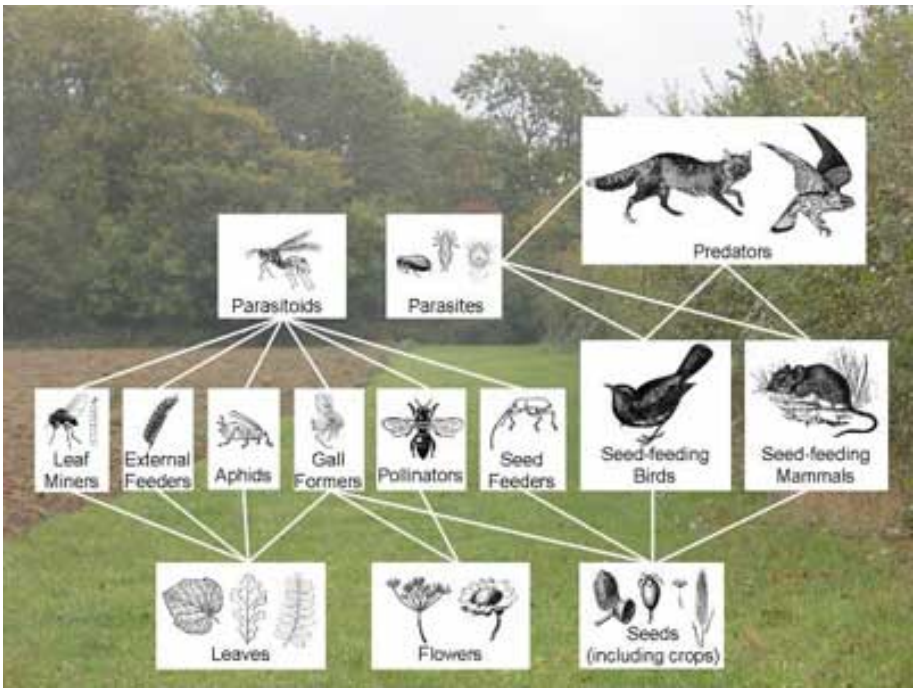

Figure 1.3: Part of a typical food web in a farm shows plant components, herbivores (pests and non-pests) and their predators/parasitoids that can be used to control them. Other animals interacting indirectly with the herbivores are birds and mammals that share a common base resource with them. Their feeding behaviour impacts indirectly the herbivores. From Memmott (2009), the Norwood Farm Project, UK. Available online at: *http://www.bio.bris.ac.uk/research/community/fwproject5.html*.

The two major groups of natural enemies used are predators and parasitoids (see e.g. Van Lenteren (2000) for greenhouse crops). However, because of their relationships with respect to the pest which is fairly similar (i.e. they both kill and feed on the pest), we will use the same terminology as Begon et al. (1996) and refer to “predator" these two types of natural enemies (see Figure 1.4).

In pushing for the wide-scale implementation of such biological control programs, field

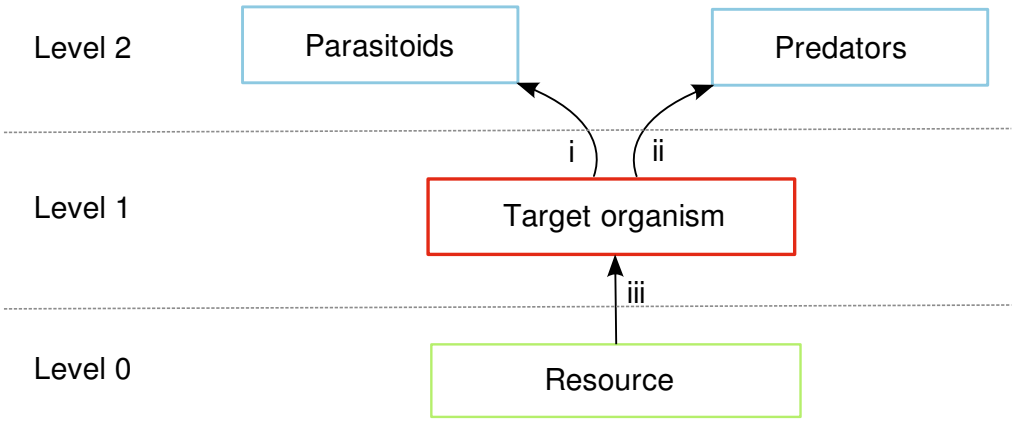

Figure 1.4: Because of the relationships with respect to the herbivory pest (Level 1), predators and parasitoids belong to the same trophic level above the pest (Level 2) (Begon et al., 1996). The crop on which the pest feeds is on a lower level (Level 0).

researchers such as Joop van Lenteren (greenhouse crop protection in the Nethelands) and Paul Van Mele (mango and citrus crop protection in Vietnam and Africa) have noted that from the growers' perspective, there are several **practical reasons** why they would be willing to switch from pesticide use to biological control.

We outline these reasons as follows[2]:

- **Reduced resistance issues.** Biological control by predators circumvents this problem simply because pests and their natural enemies evolve concurrently on a much slower evolutionary scale. To quote Van Lenteren (2007a): "once a good natural enemy, always a good natural enemy."
- **A better or the only control method available.** Sometimes a natural enemy does a better job than would a chemical pesticide. Weaver ants, for instance, are very meticulous in their handling of their prey, the mango fruit flies (Van Mele et al., 2007). In other cases, whether due to resistance issues (see above) or otherwise (pesticides have not been developed), a natural predator is the only control agent for a given pest.
- **Positive consumer perceptions to organic products.** In Europe and North America, consumers are increasingly ready to pay premiums and drive the organic market or alternate farming movement. In fact, these premiums may provide incentives to farmers in the developing world to adopt alternative methods of farming and pest control (Byers et al., 2008).
- **No premature abortion of flowers and fruits.** Due to their toxicity, some pesticides damage plants at early stages of their development. No such risk is known to exist with natural specialist predators which feed exclusively on the pest (Van Lenteren, 2007a; Gerson et al., 2003). Even for predators that are generalists – that is which eat plants as well as pests, the risk is minimal.
- **No discomfort during spraying.** In greenhouses, with high humidity levels, workers prefer alternative methods to pesticide spraying (Van Lenteren, 2000) (see also RCEP (2005)). Most likely because of the toxins they contain, pesticides often cause headaches

[2]This list is guided by Van Lenteren (2007a) and is substantiated by other sources.

and may also represent the strongest health risks (namely cancers, see e.g. Baldi and Lebailly (2007)) to the people directly manipulating them. Predators allow planters to circumvent this issue.

- **Timing convenience.** The use of natural predators is perfectly safe: unlike the application of pesticides, there is **no buffer time zone** after the release of predators. This is convenient for growers whose work is uninterrupted. In some cases, less applications of biological control agents may be required than pesticide applications - this not only saves time but also labour costs (see e.g. Stevens III et al. (2000)).
- **Lower costs of implementation.** Biological control can offer a less costly alternative to pesticides in some cases simply because biological control agents can sometimes be found locally (Van Mele et al., 2007). They can in some cases also require less applications (Stevens III et al., 2000).

1.1.4 Augmentative control

There are three control schemes for crop protection by predators ((Hoy, 1994)). The first, *conservation*, involves no release and focuses instead on increasing a resident natural enemy population by landscape or habitat management and ensuring an adequate - often alternate - food supply at low prey levels. This technique can be categorised under cultural control[3].

Classical control involves one sufficiently large release of a (usually artificially reared or non-indigenous) predator population. The ultimate aim for this release is the establishment of the predator population to provide permanent control for potential pest species. It may however result in undesired non-target effects, especially since the introduced predator species may become invasive and threaten the indigenous fauna (see e.g. Howarth (1991) for a review).

The third scheme, referred to as *augmentative control* involves repeated releases of a (usually artificially reared or non-indigenous) predator. A key feature of this scheme is that there is **no establishment of predators**. This can be deliberately done (for example to prevent invasions by the exotic predator) or simply because the predators would not survive anyway in the absence of pests. Releases then provide the only (substantial) way to replenish the populations.

In this thesis, we model augmentative predator releases. Because of the continuous replenishment of the predator population, this scheme can work for most predator species. Predators feeding voraciously on the pest species would however be more advantageous as they would result in a less costly release program.

Remark: *An augmentative release program can become classical when releases that are programmed to be large and rare (referred to as an inundative release) result unwittingly in the establishment of the predator. This is less likely to happen when releases are small and frequent (referred to as inoculative releases). In this thesis, we will consider that there is no establishment of the predator population in the absence of pest in either case.* ■

1.1.5 Predator types

Predators can be classified into two broad categories, according to their diet: specialists and generalists. In the case of predaceous mites, which represent a major group of predators for common agricultural pests, the classification can be refined into four categories which depend

[3] See also shortly the section **Integrated Pest Management, IPM**

on the behavioural traits and diet patterns, habitat sensitivity and even physical characteristics (see Ch. 26 in Gerson et al. (2003) and references therein). Under this classification, specialist predators are referred to Type I, while generalists belong to either one of Types II-IV. The choice of a predator control program (classical vs. augmentative releases, releases vs. conservation) depends on this characterisation. We highlight the key features of each category and type as follows:

Specialists

Type I feed on only one prey species, in the absence of which they die out. Group dynamics include high aggregation about a prey source. Such predators can only be used in *augmentative control programs*. Examples include *Phytoseiulus persimilis* (Athisa Henriot), *Metaseuilus occidentalis* (Nesbitt). Note also that because they are very specialised to their hosts, parasitoids such as *Encarsia formosa* (Gahan) that can only work in augmentative release programs may be considered to belong to this category.

Generalists

Generalists feed on other resources such as alternate prey species, other predators, plant components such as pollen and exudiates.

Type II are generalists that eat preferentially the target prey species (the pest). They cannibalise or prey on competitors only out of starvation. They display high aggregation about a prey source. Such predators can only provide control in augmentative release programs. Examples include *Galendromus occidentalis* (Nesbitt) (control of webbing spiders in apple orchards), *Neoseiulus fallacis* (Garman) (control of European red mite, other phytophagous mites e.g. eriophyoids in apple trees).

Types III & IV are omnivory generalists that preferentially feed on other prey than tetranychids, and consume readily pollen and plant exudiates. They also more often indulge in cannibalism and interspecific predation than do **Types I** and **II**. They display low aggregation. Some can only reproduce on specific prey (which can be a pest), so will maintain control only through an augmentative release scheme. One such example is *Typhlodromalus manihoti* (Moraes) (control of cassava green mite in Africa).

Figure 1.5: *Left.* Picture of a group of *Neoseiulus Californicus* handling their prey, the pest *Tetranychus urticae* ©Alexandre Bout. *Right. Phytoseiulus persimilis* handling its prey (right), the pest *Tetranychus urticae* ©Jarmo Holopainen.

Remark: *Diet patterns are intrinsic to a predator foraging behaviour, and consequently impacts directly a given prey uptake and hence a pest control program. In modelling terms, it has a direct effect on the* ***functional response*** *which represents the removal of a prey (in this*

thesis, the pest) by the predator. ■

See later: *The effects of predator behaviour and their diet spectrum on an augmentative release program are investigated through the models in Chapters 4 and 5.* ■

1.1.6 Integrated Pest Management (IPM)

Integrated Pest Management (IPM) involves using the whole array of pathways in the ecological niche (biological including genetic methods, conservation of the natural enemies through cultural methods) as well as direct removal of the pest (Luckmann and Metcalf, 1994; Morales-Ramos and Rojas, 2003). Figure 1.6 illustrates these different methods.

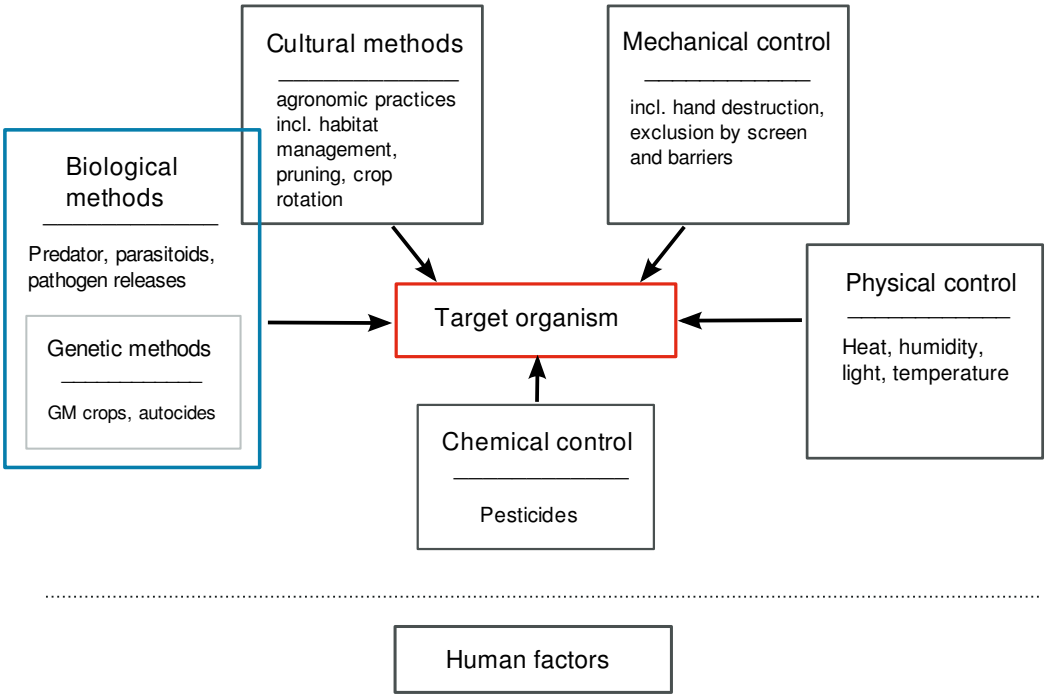

Figure 1.6: Integrated Pest Management (IPM) combines different techniques of pest control in order to decrease pesticide usage. The choice of the components or the extent to which biological, chemical or physical control methods are used for the IPM program depends on human factors such as expertise in the various components, consumer perception and awareness, and law and policy. Adapted from Luckmann and Metcalf (1994).

Remark: *Direct killing of the pests through physical methods, hand destruction, pruning , and chemical pesticides are instantaneous processes - like the augmentative releases of predators.* ■

See later: *We study the effects of pest (and predator) removal due to control methods (other than biological control) within an IPM program in Chapter 6.* ■

1.1.7 Some vital statistics

Predator species

In their review on biological control in greenhouses, Pilkington et al. (2010) report (from Van Lenteren (2007b)) that 150 species of natural enemies are available to growers worldwide. 30 species of these enemies, that is only one fifth of the available species, make up 90% of the global sales (from Bolckmans (1999)).

Outdoor crops

Resistance to pesticides may be the driving force for switching from chemical control programs to predator control. Metcalf (1994) reports that the European red mite *Paronychus ulmi* (Koda) and citrus red mite *P. citri* (McGregor) have become resistant to almost all arachnicides used in fruit production. In Egypt, cotton leafworm *Spodoptera littoralis* (Hübner) has become resistant to more than 20 insecticides, none of newly developed ones remaining effective for more than two to four years.

Conversely, a number of predators have been identified and tested in these crops and others. We note the use *Pediobius foveolatus* (Crawford) against the Mexican bean beetle in soybean protection, and *Trichogramma nubiale* (Ertle & Davis) against the European corn borer in corn protection (see e.g. Table 1, P 254 from Elzen and King (1999)).

Greenhouses

Pilkington et al. (2010) reports that up to 52,000 Ha of greenhouses operate on predator releases for pest control. This is a very small area compared to total world figures (estimated at around 2.43 million Ha, (Pilkington et al., 2010) - 2 million of which are in China (Zheng et al., 2005)).

However, we need to put this proportion in perspective: to date, 90% of the vegetable greenhouse production in the Netherlands is under IPM or biological control programs. This is probably because the effort to bridge the gap between entomological research and agronomic practice has paid off (Van Lenteren, 2007a). Given the proximity to other European countries, this know-how is likely to spread very quickly across this continent. The routine use of biological control in Almeria greenhouses in Spain (at 20,000 Ha) is probably a first step in that direction[4].

Greenhouse area is also very small compared to field crops areas, nonetheless it can make up a substantial proportion of agricultural production. In the Netherlands, 0.5% of agricultural land use dedicated to greenhouses which represent 20% of agricultural production.

Remark: *We gather from these statistics that: first, the expertise acquired in greenhouse crop protection as well as the availability of predator species on the market indicate biological control and IPM programs are likely to expand such pest control methods to a larger scale in greenhouses. Second, predator species that have not yet been exploited represent an untapped*

[4] Pilkington et al. (2010) reports that predators in Almeria are principally *Orius laevigatus* (Fieber), a generalist predatory bug, and *Amblyseius swirskii* (Athias-Henriot), a Type III predatoy mite which requires repeated releases when the crop is not a pollen producer.

potential for biological control. Third, there is also scope for a wider field application of biological control methods, though this development is likely to be slower than for greenhouses (these systems being open, predator populations are more difficult to track). The potential of biological control is apparent in figure 1.7, which shows the scale of land use for outdoor crop growth compared to greenhouse cultivations.

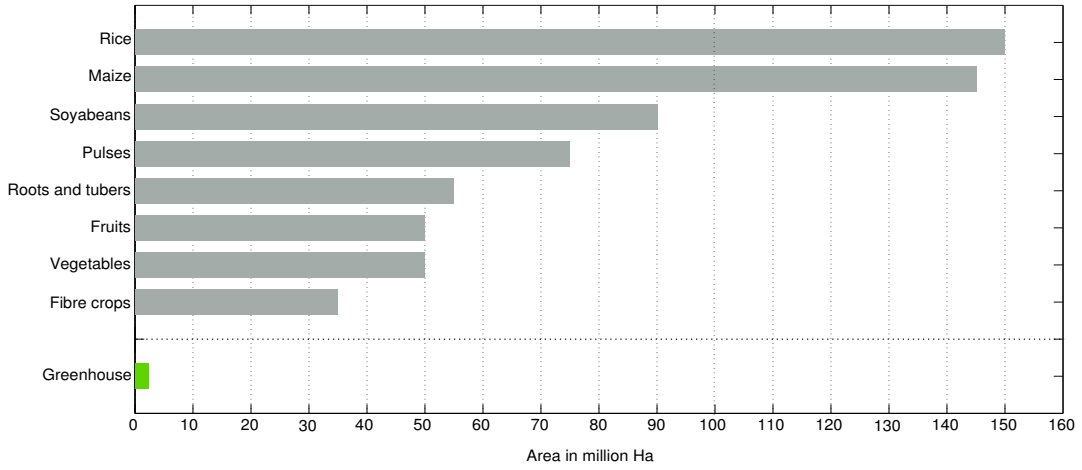

Figure 1.7: Several of the world major crops namely maize and soya beans can be protected with predator releases. Estimates in grey are based on FAO 2004 figures taken from The Economist (2005). Greenhouse crop figures are from 2005, based on Van Lenteren (2000), Zheng et al. (2005), and Pilkington et al. (2010) greenhouse crop production covers 2.43 million hectares. (Total world arable land use in 2007 was estimated at 1398 million hectares (FAO, 2009).)

■

1.2 Scope of this thesis

Goal and methodology

Based on the discussion presented in the previous section, the aims of this thesis is to provide a mathematical framework to improve current augmentative biological control (including those within an IPM programs) and develop new ones by informing experimental research related to potential predators. More specifically, this thesis aims to:

- Quantify the minimal number of biological control agents to release to eliminate pests in biological control programs;
- Identify release strategies based on the frequency and size of releases to ensure faster elimination of targeted pests;
- Provide a valuable measure of predator behaviour so as to determine if they can work as biological control agents - in particular, by quantifying interference and foraging effects.

To do so, we use qualitatively defined deterministic impulsive models so that our results can apply to the majority of predator-prey systems.

Main contributions

The main contributions of this thesis are:

1. *The qualitative modelling of the hybrid process describing an augmentative biological control program.* We provide sufficient detail so that general trends in the system behaviour may be observed and interpreted while modelling artefacts are reduced.

2. *Sector condition formulation to model arguments of the trophic functions and mortality functions in order to provide a complete qualitative description of these processes.*

3. *The global stability analysis of the zero-pest solution of the models.* The conditions are expressed as a minimal bound on the release rate that would eradicate the pest population. The global stability condition in the analysis of the various models proposed extends the local basin of attraction to the whole non-negative orthant.

4. *The practical interpretation of the stability conditions in terms of the pest control strategy.* The results of our analysis form the basis of recommendations for biological control experiments to be carried out at the French National Institute of Agricultural Reseach (INRA).

1.3 Detailed plan

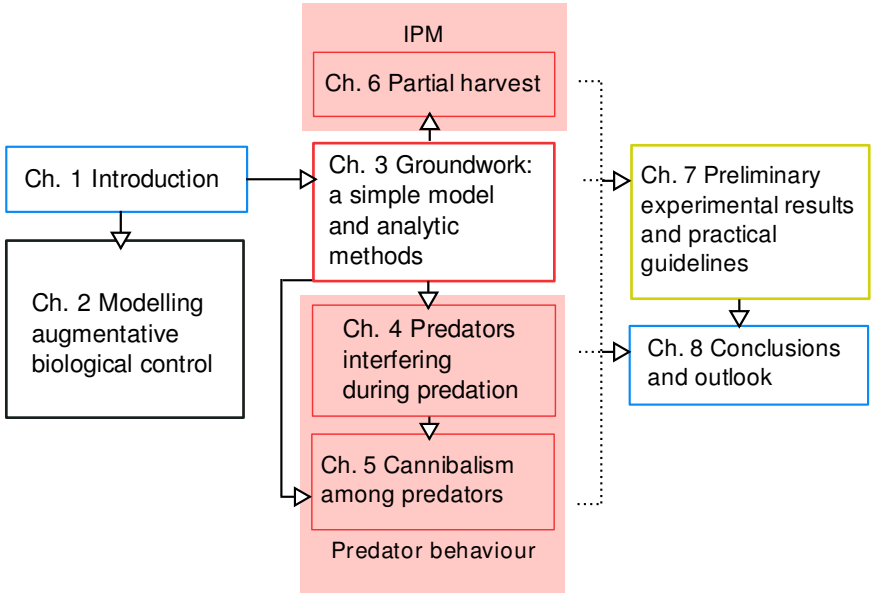

Figure 1.8: This map represents the interconnectivity of the different chapters of this thesis. The arrows indicate that information from one chapter (start of the arrow) is required for another (target of the arrow). The chapters in red represent the core of our work: the analysis of mathematical models. These chapters are motivated by Chapter 1. Chapter 2 situates it with respect to past and ongoing research trends. The Chapter 7 (in yellow) covers applications of our analyses. Chapter 8 points to potential research avenues that we have identified through our investigations.

Chapter 2. Making a choice: modelling approach This chapter presents the canonical form of the models used in this thesis to describe augmentative biological control. The modelling approach is characterised by two key features: The first are hybrid or impulsive dynamics, which depict the intrinsic predator-prey interactions as continuous processes and the periodic predator releases as discrete events. The second is the qualitative description of the predator-prey process functions. We explain in particular how modelling is about making a choice - from the number of variables and parameters that are used in the model to the mathematical framework used to describe the system. We defend our modelling choice with respect to the other models in the literature.

Chapter 3. Groundwork: a simple model and analytic methods. In this chapter, we introduce the framework used for the augmentative biological control programs described in this thesis. We present the analytic methodology used throughout our work and calculate the existence and stability condition of the zero-pest solution. These results provide the benchmark against which we compare the results of the models presented in this thesis.

Chapter 4. Predators interfering with each other during predation. In this chapter, we generalise the description of the functional response to account for intrapredator interference. We show that these behavioural characteristics have a substantial impact on the biological control program. They result in both a biological and a managerial condition for the stability of the zero-pest solution. Whether or not a stability condition of the zero-pest solution exists depends on the strength of the interference. We also study in this chapter the speed of eradication of a pest population.

Chapter 5. Cannibalism among predators. Predator cannibalise or kill conspecifiics for various reasons. In this chapter, we model three effects: territoriality, hunger and intrinsic diet. Territoriality is modelled as predator mortality enhanced by a quadratic term. The stability condition indicates that stability is favoured by small release periods (i.e. frequent releases). Hunger results cannibals which compete for conspecific victims. Finally, the last effect, intrinsic diet, takes into account intrapredator competition both in foraging for the prey and for conspecifics. This last model can in fact be extended to intraguild predation. Similarly to Chapter 4, we analyse the influence of these characteristics on the biological control program. We focus on the predator release strategy.

Chapter 6. Partial harvests. In this chapter, we look at impulsive removals of predator and pest populations that occur upon partial harvesting of crops or when pesticides (or other direct pest removal strategies in an IPM program) are in use. We consider that one of the periods of impulsive removals and of of predator releases is the integer multiple of the other. We treat the case when partial removals (harvests) are more frequent than releases and vice-versa. We show that the partial harvest frequency provides a threshold for the release frequency, below which biological control is less costly. We also look at the effect of varying partial harvest intensities on the respective predator and prey populations, and identify the cases when partial harvests disrupt control and when they aid towards eradication of pests.

Chapter 7. Practical guidelines. These guidelines are based on the mathematical analyses in Chapters 4-6. They provide suggestions for biological control experiments. We also report results from the laboratory experiments carried out at INRA[5] on intrapredator interference.

[5]The French National Institute for Agricultural Research

Chapter 8. Conclusion and outlook. We provide a summary of our results and list the publications arising from the work presented in Chapters 4-6 of this thesis. We also discuss the limitations of our results and propose potential avenues for further research.

2

Making a choice: modelling approach

Overview

The interdependency of species and their sensitivity to their surroundings underlie the complexity of biosystems. As a result, modelling such systems mathematically usually boils down to making compromises or a choice on the most relevant parameters and processes involved. This choice is to a great extent motivated by the context of the problem and the questions that the model seeks to resolve. In this chapter, where we present the canonical model studied for this thesis, we explain the process of building this model and the reasons behind the approach we use. We also take the opportunity to review the literature with respect to the two basic features of our model: its hybrid or impulsive nature and the use of qualitative descriptions for predator-prey processes.

Keywords: Qualitative modelling, impulsive control, predator-prey dynamics.

Organization of this chapter:
This chapter is structured as follows.

Section 2.1 explains how we build the model for the augmentative control program. It includes the assumptions made that lead to the modelling framework used throughout this thesis and introduces the canonical form of the model.

Section 2.2 highlights two fundamental mathematical properties of the system, namely positivity and local Lipschitz continuity of the intrinsic predator-prey processes.

Section 2.3 puts forward the need to model augmentative control with an impulsive system (rather than a purely continuous one). We comment here the impulsive control literature and the pertinence of hybridity as opposed to continuity.

Section 2.4 presents the various families of functions for each of the intrinsic predator-prey processes. It also highlights the genericity of the results obtained when using a metamodel that relies on general trends instead of explicit descriptions of processes.

Section 2.5 provides a discussion on the difficulties of modelling biological systems. It also illustrates how meta-modelling helps circumvent these inherent problems whilst also providing some insight on the general behaviour of a system.

Section 2.6 summarises the features of our model. We give a brief overview of other models in the pest control literature that use different modelling frameworks. We also tie in the aim of the biological control program we want to implement with the mathematical analysis which is required.

2.1 Building the model

2.1.1 Keeping count

The primary aim of modelling change consists of estimating and predicting a *variable* to which a numerical value can be assigned.

When studying population dynamics, the variables of interest are often population sizes which vary over time. Biological pest control models usually focus on the pest and its predator population. These two populations tend to evolve the most over the time frame considered. In these models, they are also implicitly tracked over a selected area, which can be for instance be the plantation that needs to be protected from the pest or a smaller patch within the plantation.

To determine how the populations evolve through time, these models incorporate the major processes that determine an increase and a decrease in their numbers respectively: these are natural growth and predation for the pest, and pest uptake and natural mortality for the predator. The changes in predator and pest populations are linked through the transfer from the prey to the predator population, that is the predation for the prey and the pest consumption for the predator. These two populations thus form a two-dimensional food chain that is ensconced within a far more complex food web. However, we may consider that the crop (and other prey) of the pest is implicit in the pest growth, and predation on the predator included in its natural mortality. Schematically, we may represent this system in Figure 2.1.

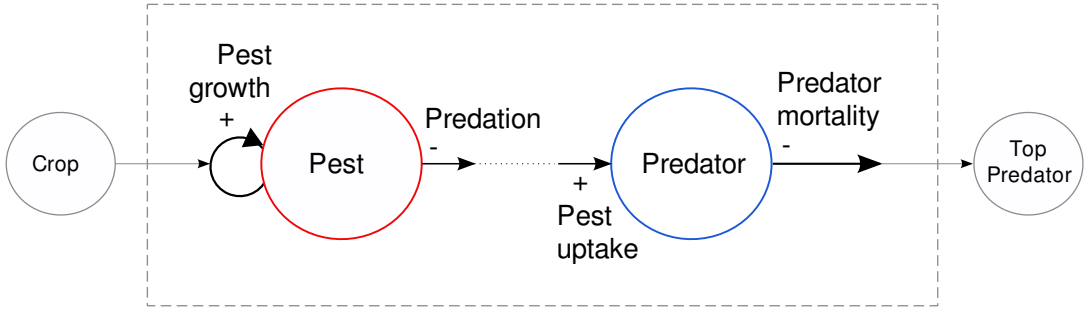

Figure 2.1: The pest (prey) and predator populations are linked by a biomass transfer from the pest to the predator through predation: this link forms the basis of the two dimensional food chain which is surrounded by the broken-lined rectangle. This chain is itself ensconced in a far more complex food web. We may however consider that the effect of this food web is implicit in the pest growth (which are determined by the plants and resources that the pest depend on) and predator mortality (which include mortality due to predation by the predator's natural enemies).

The processes involved can be described in more detail. The pest population (the prey) grows as it feeds on the plant and reproduces. The pest may also die from old age, but it is assumed that overall, the sum of births and mortality can be encompassed into a single growth term for pest population, which can be positive or negative whether actual growth or mortality dominates. Death by predation is considered separately. So, the pest population will change according to:

$$\text{Pest population change} = \text{Pest growth - Predation.} \tag{C1}$$

From the energy they acquire through the pest uptake, predators can also reproduce and increase in numbers. This energy intake takes place via pest consumption. Conversely, the predator population will decrease as predators die or escape from the system. This decrease is modelled as the predator mortality. So, the predator population will change according to:

$$\text{Predator population change} = \text{Pest consumption - Mortality.} \tag{C2}$$

The above two change equations provide an adequate description for conservation biological control programs, where an established predator population controls the pest population. In augmentative biological control programs however, a number of predators are released into the patch under observation. This creates an instantaneous surge in the predator population which also needs to be taken into account. The change equation for this particular process is given as:

$$\text{Predator pop. } \textit{after} \text{ release = Predator pop. } \textit{before} \text{ release + Size of release} \tag{C3}$$

2.1.2 Some key assumptions

Once the variables identified (here the pest and predator populations), some further assumptions need to be made. These will determine the framework that will be used to model the interactions.To translate the change equations (C1)–(C3) into a mathematical model, we list three such basic assumptions:

(A1) The populations are **uniformly distributed** across the patch or area under observation;

(A2) Abiotic parameters are uniform or can be **averaged** over this patch.

(A3) Finally, compared to the predator releases which bring an instantaneous increase in the predator populations, the processes involved intrinsically with the predator-prey interactions are considered to be **continuous**. We assume in particular that, for species that are considered in this thesis, these processes do not occur by seasonal or cyclic booms.

Processes such as reproduction and predation (and pest uptake) are dependent on the *rate of encounters* between individuals. The mathematical expressions or functions to describe them can then be derived mechanistically from the respective population concentrations or densities. By the first two assumptions (A1) and (A2), we average out and ignore spatial effects. So, the respective population densities are proportional to the population sizes.

2.1.3 Hybrid dynamics

By making the above assumptions, the continuous changes described by the change equations (C1)–(C2) can be modelled by *deterministic differential equations*[1]. Let x be the pest

[1]This is a classical approach which dates back to Lotka (1924); Volterra (1926).

population and y be the predator population. The differential equations are written as:

$$\begin{cases} \dot{x} = f(x) - g(.)y \\ \dot{y} = h(.)y - m(.)y \end{cases} \qquad (\mathcal{M}_{\text{cont.}})$$

where the function $f(x)$ represents the *pest growth rate*. The function $g(.)$ is the predation per predator (the functional response). The function $h(.)$ is the pest uptake per predator (*numerical response*). These functions ($g(.)$ and $h(.)$) are also collectively known as the trophic functions. The function $m(.)$ represents the *predator mortality rate*. Table 2.1 gives a list of the most common functions used to describe these processes. More details on the choice of these functions are given shortly.

This predator releases bring about an instantaneous change in the predator population as described in the change equation (C3). Therefore, it cannot be modelled by a continuous differential equation but by a difference equation:

$$y(t^+) = y(t) + Y,$$

where the $+$ superscript indicates the instant right after t. Y represents the size of the release. In an augmentative control program, releases are periodic. This means that the time instants at which releases take place can be indexed by the number of release periods, that is we represent them by nT where $n \in \mathbb{N}$. Releases are also of fixed size. This implies that the number of predators at each release can be calculated as the product of the predator release rate, which is the number of predators released per unit time, and the release period, which is the interval of time between two releases. We can rewrite the difference equation thus:

$$y(nT^+) = y(nT) + \mu T \quad \text{where} \quad n \in \mathbb{N} \qquad (\mathcal{M}_{\text{disc.}})$$

Remark: *This formulation is due to Mailleret and Grognard (2006) and allows two control entries for the formulation of the predator release strategy: first, the release rate μ (which can for instance represent the budget a planter invests in for his control program per year), and the release period T (which indicates how to spread the predators given a release rate).* ■

Figure 2.2 illustrates the schematics of the predator-prey dynamics involved in the augmentative control program.

2.1.4 Canonical form

The canonical form of the model combines both the differential and difference dynamics. It is written as:

$$\begin{cases} \left.\begin{array}{l} \dot{x} = f(x) - g(.)y \\ \dot{y} = h(.)y - m(.)y \end{array}\right\} \quad t \neq nT \\ y(nT^+) = y(nT) + \mu T, \quad n \in \mathbb{N} \end{cases} \qquad (\mathcal{M})$$

where the continuous differential equations form an autonomous model that describes the intrinsic predator-prey dynamics and the difference equation represents the population surge on a predator release.

Summary of common functions

Growth functions

$$f(x) = \begin{cases} rx, & \text{Malthus}^{1}, \\ rx\left(1-\frac{x}{K}\right), & \text{Verhulst}^{2}, \\ rx\ln\left(\frac{K}{x}\right), & \text{Gompertz}^{3}, \\ rxv\left(1-\left(\frac{x}{K}\right)^{1/v}\right), & \text{Generalised logistic function}^{4}, \\ rx\left(1-\frac{x}{K}\right)\left(\frac{x}{K_A}-1\right), & \text{Growth with Allee effect}^{5}, \\ \ldots \end{cases}$$

Functional responses

$$g(x) = \begin{cases} ax, & \text{Lotka-Volterra}^{6}, \\ \begin{cases} ax, & \forall x \leq \bar{x}, \\ a\bar{x}, & \forall x \geq \bar{x} \end{cases} & \text{Holling Type I}^{7}, \\ \frac{ax}{dx+c}, & \text{Holling Type II}^{7}, \\ \frac{ax^v}{dx^v+c}, & \text{Holling Type III}^{8}, \\ 1-e^{-ux}, & \text{Ivlev}^{9}, \\ \ldots \end{cases}$$

$$g(x,y) = \begin{cases} \frac{ax}{dx+(by+b_0)c}, & \text{Beddington-DeAngelis}^{10}, \\ \frac{ax}{dx+cy}, & \text{Ratio-dependent}^{11}, \\ \ldots \end{cases}$$

Numerical response

$$h(.) = eg(.)$$

Mortality

$$m(y) = \begin{cases} m, & \text{Constant natural mortality,} \\ m+qy, & \text{Mortality enhanced by territorial behaviour}^{12} \\ \ldots \end{cases}$$

$$m(.) = \begin{cases} m+\varphi(.)y, & \text{Mortality enhanced by other forms of cannibalism}^{13} \\ \ldots \end{cases}$$

Table 2.1: Pest growth, the functional response, the numerical response and the mortality function are the four main functional processes used to build predator-prey models. The references are: [1]Malthus (1798), [2]Verhulst (1838), [3]Laird (1965), [4]Richards (1959), [5]Courchamp et al. (1999), [6]Lotka (1924); Volterra (1926), [7]Holling (1961), [8]Real (1977), [9]Ivlev (1961), [10]Beddington (1975); DeAngelis et al. (1975), [11]Arditi and Ginzburg (1989), [12]Goh (1976), [13]Kohlmeier and Ebenhoh (1995).

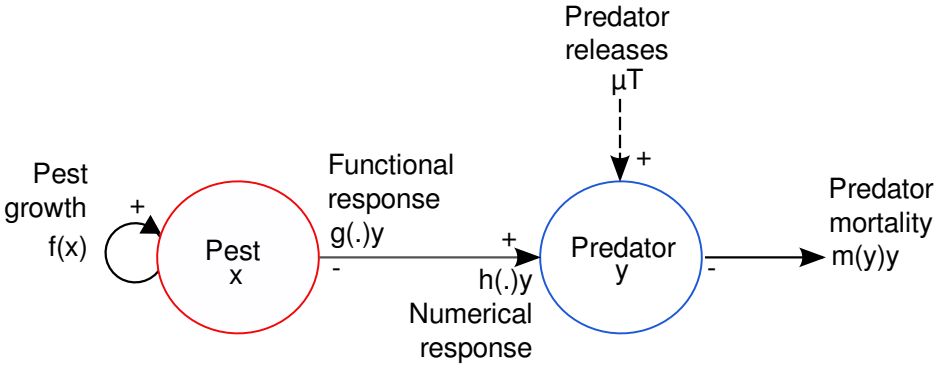

Figure 2.2: The augmentative biological control program is described by a two-dimensional impulsive model. It takes into account two types of dynamical regimes: continuous predator-prey dynamics (denoted by the continuous arrows) augmented by discrete predator releases (broken arrow).

The resulting system is *hybrid* in nature since it involves two types of dynamics. The current canonical model is in fact a subclass of hybrid systems known as *impulsive systems*.

2.2 Two mathematical properties of the impulsive model

A more general formulation for impulsive systems is given by:

$$\begin{cases} \dfrac{dx}{dt} = F(x,t), \quad t \neq \tau_k \\ x(\tau_k^+) = \theta(x(\tau_k), \tau_k) \end{cases}$$

When describing population dynamics, such models are characterised by the following two mathematical properties: positivity and local Lipschitz continuity.

Property 1: Positivity

Populations can take on only nonnegative values, that is at any given instant, they will either be zero or positive. Mathematically, these populations are represented as state variables, and they are described as forming a *positive vector*, i.e. they are restricted within the *positive orthant*. A formal definition is given below (see e.g. Mailleret (2004)):

Definition 1 (Positive vector) *A vector $x = (x_1, x_2, \ldots, x^n)^T$ is positive (denoted as $x \geq 0$) if each of the components is a non-negative real number, that is if $x_i \geq 0 \ \ \forall\, i$.*

Definition 2 (Positive orthant) *The positive n-dimensional orthant (denoted $\mathbb{R}^n_+$) is the set of all positive vectors of dimension n.*

Models that describe state values that remain nonnegative are a special class of models known as *positive systems*. A formal definition is given as:

Definition 3 (Positive system) *A dynamical system is a positive system if its state is confined to the positive orthant when the initial state is positive. That is:*

$$x(t_0) \in \mathbb{R}^n_+ \quad \Rightarrow \quad x(t) \geq 0 \ \ \forall\, t \geq t_0.$$

From the above definitions, a positive system can be identified by the following conditions:

Theorem 1 (Positive system) *A dynamical system is positive if*

$$x_i = 0 \quad \Rightarrow \quad \dot{x}_i \geq 0 \ \ \forall\, i$$

and if

$$\theta(x) \geq 0 \ \ \forall x \in \mathbb{R}^n_+$$

Remark: *Positivity is a useful property of the model. First, given that we are modelling populations, which are non-negative, a simple way to test the validity of the model is to test for its positivity. A model that is used to describe population dynamics but does not satisfy this property is clearly not appropriate for this task. Second, this property can be used to bound deviations from a given solution to the system that are tracked in order to establish stability of the given solution. More details are given in subsequent chapters.* ■

Property 2: Local Lipschitz continuity of processes

Ecological processes do not show sharp marginal increases or decreases. The growth rate $f(x)$, the trophic processes $g(.)y$ and $h(.)y$, and the mortality effect $m(y)y$ are continuous functions, with finite slopes (Solomon, 1949). This notion translates mathematically as the *local Lipschitz continuity*. Using the same notations as in Definitions 2-3, we can define this property for any two vectors of x at time t (denoted x^i and x^j respectively) (Coddington and Levinson, 1955) :

Definition 4 (Local Lipschitz continuity) *$F(x)$ is said to be locally Lipschitz continuous in x, if for every x, there exists $\epsilon > 0$ and $k > 0$ such that*

$$\forall x_i, x_j \ \ \textit{s.t.} \ \ |x_i - x| < \epsilon \ \ \textit{and} \ \ |x_j - x| < \epsilon, \ |F(x_i) - F(x_j)| \leq k|x_i - x_j|.$$

This condition provides the existence of unique solutions for a Model $\dot{x} = F(x)$. For the proof, see e.g. Coddington and Levinson (1955).

2.3 Why go impulsive?

A review of biomathematical control models

Biological systems can display intrinsically impulsive dynamics (see e.g. biological oscillators in swarms of fireflies (Buck, 1988) and crickets (Walker, 1969), stage-structured populations with dormant stages (Ghosh and Pugliese, 2004)). As in this

thesis, other biological systems can also be characterised by continuous intrinsic dynamics that are forced by an external impulsive control. The general formulation of the impulsive model, given as:

$$\begin{cases} \dfrac{dx}{dt} = F(x,t), & t \neq \tau_k \\ x(\tau_k^+) = \theta(x(\tau_k), \tau_k) \end{cases}$$

can describe a wide variety of biological dynamics. In some cases, the discrete part can represent the cumulative effects of a non-empty period of time, so that τ^+ is not necessarily the instant right after τ_k. This time-scale separation as it is called, can be used for instance to depict winter months during which insects are dormant and at the end of which only a proportion of the population has survived (Ghosh and Pugliese, 2004). It can also be used to represent the transition from the juvenile to adult stage (Geritz and Kisdi, 2004).

Mailleret and Lemesle (2009) provide an extensive review of impulsive systems in mathematical biology: these span a wide variety of problems in ecology, epidemiology, medical treatments to name but a few. We highlight here the main ideas behind this review which supports the use of impulsive modelling.

- In many situations involving such hybrid dynamics, semi-discrete models are often able to predict more accurately temporal variations than a purely continuous or a purely discrete model would.

  For instance, Gubbins and Gilligan (1997)'s host-parasitoid model that included both seasonal variations and impulsive harvesting of a lettuce crop were able to provide the best fit for their data set.

  Other works by e.g. Meng and Chen (2008); Choisy et al. (2006); Liu et al. (2006) also point out to certain complex dynamics which were not predicted by a purely continuous model, thus suggesting that in some cases, chaos may not be due to an exogenous chaotic forcing but rather related to the semi-discrete nature of a system itself.

- The semi-discrete characterisation of the model also yield more accurate thresholds given by some analytical conditions. Indeed, it is often the case that these conditions turn out to be dependent on the timing or frequency. When an inherently impulsive process is modelled continuously, estimates for similar thresholds are then erroneous. An example is the invasion by the gypsy moth, a major pest in the U.S.A, whose spread is underestimated when pulsed migration effects are not taken into account (Sharov and Liebhold, 1998; Johnson et al., 2006).

- The analysis of a desired target state in such systems also points out to the more profitable control programs between impulsive and continuous ones. Thus, Agur et al. (1993) recommended pulsed vaccination strategies rather than a continuous one; similarly, Kroll (2000) worked out that pulsed hormone treatments would work better than continuous ones as they would reduce resorption effects; in badger control Swinton et al. (1997) recommended pulsed lethal culling rather than fertility control (which has a continuous impact).

Conversely, polio vaccination was found to be more effective when carried out continuously (Wagner and Earn, 2008); pest control can under some circumstances be implemented at lower release rates if releases are carried out as frequently as physically possible (nearly continuous) (Nundloll et al., 2010a).

Remark: *Aside from a few papers (like Lakmeche and Arino (2000)'s), most of the impulsive systems studied in mathematical biology consist of an explicit description of interaction processes. In the case of predator-prey interactions, most of the models in the literature use a combination of specific growth, trophic response, and mortality functions. We discuss further the drawbacks of this method shortly in Section 2.5.* ■

2.4 Why go qualitative? – I. Structural advantages

2.4.1 Predator-prey processes

While the change equation for the predator releases is straightforward (the size of the predator population being periodically increased by the size of a release), the processes governing the continuous predator-prey dynamics are not modelled so easily.

There are two reasons for this: first, the mechanisms underlying these processes can vary as much from one species to another as from a given set of natural conditions to another (see for instance Holling (1961)). Second, the data sets collected for a given organism can also be very noisy, and can thus be fitted by functions with fundamental structural differences (Jost and Ellner, 2000). We cover more of the latter issue in Section 2.5.1.

In an attempt to provide more accurate descriptions of the intrinsic predator-prey dynamics, the literature abounds with functions for each of the processes involved.

Nonetheless, for each process, the functions need to satisfy some basic properties. We highlight these properties ('*General properties*') then give examples of functions that are most commonly used and also pertinent to the species involved in agricultural pest control programs. The examples are grouped in categories or families of functions that share some basic properties ('*Main categories*').

Figure 2.3 illustrates the different families of functions for the pest growth, functional response and mortality with respect to the population densities.

Pest growth rate, $f(x)$

General properties

The pest growth rate represents the net effect of natality and mortality of the pest population.

- 'Life begets life'. Pest reproduction and therefore the pest growth rate is a prey-dependent process. It is denoted by the function $f(x)$.

- As for other living organisms, the pest growth is zero when the population is zero. At positive pest numbers, reproduction can take place, hence growth can be positive - though it can also be negative if the natural mortality is too high.

Main categories

Growth functions take one of three forms:

1. *First form: unlimited / exponential / Malthusian growth.* This was the first growth function proposed in the literature (Malthus, 1798). It has the form:

$$f(x) = rx.$$

The parameter r is a constant which represents the net effect due to birth and mortality. In this scenario, the pest population increase is proportional to the pest population size, that is it is unlimited unless predation takes place. The growth is termed as *exponential* or *Malthusian*.

2. *Second form: limited logistic growth – overcrowding.* In nature however, it has been observed that an organism's growth is limited by the resources available per individual. So beyond a certain population size, competition due to overcrowding effects take over and growth is not proportional to the population size. Verhulst (1838) modelled this feature as the *logistic function*:

$$f(x) = rx\left(1 - \frac{x}{K}\right).$$

The parameter K is a positive constant which represents the *carrying capacity* of the environment – this is the maximal population size that can be sustained by the environment. It is easy to see that for $x < K$, we have $f(x) > 0$ which means that the population grows. Beyond K, the population will tend to decrease as growth is negative $f(x) < 0$.

Remark: *A generalised logistic function is given as* $f(x) = rx\upsilon\left(1 - \left(\frac{x}{K}\right)^{\frac{1}{\upsilon}}\right)$*, where the parameter* υ *modifies the rate at which the population reaches the carrying capacity. When* $\upsilon \to +\infty$*, the resulting growth function is another function commonly found in the literature to model tumor growth:* $f(x) = rx\ln\left(\frac{K}{x}\right)$*.* ■

3. *Third form: limited growth with Allee effect – undercrowding.* In addition to overcrowding, another growth limiting factor is the *Allee effect* (see Allee (1938) and also Stephens et al. (1999)). This phenomenon occurs at low population levels: individuals are too few to survive and reproduce. It may for instance be due to insufficient genetic diversity which makes the population susceptible to pathogens and provokes for instance population collapses. This effect has also been termed as *undercrowding*. A very simple model is given by Courchamp et al. (1999):

$$f(x) = rx\left(1 - \frac{x}{K}\right)\left(\frac{x}{K_A} - 1\right),$$

where K_A is the critical population threshold, below which the population decreases and crashes to zero.

Remark: *The Allee effect among the pests is advantageous to a biological control program: introducing sterile males or autocides in a target organism's population enhances*

the Allee effect and leads to a desired population crash. Among predators, such effects are not as desirable, especially when the control scheme is 'classical' (that is, if the predators are expected to establish after one release only). Repeated releases in an augmentative control scheme help circumvent this issue (Mailleret and Lemesle, 2009). ■

The functional response, $g(.)$

General properties

The functional response is the rate at which prey are killed per predator (Solomon, 1949). Before looking at the analytical forms proposed for this process, we may deduce that it will have the following properties:

- From its definition the functional response represents predation and therefore on the prey population size.
- In particular, the functional response is zero, i.e. no predation can take place, when there are no pests.
- When the prey population is positive, predation probably increases (and we explain how this may happen shortly).
- Predators can also either cooperate to increase the rate of per capita predation (the converse of undercrowding) or interfere with each other to reduce the per capita predation (overcrowding), or not influence the rate of per capita predation. In the first two cases, the functional response would also be predator-dependent.

Main categories

Functional response models are related to a wide range of theories motivated by ecological to evolutionary concerns – foraging strategies has direct consequences on the ecological scale, and survival thanks to some strategies but not others influence evolution. For a full review, we refer the reader to Jeshke et al. (2002). There are two broad categories of functional responses (Begon et al., 1996).

1. *First category: prey-dependence only,* $g(x)$. the functional response is dependent only on the prey density, but not on the predator density: $g(x)$. The predator population only has an *indirect* effect on the elimination of pests as the term $g(x)y$.

   Lotka (1924); Volterra (1926) proposed a linear dependence in the prey density

   $$g(x) = ax$$

   where $a > 0$ is a constant. The overall predation effect $g(x)y = axy$, a mass-action term.

   The most widely accepted and – as a result – most commonly used prey-dependent functional responses are those by Holling (1961). In his seminal paper, he proposed three types of functional responses, each derived from the mechanisms involved in the animal's physiology (especially for the Type I curve) and the foraging strategy (relevant for the Types II and III curves). Real (1977) provides a summary on these curves as follows:

i Holling Type I curves correspond to animals consuming food at a rate proportional to prey encounters, up to a saturation level limited by their gut size. Examples include filter feeders.

$$g(x) = \begin{cases} ax, & \forall\, x < \bar{x}, \\ a\bar{x}, & \forall\, x \geq \bar{x}, \end{cases}$$

where a is a positive constant.

ii Holling Type II curves depict the response of organisms that take a certain amount of time to capture and ingest their prey (search and handle in Holling (1961)'s explanation). Typically insects show such feeding patterns. The curve is a smoothed out version of the Type I curve, and can be modelled as (Holling, 1961):

$$g(x) = \frac{ax}{dx + c}$$

where $a, d, c > 0$ are constants. An alternate model for the Type II curve is given by Ivlev (1961):

$$g(x) = 1 - e^{-ux}$$

where the parameter $u > 0$ is constant.

iii Holling Type III curves are sigmoid or S-shaped. According to Holling (1965), they are characteristic of animals which show some learning behaviour in their foraging patterns. Below a certain threshold in the prey population numbers, predators do not recognise the prey as viable food source. Above this threshold, feeding rates increase rapidly until saturation. Real (1977) reports that the predators in this case "tend to learn slowly and forget the value of a food source unless they encounter it fairly often. So, when a food item is scarce, it will be eaten proportionately less often and a lag will appear in the functional response." Using an analogy with enzyme kinetics of oxyhaemoglobin described by Barcroft and Hill (1909), Real (1977) proposes a sigmoid function of the form:

$$g(x) = \frac{ax^v}{dx^v + c},$$

where $v > 0$ is a constant that determines the threshold prey populations and the sharpness of the increase at the threshold level. Sigmoid functional curves are mainly attributed to vertebrates (see Holling (1959a, 1961)). However, in a series of laboratory experiments, Hassell et al. (1977) published results on three insect predators (*Plea atomaria* (Pall) and *Notonecta glauca* (Linnaeus), two Hemiptera, and *Coccinella septempunctata* (Linnaeus), a Coleoptera) and one parasitoid (*Aphidius uzbeckistanicus* (Luzchetzki), a Hymenoptera) showing Type III responses.

An interesting point to note is that the Type III curve is a variation of the Holling II response. The Holling II curve can be rewritten as:

$$g(x) = \frac{1}{d + c/(ax)}$$

where the term $c/(ax)$ represents the time taken to locate prey, with a is the detection rate per prey. In the Type III curve, which we can rewrite as:

$$g(x) = \frac{1}{d + c/((ax^{v-1})x)},$$

due to the learning process the detection rate per prey is modified by the number of prey and is calculated as ax^{v-1}.

Remark: *The widespread use of Holling's functional responses is as much due to their successful application in models (including the fact that they match with the collected data for predation for given predator population sizes), as to the mechanistic explanations supporting them.* ■

2. *Second category: direct predator-dependence* $g(x, y)$. The net predation effect on the prey population is then $g(x, y)y$. This can happen when predators show interfering behaviour with their conspecifics that intensifies with higher predator populations and penalises the functional response.

   Alternatively, direct predator dependence of the functional response can be due to predators showing mutualistic or cooperative behaviours in attacking their prey. This phenomenon referred by Holling (1961) as group stimulation results in enhanced predation at higher predator numbers. It is similar to the Allee effect (see previous descriptions of growth functions).

   Models of the interference effects hail from two main schools of thought:

   i *Density-dependence.* One, by Beddington (1975) and DeAngelis et al. (1975) is based on a similar mechanistic construction as Holling Type II function's and takes the form:

   $$g(x, y) = \frac{ax}{dx + (by + b_0)c}. \tag{2.1}$$

   ii *Ratio-dependence.* The other approach, introduced by Arditi and Ginzburg (1989), is phenomenological, and is based on the premise that the functional response is dependent on the proportion of prey to predator, and not on the prey only. The strength of intrapredator interference depends on this *ratio* of prey to predator, and is thus referred to as *ratio-dependence*. In most papers, the ratio-dependent functional response is a modification of the Holling II response, i.e.

   $$g(x, y) = \frac{ax/y}{dx/y + c} = \frac{ax}{dx + cy}.$$

   Contrary to all others, ratio-dependent functional responses are undefined at $(0, 0)$.

See later: *The effect of predator density dependence on the augmentative biological control program is investigated in Chapter 4.* ■

The numerical response, $h(.)$

General properties

The numerical response is the rate at which the predator population increases per predator due to prey consumption. As a result, we mlight consider it to be a function of the functional response, or of the same base variables.

Main categories

Holling (1961) reports that there are three possible ways in which the predator density varies with prey.

1. The first is that it follows immediately the predation, i.e. predators are able to reproduce at any prey population level at which predation takes place. The most common formulation is that the numerical repsonse is proportional to the functional response: $h(.) = eg(.)$ where e is a constant with value within the range $0 < e \leq 1$.
2. The second is restricted by thresholds of prey density below which the functional response might be too weak for a given number of prey for reproduction of the predator to be possible. In this case, the numerical response is delayed (with respect to the prey population size).
3. The third is that the numerical response remains nil. This can happen in the case when the prey is not nutritious enough for reproduction or when there are other constraints in the predator's niche, e.g. cover limitations, which prevent it from reproducing.

The predator mortality rate, m or $m(.)$

General properties

The predator mortality rate represents the net effect of the predator birth rate and death rate. It is usually expressed as a constant, representing the fraction of the predator population that dies. In the model, the overall morality expressed as the product of the rate and the actual predator population size. So in the absence of predator, the overall mortality is nil.

Main categories

The term for the predator mortality rate can encompass the net effect of predator birth and mortality, as well as other mortality enhancing factors such as cannibalism (including overcrowding) among predators. We list the main components the term for predator mortality can contain:

1. *Fixed mortality.* In a given environment, under constant abiotic conditions, this parameter is assumed to be fixed, that is independent of predator density. The mortality is a positive constant:
$$m(y) = m$$
where $m > 0$. The net mortality effect is $m(y)y = my$.
2. *Mortality enhanced by overcrowding.* Among territorial species, the mortality rate can be enhanced to include overcrowding effects per predator. Overcrowding is directly dependent on the predator population density (Goh, 1976). So the mortality is modelled as:
$$m(y) = m + qy$$
The net mortality effect on the predator population is $m(y) = my + qy^2$. Figure **??** illustrates the two curves.

   The cannibalism term can also saturate at higher predator desnsities; this can happen as predators reach satiety. The cannibalism function then takes on a logistic shape, that is dependent in the predator density. A generic formulation is:
$$m(y, x) = m + \varphi(y)$$

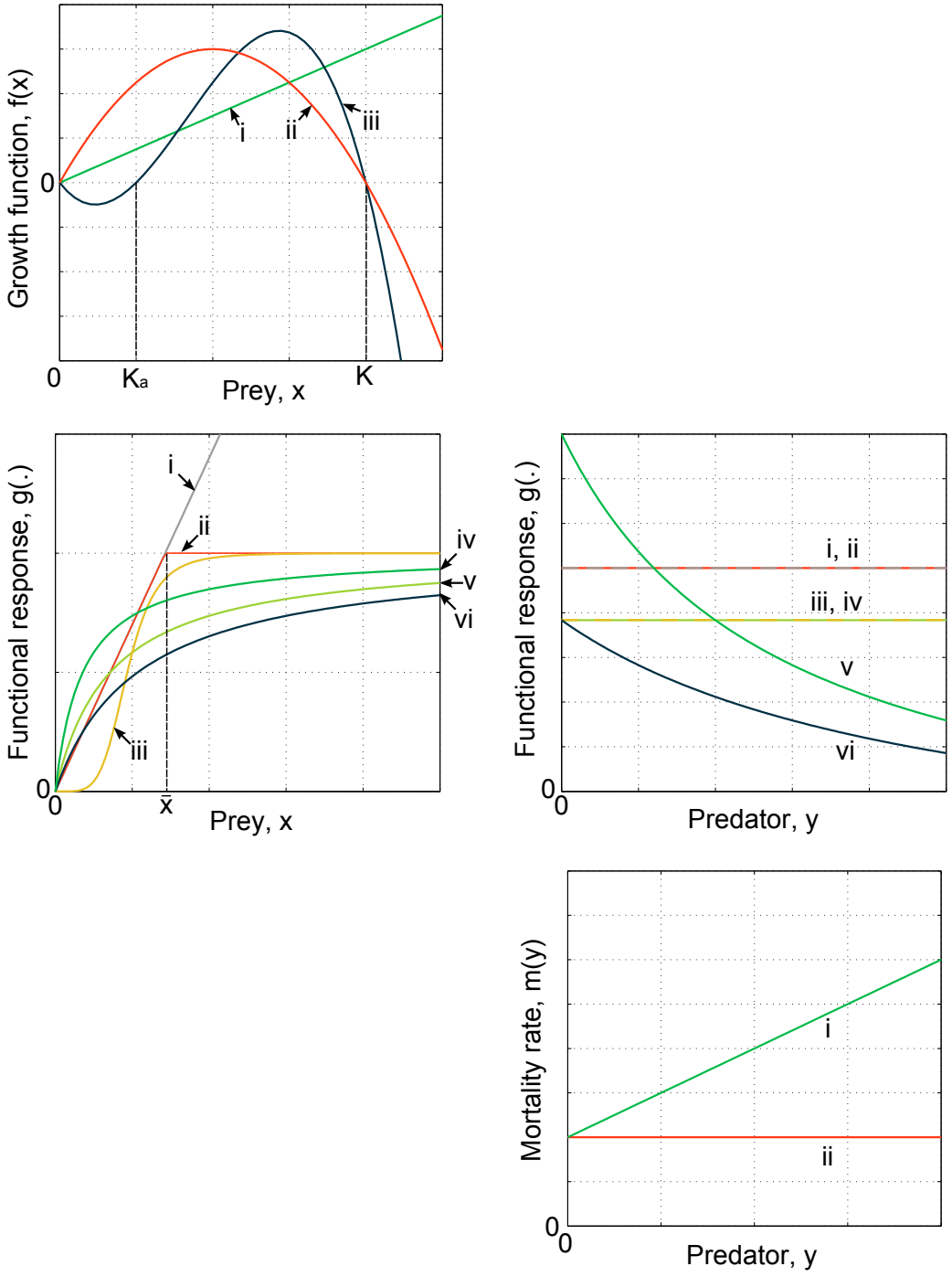

Figure 2.3: *Top left. Pest growth rates:* Curve i is Malthusian, $f(x) = rx$. Curve ii is logistic, $f(x) = rx(1 - x/K)$. Curve iii is logistic with the Allee effect, $f(x) = rx(1-x/K)(x/K_a-1)$. *Middle. Functional response:* Curve i is Lotka-Volterra, $g(x) = ax$. Curve ii - iv are the Holling Types I - III functions respectively: $\forall x \in (0, \bar{x}), g(x) = ax$ and $\forall x \geq \bar{x}, g(x) = a\bar{x}$; and $g(x) = ax/(c + x)$; $g(x) = ax^v/(dx^v + c)$. Curves i-iv are independent of predator density. Curve v is predation with Beddington-DeAngelis interference, which is predator-density dependent: $g(x, y) = ax/(dx+(by+b_0)c)$. Curve vi is predation with Arditi-Ginzburg interference, which is ratio-dependent in predators: $g(x, y) = ax/(dx + cy)$. *Bottom right. Mortality rate:* Curve i is the natural mortality rate, $m(y) = m$. Curve ii represents mortality enhanced by territorial effects, which is dependent in predator density: $m(y) = m + qy$.

3. *Mortality enhanced by prey- and predator-dependent cannibalism.* Depending on the mechanisms underlying cannibalism, and/or modelling paradigms, cannibalism can be limited by both predator and prey densities (see e.g. Kohlmeier and Ebenhoh (1995)). So we define this process generically as:

$$m(y,x) = m + \varphi(y,x)$$

See later: *Chapter 5 investigates the effect of this generic type of cannibalism on augmentative biological control in detail.* ■

2.4.2 Asymptotic behaviour

Autonomous prey-predator system

Different hypotheses on the modelling of predator-prey interactions result in different dynamical and asymptotic behaviours (see e.g. Murray (2002)). For example, the classical Lotka-Volterra predator-prey model (with Malthusian growth rate of the preys, linear mortality of the predators and mass-action predation),

$$\begin{cases} \dot{x} = rx - axy, \\ \dot{y} = \gamma axy - my, \end{cases}$$

produces sustained fluctuations of the populations densities whose amplitude is dependent on the initial population densities.

If prey growth is modelled as a logistic mapping rather than a linear one, as in the model below:

$$\begin{cases} \dot{x} = rx\left(1 - \dfrac{x}{K}\right) - axy, \\ \dot{y} = \gamma axy - my, \end{cases}$$

then, two situations are possible in both of which sustained fluctuations collapse. If the carrying capacity K is too low, the prey cannot grow sufficiently large to sustain the predator population. So predators die out while the prey density goes to the carrying capacity. If K is large, then both prey and predator populations converge asymptotically towards a positive equilibrium value provided their initial conditions are positive.

Finally, if in addition one models the predation process using a Holling II functional response,

$$\begin{cases} \dot{x} = rx\left(1 - \dfrac{x}{K}\right) - \dfrac{axy}{dx+1}, \\ \dot{y} = \gamma\dfrac{axy}{dx+1} - my, \end{cases}$$

the dynamics are even more complex. For low K values, the predator population dies out as previously. For intermediate K values, a globally stable positive equilibrium between preys and predators appears. For large K values however, this equilibrium is destabilised and there is the emergence of a limit cycle to which the model trajectories converge – a phenomenon known as “the paradox of enrichment" (Rosenzweig, 1971). The bifurcation diagram displayed in Figure 2.4 summarises the asymptotic behaviour of this model.

Remark: *The intrinsic predator-prey dynamics describe conservation biological control, in which pest eradication is neither possible nor desirable. The stability of coexisting states are favoured over that of others. The bifurcation parameter K, the carrying capacity, can be changed by habitat management.* ■

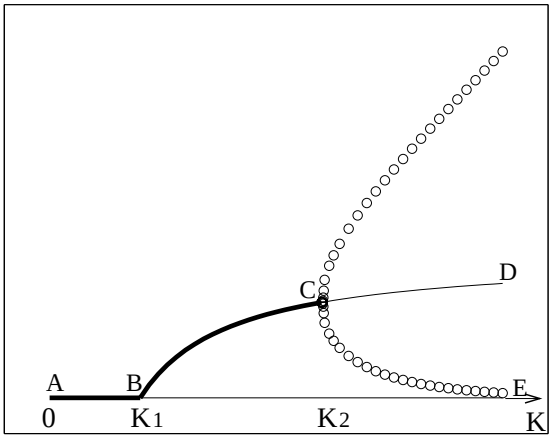

Figure 2.4: The bifurcation diagram describes the asymptotic predator population as a function of the carrying capacity K for the Rosenzweig-MacArthur model (Rosenzweig, 1971). Thick lines represent stable equilibria, thin ones unstable ones and circles the extrema of a periodic solution. A transcritical bifurcation occurs at $K = K_1$ and a Hopf bifurcation at $K = K_2$. From Zhou (2008).

With augmentative control

With an exogenous control - such as periodic predator releases - it is possible to drive the three systems and stabilise them at a desired state.

Liu et al. (2005a) for instance demonstrate that when a predator-prey system with Malthusian growth and mass action predation is impulsively controlled as in:

$$\left\{ \begin{array}{l} \left.\begin{array}{l} \dot{x} = rx - axy, \\ \dot{y} = \gamma axy - my, \end{array}\right\} \quad t \neq nT \\ y(nT^+) = y(nT) + \mu T, \quad n \in \mathbb{N} \end{array} \right. \tag{$\mathcal{M}_{\text{ex(i)}}$}$$

the system will reach a periodic pest-free state if $\mu > \frac{rm}{a}$[2]. This solution's stability implies that pest eradication is possible provided the release rate is large enough.

Likewise when pest growth is logistic, i.e. the impulsive system is given as:

$$\left\{ \begin{array}{l} \left.\begin{array}{l} \dot{x} = rx\left(1 - \frac{x}{K}\right) - axy, \\ \dot{y} = \gamma axy - my, \end{array}\right\} \quad t \neq nT \\ y(nT^+) = y(nT) + \mu T, \quad n \in \mathbb{N} \end{array} \right. \tag{$\mathcal{M}_{\text{ex(ii)}}$}$$

Liu et al. (2005b) show that the system converges to the pest-free solution if the release rate satisfies the condition $\mu > \frac{rm}{a}$.

Finally, Liu and Chen (2003) calculate for the system with logistic growth and Holling II

[2]The size of predator releases given by Liu et al. (2005a) corresponds to μT in our model formulation. So, using their result with the appropriate change of variables, we calculate the condition for pest eradication as: $\mu T > \frac{rmT}{a}$, which we see is independent of the release period T. Similar notations are given by Liu et al. (2005b) and Liu and Chen (2003), which are then adapted for subsequent models discussed in this section

predation:

$$\left\{\begin{array}{l} \dot{x} = rx\left(1-\dfrac{x}{K}\right) - \dfrac{axy}{dx+1}, \\ \dot{y} = \gamma\dfrac{axy}{dx+1} - my, \\ y(nT^+) = y(nT) + \mu T, \quad n \in \mathbb{N} \end{array}\right\} \quad t \neq nT \qquad (\mathcal{M}_{\text{ex(iii)}})$$

the pest-free solution is (locally) stable if - again - $\mu > \dfrac{rm}{a}$.

In fact for the three systems given above, we find that the control needs to satisfy an identical condition to bring about pest eradication (locally, i.e. provided the initial conditions are in the neighbourhood of the zero-pest solution). This indicates that the stability condition depends on one or more of the underlying properties inherent to pest growth and predation that are common to all three systems. A similar analysis ensues for global attractivity: though the condition is not identical in all three cases, it is possible to apply the same principle and identify a common trend.

Mailleret and Grognard (2006) thus propose a qualitative formulation satisfied by all three systems (and more) as below

$$\left\{\begin{array}{l} \dot{x} = f(x) - g(x)y \\ \dot{y} = h(x)y - my \\ y(nT^+) = y(nT) + \mu T, \quad n \in \mathbb{N} \end{array}\right\} \quad t \neq nT \qquad (2.2)$$

where the process functions satisfy the following hypotheses:

1. $f(0) = 0$ and $f'(0) > 0$;
2. $g(0) = 0$, $g'(0) > 0$, and $\forall x > 0, g(x) > 0$;
3. $h(0) = 0$ and $\forall x > 0, h(x) \geq 0$;
4. $\dfrac{f(x)}{g(x)}$ is upper bounded for $x \geq 0$ such that $S_g = \sup\limits_{x\geq 0}\left(\dfrac{f(x)}{g(x)}\right)$.

They are then able to generalise the respective conditions for local asymptotic stability global asymptotic attractivity as:

$$\mu > \frac{mf'(0)}{g'(0)} \quad \text{and} \quad \mu > m\sup_{x\geq 0}\frac{f(x)}{g(x)}.$$

It is easy to see that $f'(0)$ is identical for logistic and Malthusian growth are equal ($f'(0) = r$ in both cases). Likewise the term $g'(0)$ is equal for the mass balance term and Holling II predation ($g'(0) = a$ in both cases). Similarly, the ratio of the pest growth to that of the predation can be upper bounded for the three combinations of growth-predation functions given in the models above. Table 2.2 summarises the values this ratio takes.

See later: *A full description of the model by Mailleret and Grognard (2006) and details of analytical methods are given in Chapter 3.* ■

The first advantage of qualitative modelling approach is that it gives generic results: once we have identified the stability conditions for the solution of a generic model, the result for a specific model is recovered simply by plugging the explicit function's parameters.

		$f(x)$	
		rx	$rx(1-x/K)$
$g(x)$	ax	$\mathcal{M}_{\text{ex(i)}}$ $\dfrac{f(x)}{g(x)} = \dfrac{r}{a}$ $\sup\limits_{x\geq 0} \dfrac{f(x)}{g(x)} = \dfrac{r}{a}$	$\mathcal{M}_{\text{ex(ii)}}$ $\dfrac{f(x)}{g(x)} = \dfrac{r}{a}\left(1-\dfrac{x}{K}\right)$ $\sup\limits_{x\geq 0} \dfrac{f(x)}{g(x)} = \dfrac{r}{a}$
	$\dfrac{ax}{dx+1}$	★ $\dfrac{f(x)}{g(x)} = \dfrac{r}{a}(dx+1)$ unbounded	$\mathcal{M}_{\text{ex(iii)}}$ $\dfrac{f(x)}{g(x)} = \dfrac{r}{a}\left(1-\dfrac{x}{K}\right)(dx+1)$ $\sup\limits_{x\geq 0} \dfrac{f(x)}{g(x)} = \begin{cases} \dfrac{r}{a}, \text{ if } K \leq 1/d \\ \dfrac{r(Kd+1)^2}{4aKd}, \text{ if } K > 1/d \end{cases}$

Table 2.2: Models $\mathcal{M}_{\text{ex(i)}}$-$\mathcal{M}_{\text{ex(iii)}}$ satisfy the hypotheses listed by Mailleret and Grognard (2006), so both the local and global asymptotic stability condition for the zero-pest solution can be calculated from their generic result. The ★-combination (not considered in the examples) gives an unbounded ratio for $f(x)/g(x)$. We discuss the impact on the global attractivity in Section 3.2.5 of Chapter 3

2.5 Why go qualitative? – II. Field realities

2.5.1 Finding the right compromise

Complexity

Because of their sensitivity to a wide range of biotic as well as abiotic parameters, a complete model description of a biological system (ecosystem, genetic network, etc) would consist of an extremely large number of parameters. This presents two problems. First, to write and parameterise accurately a complete model would be a feat in itself.

Temperature, humidity, and carbon dioxide concentration are known to impact reproductive cycles, functional responses and mortality of various animals (see e.g. examples given in Chapter 7 in Cohen (1978), Gerson et al. (2003), and Weeden et al. (2007)). This inherent complexity introduces process errors which arise if an experiment is to be repeated under conditions that are not identical. It also motivates some of the criticisms against experiments in laboratory, that is under highly controlled environments (which could aid in parameterising the system), in that they do not accurately mimic field or real world conditions. These features represent the process errors.

Second, even for models of intermediate complexity, there are additional obstacles on the way: its mathematical analysis is only possible by numerical methods. Such calculations tend to be computationally intensive.

Noise

Observation errors also contribute to the difficulty in modelling the biological processes in a predator-prey system.

In many cases, this is due to the problem of *scale* both with respect to the observer (the human being) and with respect to the observed patch. For instance, tracking insects that

may barely be a fraction of millimetres wide over hectares of crops cannot be done with the same precision as counting larger animals (of size of the same order or larger than humans). Likewise, measuring fish stock accurately is impossible as the oceans span the planet without borders: here it is the size of the patch which is extremely big. The problem of scale also encompasses the idea of refuges where the living organisms hide and remain out of sight of the observer's eye. This makes the process of keeping count extremely tricky.

Sensitivity to the observer is also a source of measurement error. An observer can represent an enemy threat or physically hurt individuals of the species under obervation when manipulating them. Small organisms can be hurt or killed when manipulated roughly. This is the case with *Frankliniella occidentalis* (Pergande) and *Amblyseiulus cucumeris* (Oudemans) – which are a major plant pest and one of its biological control agents and only a fraction of millimetres in length – that are considered in our experiments reported in Chapter 7 (Vitte, 2008). They can also show defensive behaviour provoked by the presence of the observer who represents a potential enemy threat. Larson (2003) captures the issue best (see Figure 2.5). This is another source of observation error.

Figure 2.5: Human observation can be disruptive and yield inaccurate results on the behaviour of living organisms. Larson (2003) provides a humourous take on the behaviour of cows in the absence of humans.

Artefacts

Finding the right compromise between complexity and simplicity is one of the aims of mathematical modelling (Bernard, 2004)[3]. Tackling process and observation errors is, however, no

[3]We can confirm that such a compromise is achieved when predictions from a simple model match with observed behaviours. Lemesle and Gouze (2008) provides a neat example of how a simple model is identified to reproduce oscillatory dynamics that were modelled by PDEs, which could not be studied analytically.

mean feat.

Suppose, as biomathematicians, we decide to focus on a set of processes only and describe them in detail. Here there is still a risk that the analysis yields results that are related to modelling artefacts (Clark, 1990). Take for example the Holling Type II functional response (Holling, 1961). The general shape of a data cloud can also be fitted by Ivlev (1961)'s function which is instead based on satiation of predators. Emlen (1973) (in Real (1977)) offers an alternative food handling explanation, where the majority of time is spent feeding on either prey to reach a similar form. In fact, there are also probably other functions that can fit a noisy data set representing the process at each population size[4]. Inevitably the use of either function (here, Holling's or Emlen's) or either interpretation of parameters (here, Holling's or Emlen's) will have consequences on the final interpretation of the analytical results from the model.

This problem also arises when fitting models directly to time-series data on populations. Because of noise (observation or process errors) in the collected data, models that are structurally different can be fitted - after adequate parameter adjustments - to the same time-series. Jost and Arditi (2000) (see Figure 2.6) show how data artificially generated with a prey-only dependent functional response can also be fitted by a model with a ratio-dependent (that is prey and predator) functional response, and vice-versa[5]. This indicates a fundamental difficulty in identifying mechanisms underlying the functional processes that are used to build a model and interpret the results of its analysis.

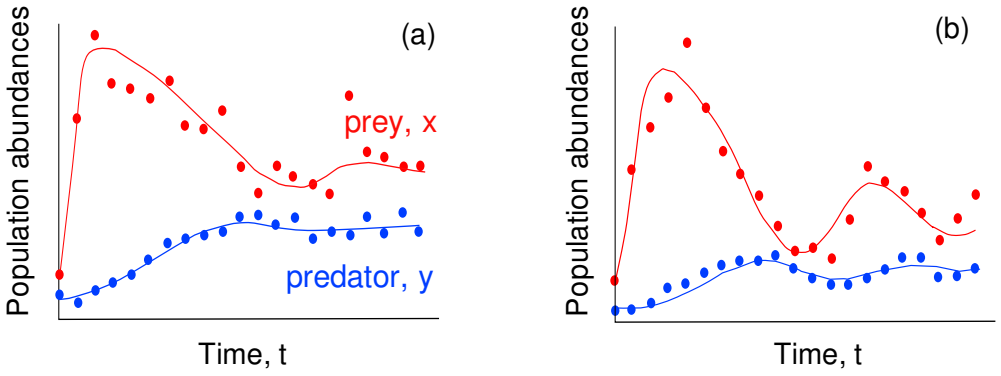

Figure 2.6: From Jost and Arditi (2000): (a) A model with ratio-dependent functional response can approximately fit data generated by a model with prey-dependent response. (b) The model with prey-dependent response can fit data generated by the ratio-dependent model.

Remark: *In order to tackle large models analytically, a biomathematician can also resort to a variety of model reduction techniques (see e.g. Beck et al. (1996)). These are not always very straightforward to implement. Other approaches exist: Cohen (1978)'s study of niche spaces resorts to graphical methods to reduce niche space and food web sizes. As he points out, this approach does not take into account the dynamical processes between the individual species of the web.* ■

[4]Examples of process data sets are given in Begon et al. (1996). We also illustrate the applications of our results in Chapter 7 with such data sets

[5]See also the article by Jost and Ellner (2000) and Jost and Arditi (2001)

2.5.2 One model to rule them all

Genericity

One way to reduce the risks of modelling artefacts in a simple model is to focus on the general trends that a process follows with respect to the state variables. Gouzé (1996) for instance remarked that biological systems were characterised by some general properties: positive variables, dynamic state changes being dependent on the sum or difference of some positive processes etc. Such a modelling approach is said to be *qualitative* or *generic*; the resultant model is referred to as a *meta-model*(D'Onofrio, 2008).

By focusing on general trends, the meta-model smoothes out uncertainties or errors in parameterising various process functions. As discussed previously in Section 2.4.2 it also yields results that apply to a wide class of models instead of being specific to one particular explicitly formulated model. Once we have identified the stability conditions for the solution of a generic model, the result for a specific model is recovered simply by plugging the explicit function's parameters.

Lakmeche and Arino (2000)'s analysis of a generic model inspired by chemotherapeutic treatment models gives generic results that are applicable to the predator-prey models with impulsive dynamics (e.g. in this thesis, Negi and Gakkhar (2007)). The use of generic functions is common in chemostat modelling: the substrate consumption is often modelled as some function of the substrate satisfying some basic properties (see e.g. Bernard (2004)).

Among predator-prey models, qualitative modelling is rare. Nonetheless, there are common attributes which can be exploited. All pest growth functions are zero at zero pest. We also note that at worst for a pest control program, marginal pest growth rate at zero pest, that is the slope of the growth rate, is positive. We can consider, as a basic description for the pest growth for our model: $f(0) = 0$ and $f'(0) \geq 0$.

Functional responses are also nil in the absence of pest (because there is no predation); they are always positive at positive pest numbers. So we can describe the prey-dependent functional response by these qualities: $g(0) = 0$ and $\forall x, \quad g(x) \geq 0$. Intrapredator interference is perceived as a general decrease in the functional response as the predator population increase (see Buffoni et al. (2005), and Chapter 4 of this thesis). The numerical function is defined similarly, and so on.

2.6 Summary of model characteristics and aims

2.6.1 Modelling features

Throughout this thesis, we use qualitatively-defined impulsive models to describe augmentative control programs. They are characterised by the four features below:

Bi-dimensionality. We assume that we are at the onset of a pest invasion and that crops or plants (the primary resource in our ecosystem) are in abundance. This means we make the assumption that the crop is implicit in the pest growth rate function. Unless stated otherwise, we consider that the predator species is a specialist, that is it feeds exclusively on the pest species. In the absence of the pest, the predators cannot sustain themselves and die (or leave the patch we model).

Implicit spatial effects. Second, we consider a homogeneous medium, the pest and predator populations are uniformly distributed across the crop or in specific 'local' patches - the size of which depend on the respective species mobility. Spatial effects such as migration to and from such patches are then implicitly taken into account in the net pest growth and predator mortality respectively else as an extension of the patch as its

populations spread uniformly. This allows us to use a lumped-parameterisation deterministic formulation.

Qualitative functions. Third, we use a qualitative description for the predator-prey processes (pest growth, trophic interaction, and predator mortality). This has two uses: on one hand it tackles uncertainty, for instance, it evens out fluctuations that can be due to abiotic factors; on the other hand, it allows us to obtain results that are valid for a wider class of function. As will be shown throughout our work, this approach already yields insightful results on various biological control systems in addition to reducing the risk of modelling artefacts (which arise when a model is too complex and explicit). More details are available in subsequent chapters where a new model is introduced.

Fixed-size periodic releases. The control of the pest is also punctuated by the sudden surge in the predator population occurring at a predator release. Releases differ from the intrinsic predator-prey dynamics in that they are not continuous but discrete in time. We consider that the size of the predator population injected into the ecosystem at each release instant is fixed, independently of the pest population size. One key aspect of our work is that we define the size of a release as the product of a *release rate* and the release *period*. The release rate is the size of the predator population released per unit time and the period is the fixed interval between two releases. This modelling choice allows for an efficient comparison between deployment strategies; indeed, when the release rate is fixed and the influence of the period is studied, no bias due to a varying number of released predators per time-unit is introduced into the analysis of the influence of the period of release.

Remark: *Our choice with respect to other modelling approaches is motivated by the aim of our model (what problem we seek to solve or what we seek to investigate and why), as well as the practicality of the chosen framework (namely the mathematical tools that are available). The biological control literature however spans other models which focus on other aspects of the ecosystem.*

Spatial effects *for instance are usually modelled by* ***partial differential equations*** *that track the population dynamics in time as well as in space (see e.g. Sherratt et al. (1997)). To include spatial effects within the ordinary differential equations (ODE) framework, Hanski (1998) developed the* ***metapopulation*** *model, which consists of visualising a landscape as a set of patches or islands, within which populations are perfectly homogeneous (see Figure 2.7). The movement across space is determined by migration from one patch to the other. Shea and Possingham (2000) tried to predict the success of repeated predator releases using the metapopulations approach: they were able to provide a numerical analysis using stochastic dynamical programming methods to assess the success of establishment of predators (the invasive species) within each patch.*

The focus of these models is on invasion dynamics; in fact, the metapopulation approach has led in particular to adaptative dynamics theory, which defines whether or not a species or mutant can take over a current local population based on how it uses the environmental resources surrounding it (see e.g. Diekmann (2004) for an introduction).

When biological control is carried out by parasitoids, the prey population can be ***stage-structured*** *in terms of susceptibles and infected. Some predators also attack the prey preferentially at specific growth stages. The pest population can then be structured into vulnerable and non-vulnerable stages. Anderson and May (1981); May and Hassell (1988) give examples of such stage-structured models.*

Many invasive species also undergo cyclic booms or cyclic increases in population, and this effect can be incorporated into a biological control model (May and Hassell, 1988; Vaidya and

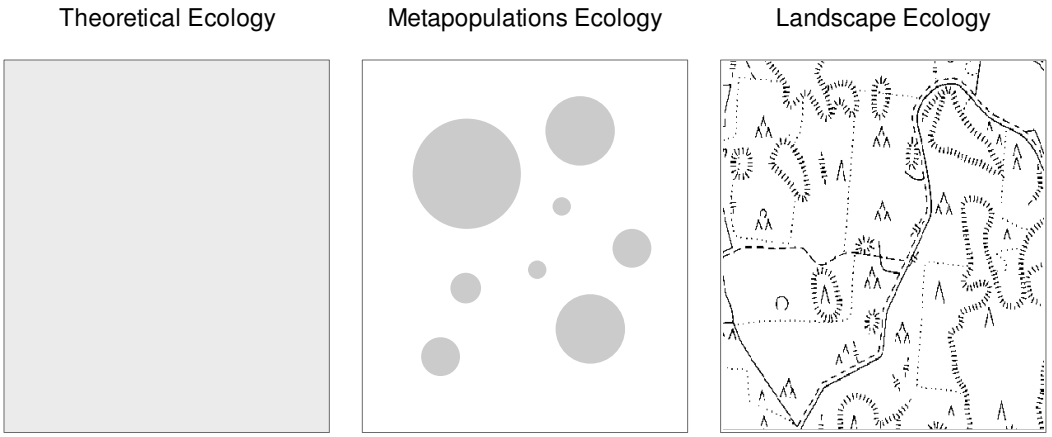

Figure 2.7: Hanski (1998) formulated the metapopulations approach to bridge the gap between theoretical ecology (which consides uniform density and homogeneous population interactions across landscape) and landscape ecology (which focuses on landscape features and not populations themselves). By considering populations spread in different patches and the effects of migrations, metapopulation models take into account implicitly the physical barriers imposed by landscape from one group (one patch) to the other; however within each patch, populations are assumed to be homogeneous.

Wu, 2008). Introducing ***delays*** *in the numerical response seems to result in a better fit in the case of predator-prey processes when the functional response shows predator dependence (Jost and Ellner, 2000).*

Stage-structuring, cyclic booms and delays add complexity to a model. The first two can be ignored because they do not match to the context and the species considered in this thesis. Population booms which usually cycle every 5-7 years (May and Hassell, 1988) are not visible for the time horizon that we consider in our biological control program (usually one season). So we can safely assume that the pest species considered in our model reproduce continuously, and are consumed continuously by their predators. On this time horizon as well as across the patch considered, we can also assume that the pest growth is continuous across the population, i.e. does not take place in cohorts, so we can model the pest population as one variable. We also assume that the predator reproduces quickly relative to the pest growth - as is the case for many voracious predaceous mites. Ignoring this delay also allows us to obtain first analytical estimates for stability conditions as outlined in this thesis.

■

2.6.2 Analysis

Throughout our work, we consider that the plantation to be protected from pest invasions consists of crops with very low or zero tolerance to pest. Examples include greenhouse crops with high cosmetic value. Alternatively, plants attacked by highly voracious invertebrates, which cause considerable damage even at low numbers, would have similarly low tolerance levels to their invasions.

In the biological control program considered in our work, *pest eradication* is therefore desirable. Mathematically, we will therefore be interested in the following properties of the

system:

The zero-pest solution. The existence of this solution, which we prove for each of the model we study, implies that provided some conditions are respected (see below), the control program may bring the pest population to zero.

The local and global stability of the zero pest solution. As far as possible, we formulate the conditions for the local (resp. global) asymptotic stability as a minimal bound on the release rate. This rate is the first control entry we have in our system. When respected, these conditions suppress pest invasions and deviations in the predator population from the periodic solution are compensated for. The global condition applies to all starting values of the pest-predator population, as much to any pest invasion size as to any predator population crash. The local condition caters for only 'small' fluctuation.

The dependence of the stability condition on the release period. This provides us with a second control feature, which allows us to identify the strategy of releases that:

1. requires the lowest release rate for pest eradication;
2. provides, for a given release rate, the fastest local convergence to the pest-free solution;
3. brings about the lowest risk of pest damage on a crop.

3

Groundwork: a simple model and analytic methods

"Draw a magical circle around them,
So that neither blight nor mildew,
Neither burrowing worm nor insect,
Shall pass o'er the magic circle. "
– From *The Song of Hiawatha*,
by H W Longfellow (1807–1882).

Overview

The aim of this Chapter is twofold: firstly, it introduces our bench model. This model depicts the fundamental biological and mechanical processes involved in an augmentative control program. Secondly, through the analysis of the zero-pest solution, this chapter presents in detail the mathematical concepts underlying the corpus of our work. We highlight the key steps involved in this analysis in order to allow the reader to grasp the underlying dynamics of the system.

Keywords: impulsive control, stability analysis, fixed-point transformation

Organisation of this chapter:
We proceed as follows.

Section 3.1 introduces a *simple* augmentative control model. This section also presents the *notations* used throughout this thesis. Moreover, based on the modelling premises, we define a set of general hypotheses on the functions involved in the system, which unless stated otherwise, apply to the variants of this model.

Section 3.2 deals with the existence and stability of the *zero-pest* or *pest-free* solution. This state is of interest to us because it represents mathematically the target of our pest

control program: pest eradication. Thus in this section, we first give a preview of the effect of applying instantaneous control to a system - or hybridising it - to obtain some insight on this solution's behaviour. In particular, we show the effect of the control on the fixed point of the intrinsic predator-prey dynamics. Then, we show, using the simple model as an example, how we may prove the existence of the pest-free solution of an impulsive control model. We also show - though this may not be possible for all models - how we explicitly calculate it. The criterion for pest eradication corresponds to the stability conditions of the zero-pest solution, which we express as a minimal condition on the predator release rate.

Section 3.3 includes a practical interpretation of our results.

The results obtained on this simple model, as well as the analytical methods used, provide the benchmark against which we compare those obtained for three of its variants studied subsequently in this thesis.

3.1 A simple model

In this thesis, we use as bench model the system presented by Mailleret and Grognard (2006). It depicts only two dimensions, namely the pest and the predator populations, denoted as x and y respectively. [1] We can represent it schematically by Figure 3.1.

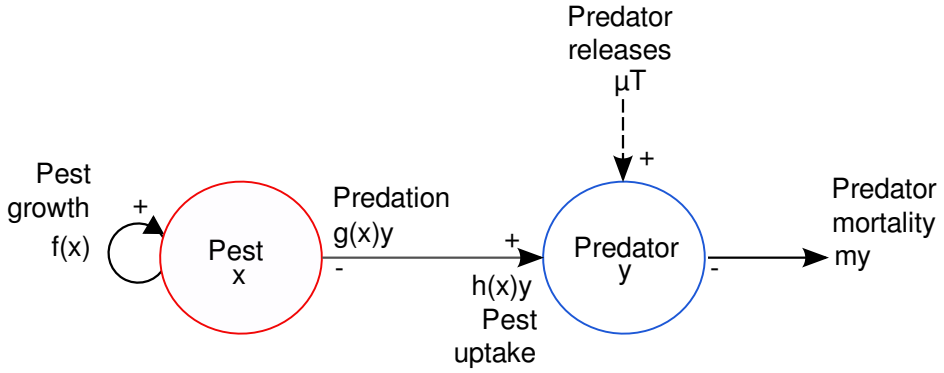

Figure 3.1: In the basic model for augmentative biological control, the biological processes considered are pest growth, the trophic responses, and predator mortality. The functional and numerical responses depend on pest density only. The mortality rate per predator is also constant. These processes are all continuous and are therefore represented by continuous arrows. By comparison, the predator releases occur at discrete moments in time. The broken arrow implies that it brings an instantaneous change (here, a surge) in the predator population size.

The intrinsic predator-prey dynamics are considered to be continuous and are therefore described using a pair of ordinary differential equations. By comparison, the predator releases occur at discrete instants in time, at which they cause a surge in the predator population. They are described by a discrete component. The resulting model is said to be hybrid, impulsive or semi-discrete because it takes into account both dynamical regimes. We write it as:

[1] The crop, on which the pest feeds, is incorporated within the pest growth.

$$\left\{\begin{array}{l} \dot{x} = f(x) - g(x)y \\ \dot{y} = h(x)y - my \end{array}\right\} t \neq nT \qquad (\mathcal{M})$$
$$y(nT^+) = y(nT) + \mu T, \quad \text{where } n \in \mathbb{N}$$

with non-negative initial conditions x_0 and y_0 at time t_0.

Continuous component (biological processes)

The function $f(x)$ represents the pest growth rate. The functional response $g(x)$ is the number of pests killed by predator or the decrease *per predator* in pest numbers due to predation. The numerical response $h(x)$ is the increase in predator biomass due to the consumed prey per predator. Recall that these functions are also referred to as *trophic functions* (see Chapter 2 and for instance Buffoni et al. (2005)). The predator mortality rate is represented by the positive constant m.

The functions $f(x)$, $g(x)$ and $h(x)$ are all locally Lipschitz continuous for $x, y \geq 0$: they have finite slopes and the solutions to the system are unique (Coddington and Levinson, 1955).

The growth and trophic functions are defined by biologically sensible properties. The first set of properties (General Hypotheses (H1)–(H3)) apply to all the models in this thesis. Note that the prime indicates the derivative of functions with respect to their argument.

General hypotheses

Pest growth at zero pest:

$$f(0) = 0 \quad \text{and} \quad f'(0) > 0 \qquad \text{(H1)}$$

Functional response:

$$g(0) = 0, \quad g'(0) > 0, \quad \text{and} \quad \forall x > 0,\ g(x) > 0; \qquad \text{(H2)}$$

Numerical response:

$$h(0) = 0 \quad \text{and} \quad \forall x > 0,\ h(x) \geq 0 \qquad \text{(H3)}$$

Through Hypothesis (H1), we consider that there is no spontaneous generation of the pest and that the pest population does not suffer from Allee effect.

The functional response is considered, unless stated otherwise, to be prey-dependent only. Hypothesis (H2) implies that there is no predation in the absence of pest, and that predation becomes immediately positive upon a pest invasion. This qualitative formulation encompasses the Holling Types I and II functions, as well as Holling III-like functions if we set $g'(0)$ to be arbitrarily low.

The numerical response is also considered to be prey-dependent only. Hypothesis (H3) implies that there is no predator population growth in the absence of pests, and that predation never has a negative effect on the predator.

We define separately the set of properties that are specific to the model presented in a given chapter or section. Here, Hypothesis (H4)) is specific to Model ($\mathcal{M}$).

Specific hypothesis

Ratio of pest growth to functional response:

$$\frac{f(x)}{g(x)} \text{ is upper bounded for } x \geq 0 \text{ such that } S_g = \sup_{x \geq 0} \left(\frac{f(x)}{g(x)} \right). \qquad \text{(H4)}$$

Hypothesis (H4) means that there is no value of x where the pest growth will overwhelmingly dominate the predation – which would otherwise make biological control impossible.

Discrete component (releases)

The augmentative releases are described by the third equation in Model ($\mathcal{M}$), with the $+$-superscript indicating the instant right after a release. The number of predators injected at these instants is defined as the product of the predator *release rate* μ and the *release period* T.

Written thus, we decouple the two characteristics of the biological control strategy to implement namely: the number of predators we are to invest in per unit time and, for a given rate, how to distribute this number over time. By doing so, we are able to compare different biological control strategies in terms of the release size and release frequency that involve the same overall number of predators.

3.2 Mathematical analysis of the simple model

3.2.1 A local preview: with or without continuous control

We may obtain some insights on the system's behaviour if we consider two regimes: one under continuous control and one without it. We write this model as:

$$\begin{cases} \dot{x} = f(x) - g(x)y \\ \dot{y} = h(x)y - my + \mu \end{cases} \tag{3.3}$$

We analyse the local behaviour of the system – setting $\mu > 0$ gives the controlled local trajectories while $\mu = 0$ corresponds to the uncontrolled system. At equilibrium,

$$\begin{cases} f(x) - g(x)y = 0 \\ h(x)y - my + \mu = 0 \end{cases} \tag{3.4}$$

From Hypotheses (H1)–(H3) on the system functions, one of the solutions of $f(x) - g(x)y = 0$ is $x = 0$. Setting $x = 0$ in the second equation of (3.4), we calculate the zero-pest equilibrium (x^*, y^*) as:

$$(x^*, y^*) = \left(0, \frac{\mu}{m}\right) \tag{3.5}$$

The Jacobian matrix, which describes the local dynamics of the system (see e.g. Dieudonné (1960)), about this fixed point is:

$$D = \begin{pmatrix} f'(0) - g'(0)y^* & 0 \\ h'(0)y^* & -m \end{pmatrix}$$

In the absence of control, $\mu = 0$, so the system equilibrium is the origin. The first eigenvalue of the Jacobian matrix is $f'(0)$ which is positive, so this point is a saddle point - with, in fact, trajectories decreasing locally along y and increasing in x.

When $\mu > 0$ however, that is control is on, the equilibrium point is positive in y. Furthermore, instead of a saddle point, because of the control entry available through $y^* = \frac{\mu}{m}$, this equilibrium point can be stabilised by providing a sufficiently large μ

$$\mu > \frac{mf'(0)}{g'(0)}, \tag{3.6}$$

which brings the eigenvalue $f'(0) - g'(0)y^*$ to a negative value. The control μ in fact drives the pest population to zero while maintaining a positive predator population.

Figure 3.2 illustrates the two trajectories of the autonomous and the controlled systems respectively.

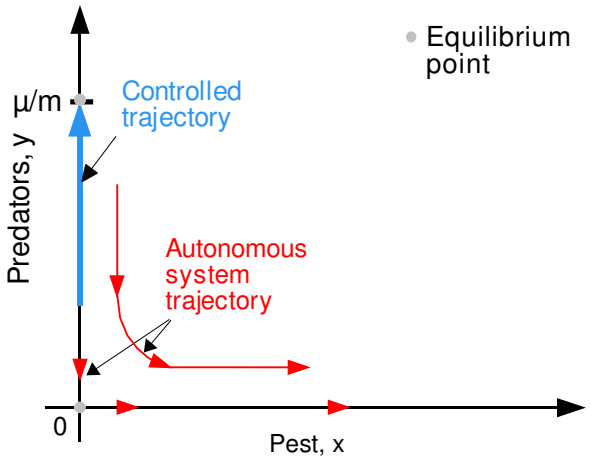

Figure 3.2: When the predator population is not replenished, the pest population will tend to increase while the predator population decreases: the origin is a saddle point. If the predator release rate is high enough, a locally stable zero-pest equilibrium exists at $(0, \mu/m)$. The arrows show the local trajectories over the state space.

Though this is an inadequate mathematical construct, we can imagine the effect of periodic predator releases in an augmentative control program as causing the system to switch alternatively from a controlled regime to the uncontrolled one. Upon a release (provided μ is large enough), the predator population momentarily increase and our calculations show that the system trajectories will be driven towards a positive-predator, zero-pest equilibrium point. Conversely, between releases the local predator dynamics run uncontrolled (i.e. $\mu = 0$) towards the origin. This would suggest the existence of a time varying zero-pest solution of the system (along the predator axis).

Releases are however discrete or instantaneous events compared to the intrinsic predator-prey dynamics. Thus using a purely continuous modelling approach, we cannot calculate the form of the zero-pest solution. Hybridising the system as ($\mathcal{M}$) introduces the release period as a parameter of the model while giving a more accurate description of the mixed continuous-discrete dynamics. This in fact allows us to evaluate the zero-pest solution, then assess the way in which the release rate required for pest eradication would depend on the release period. Note that when the release period T tends to 0, Model ($\mathcal{M}$) reduces to the continuously controlled model (3.4).

3.2.2 Positivity of the system and existence of the zero-pest solution

As stated in Section **??**, positivity is an important property of biological models, without which the practical relevance of a given model can be put in doubt. We should then analyse this for Model ($\mathcal{M}$). A classical way of showing positivity consists in using Theorem 1 by showing that the solutions cannot cross the boundaries of the positive orthant. In order to do that, let us consider the boundary where $x = 0$, where we we have:

$$\begin{cases} \dot{x} = 0 \\ \dot{y} = -my \end{cases}$$

which ensures invariance of this boundary. When $y = 0$, we have

$$\begin{cases} \dot{x} = f(x) \\ \dot{y} = 0 \end{cases}$$

which also ensures invariance of the boundary. Had we been in a situation where we only were considering continuous dynamics, Theorem 1 would have indicated that our system is positive. However, we must also take into account what happens at the impulses; since the impulses $(x(nT), y(nT)) \longrightarrow (x(nT), y(nT) + \mu T)$ also ensure the invariance of the positive orthant, system ($\mathcal{M}$) is positive.

We now define the zero-pest solution in Proposition 1, then describe in detail the calculations leading to it in the proof.

Proposition 1 (Existence of the pest-free solution)

$$(x_p(t), y_p(t)) = (0, y^* e^{-m(t \mod T)}) \tag{3.7a}$$

where

$$y^* = \frac{\mu T}{1 - e^{-mT}} \tag{3.7b}$$

is a periodic solution of Model ($\mathcal{M}$).

Proof: To prove the existence of the pest-free solution, we proceed as follows:

Step 1. Describe the zero-pest subsystem. In the absence of pests, System ($\mathcal{M}$) simplifies to

$$\begin{cases} \left.\begin{aligned} \dot{x} &= 0 \\ \dot{y} &= -my \end{aligned}\right\} t \neq nT \\ y(nT^+) = y(nT) + \mu T, \quad \text{where } n \in \mathbb{N} \end{cases}$$

Within this subsystem, the pest-dynamics solution $x = 0$ is trivial.

The predator population is periodically forced by the augmentative releases (the impulsive component), between which it decays due to its natural mortality (the continuous component). **Steps 2-4** detail how to compute y_p under these hybrid dynamics.

Step 2. Calculate the sequence of post-release y-values. Let the initial time be $t = nT^+$. Until the next release, $t \in (nT^+, (n+1)T)$ and we integrate the continuous y-dynamics to evaluate $y(t)$ as:

$$y(t) = y(nT^+)e^{-m(t-nT)} \tag{3.8}$$

At the end of the release period, $t = (n+1)T$, predators are released. So we obtain the sequence:

$$y((n+1)T^+) = y(nT^+)e^{-mT} + \mu T \tag{3.9}$$

Step 3. Calculate the limit of convergence of the sequence. We note that the coefficient

$$|e^{-mT}| < 1,$$

so the linear sequence (3.9) has an equilibrium to which it converges. By setting $y((n+1)T^+) = y(nT^+) = y^*$ in Equation (3.9), we calculate this limit as:

$$y^* = \frac{\mu T}{1 - e^{-mT}}. \tag{3.10}$$

Step 4. Formulate the periodic solution. We recall that between two releases, the continuous dynamics are determined by $\dot{y} = -my$ and the predator population value at the start of the release period. The latter is fixed by the limit y^* previously calculated in **Step 3**. This implies that, once y^* reached, the predator population will follow a periodic pattern. This periodic pattern is calculated by simply substituting y^* for $y(nT^+)$ in Equation (3.8) and replacing $(t-nT)$ by $(t \mod T)$ for a generalisation to all continuous intervals. Thus:

$$y_p(t) = y^* e^{-m(t \mod T)},$$

as given in (3.7a).

□

The limit y^* of the post-release sequence (calculated as (3.10)) can be seen as analogous to the fixed point calculated in (3.4). Readers will note however, due to the impulsive model formulation, y^* (3.10) is parameterised by the release period T. Incidentally, in the limit that $T \to 0$, that is in the limit that releases become continuous,

$$\lim_{T \to 0} \frac{\mu T}{1 - e^{-mT}} = \frac{\mu}{m},$$

that is, we recover the value in (3.4).

3.2.3 Stability of the zero-pest solution

We state the conditions required for the stability of the zero-pest solution in Theorem 2 (also given by Mailleret and Grognard (2009)). We then describe in its proof details of the analytic method used to reach this result.

Theorem 2 (Stability of the zero-pest solution) *Let $\underline{\mu}(Z) = mZ$. The zero-pest solution of Model ($\mathcal{M}$) defined in (3.7a) is locally asymptotically stable (LAS), if and only if*

$$\mu > \underline{\mu}\left(\frac{f'(0)}{g'(0)}\right) \tag{3.11a}$$

If, in addition, the following condition is satisfied,

$$\mu > \underline{\mu}\,(S_g) \tag{3.11b}$$

the solution is globally asymptotically stable (GAS) .

Proof: We calculate first the global attractivity condition in **Steps 1-5** below. Under this condition, all trajectories of the system starting in the positive orthant converge asymptotically to the zero-pest solution. We then compute the local stability condition in **Step 5 (Options 1 and 2)**. When both the global attractivity and the local stability conditions hold, **Step 7** shows how the global asymptotic stability is established, hence the proof completed. This is a classical method of analysis (see e.g. Khalil (2002) for a textbook reference).

Step 1. Describe the deviations systems dynamics. The deviation coordinates represent the difference between the actual state pair and the reference periodic solution $(0, y_p)$. We define these deviation coordinates as:

$$\tilde{x}(t) = x(t) - x_p(t) = x(t) \quad \text{and} \quad \tilde{y}(t) = y(t) - y_p(t). \tag{3.12}$$

Subjected to System ($\mathcal{M}$) dynamics and Hypotheses (H1) and (H2), the $\tilde{x}$-deviation varies as:

$$\dot{\tilde{x}} = f(x) - g(x)y = f(\tilde{x}) - g(\tilde{x})(y + y_p)$$

In a similar way, under Hypothesis (H3) and noting that $x_p = 0$, the continuous $\tilde{y}$-dynamics will vary as:

$$\begin{aligned}\dot{\tilde{y}} &= h(x)y - my - h(x_p)y_p + my_p \\ &= h(\tilde{x})(\tilde{y} + y_p) - m(\tilde{y} + y_p) + my_p \\ &= h(\tilde{x})(\tilde{y} + y_p) - m\tilde{y}\end{aligned}$$

The impulsive component of the $\tilde{y}$ dynamics vanishes because:

$$\begin{aligned}\tilde{y}(nT^+) &= y(nT^+) - y_p(nT^+) \\ &= y(nT) + \mu T - y_p(nT) - \mu T \\ &= y(nT) - y_p(nT) \\ &= \tilde{y}(nT)\end{aligned}$$

The dynamics in these new coordinates are:

$$\begin{cases} \dot{\tilde{x}} = f(\tilde{x}) - g(\tilde{x})(\tilde{y} + y_p) \\ \dot{\tilde{y}} = h(\tilde{x})(\tilde{y} + y_p) - m\tilde{y} \end{cases} \tag{3.13}$$

with initial conditions assigned as $\tilde{x}_0$ and $\tilde{y}_0$ at t_0. Note that $\tilde{x}_0$ is non-negative because $x = \tilde{x} + x_p \geq 0$, while $\tilde{y}_0 \geq -\max(y_p) = -y^*$ since $y = \tilde{y} + y_p \geq 0$.

Step 2. Divide the continuous $\dot{\tilde{x}}$-dynamics by an appropriate function, then integrate over the state and time domains. For reasons which will be apparent shortly (in the next step), this function is chosen positive.

For this simple model, we choose this function as $g(\tilde{x})$. From Hypothesis (H2), it is a positive function in $\tilde{x}$:

$$\forall \tilde{x} > 0, \quad g(\tilde{x}) > 0.$$

Dividing the $\dot{\tilde{x}}$-dynamics equation throughout by $g(\tilde{x})$ and integrating from t_0 to t, we have:

$$\int_{t_0}^{t} \frac{\dot{\tilde{x}}}{g(\tilde{x})} d\tau = \int_{t_0}^{t} \left[\frac{f(\tilde{x})}{g(\tilde{x})} - (\tilde{y} + y_p) \right] d\tau. \tag{3.14}$$

We note that $\dot{\tilde{x}}\ dt = d\tilde{x}$, so the left-hand side integral of (3.14) can be calculated on the $\tilde{x}$ domain as:

$$\int_{t_0}^{t} \frac{\dot{\tilde{x}}}{g(\tilde{x})} d\tau = \int_{\tilde{x}_0}^{\tilde{x}} \frac{1}{g(s)} ds;$$

We can then simply write the following relationship between the state and time domains:

$$\int_{\tilde{x}_0}^{\tilde{x}} \frac{1}{g(s)} ds = \int_{t_0}^{t} \left[\frac{f(\tilde{x})}{g(\tilde{x})} - (\tilde{y} + y_p) \right] d\tau. \tag{3.15}$$

Step 3. Identify the criterion for the convergence of $\tilde{x}$ to zero. We would like to identify what happens with the left-hand side of (3.15) when $\tilde{x}$ goes to zero. For a given $\tilde{x}_0$, since $g(\tilde{x})$ is locally Lipschitz continuous from Hypothesis (H2), there exists $r > 0$ such that $g(\tilde{x}) \leq r\tilde{x}$ for all $\tilde{x} \in [0, \tilde{x}_0]$; therefore,

$$\frac{1}{g(\tilde{x})} \geq \frac{1}{r\tilde{x}}.$$

Furthermore, because these functions are positive, the integrals over the state-domain when $\tilde{x} < \tilde{x}_0$ compare as:

$$\int_{\tilde{x}_0}^{\tilde{x}} \frac{1}{g(s)} ds \leq \int_{\tilde{x}_0}^{\tilde{x}} \frac{1}{rs} ds \tag{3.16}$$

When $\tilde{x} \to 0^+$,

$$\lim_{\tilde{x}\to 0^+} \int_{\tilde{x}_0}^{\tilde{x}} \frac{1}{rs} ds = -\infty.$$

Due to the comparison in (3.16), this implies that (again, in the limit that $\tilde{x} \to 0^+$):

$$\lim_{\tilde{x}\to 0^+} \int_{\tilde{x}_0}^{\tilde{x}} \frac{1}{g(s)} ds = -\infty.$$

Moreover, since $g(\tilde{x}) > 0$ for all $\tilde{x} > 0$, the integral $\int_{\tilde{x}_0}^{\tilde{x}} \frac{1}{g(s)} ds$ is finite for all $\tilde{x} > 0$. Therefore, if we can prove that

$$\lim_{t\to+\infty} \int_{\tilde{x}_0}^{\tilde{x}(t)} \frac{1}{g(s)} ds = -\infty, \tag{3.17}$$

then we can deduce that $\lim_{t\to+\infty} \tilde{x} = 0$, that is $\tilde{x}$ converges asymptotically to zero. Equation (3.17) thus provides the criterion for the convergence of $\tilde{x}$ to zero.

Step 4. Using appropriate bounds on system, identify the condition for the convergence of $\tilde{x}$ to zero. From (3.15), the criterion for the convergence of $\tilde{x}$ to zero in (3.17) is equivalent to:

$$\lim_{t\to+\infty} \int_{t_0}^{t} \left[\frac{f(\tilde{x})}{g(\tilde{x})} - (\tilde{y} + y_p) \right] d\tau = -\infty. \tag{3.18}$$

We make a small digression to identify the bounds on the elements of the integrand in Equation (3.18).

i. First, we recall Hypothesis (H4),

$$\sup_{\tilde{x}\geq 0} \frac{f(\tilde{x})}{g(\tilde{x})} \leq S_g. \tag{3.19}$$

This upper bound is time-independent.

ii. Second, from Hypothesis (H3), $\forall x,\ h(x) \geq 0$, we can bound the $\dot{\tilde{y}}$-dynamics as:

$$\dot{\tilde{y}} \geq -m\tilde{y} \tag{3.20}$$

which means that, from a simple comparison Theorem (Khalil, 2002) :

$$\tilde{y}(t) \geq \tilde{y}_0 e^{-m(t-t_0)}.$$

Furthermore, since $\tilde{y} + y_p = y$, and that (from the positivity of the system), $y \geq 0$, we have:

$$\forall\, t, \quad \tilde{y}(t) \geq -\max_{t\in[0,T]} y_p(t) = -y^*.$$

Thus, $\tilde{y}_0 \geq -y^*$. We can thus calculate a finer bound on $\tilde{y}$ as:

$$\tilde{y}(t) \geq -y^* e^{-m(t-t_0)}. \tag{3.21}$$

From the exponential form of the lower bound on $\tilde{y}(t)$, we can deduce that its integral over time will be lower bounded by a constant, which we calculate as

$$\begin{aligned} \lim_{t\to+\infty} \int_{t_0}^{t} \tilde{y}(\tau) d\tau &\geq \lim_{t\to+\infty} \int_{t_0}^{t} -y^* e^{-m(\tau-t_0)} d\tau \\ &= -\frac{y^*}{m} \lim_{t\to+\infty} (1 - e^{-mt}) \\ &= -\frac{y^*}{m}. \end{aligned} \tag{3.22}$$

Based on bounds (3.19) and (3.21), we split the time domain and the integrand in Equation (3.18) as[2]:

$$\int_{t_0}^{t}\left[\frac{f(\tilde{x})}{g(\tilde{x})}-(\tilde{y}+y_p)\right]d\tau = \left(\int_{t_0}^{(\lfloor\frac{t_0}{T}\rfloor+1)T}+\int_{(\lfloor\frac{t_0}{T}\rfloor+1)T}^{\lfloor\frac{t}{T}\rfloor T}+\int_{\lfloor\frac{t}{T}\rfloor T}^{t}\right)\left[\frac{f(\tilde{x})}{g(\tilde{x})}-y_p\right]d\tau-\int_{t_0}^{t}\tilde{y}(\tau)d\tau$$

The first and third integrals are upper bounded by S_gT. In the limit that $t\to+\infty$, the fourth integral is also upper bounded (see Equation (3.22)).

We focus on the second integral which, in that limit, can diverge. Using (3.19), we then upper bound the integrand with a T-periodic expression. We can express the integral as a multiple of smaller integrals each spanning a period T. Because of the periodicity, we can choose to integrate these smaller integrals over $t\in[0,T]$ for simplicity.

$$\begin{aligned}\int_{(\lfloor\frac{t_0}{T}\rfloor+1)T}^{\lfloor\frac{t}{T}\rfloor T}\left[\frac{f(\tilde{x})}{g(\tilde{x})}-y_p\right]d\tau &\leq \int_{(\lfloor\frac{t_0}{T}\rfloor+1)T}^{\lfloor\frac{t}{T}\rfloor T}[S_g-y_p(\tau)]d\tau\\ &= \sum_{n=\lfloor\frac{t_0}{T}\rfloor+1}^{\lfloor\frac{t}{T}\rfloor-1}\int_{nT}^{(n+1)T}[S_g-y_p(\tau)]d\tau\\ &= \left(\lfloor\tfrac{t}{T}\rfloor-\lfloor\tfrac{t_0}{T}\rfloor-2\right)\int_0^T[S_g-y_p(\tau)]d\tau\end{aligned}$$

The divergent term is the number of smaller integral required to make the larger integral, i.e. $\left(\lfloor\frac{t}{T}\rfloor-\lfloor\frac{t_0}{T}\rfloor-2\right)$. For the attractivity criterion (3.18) to be satisfied, it suffices that:

$$\int_0^T[S_g-y_p(\tau)]\,d\tau<0 \tag{3.23}$$

We then evaluate the integral of the predator solution y_p, which is parameterised by the release rate μ (see solution (3.7a)), and express Condition (3.23) as a lower bound on the release rate. Resuming our calculations, Condition (3.23) is equivalent to:

$$\begin{aligned}&\int_0^T y_p(\tau)\,d\tau>S_gT\\ \Leftrightarrow\quad &\frac{\mu T}{1-e^{-m\tau}}\int_0^T e^{-m\tau}\,d\tau>S_gT\\ \Leftrightarrow\quad &\frac{\mu T}{1-e^{-m\tau}}\left(\frac{1-e^{-mT}}{m}\right)>S_gT\\ \Leftrightarrow\quad &\mu>mS_g\end{aligned}$$

which is the condition (3.11b).

Step 5. Establish convergence of $\tilde{y}$ to zero when attractivity condition is satisfied. Since $\tilde{x}$ goes to zero, there exists a finite time t_f after which $h(\tilde{x})\leq\frac{m}{2}$ for all times. After this time, when $\tilde{y}>0$ we have

$$\dot{\tilde{y}}=h(\tilde{x})(y_p(t)+\tilde{y})-m\tilde{y}\leq h(\tilde{x})y_p(t)-\frac{m}{2}\tilde{y}<0$$

[2]For visual clarity, the following notation is used to split an integral into k integrals:

$$\int_A^B F(t)dt=\left(\int_A^{n_1}+\int_{n_1}^{n_2}+\cdots+\int_{n_k}^B\right)F(t)dt$$

where

$$\left(\int_A^{n_1}+\int_{n_1}^{n_2}+\cdots+\int_{n_k}^B\right)F(t)dt=\int_A^{n_1}F(t)dt+\int_{n_1}^{n_2}F(t)dt+\cdots+\int_{n_k}^B F(t)dt$$

Since $h(\tilde{x})y_p(t)$ goes to zero as t goes to infinity, so does also $\tilde{y}$. Conversely, suppose that $\tilde{y} < 0$: following a similar reasoning, we obtain, when $t > t_f$,

$$\dot{\tilde{y}} = h(\tilde{x})(y_p(t) + \tilde{y}) - m\tilde{y} \geq h(\tilde{x})y_p(t) - \frac{m}{2}\tilde{y} > 0$$

Once again, since $h(\tilde{x})y_p(t)$ goes to zero as t goes to infinity, so does also $\tilde{y}$. At this step, we can conclude that the solution $(0, y_p(t))$ is globally attractive for Model ($\mathcal{M}$) provided criterion (3.18) holds.

We now need to investigate the local stability of this solution. Two different methods can be used.

Step 6 (Option 1). Identify the Floquet multipliers of the system by calculating the discrete linearised mapping of $(\tilde{x}, \tilde{y})(nT^+)$ onto $(\tilde{x}, \tilde{y})((n+1)T^+)$. The local stability of a periodic solution of a nonlinear system is established using Floquet Theory (Amabili, 2008).

Recalling from Hypotheses (H1)–(H3) that

$$f(0) = 0, g(0) = 0, \text{ and } h(0) = 0,$$

the first-order dynamics in $\tilde{x}$ and $\tilde{y}$ of System ($\mathcal{M}$) is:

$$\begin{cases} \dot{x} = (f'(0) - g'(0)y_p)\tilde{x} \\ \dot{\tilde{y}} = h'(0)y_p\tilde{x} - m\tilde{y} \end{cases} \tag{3.24}$$

In matrix form, we can write the system dynamics as:

$$\begin{pmatrix} \dot{\tilde{x}} \\ \dot{\tilde{y}} \end{pmatrix} = \begin{pmatrix} f'(0) - g'(0)y_p(\tau) & 0 \\ h'(0)y_p & -m \end{pmatrix} \begin{pmatrix} \tilde{x} \\ \tilde{y} \end{pmatrix}(t)$$

Solving for the post-release mapping (which we write in matrix form):

$$\begin{pmatrix} \tilde{x} \\ \tilde{y} \end{pmatrix}((n+1)T^+) = \mathbf{B} \begin{pmatrix} \tilde{x} \\ \tilde{y} \end{pmatrix}(nT^+)$$

where the matrix

$$\mathbf{B} = \begin{pmatrix} e^{\int_{nT^+}^{(n+1)T^+}[f'(0)-g'(0)y_p(\tau)]d\tau} & 0 \\ \ddagger & e^{-m\int_{nT^+}^{(n+1)T^+} d\tau} \end{pmatrix} \tag{3.25}$$

The term ‡ is not calculated since it is not needed for our analysis. The integrands being T-periodic, we can set $n = 0$ for convenience. The mapping matrix is lower-triangular, so the spectral radius is the largest of its two diagonal terms (see e.g. Khalil (2002) for the stability of discrete multidimensional systems). We simply require first that:

$$\left| e^{-m\int_0^T d\tau} \right| < 1, \tag{3.26}$$

that is, $\left|e^{-mT}\right| < 1$. This is trivially satisfied since $m > 0$. Secondly we require that:

$$\begin{aligned} & \left| e^{\int_0^T [f'(0)-g'(0)y_p(\tau)]d\tau} \right| < 1 \\ \Leftrightarrow \quad & \int_0^T [f'(0) - g'(0)y_p(\tau)]d\tau < 0 \end{aligned} \tag{3.27}$$

On dividing by $g'(0)$ throughout, this inequality is of the same form as the global stability condition (with S_g replaced by $f'(0)/g'(0)$). We then obtain as condition:

$$\mu > \frac{mf'(0)}{g'(0)} \tag{3.28}$$

which is the LAS condition (3.11a) for System ($\mathcal{M}$); this shows that $(0, y_p(t))$ is stable iff (3.11a) holds.

The second method that can be used to show this is detailed now.

Step 6 (Option 2). Alternatively, quote directly Theorem 1 from Lakmeche and Arino (2000). One of the components of the trivial solution being zero, the periodic linear mapping is lower triangular (see matrix B (3.25)). Using this feature, Lakmeche and Arino (2000) generalise the calculation of the Floquet multipliers of a two-dimensional qualitatively defined periodic impulsive system. They also provide the local stability condition for the trivial solution. We adapt their notation thus:

$$\left\{\begin{array}{l} \left.\begin{array}{l} \dot{x} = F_2(y,x) \\ \dot{y} = F_1(y,x) \end{array}\right\} t \neq nT \\ x(nT^+) = \theta_2(y,x) \\ y(nT^+) = \theta_1(y,x), \quad \text{where } n \in \mathbb{N}. \end{array}\right. \tag{$\mathcal{L}$}$$

We reformulate Theorem 1 from Lakmeche and Arino (2000) (for the complete proof, we refer the reader to Lakmeche and Arino's paper) as:

Theorem 3 (LAS conditions, from Lakmeche and Arino (2000))
The zero-pest solution $(0, y_p)$ *of System* ($\mathcal{M}$) *is LAS if and only if*

$$\left|\frac{\partial \theta_1}{\partial y}\Big|_{(y,x)=(y_p(T),0)}\right| exp\left(\int_0^T \frac{\partial F_1}{\partial y}\Big|_{(y,x)=(y_p(\tau),0)} d\tau\right) < 1 \tag{$\mathcal{L}1$}$$

and

$$\left|\frac{\partial \theta_2}{\partial x}\Big|_{(y,x)=(y_p(T),0)}\right| exp\left(\int_0^T \frac{\partial F_2}{\partial x}\Big|_{(y,x)=(y_p(\tau),0)} d\tau\right) < 1. \tag{$\mathcal{L}2$}$$

Using this notation for system ($\mathcal{M}$), we have:

$$\left\{\begin{array}{l} \left.\begin{array}{l} \dot{y} = F_1(y,x) = h(x)y - my \\ \dot{x} = F_2(y,x) = f(x) - g(x)y \end{array}\right\} t \neq nT \\ y(nT^+) = \theta_1(y,x) = y(nT) + \mu T \\ x(nT^+) = \theta_2(y,x) = x(nT), \quad \text{where } n \in \mathbb{N}. \end{array}\right. \tag{3.30}$$

Applying Theorem 3, we compute from ($\mathcal{L}1$) the trivial condition $m > 0$ while ($\mathcal{L}2$) yields the LAS condition (3.11a).

Step 7. Show that the LAS condition completes the proof for global asymptotic stability. The expression $\underline{\mu}(Z)$ is increasing in Z:

$$\frac{\partial \underline{\mu}}{\partial Z} = m > 0.$$

We note that $\dfrac{f'(0)}{g'(0)} = \lim\limits_{x \to 0} \dfrac{f(x)}{g(x)}$, so that, from its definition in Hypothesis (H4), $S_g \geq \dfrac{f'(0)}{g'(0)}$. Consequently,

$$\underline{\mu}(S_g) \geq \underline{\mu}\left(\frac{f'(0)}{g'(0)}\right).$$

When the global attractivity condition and the LAS condition are satisfied, the proof for global asymptotic stability is complete. In this simple model, when $\mu > \underline{\mu}(S_g)$, the LAS condition is de facto satisfied, which shows that this is a sufficient condition for GAS.

□

Remark: *The function $\underline{\mu}$ represents the minimal release rate that would ensure pest eradication. In this simple model, the minimal release rate does not depend on the release period T. We discuss the insights this hybrid formulation gives us for the simple Model ($\mathcal{M}$) in Section 3.3 of the present chapter.* ■

3.2.4 A note on the computation of the global attractivity condition

Throughout this thesis, in our calculation of the global attractivity conditions, we will do as in **Steps 1-5** and divide the $\dot{\tilde{x}}$-dynamics throughout by a Lipschitz continuous function $\Psi(\tilde{x})$ as:

$$\frac{\dot{\tilde{x}}}{\Psi(\tilde{x})} = \frac{f(\tilde{x})}{\Psi(\tilde{x})} - \frac{g(.)}{\Psi(\tilde{x})}(\tilde{y} + y_p) \tag{3.31}$$

Through similar arguments as in the previous section, in order to show the convergence of $\tilde{x}$ to zero, we will then need to establish the divergence:

$$\lim_{t \to +\infty} \int_{\tilde{x}_0}^{\tilde{x}(t)} \frac{1}{\Psi(s)} ds = -\infty \tag{3.32}$$

by using the right-hand side of (3.31), but also other terms in some cases like the one exposed in Chapter 6, where harvesting introduces impulses that do not disappear upon the change of coordinates.

A model-specific requirement will then be that there is an upper bound on the ratio of the pest growth to the $\Psi(x)$-function: $\forall x \geq 0,\ \frac{f(x)}{\Psi(x)} \leq S_\Psi$, where $S_\Psi = \sup_{x \geq 0} \frac{f(x)}{\Psi(x)}$, and still be able to say something about $\frac{g(.)}{\Psi(\tilde{x})}$, so that the condition will depend on the analysis of the integral of $y_p(t)$, which contains most of the information about the choice of release rate.

Remark: *We may remark here from this brief description of our methodology that the global stability condition will depend on two main features:*

1. *the choice of the function $\Psi(\tilde{x})$, and the consequent upper bound on $f(\tilde{x})/\Psi(\tilde{x})$ – this will also determine how refined the minimal bound on the release rate required for pest eradication will be;*
2. *the y dynamics, notably on the integration of y_p, which mainly indicates the influence of the release rate.*

■

3.2.5 Interpreting the LAS and GAS conditions

The LAS condition applies to a local neighbourhood of the pest-free solution: this means that the LAS minimal release rate in (3.11a)

$$\mu > \underline{\mu}\left(\frac{f'(0)}{g'(0)}\right) = m\frac{f'(0)}{g'(0)}$$

is able to eradicate only small pest invasions. It is also resilient to small deviations from the periodic predator solution due to small predator migrations or sudden depensations related to

environmental factors. This rate would work best in a *preventive* context, that is to maintain a sentry predator population irrespective of pest detection so that an invasion is suppressed at its onset.[3]

The GAS condition (3.11b)

$$\mu > \underline{\mu}(S_g) = mS_g$$

extends the basin of attraction covered by the LAS condition to the whole (x, y) domain: this means that when the release rate respects this condition, any starting value of (x, y) will converge to the pest-free solution. This rate therefore can provide control even as a *curative* measure.

Interestingly, the GAS condition is a generalisation of the LAS condition in the limits of some function relation (here, in particular this relation is the ratio of the per capita pest growth rate to the per capita predation rate). However, though such relations between the GAS and LAS conditions make for a rather neat analytical result, they cannot be deduced from a direct inspection of the system dynamics.

The Lotka-Volterra Model

Beyond this analytical resemblance, GAS and LAS conditions are identical for some of the most classical predator-prey models. For instance, the Lotka-Volterra model

$$\left\{ \begin{array}{l} \left.\begin{array}{l} \dot{x} = rx - axy \\ \dot{y} = \gamma axy - my \end{array}\right\} t \neq nT \\ y(nT^+) = y(nT) + \mu T, \quad \forall n \in \mathbb{N} \end{array} \right.$$

yields identical conditions for local and global stability $\mu > \dfrac{mr}{a}$ since $\dfrac{f'(0)}{g'(0)} = S_g = \dfrac{r}{a}$. This is due to the intrinsically linear character of the pest-growth function and of the functional responses, and shows more that this model if over-simplified than about actual equivalence in real-life.

The Rosenzweig-MacArthur Model

We can also consider the more elaborate Rosenzweig-MacArthur model (Rosenzweig (1971, 1972))

$$\left\{ \begin{array}{l} \left.\begin{array}{l} \dot{x} = rx\left(1 - \dfrac{x}{K}\right) - \dfrac{ax}{c+x}y \\ \dot{y} = \dfrac{\gamma ax}{c+x}y - my \end{array}\right\} t \neq nT \\ y(nT^+) = y(nT) + \mu T, \quad \forall n \in \mathbb{N} \end{array} \right.$$

where the pest growth follows a classical logistic formulation, parameterised by a growth rate $r \geq 0$ and a carrying capacity $K \geq 0$. The functional and numerical responses are Holling Type II Holling (1961), defined by $a > 0$, the half-saturation constant $c > 0$, and the conversion factor $\gamma > 0$. In this case, the comparison between the two conditions is not as direct because they are in the form $\mu > \dfrac{rmc}{a}$ (LAS) and $\mu > \dfrac{rmS_g}{a}$ (GAS) with

$$S_g = \left\{ \begin{array}{ll} \dfrac{(K+c)^2}{4K} & \text{for } 0 \leq c \leq K \\ c & \text{for } c \geq K \end{array} \right.$$

Since $S_g \geq c$, we conclude that the LAS condition is, as expected, weaker than the GAS condition, but that the two are equivalent when $K \leq c$, that is when the pest carrying capacity

[3] The size of this local neighbourhood - how 'small' the invasion needs to be in order to be tackled successfully by the LAS-condition - requires further calculations.

is smaller than the half-saturation constant of the predation terms; in essence, this means that the functional response is roughly linear in the zone of interest for the model (because x stays smaller than K), which entails that local and global stability conditions are equivalent.

Unbounded $\frac{f(x)}{g(x)}$

Two possible scenarios arise:

- Suppose this ratio is unbounded only as $x \to \infty$, i.e. $\lim_{x\to\infty} \frac{f(x)}{g(x)} = \infty$. Then, the pest-free solution can be globally attractive if the numerical response of the predators ($h(x)$) increases quickly enough with the pest population. From the predator dynamics, $\dot{y} = h(x)y - my$, the predators y increase to sufficiently large numbers and are able to suppress the pest invasion by predation (the $g(x)y$ term in $\dot{x} = f(x) - g(x)y$).
- Suppose however that the ratio is unbounded at a finite pest population level, i.e. $\lim_{x\to\bar{x}} \frac{f(x)}{g(x)} = \infty$, where $\bar{x} < \infty$. In this case, if the initial pest population size at an invasion is larger than $\bar{x}$, the predators will never be able to suppress the pest population: as the pest population decreases, it either undergoes a boom at $\bar{x}$ or predation is insufficient to contain it (or both). Mathematically, the pest population cannot approach the basin of attraction beyond $\bar{x}$ and to zero. The pest cannot be eradicated.

3.3 Pest control strategy

3.3.1 The minimal predator release rate

How many predators need to be released to eradicate a pest population surge?

The predator release rate μ needs to be larger than some minimal value described by the function $\underline{\mu}$. For our simple model, the predator release rate must respect the following condition:

$$\mu > \underline{\mu}(S_g) = mS_g \quad \text{(the GAS condition)}$$

or, at smaller invasions:

$$\mu > \underline{\mu}\left(\frac{f'(0)}{g'(0)}\right) = m\frac{f'(0)}{g'(0)} \quad \text{(the LAS condition).}$$

This minimal predator release rate depends on predator mortality and the ratio of per capita pest growth to per capita predation. A higher predator mortality requires a higher release rate. Conversely, pest control is achieved at a lower cost for pest species with lower reproductive rates and predators with higher attack rates.

See also Section 3.2.5.

What happens when the predator release rate is lower than the minimal bound?

When $\mu < \underline{\mu}\left(m\frac{f'(0)}{g'(0)}\right)$, the zero-pest solution is not stable. From the matrix B in Equation (3.25), the pest population will tend to increase in size at least locally, which shows that eradication is then not possible. This actually does not mean that the pest will grow very large: one can expect a bifurcation to a periodic positive solution with small pest population densities, for μ values close to the threshold.

3.3.2 The period of release

Do we choose to make rare large releases or frequent smaller ones?

The stability conditions are independent of the release period. This suggests that a given release rate satisfying the stability condition is able to eradicate the pest population whether the total number of predators are released once or they are spread over more frequent smaller releases.

In fact, the LAS-condition is even identical to the one obtained in the continuous controlled model in Section 3.3 of the present chapter. So, does this mean that modelling the control continuously is an adequate description of an augmentative biological control program? The answer is 'no'.

The first reason is that the actual system that we are modelling is intrinsically impulsive. Using continuous modelling would yield conclusions whose translation into real-life we could not trust.

The second reason is that one of the control parameters whose value we want to fix is the period of release; if it is absent from the model, we will not be able to say anything about it. Even though the stability conditions did not give any indication as to what choice should be made for the period of release, Mailleret and Grognard (2006, 2009) showed that, in order to prevent large damage in the case where a pest-invasion would take place at the worst time between two releases, it is advised to take the period of release as small as possible. Indeed, if this period is chosen large, the released predators die out quite fast in the absence of preys, so that they are not able to counter the invasion taking place subsequently before the next release is made.

4

Predators interfering with each other during predation

Overview

There are two ways in which intrapredator interference can impact the population dynamics. The first is through interfering with the pest uptake, the second enhances predator mortality through fights or cannibalism Hassell (1978).

In this chapter we are concerned with the first type of interference: interference during the predation process. In this case, the trophic responses are best described as being directly predator-density dependent (see e.g. Begon et al. (1996)). Interference is penalising, that is predators prevent each other from feeding. So the predation per predator decreases as the predator density increases. We focus on this general trend and propose to use sector conditions from the control engineering literature (Vidyasagar, 1993) when specific predator interactions are not precisely known.

Through the analysis of our model, we answer two questions: firstly, what conditions are required for the pest population to be eliminated and secondly, what release strategy is to be employed for this eradication be as quick as possible thereby minimising the damage to crops. These concern the stability of the zero-pest solution and the local convergence to that solution respectively.

Keywords: intrapredator interference, Beddington-DeAngelis, ratio-dependent functional response, sector conditions.

Organization of this chapter:

This chapter is structured as follows.

Section 4.1 gives specific instances of the intrapredator competition occurring among natural enemy species involved in biological control. In particular, we highlight how this competition can directly interfere with the predators' efficacy to kill the prey, and hence, is to be incorporated within the trophic responses in predator-prey models.

Section 4.2 introduces our model which includes generic trophic responses that are directly dependent on the predator population. We use sector conditions to bound nonlinear parameters of these functions: this caters for uncertainties in the predator-prey behaviour. We argue that it can also account for local effects, that is slight changes in behavioural patterns occurring over short ranges.

Section 4.3 demonstrates the existence of the periodic pest-free solution driven by the predator releases and compute the conditions required for its stability: one is related to the biology of the predator species while the other defines a minimal predator release rate that guarantees pest eradication. We treat separately the case where the parameters of the trophic functions are affine mappings and the case where these mappings are nonlinear and bounded within sector conditions. In the nonlinear case, we are only able to specify an upper threshold for the minimal release rate, above which the zero-pest solution is stable; and a lower threshold below which a release rate is insufficient to stabilise the zero-pest solution.

In the next sections, we treat two features of our analysis in more detail:

Section 4.4 establishes that the minimal predator release rate is an increasing function of the release period.

Section 4.5 shows that the speed of eradication decreases with the release period.

In each section, the results are presented first for the case when parameters of the trophic responses are affine, which is a simpler version than the one allowed by a more general formulation. These results are then extended to the case when the parameters are nonlinear and are bounded within sectors.

Section 4.6 provides a review of our results.

4.1 The premises

Impact of interference on predator access to prey

Intrapredator interference during predation is well-documented in the biological control literature. In instances where it does not cause the predator to disperse, only the trophic response (in particular, the functional response) is affected, and not predator mortality (which includes fleeing rate). There are two types of behaviours which suppress the trophic responses at higher predator numbers.

The first is mechanistic: the presence of higher densities of conspecifics results in a higher interaction with them - for no particular reason, and is observed as collisions - and decreases the time a predator could spend searching for and handling a prey. Such interference is apparent, for example, among predatory mites that are widely used against various pests in orchards and greenhouse crops (Everleigh and Chant, 1982; Gerson, 2007; Skirvin et al., 2007).

The second is a consequence of energistic requirements. Several insect species of the Hymenoptera and Coleoptera order emit volatile products at high population densities. We cite a few examples: Damon (2000) reports that adult *Cephalonomia stephanoderis* (a parasitoid of the coffee berry borer) emit an aromatic substance in confined spaces. Yasuda et al. (2000) observed that the females of the predatory ladybirds *Harmonia axyridis* (predators of several pest species) emits oviposition deterring volatile pheromones. Damon (2000) argues that this process has a high energetic cost as a consequence of which, the organism is less efficient in prey searching and handling, or in controlling pests.

Nachman (2006); Umbanhowar and Maron (2003) and several others assert that interference effects are not as apparent in an open field (where predators can disperse to avoid each other) as they are in laboratory conditions. According to Damon and Valle (2002) however, such effects cannot be ignored in a biological control program: this is because control agents are usually carried in boxes in which they are densely packed and where the emission of such pheromones is high.

Direct predator-dependence of the trophic response

Holling (1959a) built the functional response mechanistically (see Chapter 2). His calculations were based on the time taken for a predator to search for and handle a prey. He obtained a function of the form:

$$g(x) = \frac{ax}{bx+1},$$

which is known as the Holling Type II functional response. The functional response is not predator dependent (Begon et al., 1996). The total prey removal is then given by $g(x)y = \frac{axy}{bx+1}$ (a and b are parameters).

In fact, though he did not himself formulate a predator-dependent functional response, Holling (1959b, 1961) acknowledged (from the data in his bibliographical research) that predators interact with each other at varying degrees at different densities. He remarked that this was likely to affect prey handling and searching, and thus the functional response form. Following Holling's mechanistic reasoning to calculate the Holling II functional response, Beddington (1975) and DeAngelis et al. (1975) postulated that in addition to encountering their prey, predators were also likely to collide with each other. They added a time component that describes the time predators 'lose' in interfering with each other and formulated the eponymous function (the Beddington-DeAngelis functional response) as:

$$g(x,y) = \frac{ax}{bx+cy+1},$$

where a, b and c are parameters.

Arditi and Ginzburg (1989) later suggested that it was only the *ratio* of the prey density to the predator density, and not the actual populations densities, that best captured the interference effect. As a result, Arditi and Ginzburg (1989) modified the Holling II function by changing the prey density argument into the per predator capita food availability. They proposed a response of the form

$$g(x/y) = \frac{a(x/y)}{b(x/y)+1} = \frac{ax}{bx+y} \tag{4.1}$$

This formulation is said to be *ratio-dependent*. The support for such a model has been rather strong because it is in good agreement with experimental data (e.g. Akcakaya et al. (1995); Skalski and Gilliam (2001)). But the singularity the model displays at the origin and the lack of mechanistic support has been subject to a fierce debate in the ecological modelling community (see e.g. Abrams (1994); Gleeson (1994); Sarnelle (1994)).

Buffoni and Gilioli (2003); Buffoni et al. (2005) generalised the analysis of density-dependent (Beddington-DeAngelis) and ratio-dependent functional responses by parameterising the argument of the trophic functions thus:

$$g\left(\frac{ax}{\theta by + (1-\theta)}\right), \tag{4.2}$$

with the parameter θ taking its values between 0 and 1. They qualitatively defined the response as:

$$g(0) = 0, \quad \lim_{s\to+\infty} g(s) \to \text{constant (positive)}, \quad \forall\, s,\ g'(s) > 0 \tag{4.3}$$

and pointed out that a Holling Type II function respects these criteria. In fact, under a mapping of the form $g(s) = \frac{s}{s+1}$ (which is a simple version of the Holling II mapping), this functional response becomes:

$$g\left(\frac{ax}{\theta by + (1-\theta)}\right) = \frac{ax}{ax + \theta by + (1-\theta)} \tag{4.4}$$

In the limit that $\theta = 0$, the expression in (4.4) is a Holling Type II response, and when $\theta = 1$, it takes a ratio-dependent form. The intermediate values of $0 < \theta < 1$ give the Beddington-DeAngelis functional response. So, the qualitative description of the function by Buffoni et al. (2005) generalises all three formulations.

In the present chapter, we generalise even further Buffoni et al. (2005)'s formulation, by considering qualitative functions to describe the parameters, that is:

$$g\left(\frac{\alpha(x)}{\beta(y)}\right) \tag{4.5}$$

where $\alpha(x)$ and $\beta(y)$ are generally increasing in x and y respectively. We explain the basis for this generalisation in the next section.

Remark: *In the functions described above, $g(x, y)$ is decreasing in y. Yet, although predator interference is taken into account, the total prey removal $g(x, y)y$ for both Beddington-DeAngelis and ratio-dependent interference is still increasing in y. This kind of interference is therefore said to be 'moderate'. We come back to this point in the Section 4.6 of this chapter.* ■

Interference with impulsive control

To the best of our knowledge, impulsive models that also take into account density-dependent predation consider only explicit Beddington-DeAngelis. Zhang and Chen (2006) and Zhang et al. (2008) analyse the local (but not global) stability of the pest-free solution, and focus on coexisting states.

Negi and Gakkhar (2007) investigate the behaviour of a system where the predator is subjected to impulsive proportional harvesting. There are no additive impulses modelling releases. They establish the global stability condition for the trivial prey-only state, but this result cannot be mapped directly to a system with additive releases such as the one used to describe augmentative biological control. In Nundloll et al. (2010b), we calculate global stability conditions for the zero-pest solution in an impulsive control program, but the model only considers an explicit Beddington-DeAngelis functional response.

The more general formulation by Buffoni et al. (2005) cater only for conservation biological control programs, where the dynamics are purely continuous and thus do not consider the influence of impulsive releases on the model dynamics.

In this chapter, we generalise Buffoni et al. (2005)'s formulation and extend the analysis for the *density-dependent case* to the zero-pest solution within an impulsive framework. Thus, we investigate how predator releases affect a model that incorporates a general description of intrapredatory interference.

Remark: *In the final paper published during this thesis (Nundloll et al., 2010a), we have been able to extend our results to include the generalised version of ratio-dependence, thus completing the analysis of the zero-pest solution for the generalised model by Buffoni et al. (2005). Due to time constraints however, it was not possible to update the calculations in this chapter.* ■

4.2 The model

Generic qualitative formulation

We adopt the general framework proposed in Chapter 3, but use trophic responses that are directly dependent on the predator density to include the penalising intrapredator interference effects (see Figure 4.1).

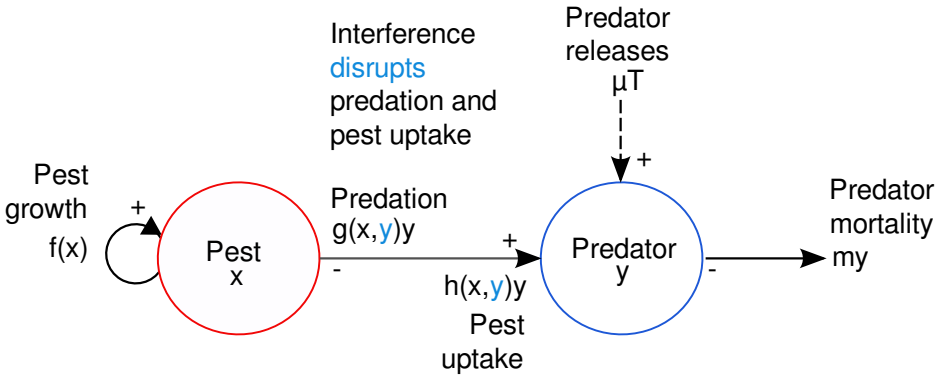

Figure 4.1: The trophic responses are directly dependent on the predator density. Furthermore, when predators interfere with each other in prey access, the transfer of biomass from prey to predator is reduced.

Using the formulation in (4.5), we therefore modify the augmentative control model as:

$$\left\{\begin{array}{l} \dot{x} = f(x) - g\left(\frac{\alpha(x)}{\beta(y)}\right) y \\ \dot{y} = h\left(\frac{\alpha(x)}{\beta(y)}\right) y - my \\ y(nT^+) = y(nT) + \mu T, \end{array}\right\} t \neq nT \quad \text{where } n \in \mathbb{N} \tag{$\mathcal{M}_b$}$$

with non-negative initial conditions x_0 and y_0.

To ensure unicity of trajectories, the growth function $f(x)$ and predation functions $g\left(\frac{\alpha(x)}{\beta(y)}\right) y$ and $h\left(\frac{\alpha(x)}{\beta(y)}\right) y$ are assumed all locally Lipschitz continuous for $x, y \geq 0$. As previously, m represents the mortality rate of the predators. It is a positive constant, independent of the predator population size. We recall the general hypotheses from Chapter 3 on the growth and trophic responses as:

General hypotheses

$$f(0) = 0, \quad f'(0) > 0; \tag{H1}$$

$$g(0) = 0, \quad g'(0) > 0, \quad \forall s > 0, \quad g(s) > 0; \tag{H2*}$$

$$h(0) = 0, \quad \forall s > 0, \quad h(s) \geq 0 \tag{H3*}$$

The trophic responses are parameterised by $s = \frac{\alpha(x)}{\beta(y)}$; so the prime of the functional response indicates the derivative with respect to s. The mapping $\alpha(x)$ verifies linear sector conditions, that is $\forall\, x \geq 0$

$$a_1 x \leq \alpha(x) \leq a_2 x. \tag{4.7}$$

Likewise, $\beta(y)$ are assumed to verify affine sector conditions as follows: $x, y \geq 0$.

$$b_1 y + b_0 \leq \beta(y) \leq b_2 y + b_0. \tag{4.8}$$

When the coefficient b_0 equals zero, the argument is ratio-dependent, while a strictly positive $b_0 > 0$ results in a density-dependent argument. The other coefficients are all strictly positive, i.e. $a_i, b_i > 0$. In this chapter *we shall stick to a positive* b_0; in this situation it is fairly easy to show through the reparameterisation of a_1 and b_1 that we can consider the case $b_0 = 1$ without any loss of generality. Hence, (4.8) becomes:

$$b_1 y + 1 \leq \beta(y) \leq b_2 y + 1. \tag{4.9}$$

We refer to Nundloll et al. (2010a) for the study of $b_0 = 0$.

Remark: *The previous hypotheses for the basic model on the trophic responses, i.e. Hypotheses (H2*)-(H3*), now hold for the composite argument* $\frac{\alpha(x)}{\beta(y)}$. *Together with the sector conditions (4.7)-(4.9), these hypotheses imply that there is no predation in the absence of pests. Predation becomes immediately positive upon a pest invasion, then remains positive for all positive pest population. The numerical response of the predators is on its part only assumed to be non-negative in the presence of pests.* ■

In our analysis of the zero-pest solution of Model $\mathcal{M}_b$, we will make use of the two biologically sensible assumptions concerning the pest growth rate. We list them as Hypotheses (H5)-(H6) below:

Specific hypotheses

Limited pest growth:

$$\exists\, \bar{x} \text{ such that } \forall\, x \geq \bar{x}, \quad f(x) < 0; \tag{H5}$$

Specific notation

Limited per capita pest growth:

$$f(x)/x \text{ is upper bounded } \forall\, x \geq 0, \text{such that } S = \sup_{x \geq 0}(f(x)/x); \tag{H6}$$

Hypothesis (H5) implies that the population cannot be sustained beyond $\bar{x}$. This can be because the resources available to the pest population are limited, or alternatively, that the pest population's ability to exploit the resources (notably, the crop) available to them is limited. This is the basic characteristic of logistic and Gompertz growth (see e.g. Murray (1989)). So when the population is above $\bar{x}$, it decreases back under $\bar{x}$. In fact, we express this behaviour of the system mathematically by Proposition 3.

The notation (H6) simply highlights that the pest growth rate per pest individual $f(x)/x$ cannot be infinite at any density x. This is a directly due to the local Lipschitz continuity of the pest growth function.

We note that with Hypotheses (H1)-(H6) and conditions (4.7)-(4.9), ($\mathcal{M}_b$) is a positive system. Its variables stay in the non-negative orthant if initiated therein. We refer the reader to Theorem 1 in Chapter 2 for a formal mathematical explanation .

Sector conditions

The use of sector conditions caters for a nonlinear parameterisation of the trophic responses. Figure 4.2 shows three examples of nonlinear $\alpha(x)$ and $\beta(y)$ functions bounded within a sector defined as in conditions (4.7) and (4.9). The Lipschitz square-like fluctuations depicted for both functions provide a rather extreme example for the purpose of our analysis: we do not expect them to occur in nature.

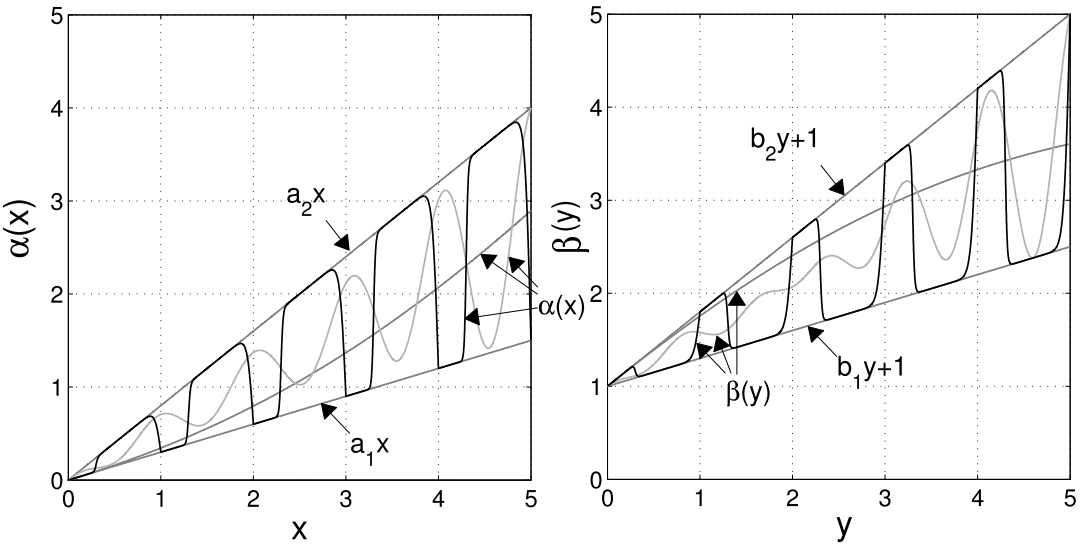

Figure 4.2: Sector conditions (4.7)(left figure) and (4.9)(right figure) bound nonlinearities in the $\alpha(x)$ and $\beta(y)$ parameters. The square variations probably do not occur in nature but are used as a textbook example for the purpose of our analysis.

To explain the advantages of this formulation, it makes more sense biologically to consider that the trophic responses are increasing functions of their argument, that is:

$$\forall \ s > 0, \quad g'(s) > 0 \ \text{ and } \ h'(s) > 0. \tag{H6}$$

There are two reasons why we feel that our description is more exhaustive than Buffoni et al. (2005)'s. The first is that the degree of interference may fluctuate at a given population size. This may be due to some climate forcing or seasonal patterns: during reproductive periods, the stress for territorial defence may be higher. Pheromones would then be emitted in higher quantities, which means that predation would be more heavily penalised - this for the same number of predators. Denoting $\tilde{g}\left(\frac{\alpha(x)}{\beta(y)}, t\right)$ the seasonally forced functional response of the predators, provided there exist sectors that lower and upper bound this function, that is, provided there exist a_1, a_2, b_1 and b_2 such that for all time t:

$$g\left(\frac{a_1 x}{b_2 y + 1}\right) \leq \tilde{g}\left(\frac{\alpha(x)}{\beta(y)}; t\right) \leq g\left(\frac{a_2 x}{b_1 y + 1}\right).$$

Then, the sector formulation, and thus the results we derive next, would also encompass such time dependent functions.

Fluctuations may also be due to momentary decreases of interfering behaviour due to cooperation (among predators) in searching for prey. Interference could intensify only during prey handling. The exact behaviour of the predators (and the prey individuals) can vary at for a given predator population size. The sectors bound this variation.

Secondly, the nonlinearity may provide a more precise way for the trophic response to capture local effects, which occur over short ranges of predator and prey population sizes. When the functions $\alpha(x)$ and $\beta(y)$ are highly nonlinear, they modulate in turn the trophic response with respect to the pest and predator populations. For instance, a dip in the $\beta(y)$ function can be due to intrapredator cooperation, which dominates (locally) interference. Many species of arthropods attack their prey in small groups especially if the prey is too large compared to their size. Similarly, fluctuations in the mapping $\alpha(x)$ can describe aggregative effects of pest

individuals which may either facilitate predation (groups of prey are more easily located than solitary prey individuals by predators, see e.g. Krause and Ruxton (2002); Saito (1997)) or antipredator behaviour (webs of spider mite colonies of critical sizes protect them from their predators, see e.g. Mori and Saito (2004); Saito (1997)).

Simply put, the mappings $\alpha(x)$ and $\beta(y)$ capture the details, while the sectors focus on the general trends. It is the general trends that motivate our model. The trophic functions argument follows a generally increasing trend with respect to x as given by $\alpha(x)$, so under Hypothesis (H6) the trophic uptake generally tends to increase with x. Conversely, increasing predator numbers penalises this argument according to the strength of intraspecific competition given by $\beta(y)$, so that it generally tends to decrease with y. These two characteristics of the functional response are typical of the type of interference we consider in this chapter. Knowing the upper and lower bounds allow us to refine our result, namely to obtain limits of stability and instability of the periodic zero-pest solution.

This phenomenological description aims to minimise the risks of modelling artefacts linked to the use of specific forms for the modelling of the interference, while giving enough information on the general properties of the system. It provides sufficient detail for realistic interpretations of the results and the formulation of practical guidelines.

Remark: *We note here that if the trophic responses are Type II Holling curves, that is $g(s)$ is of the form $\frac{s}{s+1}$, and if $\alpha(x)$ and $\beta(y)$ are affine, that is $a_1 = a_2$ in (4.7), $b_1 = b_2$ in (4.9), one recovers a classical Beddington-DeAngelis form. Indeed:*

$$g\left(\frac{\alpha(x)}{\beta(y)}\right) = \frac{\frac{ax}{1+by}}{\frac{ax}{1+by}+1} = \frac{ax}{ax+by+1}$$

Figure 4.3 shows the effect of varying the functions α and β with respect to x and y and the consequences on the trophic responses. ■

Absolute stability

The use of sector conditions is common in the control engineering and neural networks literature: as we showed, these conditions bound nonlinearities between two linear functions. The stability results obtained for this kind of formulation are termed **absolute stability**, that is the considered system is stable **for any nonlinearity** that satisfies the sector conditions. We refer the reader to Luré and Postnikov (1944) for the founding paper, Liberzon (2006) for a review, Vidyasagar (1993); Khalil (2002) for textbook presentations and, finally, Duan et al. (2008) for current research on the topic.

4.3 The zero-pest solution

Model ($\mathcal{M}_b$) has a zero pest solution which we express in Proposition 2 below.

Proposition 2 (Existence, intrapredatory competition) *Let Hypotheses H5-H6 and conditions (4.7)–(4.9) be satisfied. Then,*

$$(x_p(t), y_p(t)) = (0, y^* e^{-m(t \mod T)}) \tag{4.11a}$$

where

$$y^* = \frac{\mu T}{1 - e^{-mT}} \tag{4.11b}$$

is a periodic solution of model ($\mathcal{M}_b$).

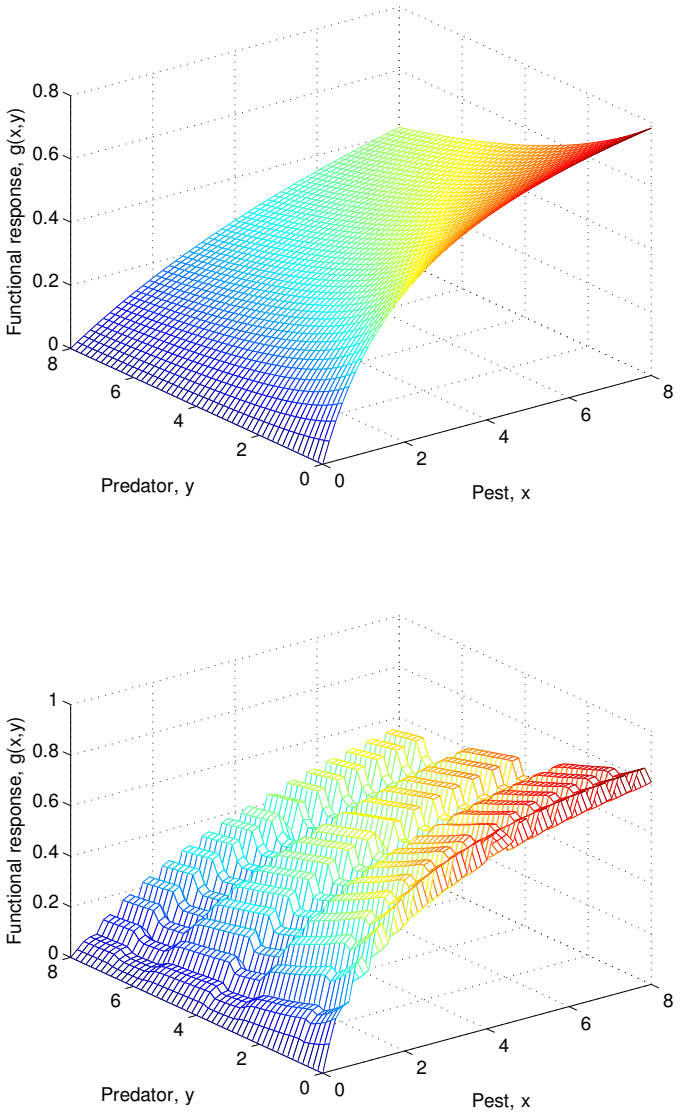

Figure 4.3: Nonlinear parameters distort the functional response. The two figures are plots of the a Holling Type II function $g(s) = \frac{s}{s+1}$, where $s = \frac{\alpha(x)}{\beta(y)}$ in order to incorporate predator dependence. In the figure at the top, $\alpha(x)$ and $\beta(y)$ are linear and affine respectively. In the figure at the bottom $\alpha(x)$ is sill linear and $\beta(y)$ follows the Lipchitz square like function depicted on figure 4.2 (right).

The reader is referred to the proof of Proposition 1 in Chapter 3, which is similar.

We treat separately the case when the parameterisation of the trophic responses is affine (as in Buffoni et al. (2005)), and the case when it is nonlinear (which requires the use of sector conditions).

Before presenting the results of our analyses, we state two preliminary propositions on pest growth and on the functional response which will be useful in our calculations.

Proposition 3 (Limited pest growth) *Let Hypotheses (H1)–(H6) and Conditions (4.7)–(4.9) be satisfied. Then there exists* $t_f(x_0) \geq 0$ *such that* $\frac{\alpha(x(t))}{\beta(y(t))} \in [0, a_2\bar{x}]$ *for* $t \geq t_f(x_0)$.

Proof: First suppose $x_0 \leq \bar{x}$. Since $f(\bar{x}) < 0$, $\dot{x}(x = \bar{x}) < 0$. Therefore, $\forall\ t \geq 0$, $x(t, x_0) \leq \bar{x}$. Next, suppose $x_0 > \bar{x}$. $g(.) \geq 0$ for $x, y \geq 0$ and $f(x) < 0$ for $x \geq \bar{x}$, $\dot{x} = f(x) - g(.)y \leq \max_{\bar{x} \leq x \leq x_0} f(x)$. This is a negative constant, so that x decreases into $x \in [0, \bar{x}]$ in finite time $t_f(x_0)$. This implies that $\frac{\alpha(x)}{\beta(y)} \leq \frac{a_2\bar{x}}{1} \in [0, a_2\bar{x}]$ for $t \geq t_f(x_0)$. □

Proposition 4 (Lower bound on the functional response) *Let* $\sigma(s, q) = g'(0)\frac{qs}{q+s}$ *where* $q > 0$. *Then for all* $K > 0$, *there exists* q *such that* $\forall s \in [0, K]$, $\sigma(s, q) \leq g(s)$, *and* $\sigma'(0, q) = g'(0)$.

Proof: The proof for the existence of $\sigma(s, q)$ is constructed in two parts on the interval $[0, K]$. We note that

$$\sigma'(0, q) = g'(0)$$

Let $q^* > 0$ be chosen such that

$$\sigma''(0, q^*) = \frac{-2g'(0)}{q^*} < g''(0)$$

Then

$$\begin{aligned} \sigma(s, q^*) - g(s) &= \sigma(0, q^*) + \sigma'(0, q^*)s + \frac{\sigma''(0, q^*)}{2}s^2 \\ &\quad -g(0) - g'(0)s - \frac{g''(0)}{2}s^2 + O(s^3) \\ &= \frac{s^2}{2}(\sigma''(0, q^*) - g''(0)) + O(s^3) \end{aligned}$$

Therefore for the chosen q^* there exists $\epsilon > 0$ such that $\forall\ s \in [0, \epsilon)$, $\sigma(s, q^*) - g(s) \leq 0$. Furthermore, $\sigma(s, q)$ is an increasing function in q so that $\forall s \in [0, \epsilon)$, $\forall q \in (0, q^*]$, $\sigma(s, q) \leq g(s)$. Next, we consider the interval $s \in [\epsilon, K]$. Here, since for any given s, $\lim_{q \to 0^+} \sigma(s, q) = 0$, it suffices to choose a sufficiently small $q \leq q^*$ such that

$$\max_{s \in [\epsilon, K]} \sigma(s, q) \leq \min_{s \in [\epsilon, K]} g(s)$$

and thus

$$\sigma(s, q) \leq g(s)\ \ \forall s \in [0, K]$$

□

Figure 4.4 illustrates Proposition 4, in particular how the lower bound behaves with varying q: for any $g(.)$ it suffices to decrease sufficiently q to obtain a $\sigma(s, q)$ that satisfies Proposition 4.

We also introduce the following notation for the bounds on the predator release rates:

$$\underline{\mu}(Q, \lambda, T) = Q\left(\frac{1 - e^{-\lambda m T}}{e^{-\lambda m T} - e^{-mT}}\right)\left(\frac{1 - e^{-mT}}{T}\right). \tag{4.12}$$

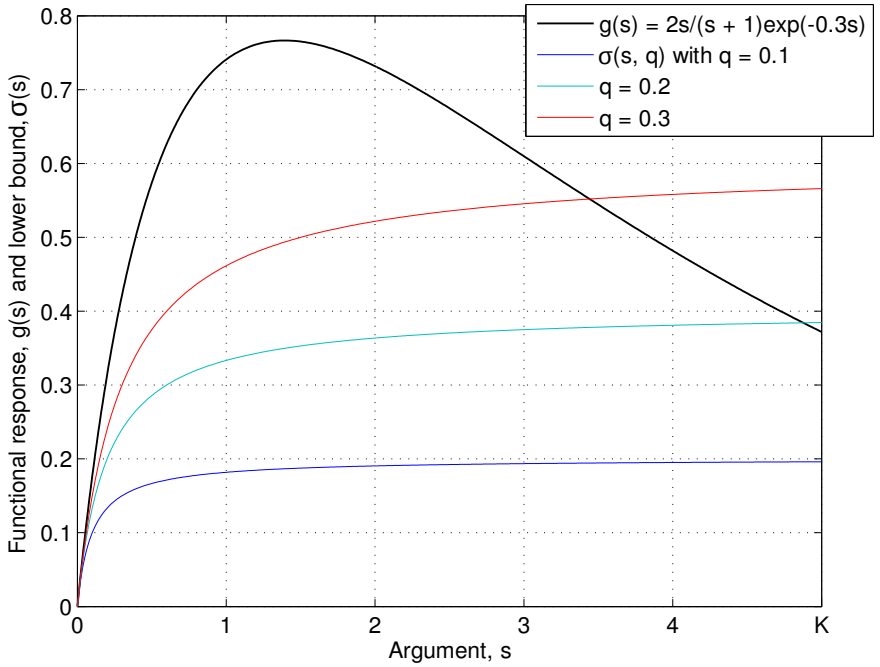

Figure 4.4: The parameter q determines the deceleration of the lower bound σ with respect to s. The smaller the q the larger the deceleration, and σ is able to provide a lower bound.

When $\lambda < 1$ and $Q > 0$, we note that

$$\frac{\partial \underline{\mu}}{\partial Q} = \frac{(1 - e^{-\lambda m T})(1 - e^{-mT})}{(e^{-\lambda mT} - e^{-mT})T} > 0, \tag{4.13}$$

and

$$\frac{\partial \underline{\mu}}{\partial \lambda} = \frac{Q(1 - e^{-mT})^2 mT e^{-\lambda mT}}{T(e^{-\lambda mT} - e^{-mT})^2} > 0. \tag{4.14}$$

4.3.1 Stability with affine parameterisation

In the affine case, the functions read $\alpha(x) = ax$ and $\beta(y) = by + 1$, where a, b are positive constants. This formulation corresponds to that of Buffoni et al. (2005) for density-dependent trophic responses; our analysis extends his work to the case of impulsive biological control. It is a generalisation of the Beddington-DeAngelis trophic response. The results of this section therefore encompass those obtained by Zhang and Chen (2006) and Nundloll et al. (2010b) on biological control with explicit Beddington-DeAngelis functions.

The stability conditions are described by the following theorem.

Theorem 4 (Stability, affine parameterisation) *The solution* $(0, y_p(t))$ *of* $(\mathcal{M}_b)$ *is LAS if and only if*

$$f'(0) < \frac{ag'(0)}{b} \tag{4.15a}$$

and

$$\mu > \frac{1}{b}\left(\frac{1 - e^{-m\frac{bf'(0)}{ag'(0)}T}}{e^{-m\frac{bf'(0)}{ag'(0)}T} - e^{-mT}}\right)\left(\frac{1 - e^{-mT}}{T}\right) = \underline{\mu}\left(\frac{1}{b}, \frac{bf'(0)}{ag'(0)}, T\right) \tag{4.15b}$$

Furthermore, let q be chosen as in Proposition 4 with $K = a\bar{x}$. Then, the solution is GAS if

$$S < \frac{ag'(0)}{b} \tag{4.15c}$$

and

$$\mu > \frac{q + a\bar{x}}{qb}\left(\frac{1 - e^{-m\frac{bS}{ag'(0)}T}}{e^{-m\frac{bS}{ag'(0)}T} - e^{-mT}}\right)\left(\frac{1 - e^{-mT}}{T}\right) = \underline{\mu}\left(\frac{q + a\bar{x}}{qb}, \frac{bS}{ag'(0)}, T\right) \tag{4.15d}$$

Proof: For convenience, we start by calculating the local stability conditions. We apply directly Theorem 1 from Lakmeche and Arino (2000), as described in Chapter 3 of this thesis. We obtain as necessary and sufficient conditions:

$$e^{-m\int_0^T d\tau} < 1$$

and

$$e^{\int_0^T \left[f'(0) - \frac{g'(0)\alpha'(0)y_p(\tau)}{\beta(y_p(\tau))}\right]d\tau} < 1.$$

The former is trivial while the latter yields

$$\int_0^T \frac{\alpha'(0)y_p(\tau)}{\beta(y_p(\tau))}d\tau > \frac{f'(0)}{g'(0)}T. \tag{4.16}$$

We note that in this equation $\alpha'(0) = a$ and $\beta(y_p(\tau)) = by^*e^{-m\tau} + 1$, so we can rewrite it as:

$$\int_0^T \frac{ay^*e^{-m\tau}}{by^*e^{-m\tau} + 1}d\tau > \frac{f'(0)}{g'(0)}T.$$

On evaluating the integral and rearranging, we obtain:

$$\begin{aligned} -\frac{a}{mb}\ln\left(\frac{by^*e^{-mT} + 1}{by^* + 1}\right) &> \frac{f'(0)}{g'(0)}T, \\ \Leftrightarrow by^*e^{-mT} + 1 &> by^*e^{-m\frac{bf'(0)}{ag'(0)}T} + e^{-m\frac{bf'(0)}{ag'(0)}T}, \\ \Leftrightarrow \left(e^{-m\frac{bf'(0)}{ag'(0)}T} - e^{-mT}\right)by^* &> \left(1 - e^{-m\frac{bf'(0)}{ag'(0)}T}\right). \end{aligned} \tag{4.17}$$

The right-hand side of the above inequality is positive, which requires that the left-hand side be too. Thus for the stability condition to hold we need that:

$$e^{-m\frac{bf'(0)}{ag'(0)}T} - e^{-mT} > 0,$$

or (on rearranging) that:

$$\begin{aligned} -m\frac{bf'(0)}{ag'(0)}T &> -mT, \\ \Leftrightarrow f'(0) &< \frac{ag'(0)}{b}. \end{aligned}$$

This is the necessary condition (4.15a). The inequality (4.17) can be rewritten as:

$$y^* > \frac{1}{b}\left(\frac{1 - e^{-m\frac{bf'(0)}{ag'(0)}T}}{e^{-m\frac{bf'(0)}{ag'(0)}T} - e^{-mT}}\right). \tag{4.18}$$

Since from Equation (4.11b), $y^* = \dfrac{\mu T}{1-e^{-mT}}$, we obtain, by substitution in (4.18):

$$\mu > \frac{1}{b}\left(\frac{1-e^{-m\frac{bf'(0)}{ag'(0)}T}}{e^{-m\frac{bf'(0)}{ag'(0)}T}-e^{-mT}}\right)\left(\frac{1-e^{-mT}}{T}\right),$$

which is condition (4.15b).

As in Chapter 3, to establish the condition for global stability of Solution (4.11a), we define a new system of coordinates $\tilde{x} = x - x_p$ and $\tilde{y} = y - y_p$ which describes the departure from the pest-free solution. This gives the equivalent system:

$$\begin{cases} \dot{\tilde{x}} = f(x) - g(s)y = f(\tilde{x}) - g(s)(\tilde{y}+y_p) \\ \dot{\tilde{y}} = h(s)y - my + my_p = h(s)(\tilde{y}+y_p) - m\tilde{y} \end{cases} \tag{4.19}$$

where $s = \frac{\alpha(x)}{\beta(y)} = \frac{a\tilde{x}}{b(\tilde{y}+y_p)+1}$. The impulsive component vanishes.

Let the initial conditions be $(\tilde{x}_0, \tilde{y}_0)$ at $t = t_0$. Integrating $\frac{\dot{\tilde{x}}}{\tilde{x}}$, we get

$$\int_{\tilde{x}_0}^{\tilde{x}(t)} \frac{1}{u}du = \int_0^t \left[\frac{f(\tilde{x})}{\tilde{x}} - \frac{g(s)}{\tilde{x}}(\tilde{y}+y_p)\right]d\tau$$

Similarly to Chapter 3, to show that $\lim_{t\to+\infty}\tilde{x} = 0$, we demonstrate the divergence of $\int_{\tilde{x}_0}^{\tilde{x}(t)}\frac{1}{u}du$ to $-\infty$ as $t \to +\infty$. For t large enough, we can write:

$$\int_{\tilde{x}_0}^{\tilde{x}(t)} \frac{1}{u}du = \left(\int_{t_0}^{\left(\lfloor\frac{t_f}{T}\rfloor+1\right)T} + \int_{\left(\lfloor\frac{t_f}{T}\rfloor+1\right)T}^{\lfloor\frac{t}{T}\rfloor T} + \int_{\lfloor\frac{t}{T}\rfloor T}^{t}\right)\left[\frac{f(\tilde{x})}{\tilde{x}} - \frac{g(s)}{\tilde{x}}(\tilde{y}+y_p)\right]d\tau, \tag{4.20}$$

with t_f defined as in Proposition 3. The first and third terms in (4.20) are bounded for all t. The second integral, however, is not necessarily so. More specifically, we need for stability that:

$$\lim_{t\to+\infty}\int_{\left(\lfloor\frac{t_f}{T}\rfloor+1\right)T}^{\lfloor\frac{t}{T}\rfloor T}\left[\frac{f(\tilde{x})}{\tilde{x}} - \frac{g(s)}{\tilde{x}}(\tilde{y}+y_p)(\tau)\right] = -\infty \tag{4.21}$$

At this point it is useful to note that from Proposition 4, we can write

$$\frac{g(s)}{\tilde{x}}(\tilde{y}+y_p) \geq \frac{\sigma(s,q)}{\tilde{x}}(\tilde{y}+y_p) = \frac{qg'(0)a(\tilde{y}+y_p)}{qb(\tilde{y}+y_p)+q+a\tilde{x}}$$

which is an increasing function of $(\tilde{y}+y_p)$ and a decreasing one of $\tilde{x}$.

Because of the positivity of the system (and recalling the inequality (3.21)), y can be lower bounded as:

$$y_p(t) + \tilde{y}(t) \geq y^*(e^{-m(t \mod T)} - e^{-m(t-t_0)}).$$

From the proof of Proposition 3, we also note that $\tilde{x} \in [0, \bar{x}]$, as t is sufficiently large.

Therefore, from these two bounds, we can write

$$\frac{g(s)}{\tilde{x}}(\tilde{y}+y_p) \geq \frac{ag'(0)qy^*(e^{-m(t \mod T)} - e^{-m(t-t_0)})}{qby^*(e^{-m(t \mod T)} - e^{-m(t-t_0)}) + q + a\bar{x}} \tag{4.22}$$

Recall from Hypothesis (H6) that $S = \sup_{x\geq 0}(f(x)/x)$. So, from this property and equation (4.22) we can upper bound the second term of the inequality (4.20) as

$$\int_{\left(\lfloor\frac{t_f}{T}\rfloor+1\right)T}^{\lfloor\frac{t}{T}\rfloor T}\left[\frac{f(\tilde{x})}{\tilde{x}} - \frac{g(s)}{\tilde{x}}(\tilde{y}+y_p)(\tau)\right]d\tau \leq \sum_{n=\lfloor\frac{t_f}{T}\rfloor+1}^{\lfloor\frac{t}{T}\rfloor-1} I(n) \tag{4.23}$$

where

$$\begin{aligned} I(n) &= \int_{nT}^{(n+1)T} \left[S + \frac{ag'(0)}{bm} \frac{mqby^* e^{-m(\tau-nT)}(1-e^{-m(nT-t_0)})}{qby^* e^{-m(\tau-nT)}(1-e^{-m(nT-t_0)}) + q + a\bar{x}} \right] d\tau \\ &= ST + \frac{ag'(0)}{bm} \left[\ln \left(qby^* e^{-m(\tau-nT)}(1-e^{-m(nT-t_0)}) + q + a\bar{x} \right) \right]_{nT}^{(n+1)T} \\ &= ST + \frac{ag'(0)}{bm} \ln \left(\frac{qby^* e^{-mT}\left(1-e^{-m(nT-t_0)}\right) + q + a\bar{x}}{qby^*\left(1-e^{-m(nT-t_0)}\right) + q + a\bar{x}} \right). \end{aligned}$$

To satisfy (4.21), knowing (4.23), it would suffice that:

$$\lim_{t\to+\infty} \sum_{n=\lfloor \frac{t_f}{T} \rfloor + 1}^{\lfloor \frac{t}{T} \rfloor - 1} I(n) = -\infty. \tag{4.24}$$

Two properties of the function $I(n)$ suggest that this condition may be satisfied. First, $I(n)$ is decreasing in n: the argument of the logarithm is a fraction of two affine functions of $\left(1-e^{-m(nT-t_0)}\right)$ with the same intercept. The numerator affine function has a smaller slope than the denominator affine function, thus the argument of the logarithm is decreasing in $\left(1-e^{-m(nT-t_0)}\right)$, which is by itself increasing in n. Thus $I(n)$ is decreasing in n.

Second, it has a limit when $n \to +\infty$ exists[1]; we calculate it as:

$$\begin{aligned} \lim_{n\to+\infty} I(n) &= ST + \frac{ag'(0)}{bm} \lim_{n\to+\infty} \ln \left(\frac{qby^* e^{-mT}(1-e^{-m(nT-t_0)}) + q + a\bar{x}}{qby^*(1-e^{-m(nT-t_0)}) + q + a\bar{x}} \right) \\ &= ST + \frac{ag'(0)}{bm} \ln \left(\frac{qby^* e^{-mT} + q + a\bar{x}}{qby^* + q + a\bar{x}} \right) \end{aligned}$$

Based on these two properties, in order for Equation (4.24) to be satisfied, we require that

$$\lim_{n\to\infty} I(n) < 0,$$

that is:

$$ST + \frac{ag'(0)}{bm} \ln \left(\frac{qby^* e^{-mT} + q + a\bar{x}}{qby^* + q + a\bar{x}} \right) < 0.$$

Rearranging the above inequality yields

$$(e^{-m\frac{bS}{ag'(0)}T} - e^{-mT}) qby^* > (q + a\bar{x}) \left(1 - e^{-m\frac{bS}{ag'(0)}T}\right) \tag{4.25}$$

As previously for inequality (4.17), because the right-hand side of (4.25) is positive, we need that

$$(e^{-m\frac{bS}{ag'(0)}T} - e^{-mT}) > 0.$$

which gives the first condition (4.15c). Finally, recalling (4.11b) and rearranging (4.25) so that μ is the subject of formula yields

$$\mu > \frac{q + a\bar{x}}{qb} \left(\frac{1 - e^{-m\frac{bS}{ag'(0)}T}}{e^{-m\frac{bS}{ag'(0)}T} - e^{-mT}} \right) \left(\frac{1 - e^{-mT}}{T} \right)$$

which is the GAS condition (4.15d). Proving the convergence of $\tilde{y}$ to zero as well is straightforward and similar to the one in Chapter 3.

[1] This limit can be taken because the log function and hence $I(n)$ is continuous in n.

We notice that the LAS and GAS conditions μ have a similar form. Using the notation (4.12), we can rewrite the LAS condition (4.15b) and the GAS condition (4.15d) as:

$$\mu > \underline{\mu}\left(\frac{1}{b}, \frac{bf'(0)}{ag'(0)}, T\right) \tag{4.26}$$

and

$$\mu > \underline{\mu}\left(\frac{q+a\bar{x}}{qb}, \frac{bS}{ag'(0)}, T\right) \tag{4.27}$$

respectively. We recall that $\underline{\mu}(Q, \lambda, T)$ is increasing in Q and λ. Since $\frac{q+a\bar{x}}{qb} > \frac{1}{b}$, and $\frac{bS}{ag'(0)} > \frac{bf'(0)}{ag'(0)}$, the lower bound in (4.15b) is smaller than the lower bound in (4.15d). This means that when condition (4.15d) is satisfied, the solution is also locally asymptotically stable. It also implies that when Condition (4.15d) holds, the solution is globally asymptotically stable. □

The calculations for the affine case serve as a basis for the analysis when α and β are nonlinear.

The proof on affine α and β yields two types of conditions for the stability of the pest-free solution: The first concerns the biological processes and species behaviour (Conditions (4.15a) and (4.15c)). We will refer to these conditions as the *biological* conditions. When expressed in terms of b, as:

$$b < \frac{f'(0)}{ag'(0)} \quad \text{and} \quad b < \frac{S}{ag'(0)},$$

this condition informs the selection of the predator species.

The second set of conditions concerning the minimal release rate is managerial (Conditions (4.15b) and (4.15d)). Because of the minimal release rate's dependence in the release period T, these conditions can be analysed further to identify the effect of various release strategies on the minimal release rate. We discuss this shortly in the next section.

Finally, we note that the *managerial* conditions exists *only if* the biological condition is satisfied. The local biological condition means that if the predators interfere too much with their conspecifics, no control will work even if the invasion is negligible.

4.3.2 Stability with nonlinear parameterisation

When the α and β functions are nonlinear, we bound them within sectors given by (4.7) and (4.9).

As previously, the non-trivial local stability condition is obtained as (4.16), that is

$$\int_0^T \frac{\alpha'(0)y_p(\tau)}{\beta(y_p(\tau))}d\tau > \frac{f'(0)}{g'(0)}T$$

Due to the sector conditions, the left-hand side of the inequality is bounded as:

$$\int_0^T \frac{a_1 y_p(\tau)}{b_2 y_p(\tau)+1}d\tau \leq \int_0^T \frac{\alpha'(0)y_p(\tau)}{\beta(y_p(\tau))}d\tau \leq \int_0^T \frac{a_2 y_p(\tau)}{b_1 y_p(\tau)+1}d\tau \tag{4.28}$$

Therefore, for *absolute stability*, it is clear that considering the lower bound in (4.28) will yield a sufficient condition for local stabiliy; that is, only the lower bound of α and the upper bound of β are of interest. This gives

$$\int_0^T \frac{a_1 y_p(\tau)}{b_2 y_p(\tau)+1}d\tau > \frac{f'(0)}{g'(0)}T$$

The analysis for the local stability of (4.11a) follows as in the previous subsection except that a and b are now replaced by a_1 and b_2.

The procedure to find the global stability condition is also similar to the linear case. The key point to note is that the σ-function from Proposition 4 is bounded as

$$\sigma(s,q) \geq g'(0)\frac{qa_1x}{qb_2y+q+a_1x} \tag{4.29}$$

provided q is chosen appropriately to satisfy Proposition 4, with $K = a_2\bar{x}$. The lower bound on σ as given in (4.29) is sure to lower bound $g(s)$. The analysis for global stability therefore follows as in the linear section, except that a_1 and b_2 are substituted in for a and b respectively. As for the local stability, due to the sector conditions (4.7) and (4.9), we thus obtain only sufficient conditions.

Theorem 5 summarises the conditions for the nonlinear parameterised system.

Theorem 5 (Stability, nonlinear parameterisation) *Let q be chosen as in Proposition 4 with $K = a_2\bar{x}$. The solution $(0, y_p(t))$ of ($\mathcal{M}_b$) is LAS if*

$$f'(0) < \frac{a_1g'(0)}{b_2} \tag{4.30a}$$

and

$$\mu > \underline{\mu}\left(\frac{1}{b_2}, \frac{b_2f'(0)}{a_1g'(0)}, T\right) \tag{4.30b}$$

Furthermore, the solution is GAS if

$$S < \frac{a_1g'(0)}{b_2} \tag{4.30c}$$

and

$$\mu > \underline{\mu}\left(\frac{q+a_1\bar{x}}{qb_2}, \frac{b_2S}{a_1g'(0)}, T\right) \tag{4.30d}$$

We note that the minimal rates stated above are only sufficient conditions. Due to the sector conditions, a given μ value below the minimal release rate at a given T value does not necessarily result in an unstable pest-free solution. The instability condition is in fact given by the upper bound of the α function and the lower one of the β function as follows.

Proposition 5 (Instability, nonlinear parameterisation) *The solution $(0, y_p(t))$ of system ($\mathcal{M}_b$) is unstable if*

$$f'(0) \geq \frac{a_2g'(0)}{b_1} \tag{4.31a}$$

or if

$$\mu < \underline{\mu}\left(\frac{1}{b_1}, \frac{b_1f'(0)}{a_2g'(0)}, T\right) \tag{4.31b}$$

Proof: The conditions for local instability obtained from Lakmeche and Arino (2000) are $e^{-m\int_0^T d\tau} > 1$ or

$$e^{\int_0^T\left[f'(0)-\frac{g'(0)\alpha'(0)y_p(\tau)}{\beta(y_p(\tau))}\right]d\tau} > 1.$$

The former is physically impossible. After some computations, the latter however can be written as

$$\int_0^T \frac{\alpha'(0)y_p(\tau)}{\beta(y_p(\tau))}d\tau < \frac{f'(0)}{g'(0)}T$$

Recalling (4.28), this instability condition is necessarily satisfied if

$$\int_0^T \frac{a_2 y_p(\tau)}{b_1 y_p(\tau)+1} d\tau < \frac{f'(0)}{g'(0)} T$$
$$\Leftrightarrow \quad \left(e^{-m\frac{b_1 f'(0)}{a_2 g'(0)}T} - e^{-mT}\right) b_1 y^* < \left(1 - e^{-m\frac{b_1 f'(0)}{a_2 g'(0)}T}\right) \tag{4.32}$$

We note at this point that if (4.31a) holds, the left-hand side of inequality (4.32) is negative or zero while the right-hand side is positive, so that (4.32) is satisfied. Otherwise, we obtain, on rearranging the inequality (4.32),

$$\mu < \frac{1}{b_1}\left(\frac{1 - e^{-m\frac{b_1 f'(0)}{a_2 g'(0)}T}}{e^{-m\frac{b_1 f'(0)}{a_2 g'(0)}T} - e^{-mT}}\right)\left(\frac{1-e^{-mT}}{T}\right)$$

which is condition (4.31b). □

We obtain similar conditions (a biological and a managerial condition) in the case when parameterisation is nonlinear. When the parameterisation of α and β is affine however, the definition of the minimal rate, that is the local stability condition on μ, is a necessary and sufficient condition. In the nonlinear case, since we only have sufficient conditions for stability and instability, when

$$\underline{\mu}\left(\frac{1}{b_1}, \frac{b_1 f'(0)}{a_2 g'(0)}, T\right) < \mu < \underline{\mu}\left(\frac{1}{b_2}, \frac{b_2 f'(0)}{a_1 g'(0)}, T\right) \tag{4.33}$$

no conclusion can be drawn for the local stability of the pest-free solution.

4.4 The minimal predator release rate

We investigate the effects of the biological processes and the release period T on the minimal release rate, first when the parameterisation is affine, then when it is nonlinear. The expression for the minimal release rate required for the eradication of pests is obtained from the stability analysis presented previously.

4.4.1 Affine parameterisation

Dependence in biological processes

The product term $ag'(0)$ can be interpreted as the 'effective' marginal predation at zero pest. It represents the attack rate of the predators, the coefficient a specifies this rate *with respect to the prey species*. We can consider a as an index of the prey's exposure or vulnerability.

The dependence of the minimal release rate $\underline{\mu}$ for pest eradication with respect to the term $ag'(0)$ is found to be monotonic decreasing: recall that λ in both the LAS and GAS conditions (given in (4.26) and (4.27) respectively) is inversely proportional to $ag'(0)$:

$$\lambda = \frac{bf'(0)}{ag'(0)} \quad \text{and, } respectively, \quad \lambda = \frac{bS}{ag'(0)}.$$

So $\dfrac{\partial \lambda}{\partial (ag'(0))} < 0$. Since from (4.14) $\dfrac{\partial \underline{\mu}}{\partial \lambda} > 0$, we have:

$$\frac{\partial \underline{\mu}}{\partial (ag'(0))} = \frac{\partial \underline{\mu}}{\partial \lambda}\frac{\partial \lambda}{\partial (ag'(0))} < 0.$$

Hence a higher attack rate (or 'effective' marginal predation at zero pest) of the predators lowers the release rate required for pest elimination.

Analysing the influence of the interference strength b requires a few more computations. We proceed by inspecting the inequality (4.16). When the parameterisation of $\alpha(x)$ and $\beta(y)$ is affine, we can write this inequality as:

$$\int_0^T \frac{ay^* e^{-m(\tau \mod T)}}{by^* e^{-m(\tau \mod T)} + 1} d\tau > \frac{f'(0)}{g'(0)} T.$$

where:

$$y^* = \frac{\mu T}{1 - e^{-mT}}$$

We note, that the integral on the left-hand of the inequality is decreasing in the interference strength b. When b is high, y^* needs to be increased in order to compensate for the depression in the integral value due to b, and hence ensure that conditions for the stability of the pest-free solution are satisfied. Since y^* is directly proportional to μ, we can infer that a higher interference strength requires a higher minimal rate for pest eradication.

Dependence in the release period

We also find that the minimal rate is a function of the release period T. When the arguments of the trophic responses are considered to be affine as in subsection 4.3.1, the calculation of $\frac{\partial \underline{\mu}}{\partial T}$ yields the following observation:

Theorem 6 (Dependence in T) *For $0 < \lambda < 1$, $\underline{\mu}(Q, \lambda, T)$ is increasing in T for $T > 0$ and $\lim_{T \to \infty} \underline{\mu}(Q, \lambda, T) = +\infty$.*

The proof is presented below.

Proof: The function can be rewritten as

$$\underline{\mu}(Q, \lambda, T) = Qm \frac{2 \sinh\left(\frac{\lambda m T}{2}\right) \sinh\left(\frac{mT}{2}\right)}{\sinh\left(\frac{(1-\lambda) mT}{2}\right) mT} = Qm \frac{2}{\left(\coth\left(\frac{\lambda m T}{2}\right) - \coth\left(\frac{mT}{2}\right)\right) mT}$$

Its derivative is calculated as

$$\begin{aligned} \frac{d\underline{\mu}}{dT} &= 2Qm^2 \frac{-\left(\coth\left(\frac{\lambda m T}{2}\right) - \coth\left(\frac{mT}{2}\right)\right) + \left(\frac{\frac{\lambda m T}{2}}{\sinh^2\left(\frac{\lambda m T}{2}\right)} - \frac{\frac{mT}{2}}{\sinh^2\left(\frac{mT}{2}\right)}\right)}{m^2 T^2 (\coth\left(\frac{\lambda m T}{2}\right) - \coth\left(\frac{mT}{2}\right))^2} \\ &= \frac{2Q\left(\Gamma\left(\frac{\lambda m T}{2}\right) - \Gamma\left(\frac{mT}{2}\right)\right)}{T^2 (\coth\left(\frac{\lambda m T}{2}\right) - \coth\left(\frac{mT}{2}\right))^2} \end{aligned}$$

where

$$\Gamma(u) = \frac{u}{\sinh^2 u} - \coth u$$

The denominator is positive so the sign of $\frac{d\underline{\mu}}{dT}$ will depend on that of the numerator. We note that

$$\frac{d\Gamma}{du} = \frac{2}{\sinh^2 u}(1 - u \coth u)$$

is negative for all $u \geq 0$. $\Gamma(u)$ is therefore a decreasing function of u. Since $\lambda < 1$, we deduce that

$$\Gamma\left(\frac{\lambda m T}{2}\right) > \Gamma\left(\frac{mT}{2}\right)$$

so that

$$\frac{d\underline{\mu}}{dT} > 0$$

Finally,

$$\lim_{T\to+\infty} \underline{\mu} = \lim_{T\to+\infty} \frac{Q(1-e^{-mT})(1-e^{-\lambda mT})}{(1-e^{-(1-\lambda)mT})} \frac{e^{\lambda mT}}{T} = +\infty$$

□

This result applied to (4.15b) and (4.15d) has two possible interpretations. First, more frequent releases will require a smaller minimal rate to succeed in driving the pest population to zero. Second, a given rate μ^* will work up to a threshold release period T^*, which is defined such that

$$\underline{\mu}\left(\frac{1}{b}, \frac{af'(0)}{bg'(0)}, T^*\right) = \mu^*$$

Beyond this period, as the zero-pest solution becomes unstable, pest invasion occurs.

4.4.2 Nonlinear parameterisation

Dependence in biological processes

When the parameterisation of $\alpha(x)$ and $\beta(y)$ is nonlinear, only general trends in the behaviour of the release rate with respect to the biological processes can be identified. While the precise value of the minimal release rate, which we shall denote by $\tilde{\mu}(T)$, is unknown, we know however that it is bounded as in (4.33). Each of the bounds have the same form as for the affine case: each of the minimal release rate bounds will decrease with $a_i g'(0)$ and increase with b_i. The actual $\tilde{\mu}(T)$ will therefore generally tend to increase with respect to each $a_i g'(0)$ and b_i within a tube defined by these two bounds.

Dependence in the release period

Likewise, each bound is increasing in T. The actual $\tilde{\mu}(T)$ will therefore generally tend to increase with respect to the release period T within a tube defined by these two bounds. As in the affine case, increasing the release period for a given release rate μ^* will at some point destabilise the pest-free solution. Due to the sector formulation, we are able to calculate that the rate μ^* will work up to a release period $\underline{T^*}$, but not beyond a $\overline{T^*} > \underline{T^*}$, defined respectively such that

$$\underline{\mu}\left(\frac{1}{b_2}, \frac{a_1 f'(0)}{b_2 g'(0)}, \underline{T^*}\right) = \mu^* \text{ and } \underline{\mu}\left(\frac{1}{b_1}, \frac{a_2 f'(0)}{b_1 g'(0)}, \overline{T^*}\right) = \mu^*$$

A graphical representation of this result is presented in Figure 4.5. Here we have considered $\alpha(x)$ to be affine and $\beta(y)$ a Lipschitz square-like wave similar to the one given in Figure 4.2. The shaded tube is limited by the bounds resulting from the sector formulation and contains the actual $\tilde{\mu}(T)$ curve for the pair of $\alpha(x)$ and $\beta(y)$ functions considered. Note that in this example the actual $\tilde{\mu}(T)$ coincides with $\underline{\mu}\left(\frac{1}{b_2}, \frac{a_1 f'(0)}{b_2 g'(0)}, T\right)$ for small T and hedges close to $\underline{\mu}\left(\frac{1}{b_1}, \frac{a_2 f'(0)}{b_1 g'(0)}, T\right)$ for intermediate T values. This somewhat validates the quality of the bounds obtained on $\tilde{\mu}(T)$ from the chosen sectors.

4.5 The pest evolution rate

Classical indicators of pest damage are often based on pest population thresholds at a given time moment (economic injury level, economic threshold (Stern et al., 1959), and even aesthetic threshold levels for ornamental crops (Schumacher et al., 2006)).

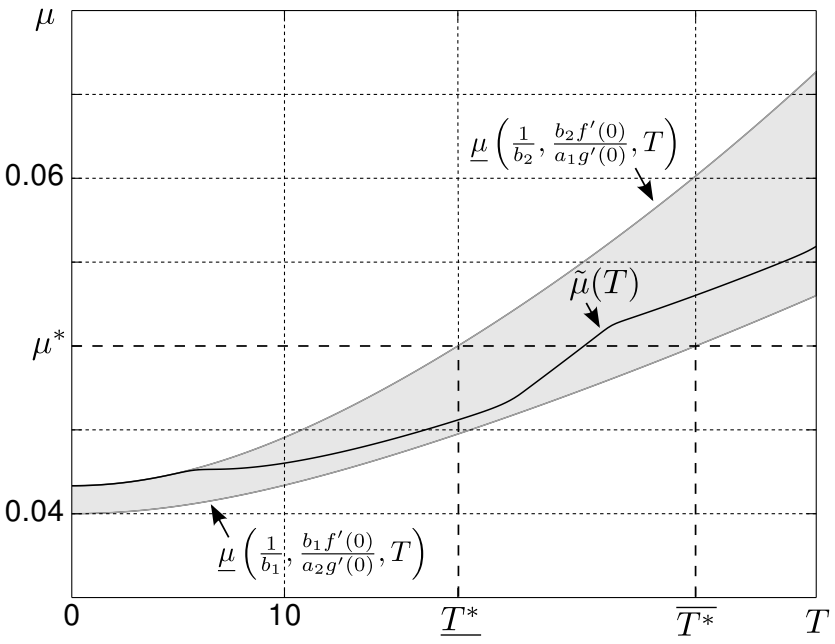

Figure 4.5: The minimal release rate $\tilde{\mu}(T)$ is numerically computed from an affine $\alpha(x)$ and a Lipschitz square-like $\beta(y)$ shown on Figure 4.2. The shaded area is the tube defined by (4.33) to which $\tilde{\mu}(T)$ belongs. The threshold $\underline{T^*}$ (*resp.* $\overline{T^*}$) under which (*resp.* over) the pest-free solution is guaranteed to be locally stable (*resp.* unstable) is also shown for $\mu^* = 0.05$.

However, pest populations cause continuous damage over a period of time and the extent of their impact is the result of *cumulative population effects*, measured in “insect-day" (Ruppel, 1983; Carey, 1993). Pest damage is therefore best captured by the integral of the density of pests over time, rather than by the actual pest population density at some time moment.

So, when designing the biological control program, a way to reduce the damage caused by pests on the crop is to ensure that the pest eradication is as fast as possible. We may define a mathematical expression for the rate of evolution around the pest-free state as follows. At first order in $(x, y - y_p)$, the x-dynamics are

$$\dot{x} = \left(f'(0) - g'(0) \frac{\alpha'(0)}{\beta(y_p(t))} \right) x.$$

Because of the periodicity of y_p in T, we have

$$\forall t \;\; x(t + kT) = x(t) e^{\phi(T) kT},$$

where the exponent

$$\phi(T) \;\; = \;\; \frac{1}{T} \int_0^T \left(f'(0) - g'(0) \frac{\alpha'(0)}{\beta(y_p(\tau))} y_p(\tau) \right) d\tau \tag{4.34}$$

is the mean rate of evolution of x around the pest-free solution $(0, y_p)$.

We note that $\phi < 0$ when the solution is locally stable, so that x converges to zero. For all positive ϕ, x diverges from zero. Likewise, we can say from the sign of ϕ whether x converges

to zero or not: a negative ϕ implies that pests are eliminated, while a positive ϕ implies that the pest population will persist at some positive value.

Furthermore, the mean speed of evolution of x around $(0, y_p)$ depends on the magnitude of ϕ: the larger its magnitude, the higher the speed of evolution (divergence or convergence).

These two points on the sign and magnitude of ϕ are important in the interpretation of the subsequent results.

We note that this evolution rate ϕ is dependent, via y_p, on the release rate μ. It is also dependent on the release period, related to the release program.

4.5.1 Affine parameterisation

Dependence in the release rate

We can identify this dependence by inspection of Equation (4.34). When $\beta(y_p)$ is affine, $\frac{y_p}{\beta(y_p)} = \frac{y_p}{by_p+1}$ increases with y_p and therefore increases with μ. Consequently, the rate ϕ decreases with the predator release rate used. When the pest-free solution is stable, ϕ is negative.

Therefore, as ϕ is more negative when higher predator release rates are implemented, using as high a release rate as possible ensures a faster pest eradication.

Remark: *In practice however, due to financial constraints, this rate has a maximal fixed value, which in turn limits the speed of pest elimination, at a given release period.* ■

Dependence in the release period

Here, for a given release rate μ, the question is how to deploy the predators in time in order to maximise the speed of eradication. When the arguments of the trophic responses are considered to be affine as in subsection 4.3.1, the convergence rate $\phi(T)$ is calculated as

$$\phi(T) \quad = \quad f'(0) + \frac{g'(0)a}{bmT} \ln\left(\frac{by^* e^{-mT} + 1}{by^* + 1}\right) \tag{4.35}$$

In the affine case, the following theorem applies.

Theorem 7 (Rate of evolution, affine parameterisation) *Suppose that local stability conditions are satisfied. Then for a given μ, the average rate of evolution of x to the pest-free solution is an increasing function of the release period.*

Proof: Recalling (4.11b), for a given μ, we can write

$$\phi(T) = f'(0) + \frac{g'(0)a}{bmT} \ln\left(\frac{b\mu T e^{-mT} + 1 - e^{-mT}}{b\mu T + 1 - e^{-mT}}\right) \tag{4.36}$$

This equation can be rearranged with μ as the subject of the formula and expressed using the $\underline{\mu}$-function as follows:

$$\mu = \frac{1}{b}\left(\frac{1 - e^{-\frac{b}{a}\left(\frac{f'(0)-\phi(T)}{g'(0)}\right)mT}}{e^{-\frac{b}{a}\left(\frac{f'(0)-\phi(T)}{g'(0)}\right)mT} - e^{-mT}}\right)\frac{1-e^{-mT}}{T} = \underline{\mu}\left(\frac{1}{b}, \frac{b}{a}\left(\frac{f'(0)-\phi(T)}{g'(0)}\right), T\right)$$

To determine the sign of $\frac{\partial \phi}{\partial T}$, we make use of the chain rule:

$$\frac{\partial \mu}{\partial T} = \frac{\partial \underline{\mu}}{\partial T} + \frac{\partial \underline{\mu}}{\partial \phi}\frac{\partial \phi}{\partial T} \tag{4.37}$$

For a given μ, $\frac{\partial \mu}{\partial T} = 0$. We note that independently of the sign of $\phi(T)$, from its definition in (4.35) and since $by^* > 0$, for $T > 0$

$$\frac{b}{a}\left(\frac{f'(0) - \phi(T)}{g'(0)}\right) = -\frac{1}{mT}\ln\left(\frac{by^* e^{-mT} + 1}{by^* + 1}\right) < -\frac{1}{mT}\ln(e^{-mT}) = 1$$

Furthermore, since $0 < \frac{by^* e^{-mT}+1}{by^*+1} < 1$, we can deduce that

$$-\frac{1}{mT}\ln\left(\frac{by^* e^{-mT} + 1}{by^* + 1}\right) > 0$$

Therefore we can establish that

$$0 < \frac{b}{a}\left(\frac{f'(0) - \phi(T)}{g'(0)}\right) < 1$$

So, we can apply Theorem 6, with $\lambda = \frac{b}{a}\left(\frac{f'(0) - \phi(T)}{g'(0}\right)$, and deduce that $\frac{\partial \mu}{\partial \overline{T}} > 0$. Furthermore

$$\frac{\partial \mu}{\partial \phi} = -\frac{m}{ag'(0)} \frac{(1 - e^{-mT})^2 \, e^{-\frac{b}{a}\left(\frac{f'(0)-\phi(T)}{g'(0)}\right)mT}}{\left(e^{-\frac{b}{a}\left(\frac{f'(0)-\phi(T)}{g'(0)}\right)mT} - e^{-mT}\right)^2} < 0$$

So for (4.35) to be satisfied, since

$$\frac{\partial \phi}{\partial T} = -\frac{\partial \mu}{\partial T} \Big/ \frac{\partial \mu}{\partial \phi},$$

we deduce that $\frac{\partial \phi}{\partial T} > 0$. □

When the zero-pest solution is stable, $\phi(T)$ is negative and the pest population asymptotically tends towards zero. The above result therefore means that the rate of evolution towards zero becomes less negative, that is its magnitude decreases, as the period of release is increased. The speed of convergence towards the pest-free solution decreases until zero.

Figure 4.6 illustrates this result. The convergence of x to zero for three different release periods is shown. In all three cases, μ is identical and sufficiently high to ensure the stability of this solution. So ϕ is negative and the convergence of x to zero is faster for smaller release periods.

The proof of Theorem 7 actually tells us more than Theorem 7 itself. For positive $\phi(T)$, that is when the stability of the pest-free system is not guaranteed, increasing the period of releases increases the magnitude of ϕ. In fact, it is the rate of invasion that is increased. We remark here that keeping T small is favourable even if ϕ is positive since it would be delaying the pests invasion.

4.5.2 Nonlinear parameterisation

When the parameterisation of $\alpha(x)$ and $\beta(y)$ is nonlinear, we can determine the rate of evolution to be enclosed within a tube, the upper and lower bounds of which are related to the sector conditions. Recalling (4.28), that is:

$$\int_0^T \frac{a_1 y_p(\tau)}{b_2 y_p(\tau) + 1} d\tau \leq \int_0^T \frac{\alpha'(0) y_p(\tau)}{\beta(y_p(\tau))} d\tau \leq \int_0^T \frac{a_2 y_p(\tau)}{b_1 y_p(\tau) + 1} d\tau$$

we can bound ϕ defined in (4.34) as

$$\phi(T; a_2, (b_1 y_p + 1)) \leq \phi(T; \alpha'(0), \beta(y_p)) \leq \phi(T; a_1, (b_2 y_p + 1)) \tag{4.38}$$

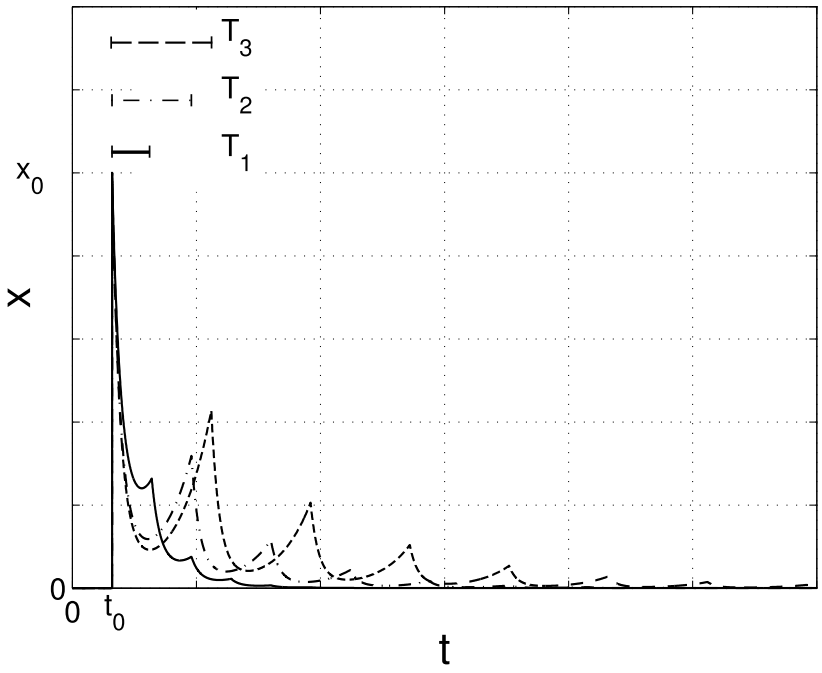

Figure 4.6: Pest convergence for a given μ at different release periods, $T_1 < T_2 < T_3$. As the period of release is increased, the pest population converges less quickly to zero.

Dependence in the release rate

We follow a similar reasoning as in the affine case. Each of the bounds imposed in (4.38) have the same form as (4.35) and will be decreasing in the release rate μ. So we can deduce that ϕ will follow a generally decreasing trend with respect to the release rate.

Dependence in the release period

From the proof of Theorem 7, both $\phi(T; a_2, (b_1 y_p + 1))$ and $\phi(T; a_1, (b_2 y_p + 1))$ are increasing in T. From the bounds imposed in (4.38), we can deduce that $\phi(T)$ will follow a generally increasing trend with respect to the release period T. The actual speed of convergence towards the pest-free solution will tend to decrease. Similarly to the behaviour of μ with respect to T described above, the value of T at the actual crossover of ϕ is unknown. The threshold values $\overline{T^*}$ and $\underline{T^*}$ are however obtained as

$$\phi(\underline{T^*}; a_1, (b_2 y_p + 1)) = 0 \quad \text{and} \quad \phi(\overline{T^*}; a_2, (b_1 y_p + 1)) = 0$$

Figure 4.7 shows the variation of the rate of evolution ϕ with respect to the release period T, with the release rate fixed at $\mu^* = 0.05$. As for the simulation to calculate $\tilde{\mu}(T)$ in the previous section, $\alpha(x)$ is affine and $\beta(y)$ Lipschitz square-like. All parameters are identical. The shaded tube is limited by the bounds obtained from the sector conditions and contains the actual ϕ that was computed numerically. The two thresholds on the release period ($\underline{T^*}$ and $\overline{T^*}$) are equal to the ones in Figure 4.5. Once again, for this example, we note that the actual ϕ coincides with the upper bound $\phi(\overline{T^*}; a_1, (b_2 y_p + 1))$ for small T and approaches close to $\phi(\underline{T^*}; a_2, (b_1 y_p + 1))$ for intermediate T values. The quality of our bounds is demonstrated.

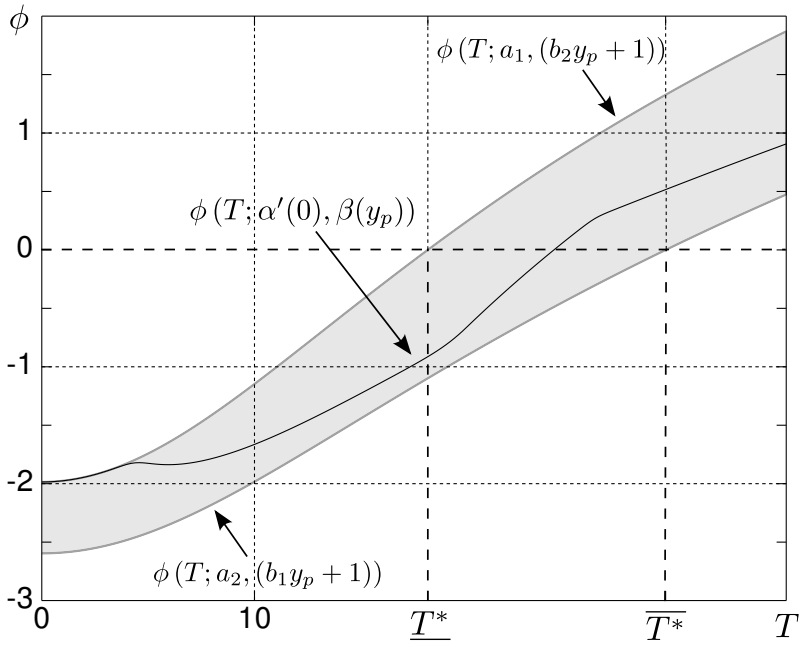

Figure 4.7: The mean rate of evolution $\phi(T)$ is numerically computed from $\mu = 0.05$, an affine $\alpha(x)$ and a Lipschitz square-like $\beta(y)$ shown on Figure 4.2. The shaded area is the tube defined by (4.38) to which ϕ belongs. The threshold $\underline{T^*}$ (*resp.* $\overline{T^*}$) under which (*resp.* over) the pest-free solution is guaranteed to be locally stable (*resp.* unstable) is also shown.

4.6 The pest control strategy

We are able to obtain, from the stability analysis of the pest-free solution, two types of conditions: the first one of biological nature and the second in terms of implementation of the biological control program that would inform the choice of the predator species and the strategy of its release respectively.

4.6.1 Choice of the predator species

Which predator species makes a successful biological control agent?

The biological condition determines an upper threshold on the interference parameter b, beyond which no control would be possible - this even if the invasion of pests were to be very small. That is,

$$b < \frac{ag'(0)}{f'(0)}$$

Thus to be a successful biological control agent, a predator species needs to have a sufficiently low intraspecific interfering behaviour b. A high attack rate $g'(0)$, compared to the term $\frac{f'(0)}{a}$, also favours biological control program. The term $\frac{f'(0)}{a}$ can on its part be interpreted as the 'effective pest growth rate', since it is the pest growth weighed by a, which is similar to pest exposure or vulnerability index with respect to the predator.

Moreover, we have shown that similar conclusions can be derived in the more general case where interference can only be described through the sector condition framework, and not by a more precise analytical formulation. The information obtained through this phenomenological modelling approach clarifies the interplay of the pest growth versus the predation capacity of the predator which may be weighed down by interference effects and enhanced by the pest's exposure.

4.6.2 The predator releases

How many predators need to be released to eradicate a pest population surge?

Provided the biological condition is satisfied, our calculations show that the rate of predator release μ must be larger than a minimal threshold to guarantee the stability of the pest-free solution. This threshold is expressed as

$$\mu > \underline{\mu}(Q, \lambda, T)$$

where the terms $Q > 0$ and $0 < \lambda < 1$ are constants defined by intrinsic biological properties of the interacting species, and also depend on the gravity of the invasion.

When the parameterisation is affine, a given rate that is smaller than this threshold value results in the pest-free solution being unstable.

When the parameterisation is nonlinear, we cannot pinpoint the exact threshold value of the minimal release rate. We obtain two distinct thresholds: first, a lower bound on the rate for stability. Above this bound, stability is guaranteed. Secondly, we obtain an upper bound for instability of the pest-free solution. Below this bound, the pest is guaranteed to persist. Both conditions are only sufficient. The actual threshold is between the two bounds.

Nonetheless, a robust result from our analysis is that the more predator releases are invested in, i.e. the larger the μ, the higher the chances that the biological control program will be successful. We shall however see briefly in Section 4.6.3 that, although this result sounds intuitive, it does not necessarily hold for any interference type.

Why choose small frequent releases over large rare ones?

The release strategy of the pest management program can be optimised in two aspects. Firstly, to minimise the financial costs of a biological program related to predator purchase, we would like to minimise the threshold release rate for pest eradication $\underline{\mu}(\lambda, Q, T)$. Secondly, as explained in Section 4.5, we would like to minimise the damage caused by pests on the crop and thus maximise the the speed of convergence of the pest population to zero after an invasion.

Our computations of the release rate condition's dependence on T reveals that small frequent releases lower the minimal release rate required for pest eradication and jointly increases the speed of convergence of the pest population towards zero. This result is strictly correct in the linear parameterisation context, and still remains generically true in the more general sector conditions framework.

This means that in practice, small frequent releases are to be favoured over large less frequent ones. Interestingly, by using a *minimal risk of damage* approach, Mailleret and Grognard (2006, 2009) obtained similar recommendations on a predator-prey model without predator interference. Such corroborating results lend support to the validity of the strategy in real-life.

See later: *We had the opportunity to test this recommendation for a reduced experimental batch and report this work in Chapter 7. This preliminary experimental evidence seems to match with the prediction: more frequent releases produced a faster eradication. However, things may not be that simple (see Section 4.6.3, 'Additional questions') and require more experimental work both on the strength of interference among individuals of a species for validation and frequency of releases.* ■

Can the pest eradication be accelerated?

We saw during our calculations that a higher predator release rate increased the speed of eradication. It is possible to split the predator release regime into two. Having massive release at the start of an invasion (the first regime, also at a higher release rate) can provide a fast reduction in the pest population. If this reduction is sufficient, i.e. it reduces the pest population size to the local basin of attraction of the system, we can follow up by a smaller release rate (local stability condition) that is spread over more frequent releases (the second regime) in order to guarantee eradication and keep the risk of damage at low levels. Adapting the pest control program to pest invasions as such depends on how easy it is to detect them. Our model does not tackle explicitly tackle adaptability to invasions, but is only able to provide a first guess on the strategy to follow.

4.6.3 Additional questions: beyond sector modelling

The sector formulation in this chapter generate a maximal family of models that yield two types of stability conditions for the zero-pest solution: the first is a biological and necessary condition, and the second, a managerial one. This observation indicates that the result of the stability analysis is not related to the explicit form of the trophic responses (such as the Beddington-DeAngelis function, which yields these two types of conditions), but rather, to the structure of the trophic response's parameters.

In our final paper (Nundloll et al., 2010a), we presented the structural changes that occur to the stability of the zero-pest solution when the parameters of the trophic response cannot be be enclosed within sectors. We highlighted the need to discriminate between three levels of interference:'within sectors or moderate', 'too weak', and 'too strong'. We were also able to generalise completely Buffoni et al. (2005)'s formulation for both density and ratio-dependence.

We summarize these additional results in the answers to the three questions below:

What if the intrapredator interference are due to ratio-dependence effects?

The ratio dependence case correponds to $b_0 = 0$ in equation (4.8). We were able to prove that although a biological condition of the form (4.15a) is still required for the pest-free solution to be stable, it is actually the only condition required for stability: the pest-free solution is stable for any positive release rate μ. This result is consistent with the stability of the origin of the unforced ratio-dependent system (Kuang and Beretta, 1998), which states that, if the equivalent of our biological condition is satisfied, and if the predators cannot go extinct while preys are present, then the origin (pest-free solution, when there are no releases) is stable.

What happens if interference is *extremely* weak?

Mathematically, this implies that the $\beta(y)$ function cannot be lower bounded by an increasing linear function. In the weak interference case, it was shown that no biological necessary condition like (4.15a) needs to be satisfied in order for the pest population to be stable: as in the non-interfering predator case (see Chapter 3), there always exist a large enough predator release rate that guarantees the pests can be eradicated. In this situation, the interference is actually weaker than the density effect on the predation: the more predators are present, the more efficient the total predation is.

What happens if interference is *too* strong?

The strong interference case implies mathematically that the $\beta(y)$ function cannot be upper bounded by an increasing linear function. As a result, things are a little bit different. We concluded that the pest free solution can be stable over intermediate values of the release rate μ only: in particular if μ is too large, biological control is disrupted. This contrasts with our result in the presence of sector conditions where, when a biological condition is satisfied, increasing the release rate ensures that the zero-pest solution is stable. Here, the interference is stronger than the density effect: the larger the predator-density, the less efficient the effective predation $g(x, y)y$ is.

5
Cannibalism among predators

Overview

Other than interfering with predation or pest uptake[1], intrapredator encounters can also impact the mortality rate of the predator population.

We will use the terminology adopted by Claessen et al. (2004) to define cannibalism as all the processes that enhance mortality - including those that are not cannibalistic in the biological sense of the term. As we will explain shortly, our use of the term '*cannibalism*' includes killing without eating or the elimination of individuals from the patch under consideration via fleeing. We also extend this definition to include interpredator feeding within a guild of predators as proposed by Kohlmeier and Ebenhoh (1995).

Cannibalism implies that predators get depleted faster, that is fewer predators than anticipated are present to control pests. This means that the augmentative biological control program will have to compensate for this predator behaviour. In this chapter, we analyse three models, which depict different circumstances under which predators cannibalise, and the impact on the pest control strategy.

Keywords: enhanced mortality, feeding strategy, intraguild predation.

Organization of this chapter:

This chapter is organized as follows.

Sections 5.1 introduces the notion of cannibalism as it is used in biomathematics. It presents a general model for this behaviour, variants of which are studied in more detail in the next three sections.

Section 5.2 covers in detail the first variant of the general model which describes cannibalism arising from *territorial* effects. This includes a linear as opposed to a constant mortality rate, which introduces some new challenges at the analytical level.

[1] See Chapter 4 for the effects of intrapredator interference that impacts predation on an augmentative biological control program.

Section 5.3 deals with *hunger-related* cannibalism. Here, the model is bounded between two systems that have the same form as our benchmark model in **Chapter 3**.

Section 5.4 presents a model of cannibalism that arises due to *dietary requirements*. This model is bounded as the competition model in **Chapter 4** with the added cannibalism term. We use sector conditions to describe the function arguments.

In each section, we analyse the existence and stability of the zero-pest solution. For the latter two models, we focus on the key points of our analysis which justify the choice of these bounds on them.

Section 5.5 provides a comparison among the minimal release rates for pest eradication obtained in the preceding sections. We study in detail the form of the minimal rate solution for *territoriality*, and derive the characteristic features of the *hunger*-related model and the *diet*-related ones based on our work in **Chapters 3** and **4**.

5.1 Understanding cannibalism

5.1.1 Cannibalism in the biomathematical jargon

Cannibalism is the process whereby an individual kills and feeds on its conspecifics. It impacts directly the mortality of a population. If we follow the biomass transfers during cannibalism however, we see that the consequences of this killing and feeding differ across different species or under different environmental conditions. Cannibalism among predators does not necessarily modify the pest uptake by the predators. Conversely, the biomass recovered through the consumption of conspecifics may not get transformed into more predators. As Polis (1981) and Claessen et al. (2004) point out, this means that cannibalism will also influence the functional and numerical responses only under a particular set of circumstances.

Likewise, we can also describe two other instances whereby mortality of the predator population is enhanced by intrapredator interactions: the first is that one or one group of predators (which we refer to as the victim of the encounter) *flees* the patch upon meeting another (the winner of the encounter). Another instance is that the victim *gets killed* by the winner but not consumed.

Mathematically, this then implies that, in some instances, the modelling of cannibalism - or at least some forms of it - is strictly identical to the modelling of predators getting killed but not eaten by their conspecifics or simply fleeing them.

Because of these similarities from a mathematical point of view, the term cannibalism is therefore used to define all the processes that enhance mortality, including those that are not - biologically speaking - actually so (Claessen et al., 2004). Schematically, it can be represented as in Figure 5.1.

In this chapter, we look at three mechanisms underlying cannibalism, each of which is matched to a specific model. They are:

1. Territoriality,
2. Hunger,
3. Diet (which we also extend to intraguild predation effects).

5.1.2 Impact of cannibalism on augmentative pest control

When predators are killed through cannibalism, it is reasonable to assume that predation on the pest is reduced. Here's why: first, in most reported cases, the energy or biomass of the defeated conspecific cannot be fully recovered. Even during consumption of the victim,

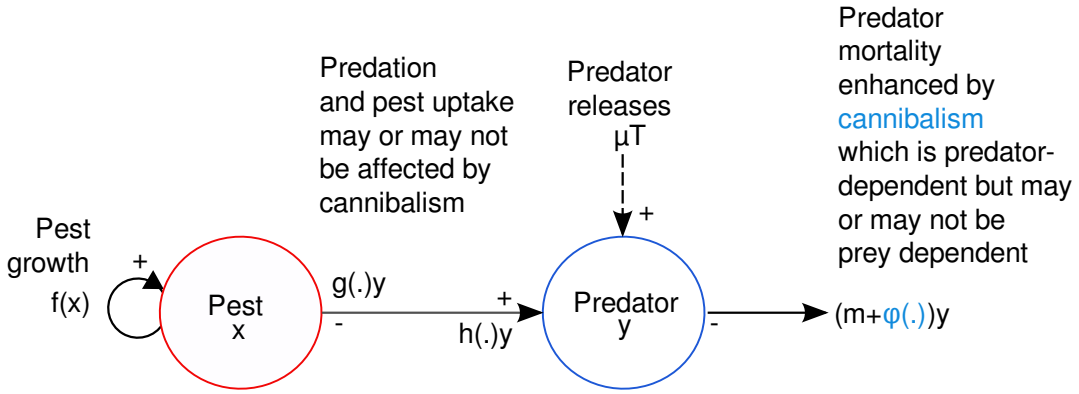

Figure 5.1: Cannibalism enhances predator mortality, but may or may not affect predation and pest uptake.

part of the carcass - which forms a fraction of the biomass - is wasted (see for instance the reviews by Polis (1981) and Claessen et al. (2004) and the references therein). Likewise, it is possible that the nutrients recovered during cannibalism are not sufficient for reproduction, so the numerical response can remain unaffected. This implies that whether as an immediate consequence or over a subsequent generation, the number of predators is decreased.

When this happens in the case of competitive predators, which compete for the pest as prey, the per capita predation (represented by the functional response) can increase as predators have more prey per surviving cannibalist. Nonetheless, because of saturation effects that are fixed by stomach size, a decrease in the total number of predators results in a decrease in predation.

So cannibalism can be expected to have detrimental impact on pest control.

5.1.3 General model of cannibalism

As was mentioned in Section 5.1.1, cannibalism affects directly predator mortality. Depending on the circumstances provoking this behaviour, it may also modify the trophic response. Consequently, we generalise a model of cannibalism as:

$$\left\{\begin{array}{l} \left.\begin{array}{l} \dot{x} = f(x) - g(x,y)y \\ \dot{y} = h(x,y)y - my - \varphi(y,x)y \end{array}\right\} t \neq nT \\ y(nT^+) = y(nT) + \mu T, \quad \forall n \in \mathbb{N} \end{array}\right. \tag{$\mathcal{M}_q$}$$

where $\varphi(y,x)y$ represents cannibalism.

We recall the general biologically plausible hypotheses (H1)–(H3‡) on the basic predator-prey processes:

General hypotheses on predator-prey processes

Pest growth at zero pest:

$$f(0) = 0 \quad \text{and} \quad f'(0) > 0 \tag{H1}$$

Functional response: $\forall y \geq 0$:

$$g(0,y) = 0, \quad \forall x \geq 0, \ \frac{\partial g}{\partial x}(x,y) > 0, \quad \text{and} \quad \forall x > 0, \ g(x,y) > 0; \tag{H2‡}$$

Numerical response: $\forall y \geq 0$:

$$h(0,y) = 0 \quad \text{and} \quad \forall x > 0, \ h(x,y) \geq 0 \tag{H3‡}$$

The trophic responses $g(x, y)$ and $h(x, y)$ can be solely prey-dependent or dependent on both prey and predator densites (in case of interferences similar to those presented in Chapter 3).

The function $\varphi(y, x)$ which represents pure cannibalism has the following characteristics:

General hypothesis on the cannibalism function

Cannibalism function:

$$\forall\, x, y \geq 0,\ \varphi(y, x) \geq 0 \tag{H7}$$

The hypothesis (H7) on the cannibalism function implies that cannibalism is non-negative in the presence of predators. The function $\varphi(.)$ may only depend on predator density or both on prey and predator population densities (it may even be constant, but that would lead to a simple increased mortality rate).

The models differ in the dependency of the trophic responses and the cannibalism function on the respective population densities. This dependency arises from the behavioural mechanisms leading predators to cannibalise. Table 5.1 summarises the consequences of the various kinds of cannibalism on the trophic responses and cannibalism function of Model ($\mathcal{M}_q$). More details are given in each of the sections.

Model	Territoriality (i)	Hunger (ii)	Diet (iii)
Functional response	$g(x)$	$g(x)$	$g(x, y)$
Numerical response	$h(x)$	$h(x)$	$h(x, y)$
Cannibalism function	$\varphi(y)$	$\varphi(y, x)$	$\varphi(y, x)$

Table 5.1: Direct and indirect impacts of cannibalism. Intrapredator competition and alternate prey determine the dependence of the trophic responses and the cannibalism function on the prey and predator densities.

5.2 Mechanism I: Territoriality

5.2.1 The premises

Territorial behaviour among ladybirds has been observed by Agarwala et al. (2003); they mark their territory by their feces to deter conspecifics from foraging: this forced fleeing from the prey patch can also be modelled as enhanced mortality, since it corresponds to a decrease of the predator density on the patch. Cannibalism is a strategic behaviour: individuals cannibalize potential enemies to defend resources such as space and ultimately increase chances of reproductive success.

Some species such as *Phytoseiulus persimilis* (Athias-Henriot) which are natural enemies of *Tetranychus urticae* (Koch). *P. persimilis* are extremely voracious and do not discriminate, at high predator numbers, between the prey and conspecifics (Gerson et al., 2003). Here cannibalism appears to be a direct consequence of overcrowding but not territoriality per se.

In both cases, cannibalism is the result of density-related encounters. Higher densities of predators increase the rate of conspecific encounters, hence the rate of territorial disputes. Likewise, cannibalism is not limited by the pest population, nor is predation of the pest limited by the presence of an alternate food source represented by the potential cannibal victims among predators. So, we simply consider the cannibalism function as a term dependent in

the predator density only ($\varphi(y)$ with $\frac{d\varphi}{dy} > 0$) without any impact on the trophic responses. This has been proposed before in the literature Rosenzweig (1972); Kuang et al. (2003); Ruan et al. (2007); Deng et al. (2007), with Goh (1976) specifically attributing it to overcrowding.

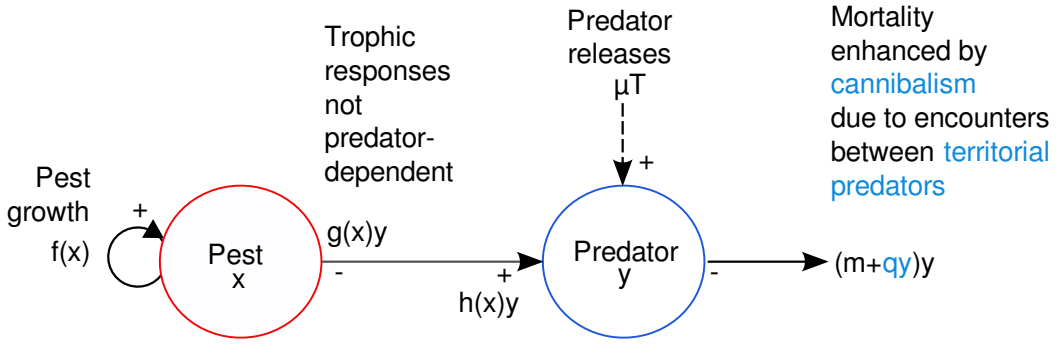

Figure 5.2: Territoriality effects enhance predator loss. This is represented by a mass-balance term which describes asymmetric encounters upon which the weaker conspecific flees the stronger one of the two.

5.2.2 The model and specific hypotheses

The following model is used to depict territorial effects:

$$\left\{\begin{array}{l} \left.\begin{array}{l} \dot{x} = f(x) - g(x)y \\ \dot{y} = h(x)y - my - qy^2 \end{array}\right\} t \neq nT \\ y(nT^+) = y(nT) + \mu T, \quad \forall n \in \mathbb{N} \end{array}\right. \qquad (\mathcal{M}_{q(i)})$$

In this case, we chose to model territorial effects as qy^2 since it corresponds to cannibalism taking place every time two predators meet (Deng et al., 2007). The factor q represents the loss incurred in predator biomass at predator-to-predator encounters. In the case where the encounter is particularly asymmetric (for instance, due to size - small versus large predator -, or mobility - immobile larval stage versus highly active adult stage), the factor represents the fraction of the predator population lost (dead or escaped) as victims of cannibalistic combat. We see here that this form of cannibalism is very strong: when the predator density is very high, cannibalism causes its quick decrease.

We now make the following assumptions:

Specific hypotheses during overcrowding

Ratio of pest growth to functional response:

$$\frac{f(x)}{g(x)} \text{ is upper bounded for } x \geq 0 \text{ such that } S_g = \sup_{x \geq 0}\left(\frac{f(x)}{g(x)}\right). \qquad \text{(H4)}$$

5.2.3 Existence of the zero-pest solution

The nonlinear predator mortality has a direct impact on the form of the predator dynamics in the pest-free periodic solution. We state the result before detailing the computation of this solution in the proof further below.

Proposition 6 (Existence, elementary cannibalism) *Model ($\mathcal{M}_{q(i)}$) possesses a pest-free solution*

$$(x_p(t), y_p(t)) = \left(0, \frac{my^* e^{-m(t \mod T)}}{m + (1 - e^{-m(t \mod T)})qy^*}\right), \tag{5.2a}$$

where

$$y^* = \frac{1}{2}\left(\mu T - \frac{m}{q} + \sqrt{\left(\mu T - \frac{m}{q}\right)^2 + \frac{4\mu m T}{q(1 - e^{-mT})}}\right). \tag{5.2b}$$

Proof: In the absence of pests, $x(t_0) = 0$ and system ($\mathcal{M}_{q(i)}$) is simplified to

$$\begin{cases} \dot{x} = 0 \\ \dot{y} = -(m + qy)y \\ y(nT^+) = y(nT) + \mu T \end{cases} \tag{5.3}$$

As in the previous models, $x_p(t) = 0$ is trivially found. We consider the time interval between two releases $t \in (nT^+, (n+1)T)$ to calculate the variation of $y_p(t)$ under the continuous regime. The quadratic component in the mortality requires some additional computations in the integration of the $\dot{y}$ dynamics. We start by separating variables and obtain:

$$\begin{aligned} & \int_{y(nT^+)}^{y(t)} \frac{1}{(m + qs)s} ds = \frac{1}{m} \int_{y(nT^+)}^{y(t)} \left(\frac{1}{s} - \frac{q}{m + qs}\right) ds = -\int_{nT^+}^{t} d\tau \\ \Leftrightarrow \quad & \ln\left(\frac{y(t)}{y(nT^+)}\left(\frac{m + qy(nT^+)}{m + qy(t)}\right)\right) = -m(t - nT) \\ \Leftrightarrow \quad & y(t) = \frac{my(nT^+)e^{-m(t-nT)}}{m + (1 - e^{-m(t-nT)})qy(nT^+)}. \end{aligned} \tag{5.4}$$

After release at $t = (n+1)T$, we can formulate the sequence

$$y((n+1)T^+) = \frac{my(nT^+)e^{-mT}}{m + (1 - e^{-mT})qy(nT^+)} + \mu T \tag{5.5}$$

Since this discrete nonlinear system is positive and $\frac{d}{dy}\left(\frac{mye^{-mT}}{m + (1 - e^{-mT})qy}\right) \leq e^{-mT} < 1$, this system has a unique and stable positive equilibrium

$$y^* = y((n+1)T^+) = y(nT^+),$$

which can be computed as (5.2b). The existence of (5.2a) is thus proved. □

5.2.4 Stability of the zero-pest solution

The conditions required for the stability involves at some point comparing the upper bound on the per capita pest growth to predation rate with the periodic predator variation which is a function of the release rate μ. Because of the quadratic mortality however, the predator reference fixed point y^* is a highly nonlinear mapping of this release rate. We may only provide an implicit stability condition in terms of this release rate. Theorem 8 states the result of our analysis.

Theorem 8 (Stability, elementary cannibalism) *The solution $(x(t), y(t)) = (0, y_p(t))$ of Model ($\mathcal{M}_{q(i)}$) is LAS if and only if*

$$\ln\left(1 + \frac{q}{m}(1 - e^{-mT})y^*\right) > \frac{f'(0)}{g'(0)}qT \tag{5.6a}$$

and is GAS if, in addition,

$$\ln\left(1+\frac{q}{m}(1-e^{-mT})y^*\right) > S_g qT \tag{5.6b}$$

*where S_g is as defined in Hypothesis (**??**).*

Proof: To prove the condition for local stability, we evoke once again Theorem 1 from Lakmeche and Arino (2000), and obtain the necessary and sufficient conditions $e^{-\int_0^T (m+qy_p(\tau))d\tau} < 1$, which is trivial, and $e^{\int_0^T f'(0)-g'(0)y_p(\tau)d\tau} < 1$. Recalling $y_p(t)$ from (5.2a), the latter yields

$$\begin{aligned}
&\int_0^T [f'(0)-g'(0)y_p(\tau)]d\tau < 0\\
\Leftrightarrow\quad &\int_0^T \frac{my^*e^{-m\tau}}{m+(1-e^{-m\tau})qy^*}d\tau > \frac{f'(0)}{g'(0)}T\\
\Leftrightarrow\quad &\tfrac{1}{q}\ln\left(m+q(1-e^{-mT})y^*\right)-\tfrac{1}{q}\ln(m) > \frac{f'(0)}{g'(0)}T\\
\Leftrightarrow\quad &\ln\left(1+\frac{q}{m}(1-e^{-mT})y^*\right) > \frac{f'(0)}{g'(0)}qT
\end{aligned} \tag{5.7}$$

which is the LAS condition (5.6a).

Next, to obtain the global stability condition, we consider the deviations from the periodic solution as $\tilde{x}=x-x_p$ and $\tilde{y}=y-y_p$ respectively. The equivalent system, which is continuous as the release effect cancels, is given as[2]:

$$\begin{cases}\dot{\tilde{x}} = f(\tilde{x})-g(\tilde{x})(\tilde{y}+y_p)\\ \dot{\tilde{y}} = h(\tilde{x})(\tilde{y}+y_p)-m\tilde{y}-2qy_p\tilde{y}-q\tilde{y}^2\end{cases} \tag{5.8}$$

Let the starting coordinates be $\tilde{x}_0$ and $\tilde{y}_0$ at time t_0. As previously, the global asymptotic stability of the pest-free solution is proved by first showing the convergence of $\tilde{x}$ and $\tilde{y}$ to zero.

We start by looking at the $\tilde{x}$ dynamics. As previously, we divide the $\dot{\tilde{x}}$-equation throughout by $g(\tilde{x})$. If we separate state $\tilde{x}$ and time domains, then integrate from t_0 up to an instant t, we obtain:

$$\int_{\tilde{x}_0}^{\tilde{x}(t)}\frac{1}{g(s)}ds = \int_{t_0}^{t}\left[\frac{f(\tilde{x})}{g(\tilde{x})}-(\tilde{y}+y_p)\right]d\tau$$

Following what we did in the previous chapters, convergence of $\tilde{x}$ to zero is shown by proving

$$\lim_{t\to+\infty}\int_{\tilde{x}_0}^{\tilde{x}(t)}\frac{1}{g(s)}ds = \lim_{t\to+\infty}\int_{t_0}^{t}\left[\frac{f(\tilde{x})}{g(\tilde{x})}-(\tilde{y}+y_p)\right]d\tau = -\infty. \tag{5.9}$$

As in the previous chapters, we focus on the time-domain integral, which we split as:

$$\begin{aligned}
&\int_{t_0}^{t}\left[\frac{f(\tilde{x})}{g(\tilde{x})}-(\tilde{y}+y_p)\right]d\tau\\
&\qquad=\left(\int_{t_0}^{(\lfloor\frac{t_0}{T}\rfloor+1)T}+\int_{(\lfloor\frac{t_0}{T}\rfloor+1)T}^{\lfloor\frac{t}{T}\rfloor T}+\int_{\lfloor\frac{t}{T}\rfloor T}^{t}\right)\left[\frac{f(\tilde{x})}{g(\tilde{x})}-y_p\right]d\tau-\int_{t_0}^{t}\tilde{y}\ d\tau.
\end{aligned} \tag{5.10}$$

[2]The calculations are detailed as follows

$$\begin{cases}\dot{\tilde{x}} = \dot{x}-\dot{x_p} = f(\tilde{x})-g(\tilde{x})(\tilde{y}+y_p)\\ \dot{\tilde{y}} = \dot{y}-\dot{y}_p = h(x)y-my-qy_p^2+my_p+qy_p^2\\ \qquad\quad = h(\tilde{x})(\tilde{y}+y_p)-m(\tilde{y}+y_p)-q(\tilde{y}+y_p)^2+my_p+qy_p^2\\ \qquad\quad = h(\tilde{x})(\tilde{y}+y_p)-m\tilde{y}-q\tilde{y}^2-2qy_p\tilde{y}\end{cases}$$

We make at this point a small digression to show that the integral $\int_{t_0}^{t} \tilde{y}\ d\tau$ is bounded as t goes to infinity. Since x and y are always non-negative

$$\dot{\tilde{y}}(t) \geq -(m + 2qy_p + q\tilde{y})\tilde{y} = -(m + qy_p + qy)\tilde{y} \tag{5.11}$$

There are two possible lower bounds on the variation of $\tilde{y}$ depending on the initial values $\tilde{y}_0$. Indeed, the positivity of $\tilde{y}$ can be seen from (5.11), so that when $\tilde{y}_0 \geq 0$, $\tilde{y}(t) \geq 0$ for all $t \geq 0$. Conversely, when $\tilde{y}_0 < 0$, $\dot{\tilde{y}}(t) \geq -(m+qy_p+qy)\tilde{y} \geq -m\tilde{y}$, so that $\tilde{y}(t) \geq \tilde{y}_0 e^{-m(t-t_0)}$. Therefore

$$\tilde{y}(t) \geq \min\left(0, \tilde{y}_0 e^{-m(t-t_0)}\right)$$

So, $\int_{t_0}^{t} \tilde{y}\ d\tau$ is lower-bounded with $\int_{t_0}^{t} \min\left(0, \tilde{y}_0 e^{-m(\tau-t_0)}\right) d\tau$ whose limit as $t \to \infty$ is finite.

Back to the other components of the split integral in (5.10), we note that the first and third integral span at most over one period. The integrand is upper bounded by S_g, so these integrals are each upper bounded by $S_g T$.

The magnitude of the second integral however, grows indefinitely as t goes to infinity. By considering the periodic upper bound of the integrand $(S_g - y_p)$, we may upper bound this integral by a sum of T-integrals each spanning over a complete period $(0, T)$. Equation (5.9), that shows convergence of $\tilde{x}$ to zero is then satisfied if:

$$\lim_{t\to+\infty} \int_{(\lfloor \frac{t_0}{T} \rfloor + 1)T}^{\lfloor \frac{t}{T} \rfloor T} \left[\frac{f(\tilde{x})}{g(\tilde{x})} - y_p\right] d\tau$$
$$\leq \lim_{t\to+\infty} \left(\lfloor \frac{t}{T} \rfloor - \lfloor \frac{t_0}{T} \rfloor - 2\right) \int_0^T [S_g - y_p]\ d\tau = -\infty \tag{5.12}$$

For convergence, it therefore suffices that the sign of the T-integral be negative, which we may then compute to give condition (5.6b), as follows:

$$\begin{aligned} & \int_o^T [S_g - y_p]\ d\tau < 0 \\ \Leftrightarrow \quad & \int_0^T \frac{my^* e^{-m\tau}}{m + (1 - e^{-m\tau})qy^*} d\tau > S_g T \\ \Leftrightarrow \quad & \ln\left(1 + \frac{q}{m}(1 - e^{-mT})y^*\right) > S_g q T \end{aligned}$$

Having shown global convergence to the pest-free solution, we finally notice that, when condition (5.6b) is satisfied, the local stability condition (5.6a) also is, so that condition (5.6b) implies global asymptotic stability. □

Remark: *On inspecting conditions (5.6a) and (5.6b), we see that the pest-free solution will be stabilised by a low pest growth and a high predation rate - indeed, both lower the right-side of the inequality. The effect of predator mortality is less apparent (because of its influence on y^*) but numerical simulations show that the left-hand side of the inequality is monotonic decreasing with m. This means that low predator mortality stabilises the pest-free solution. These observations are not surprising: low pest growth implies slowly developing preys, high predation rate implies efficient control by the predator, and low mortality means longer-living predators. These parameter conditions indicate the ease with which pest control can be carried out and thus, as expected, point towards a small minimal release rate.* ■

5.2.5 The minimal release rate

The formulation (5.6a) and (5.6b) are not explicit enough for a practical read-off of the mathematical result. The dependence of the left-hand side of these inequalities in μ and the non-dependence of the right-hand side only indicate that provided μ is large enough,the stability condition can be satisfied. However, the release rate μ and the release period T are embedded in a highly nonlinear way in y^*, so unlike previous models, it is not possible to express the minimal release rate at all T by simply rearranging condition (5.6a) or (5.6b). To extract μ, we can however consider two distinct situations, one for T large and one for T small.

Large release period

In this case, we note that

$$y^* = \frac{1}{2}\left(\mu T - \frac{m}{q} + \sqrt{\left(\mu T - \frac{m}{q}\right)^2 + \frac{4\mu m T}{q(1-e^{-mT})}}\right)$$

is essentially linear in T because the first term under the square root dominates the second one. For $T \to +\infty$, we then have

$$y^*(T) = \mu T + o(\mu T)$$

so that, for example, for the local stability condition (5.6a) to be satisfied, we need

$$\ln\left(1 + \frac{q}{m}(\mu T + o(\mu T))\right) > \frac{f'(0)}{g'(0)} qT. \tag{5.13}$$

For a given μ, the left-hand-side is essentially the logarithm of a linear function in T, so that this condition cannot stay satisfied for large T: neither the local (5.6a) nor the global stability (5.6b) conditions are fulfilled for T large.

Small release period

We may now shift our attention to the case when releases are 'frequent enough', i.e. in the limit that $T \to 0^+$. This case is particulary relevant, since we have seen in the previous chapters that small release periods are advocated most of the time. In that case, we can prove the following corollary to Theorem 8.

Corollary 1 (Release rate, cannibalism due to overcrowding) *Provided T is small enough, the periodic solution $(x(t), y(t)) = (0, y_p(t))$ of Model ($\mathcal{M}_{q(i)}$) is LAS if*

$$\mu > \underline{\mu}_{q_a} = \frac{f'(0)}{g'(0)}\left(\frac{f'(0)q}{g'(0)} + m\right) \tag{5.14a}$$

and is GAS if

$$\mu > \underline{\mu}_{q_b} = S_g(S_g q + m) \tag{5.14b}$$

Proof: We define $\Omega(T, y^*(T)) = \ln\left(1 + \frac{q}{m}(1 - e^{-mT})y^*(T)\right)$ and note that both sides of (5.6a), $\Omega(T, y^*(T))$ and $\frac{f'(0)}{g'(0)}qT$, cancel at $T = 0$ because $y^*(T)$ stays bounded for T small. Taking the Taylor expansion of (5.6a) then yields

$$\frac{d}{dT}\Omega(T, y^*(T))T_{|0} + o(T) > \frac{f'(0)}{g'(0)}qT$$

so that the condition is satisfied for T small if

$$\frac{d}{dT}\Omega(T, y^*(T)) > \frac{f'(0)}{g'(0)}q$$

in the limit as $T \to 0^+$, with

$$\lim_{T\to 0^+}\frac{d}{dT}\Omega(T, y^*(T)) = \lim_{T\to 0^+}\frac{\partial\Omega}{\partial T} + \lim_{T\to 0^+}\frac{\partial\Omega}{\partial y^*}\frac{\partial y^*}{\partial T} \tag{5.15}$$

We first calculate $\lim_{T\to 0^+} y^*(T) = \frac{1}{2q}\left(-m + \sqrt{m^2+4q\mu}\right)$. Hence

$$\begin{aligned}\lim_{T\to 0^+}\frac{\partial\Omega}{\partial T} &= \lim_{T\to 0^+}\frac{me^{-mT}\frac{q}{m}y^*(T)}{1+(1-e^{-mT})\frac{q}{m}y^*(T)}\\ &= \frac{1}{2}\left(-m+\sqrt{m^2+4q\mu}\right)\end{aligned}$$

and $\lim_{T\to 0^+}\frac{\partial\Omega}{\partial y^*}\frac{\partial y^*}{\partial T} = 0$[3].

So, the stability condition (5.15) becomes

$$\begin{aligned}&\lim_{T\to 0^+}\frac{d}{dT}\Omega(T, y^*(T)) = \frac{1}{2}\left(-m+\sqrt{m^2+4q\mu}\right) > \frac{f'(0)q}{g'(0)}\\ \Leftrightarrow\quad &\mu > \frac{\left(2\frac{f'(0)q}{g'(0)}+m\right)^2 - m^2}{4q} = \frac{f'(0)}{g'(0)}\left(\frac{f'(0)q}{g'(0)}+m\right)\end{aligned}$$

[3]This is calculated as follows: the second expression on the right-hand side of (5.15) is a product of two partial derivatives:

$$\frac{\partial\Omega}{\partial y^*} = \frac{q(1-e^{-mT})}{m+q(1-e^{-mT})y^*}$$

and

$$\frac{\partial y^*}{\partial T} = \frac{\mu}{2} + \frac{1}{2q}\left(\frac{q\mu^2 T - m\mu + 2m\mu\frac{1-e^{-mT}-mTe^{-mT}}{(1-e^{-mT})^2}}{\sqrt{\left(\frac{m}{q}-\mu T\right)^2 + \frac{4m\mu}{q}\frac{T}{(1-e^{-mT})}}}\right)$$

In the limit as $T \to 0^+$, the product $\frac{\partial\Omega}{\partial y^*}\frac{\partial y^*}{\partial T}$ can be written as the sum of three terms as follows

$$\begin{aligned}\lim_{T\to 0^+}\frac{\partial\Omega}{\partial y^*}\frac{\partial y^*}{\partial T} &= \frac{q\mu}{2}\lim_{T\to 0^+}\left(\frac{1-e^{-mT}}{m+q(1-e^{-mT})y^*(T)}\right)\\ &+\frac{q\mu^2}{2}\lim_{T\to 0^+}\left(\frac{(1-e^{-mT})T}{\left(m+q(1-e^{-mT})y^*(T)\right)\sqrt{\left(\frac{m}{q}-\mu T\right)^2+\frac{4m\mu}{q}\frac{T}{(1-e^{-mT})}}}\right)\\ &+\frac{m\mu}{2}\lim_{T\to 0^+}\left(\frac{(1-e^{-mT})\left(-1+2\frac{1-e^{-mT}-mTe^{-mT}}{(1-e^{-mT})^2}\right)}{\left(m+q(1-e^{-mT})y^*(T)\right)\sqrt{\left(\frac{m}{q}-\mu T\right)^2+\frac{4m\mu}{q}\frac{T}{(1-e^{-mT})}}}\right)\end{aligned}$$

It is easily seen that the denominators are bounded away from zero when $T \to 0^+$, since $m + q(1 - e^{-mT})y^*(T) \geq m$ and

$$\sqrt{\left(\frac{m}{q}-\mu T\right)^2 + \frac{4m\mu}{q}\frac{T}{(1-e^{-mT})}} = 2y^*(T) - \mu T + \frac{m}{q}$$

which is larger than $\frac{m}{2q}$ when T is small. We are then left with considering the numerators and checking their convergence to zero: it can be seen directly that this is the case for the first two terms. For the third term, we note that

$$\lim_{T\to 0^+}\frac{1-e^{-mT}-mTe^{-mT}}{(1-e^{-mT})^2} = \lim_{T\to 0^+}\frac{mT}{2(1-e^{-mT})} = \lim_{T\to 0^+}\frac{1}{2e^{-mT}} = \frac{1}{2}$$

This brings the numerator of the third term to zero, which proves that $\frac{\partial\Omega}{\partial y^*}\frac{\partial y^*}{\partial T} = 0$.

which is condition (5.14a). The global condition (5.14b) on μ follows from the fact that the inequality (5.6b) has exactly the same form as (5.6a), with $\frac{f'(0)}{g'(0)}$ substituted by S_g. □

This result is consistent with what was shown in Chapter 3 since, when q goes to 0, model ($\mathcal{M}_{q(i)}$) becomes model ($\mathcal{M}$) and the stability conditions coincide.

5.3 Mechanism II: Hunger and fighting for survival

5.3.1 The premises

Hunger forces the predators to expand their dietary spectrum. The preferred food source of the predators is the pest and predators cannibalize only when the pest population is low. This is referred to as a lifeboat mechanism (Polis, 1981).

The rove beetle, *Aleochara bilineata* (Gyllenhaal) (Coleoptera: Staphylinidae), a predator of root maggots is known to eat their own eggs and attack other adults when food levels are low (Weeden et al., 2007). Some generalist predaceous mites such as *Neoseiulus californicus* (McGregor), feed preferentially on pest species, and cannibalize only out of necessity, that is at low pest levels Gerson et al. (2003). In fact, cannibalism does not allow it to lay eggs: this means that there is no evolutionary benefit to it, so it should limit its occurence to emergency survival cases (Walzer et al. (1999)). In population dynamical terms, the cannibalism term is both prey and predator limited. Conversely, predation on the pest remains unaffected.

5.3.2 The model and specific hypotheses

This type of cannibalism is a fight for survival, so we will consider that predators also compete among themselves for the cannibalised victims. We can then consider the cannibalism function to be limited by the pest densities ($\varphi(y,x)$ with $\frac{d\varphi}{dx} < 0$) and bounded ($\frac{d\varphi}{dy} > 0$ and $\varphi(y,0) < C$: the more conspecifics the predator has, the more it will cannibalize, up to a certain point given by the limited handling time). This cannibalism term has a similar form as the one by Kohlmeier and Ebenhoh (1995). We can represent the system schematically as in Figure 5.3.

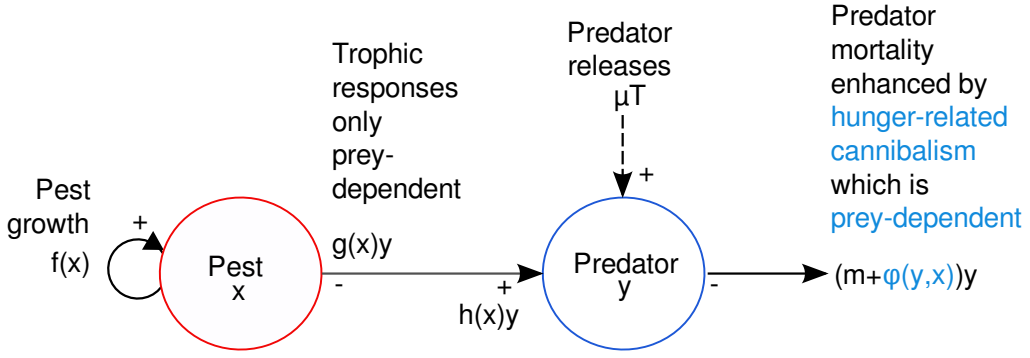

Figure 5.3: Predators cannibalising out of hunger result in enhanced mortality. Hunger effects result in a prey-dependent cannibalism function.

We write the model as:

$$\left\{\begin{array}{l} \left.\begin{array}{l} \dot{x} = f(x) - g(x)y \\ \dot{y} = h(x)y - \varphi\left(y, x\right) y - my \end{array}\right\} t \neq nT \\ y(nT^+) = y(nT) + \mu T, \quad \forall n \in \mathbb{N}, \end{array}\right. \tag{$\mathcal{M}_{q(ii)}$}$$

with the following Hypotheses:

Specific hypotheses for hunger-related cannibalism
Ratio of pest growth to the functional response:

$$\frac{f(x)}{g(x)} \text{ is upper bounded for } x \geq 0 \text{ such that } S_g = \sup_{x \geq 0}\left(\frac{f(x)}{g(x)}\right). \tag{H4}$$

The cannibalism term is bounded

$$\frac{\partial \varphi}{\partial y}(y, 0) \geq 0 \text{ and } \exists C > 0, \forall y \geq 0 : 0 \leq \varphi\left(y, 0\right) \leq C \tag{H8}$$

In essence, $\varphi(y, x)$ is a decreasing function of x since we presented cannibalism as hunger-driven in this case: in the presence of few prey, cannibalism takes place while, if food is abundant, no cannibalism should occur. However, this hypothesis on the dependency of φ with respect to x does not need to be made for the mathematical proof.

On the other hand, the larger the predator population is, the more cannibalistic preys are available, so that the larger the cannibalism rate per predator is; this translates into $\frac{\partial \varphi}{\partial y}(y, 0) \geq 0$ in Hypothesis (H8). The boundedness of $\varphi(y, 0)$ is due to the classical saturation phenomenon that is usually modelled by Holling type II functional responses (due to satiation or limitation because of the food handling time).

Remark: *This form of cannibalism is weaker than the one presented in the previous section: the present form has a bounded cannibalism rate, while this rate was linear in y in the previous case, which would result in massive cannibalism for large values of y.* ■

5.3.3 Existence of the zero-pest solution

The zero-pest subsystem of Model ($\mathcal{M}_{q(ii)}$) is given as:

$$\left\{\begin{array}{l} \left.\begin{array}{l} \dot{x} = 0 \\ \dot{y} = -\varphi\left(y, 0\right) y - my \end{array}\right\} t \neq nT \\ y(nT^+) = y(nT) + \mu T, \quad \forall n \in \mathbb{N}, \end{array}\right. \tag{5.16}$$

Since we do not know exactly the φ function, we cannot give an explicit form of a periodic pest-free condition. Through the previous hypotheses, we will however be able to show that it is unique and give an upper and lower bound to it. The result is stated in Proposition 7 and the calculations detailed in the proof.

Proposition 7 (Existence, cannibalism due to hunger) *Let Hypotheses (H1)–(H3‡), (H7), and (H8) be satisfied. Then Model ($\mathcal{M}_{q(ii)}$) possesses a unique periodic pest-free solution whose y coordinate is bounded as*

$$\frac{\mu T}{1 - e^{-(C+m)T}} e^{-(C+m)(t \mod T)} \leq y_p(t) \leq \frac{\mu T}{1 - e^{-mT}} e^{-m(t \mod T)} \tag{5.17}$$

Proof: We first tackle unicity of the pest-free solution. Following the steps in the proof of Proposition 6, we consider the time interval between two releases $t \in [nT^+, (n+1)T]$ to calculate the variation of $y(t)$ over the continuous interval. Separating variables, we obtain:

$$\int_{y(nT^+)}^{y(t)} \frac{1}{(\varphi(s,0)+m)s} ds = -\int_{nT^+}^{t} d\tau.$$

For a periodic solution $y_p(t)$ of period T, there exists y^* such that $y^* = y((n+1)T^+) = y(nT^+)$. Furthermore, the discrete component imposes that at the end of the period, just before a release,

$$y_p((n+1)T) = y_p(nT^+) - \mu T = y^* - \mu T.$$

To establish unicity of the periodic solution, we must then show explicitly that the y^* value that solves

$$\int_{y^*}^{y^*-\mu T} \frac{1}{(\varphi(s,0)+m)s} ds = -T, \tag{5.18}$$

is unique.

To do so, we demonstrate that the left-hand side integral is bijective in y^*. Let this integral be denoted as $H(y)$:

$$H(y) = \int_{y}^{y-\mu T} \frac{1}{(\varphi(s,0)+m)s} ds$$

Because of the positivity of the system, $y - \mu T \geq 0$, so we consider only $y \geq \mu T$. $H(y)$ is well-defined, continuous and is calculated to be an increasing (though negative) function in y[4]:

$$\begin{aligned} H'(y) &= \frac{1}{(\varphi(y-\mu T,0)+m)(y-\mu T)} - \frac{1}{(\varphi(y,0)+m)y} \\ &= \frac{(\varphi(y,0)+m)y - (\varphi(y-\mu T)+m)(y-\mu T)}{(\varphi(y-\mu T,0)+m)(y-\mu T)(\varphi(y,0)+m)y} \\ &= \frac{(\varphi(y,0)-\varphi(y-\mu T,0))y + (\varphi(y-\mu T,0)+m)\mu T}{(\varphi(y-\mu T,0)+m)(y-\mu T)(\varphi(y,0)+m)y} > 0 \end{aligned}$$

since from Hypothesis (H8), the cannibalism function φ is increasing in y. Recalling from Hypothesis (H8) that the cannibalism term is bounded, we can write:

$$-\frac{1}{m}\ln\left(\frac{y}{y-\mu T}\right) \leq H(y) \leq -\frac{1}{C+m}\ln\left(\frac{y}{y-\mu T}\right).$$

It is easy to see within these bounds that

$$\lim_{y\to\mu T} H(y) = -\infty \quad \text{and} \quad \lim_{y\to+\infty} H(y) = 0.$$

So, $H(y)$ is a strictly increasing (and negative) function over the domain $y \in (\mu T, +\infty]$, i.e. it is bijective. We can further deduce that $\forall \alpha < 0$, $\exists\ y \in (\mu T, +\infty]$ such that $H(y) = \alpha$. So for $\alpha = -T$, there exists a unique $y^* \in (\mu T, +\infty]$ such that $H(y^*) = -T$, i.e. that solves (5.18). This means that there is a unique pest-free periodic solution to ($\mathcal{M}_{q(ii)}$).

We then show that the solution y_p is constrained by the bounds defined in (5.17) as follows. Using (H8), we have that

$$-(C+m)y \leq \dot{y} = -\varphi(y,0)\, y - my \leq -my$$

which yields, for $nT \leq t \leq (n+1)T$, and for a given initial condition $y(nT^+)$ at time nT^+:

$$y(nT^+)e^{-(C+m)(t-nT)} \leq y(t) \leq y(nT^+)e^{-m(t-nT)}$$

[4]It is also possible to show this behaviour by inspection: From Hypothesis (H8), the argument of the integral is a decreasing function (considering only values of $y \geq \mu T$). The left-hand-side is therefore an increasing (negative) function of y.

so that the post-release y-sequence is then given as:

$$y(nT^+)e^{-(C+m)T} + \mu T \leq y((n+1)T^+) \leq y(nT^+)e^{-mT} + \mu T$$

It is then easily seen that the fixed point of the sequence is then also bounded between the fixed-points of the left and right sequences:

$$\frac{\mu T}{1-e^{-(C+m)T}} \leq y^* \leq \frac{\mu T}{1-e^{-mT}} \tag{5.19}$$

Hence, (5.17) is satisfied. □

5.3.4 Stability of the zero-pest solution

Here, the bounds provide a lower threshold below which the solution is unstable and an upper threshold above which the solution is guaranteed to be stable. We focus on this sufficient condition. The result is stated in Theorem 9 and the calculation details follow in its proof.

Theorem 9 (Stability, hunger effects) *The solution* $(x(t), y(t)) = (0, y_p(t))$ *of Model* ($\mathcal{M}_{q(ii)}$) *is LAS if*

$$\mu > (C+m)\frac{f'(0)}{g'(0)} \tag{5.20a}$$

and is GAS if, in addition,

$$\mu > (C+m)S_g \tag{5.20b}$$

Moreover, it is unstable if

$$\mu < m\frac{f'(0)}{g'(0)} \tag{5.21}$$

Proof: We evoke Theorem 1 from Lakmeche and Arino (2000) to identify the LAS condition. As usual, this theorem yields a first condition

$$e^{\int_0^T \left[-\frac{\partial \varphi}{\partial y}(y_p,0)y_p - \varphi(y_p,0) - m\right]d\tau} < 1$$

which is trivial because $\frac{\partial \varphi}{\partial y}(y,0) \geq 0$ and $\varphi(y,0) \geq 0$. A second nontrivial condition is calculated as:

$$e^{\int_0^T [f'(0) - g'(0)y_p]d\tau} < 0$$

Upper-bounding the left-hand-side by replacing y_p with its lower-bound from (5.17), we obtain a sufficient condition for stability, which becomes, upon rearranging the terms,

$$\mu > (C+m)\frac{f'(0)}{g'(0)},$$

which is condition (5.20a).
Similarly, $e^{\int_0^T [f'(0) - g'(0)y_p]d\tau} < 0$ is a condition for instability, which, when satisfied when replacing y_p with its upper-bound, gives the sufficient condition (5.21) for instability

$$\mu < m\frac{f'(0)}{g'(0)}$$

To evaluate the GAS condition, we consider, as in our previous models, the deviation of

$$\begin{cases} \dot{\tilde{x}} = f(x) - g(x)y = f(\tilde{x}) - g(\tilde{x})(\tilde{y} + y_p) \\ \dot{\tilde{y}} = h(\tilde{x})(\tilde{y} + y_p) - \varphi(\tilde{y} + y_p, \tilde{x})(\tilde{y} + y_p) + \varphi(y_p, 0)y_p - m\tilde{y}. \end{cases} \tag{5.22}$$

From here on, we concentrate on the evolution of the $\tilde{x}$ variable, and follow the footsteps of the proof of Theorem 2 in which the x dynamics are identical to the ones here. Identical developments lead to (3.23)

$$\int_0^T [S_g - y_p(\tau)]\, d\tau < 0.$$

Since we do not have an explicit expression of $y_p(t)$, this sufficient condition for global convergence to the pest-free solution is satisfied if it is so while replacing y_p with its lower-bound. We then have the condition

$$S_g T - \int_0^T \left[\frac{\mu T}{1 - e^{-(C+m)T}} e^{-(C+m)(t \mod T)} \right] < 0$$

In computing the integral and isolating μ, we obtain condition (5.20b)

$$\mu > (C + m) S_g$$

for global convergence of x to 0. Convergence of y to y_p follows from similar arguments as in previous chapters. Noticing that (5.20a) is satisfied when (5.20b) is concludes the proof of GAS.

□

5.4 Mechanism III: Diet, alternate prey & intraguild predation

5.4.1 The premises

Using one predator species

Part of the predator population form an integral component of the predator diet. Predators cannibalize to recover nutrients not commonly found in their environment. In other words, both the prey and the cannibal victims provide alternate diet sources to the predator, so that both take part to the same extent in the predator repletion or the limited handling time predators can devote to feeding. Cannibalism influences the way predators consume the preys (unlike other forms of cannibalisms which do not tend to perturb predation). On the basis of heuristics presented in Kohlmeier and Ebenhoh (1995), this model may also be used to describe *intraguild predation*: the predation of one species on another is modelled as cannibalism within the predator guild.

When diet motivates cannibalism, this means that the predator preys equally on the pest and vulnerable conspecifics so the presence of either food source will hinder the exploitation of the other. The presence of pests lowers cannibalism while the presence of vulnerable conspecifics reduces pest uptake: the conspecifics are actual desired food for the predators and not only a means for survival in case of absence of preys. Alternatively, this can be a model of interfering predator behaviour involving both *cannibalism* and *intrapredator competition* for the prey and cannibal victims.

Schausberger (2003) reports that this behaviour is common among specialist predatory mites like *Phytoseiulus persimilis* and *Phytoseiulus macropilis* (Banks) which have a limited diet range. In particular, he reports from Walzer et al. (1999) and Schausberger and Croft (2000) that both these species develop on an exclusive cannibalistic diet as fast as on their primary prey, spider mites.

Using a guild of predators

Several species of predators, that is a guild of predators rather than one predator species, are sometimes used to control a pest. Reasons vary as to why this is done: predators are used in tandem to control wider range of pest densities than if each is used alone. For instance, phytoseiids which are more aggressive predators when pest density is high are often used with stigmaeidae which are more efficient at low pest populations (Gerson, 2007). Some predator species also attack specific life stages of the pest and are used with other predators that are effective predators of other life stages. Also, different species of predators have different resistance patterns to pesticides, and using both improves chance of survival of at least one group after chemical application.

Within the predator guild, one of the predator species (an IG predator) usually also predates on another (an IG prey), a process referred to as *intraguild predation* (IGP). Examples of species involved in IGP systems are the phytoseiid mite *Neoseiulus cucumeris* (Oudemans), which feeds on the thrips *Franklinothrips vespiformis* (Crawford), and their shared prey, *Frankliniella occidentalis* (Pergande), in rose greenhouses (Hervouet, 2006).

Kohlmeier and Ebenhoh (1995) proposed a similar model within the fisheries context where they group several species of predator within one category (or state variable). Within this framework, competition for the different predator species for a shared prey and inter-predator predation correspond to intragroup competition and a pseudo-cannibalism respectively. We may use a similar approach in intraguild predation (IGP) networks arising in biological control programs (see Rosenheim et al. (1995); Holt and Polis (1997); Bampfylde and Lewis (2007); Briggs and Borer (2005) for biological instances and modelling approaches to intraguild predation).

In the predator group, we may then consider two natural enemy species: the intraguild predator and the intraguild prey, which we group as one predator. The intraguild predator feeding on the intraguild prey counts as *cannibalism*. The released predators consist of an unspecified mix of both species or only one of them (see Figure 5.4). The model for apparent cannibalism from the grouping of IG predators and IG preys into one predator class is identical to that of pure diet-related cannibalism. So the results of our analyses for such a diet-related cannibalism model are in fact applicable to both cases.

5.4.2 The model and specific hypotheses

We can represent schematically diet-related cannibalism as in Figure 5.5:

We consider in our model that cannibalism and prey predation will then each be limited by the presence of the other food source ($g(x,y)$ with $\frac{dg}{dy} < 0$ and $\varphi(y,x)$ with $\frac{d\varphi}{x} < 0$). We use sector conditions to bound the arguments of the trophic response as in Chapter 4. Thus we propose a generalisation of Kohlmeier and Ebenhoh (1995)'s formulation as:

$$\left\{\begin{array}{l} \dot{x} = f(x) - g\left(\frac{ax}{1+by}\right) y \\ \dot{y} = h\left(\frac{ax}{1+by}\right) y - \varphi(y,x)y - my \\ y(nT^+) = y(nT) + \mu T, \quad \forall n \in \mathbb{N} \end{array}\right\} t \neq nT \qquad (\mathcal{M}_{q(iii)})$$

where $a, b > 0$. This model is very similar to Model ($\mathcal{M}_b$) with the addition of a cannibalism term $\varphi(y,x)$. The forms of the functional and numerical responses have a slightly different justification: with the conspecifics being part of the diet of the predators, a high density of predators implies that the predators satisfy their need for food by eating their conspecifics, so that they do not eat the preys much. This behaviour, which is intrinsically related to cannibalism, mathematically translates into interference between predators for the access to the preys.

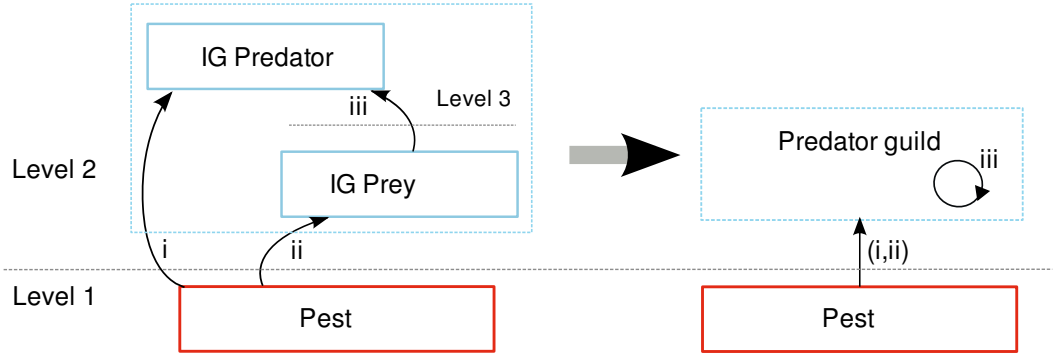

Figure 5.4: Intraguild predation is often observed among predator species used to control pests. In addition to the predation on the prey, and the competition between the predators for the prey, one predator species (usually a generalist species) can predate on the other. In his fisheries model involving numerous species of predator (and prey), Kohlmeier and Ebenhoh (1995) propose to merge predators in one group and model the trophic interactions among them as cannibalism. This simplification provides a preliminary approach in modelling the use of multiple predators and the effect of intraguild predation in an augmentative control program.

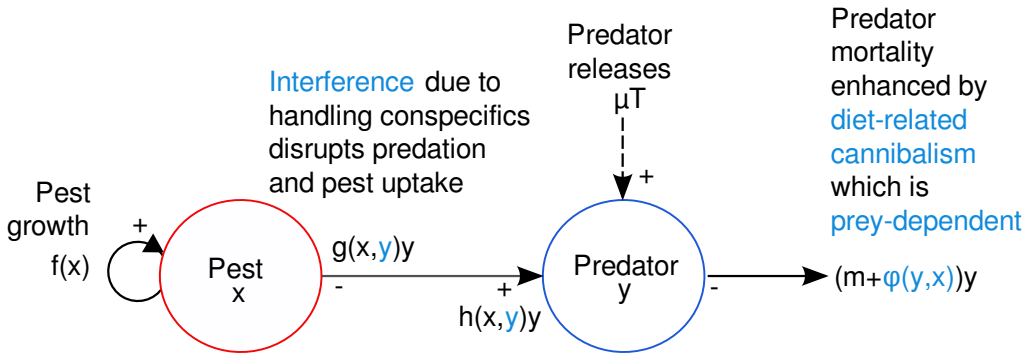

Figure 5.5: Diet-related cannibalism impacts both the trophic responses and the mortality of predators. The trophic responses are penalised by conspecific handling and searching. Cannibalism is also mitigated due to pest handling by the predator. In an intraguild predation model, the trophic responses represent the transfer from the pest population to the predator guild which consists of more than one predator species. The cannibalism function represents the consumption of the intraguild prey species by the intraguild predator species.

Specific hypotheses for diet-related cannibalism or IGP
Limited pest growth:

$$\exists\, \bar{x} \;\text{ such that } \forall\, x \geq \bar{x}, \quad f(x) < 0; \tag{H5}$$

Limited per capita pest growth:

$$f(x)/x \;\text{ is upper bounded } \forall\, x \geq 0, \text{such that } \; S = \sup_{x \geq 0}(f(x)/x); \tag{H6}$$

Bounded cannibalism:

$$\frac{\partial \varphi}{\partial y}(y, 0) \geq 0 \text{ and } \exists C > 0, \forall y \geq 0 : 0 \leq \varphi\,(y, 0) \leq C \tag{H8}$$

Hypotheses (H5) and (H6) are the ones that were already necessary for model $\mathcal{M}_b$ describing interference for access to the preys through sector conditions. We do not go through the length of describing the present result with sector conditions and limit it to the specific interference forms $g\left(\frac{ax}{1+by}\right)$ and $h\left(\frac{ax}{1+by}\right)$. An extension to sectors similar to that presented in Chapter 4 could readily be made.

5.4.3 Existence and stability of the zero-pest solution

It is straightforward to see that the zero-pest solution has the same form as for hunger-motivated cannibalism since the zero-pest subsystem is identical in both cases. We only state this solution below; for the proof please see Proposition 7.

Proposition 8 (Existence, diet and intraguild predation)
Let general hypotheses (H1), (H2‡) and (H3‡), and specific hypotheses (H7), and (H8) be satisfied. Then Model ($\mathcal{M}_{q(iii)}$) possesses a unique periodic pest-free solution whose y coordinate is bounded as

$$\frac{\mu T}{1 - e^{-(C+m)T}} e^{-(C+m)(t \mod T)} \leq y_p(t) \leq \frac{\mu T}{1 - e^{-mT}} e^{-m(t \mod T)} \tag{5.23}$$

We have seen in **Section 5.3** that the stability conditions were identical between the "Hunger cannibalism" and the simple model of Chapter 3. In the current case, we see that the same holds when comparing "diet cannibalism" and the interference model of Chapter 4, so that the Theorem is similar, except that the lower-bound of y_p needs to be considered in order to compute the conditions

Theorem 10 (Stability, Diet and intraguild predation)
The solution $(x_p(t), y_p(t)) = (0, y_p(t))$ of Model ($\mathcal{M}_{q(iii)}$) is LAS if

$$f'(0) < \frac{ag'(0)}{b} \tag{5.24a}$$

and if

$$\mu > \frac{1}{b}\left(\frac{1 - e^{-(C+m)\frac{bf'(0)}{ag'(0)}T}}{e^{-(C+m)\frac{bf'(0)}{ag'(0)}T} - e^{-(C+m)T}}\right)\frac{1 - e^{-(C+m)T}}{T} \tag{5.24b}$$

The solution is unstable if

$$f'(0) > \frac{ag'(0)}{b} \tag{5.24c}$$

or

$$\mu > \frac{1}{b}\left(\frac{1 - e^{-m\frac{bf'(0)}{ag'(0)}T}}{e^{-m\frac{bf'(0)}{ag'(0)}T} - e^{-mT}}\right)\frac{1 - e^{-mT}}{T} \tag{5.24d}$$

Proof: The proof is the same as that of Theorem 4, except that, when reaching equation (4.16)

$$\int_0^T \frac{\alpha'(0)y_p(\tau)}{\beta(y_p(\tau))}d\tau > \frac{f'(0)}{g'(0)}T,$$

which is a neccessary and sufficient condition for Local Asymptotic Stability, we cannot compute it directly and, since the argument of the integral is increasing in y_p, we can replace $y_p(t)$ by its lower-bound from (5.23) to obtain a sufficient condition for stability. The remainder of the proof is unchanged and directly yields (5.24a) and (5.24b).

The instability conditions is in the line of Proposition 5 where we need to replace y_p with its upper-bound in the proof to obtain (5.24c) and (5.24d).

□

Remark: *The extra term obtained because of the nonlinear cannibalism term has no effect on the biological conditions: as shown after the proof of Theorem 4 this biological condition is strictly related to the $\dot{x}$ dynamics and the capacity of the predators to control the preys, independently of the predator dynamics. The managerial instability condition (5.24d) is identical to the one without cannibalism. It is normal that it is not any better, since cannibalism will reduce the predator population; however, since our lower-bound on cannibalism is 0, there is no basis for the instability condition to be worse.*
The minimal stability rate resulting from our analysis has the same form as the minimal release rate in Chapter 4, with the natural mortality of the predators enhanced by cannibalism or the consumption of the intraguild prey by the intraguild predator; larger releases have to be carried out in order to counter the additional predator mortality related to cannibalism; since cannibalism is here upper-bounded by a linear function, the general form of the condition is unchanged. ■

5.4.4 The Kohlmeier- Ebenhöh model

We have justified the introduction of this family of models by relating it to the model proposed in Kohlmeier and Ebenhoh (1995), which we will now use to particularise the result that we just obtained:

$$\left\{\begin{array}{l} \dot{x} = rx\left(1-\dfrac{x}{K}\right) - \dfrac{\gamma x}{1+\nu(x+\eta y)}y \\ \dot{y} = \dfrac{\gamma'(x+\eta y)-\gamma\eta y}{1+\nu(x+\eta y)}y - my \\ y(nT^+) = y(nT) + \mu T, \quad \forall n \in \mathbb{N} \end{array}\right\} t \neq nT \tag{5.25}$$

where the preys have a logistic growth, the available food for the predator is $x+\eta y$ (with η the preference of the predator for cannibalism), and γ/ν and γ'/ν are respectively the maximal food uptake rate and the maximum assimilation rate (with $\gamma' < \gamma$).

In order to apply Theorem 10, we need to put model (5.25) in the framework of model ($\mathcal{M}_{q(iii)}$). Prey growth $f(x) = rx\left(1-\dfrac{x}{K}\right)$ (so that $S = r$), predator mortality $-my$ and cannibalism $\varphi(y,x) = \dfrac{\gamma\eta y - \gamma'\eta y}{1+\nu(x+\eta y)}$ are easily identified. This last term satisfies (H8) since $\varphi(y,0) = \dfrac{\gamma\eta y - \gamma'\eta y}{1+\nu\eta y} \leq \frac{\gamma-\gamma'}{\nu} \triangleq C$ and $\frac{d\varphi}{dy}(y,0) > 0$. The functional response is then obtained by taking $g(s) = \frac{\gamma}{\nu}\frac{s}{s+1}$, $a = \nu$ and $b = \nu\eta$. Conditions (5.24a) and (5.24b) then write as

$$r < \frac{\gamma}{\nu\eta} \tag{5.26}$$

and

$$\mu > \frac{1}{\nu\eta}\left(\frac{1-e^{-(\frac{\gamma-\gamma'}{\nu}+m)\frac{\nu\eta r}{\gamma}T}}{e^{-(\frac{\gamma-\gamma'}{\nu}+m)\frac{\nu\eta r}{\gamma}T}-e^{-(\frac{\gamma-\gamma'}{\nu}+m)T}}\right)\frac{1-e^{-(\frac{\gamma-\gamma'}{\nu}+m)T}}{T}$$

5.5 The pest control strategy

5.5.1 Predator releases

When cannibal predators are territorial

When predators are territorial, we have shown that, for a given release period, pest-eradication could be achieved for a large enough release rate. Necessary condition (5.13) points more precisely to the fact that, the larger the T, the larger μ will need to be because the right-hand-side increases linearly in T, while the left-hand-side only increases logarithmically, which should be compensated by μ. This point at the necessity of not taking T too large to keep μ in check. Similarly, if μ is fixed, T has a maximal value that it can take without violating (5.13). All this is quite natural indeed; taking large values of T implies that large quantities of predators are released at the same time; this produces intensive cannibalism right after the release, and thus wastes biocontrol agents.

When cannibalism is hunger-driven

Not much is different with respect to what was shown in Chapter 3: we can only bound hunger effects linearly and therefore obtain a T-independent bound on the release rate required for pest eradication. The cannibalism effect only enhances mortality, so that the bound is large than in the absence of cannibalism, but it does not fundamentally changes. An optimised minimal risk strategy as in Mailleret and Grognard (2006, 2009) would require small frequent releases as was shown in Section 3.3.2.

When cannibalism is part of the diet

The same analysis as for the simple model in **Chapter 3** is conducted. As for the IGP model or when cannibalism is motivated by dietary requirements, the minimal release rate has the same form as the interference model, with the mortality effect enhanced by cannibalism. Smaller release periods are to be favoured as they lower the minimal release rate and increase the speed of pest eradication. From that point of view, this form of cannibalism is similar to the territorial one. The main difference lies in the fact that, in the case of territoriality, for any T, one can always find μ large enough such that pest-eradication, even if it means wasting a lot of them in cannibalistic encounters, while in the present case, there is a biological condition that needs to be satisfied for that to be possible. This is visible as the biological condition for the Kohlmeier-Ebenhöh model (5.26): if the slope of the functional response at low pest densities ($\frac{\gamma}{\nu}$) is too weak or - and this is quite natural - if the predators have a too large preference in consuming conspecifics rather than preys (large η), no pest-eradication can be achieved.

5.5.2 The selection of the predator species

Low cannibalism levels

As expected, we should favour biocontrol agents that have small cannibalism levels: this is directly apparent on conditions (5.20a) and (5.20b) for the hunger related cannibalism: the

lower the cannibalism parameter, the smaller the predator release rate required. This is also directly clear in the overcrowding case with T small by inspecting the influence of q on (5.14a)-(5.14b).

Low cannibalism preference

As we have seen, the cannibalism preference is central in the possibility of controlling the pest while using a cannibalist predator that has its conspecifics in its own diet or an IGP system: quite naturally, we should favour predators whose diet favor the preys or IGP system whose IG predators favor the pests over the IG prey.

6
Partial harvest effects

Overview

Over their growing period, many agricultural crops, such as greenhouse grown flowers and vegetables, are partially harvested on a regular basis (see e.g. Gerson and Weintraub (2007); Schumacher et al. (2006) for such crops). This practice can remove a proportion of the insects - including the predatory ones - present throughout the plantation.

In this chapter, we explicitly account for the discrete nature of crop harvesting and, like predator releases, model it as an *instantaneous change* occuring *periodically*. We consider the harvest period as a reference since it is set by crop growth and market constraints. We investigate in particular how the frequency of releases is to be varied with respect to the harvesting frequency to minimise the minimal budget value.

Keywords: Periodic harvests, Integrated Pest Management (IPM), multiple integer periods

Organization of this chapter:

This chapter is organised as follows:

Section 6.2 describes the model and its biological premises.

Section 6.3 presents two forms of the pest-free solution: first in the case when releases occur more often than partial harvests, and second, when they are less frequent.

Section 6.4 identifies the conditions for the global and local stability of the pest-free solutions.

Section 6.5 investigates the consequences of the previous mathematical results on the strategy of pest control to employ. We look first at how the release period relative to the partial harvest influences the minimal predator release required for pest eradication. Then, we identify the partial harvesting parameters that would either contribute to or interfere with pest control by the predator releases.

Section 6.6 provides a summary of the mathematical results presented in this chapter and their practical interpretation, and the perspectives for extending the analysis to the case where neither period (release or partial harvest) is the integer multiple of the other.

6.1 The premises

Partial population removals

Harvest has already been shown in Volterra (1978); Negi and Gakkhar (2007) among others to influence, even counter-intuitively, predator-prey dynamics. Volterra (1978) for instance shows that adding a continuous harvest rate to both prey and predator populations would benefit the prey population and repress the predator population.

The effect of partial harvesting is similar to that of pesticide usage proposed by Liu et al. (2005b) in their Integrated Pest Management (IPM) strategy. Like predator releases, it is modelled as an *instantaneous change* occuring *periodically*. They investigate in particular how the frequency of releases is to be varied with respect to the harvesting frequency to minimise the minimal budget value.

There are two aspects that are taken into account in including partial harvesting into our model:

1. Firstly, both the predator and pest populations are subjected to partial harvesting when it occurs, which involves a proportional decrease in the respective populations. In particular, since pest and predator are likely to occupy different parts of a plant, we allow the strength of the removal to be different for each population.The effect of partial harvesting is similar to that of pesticide usage proposed by Liu et al. (2005b) in their Integrated Pest Management (IPM) strategy.

2. Secondly, one period (the predator releases or the harvest one) is taken as the integer multiple of the other. This feature is a key for calculating the stability condition to obtain the minimal budget value and include the case where the frequencies of predator releases and of crop harvests are not the same. Although accounting for different discrete phenomena occuring at the same frequency but not at the same instants is quite common (see *e.g.* Liu et al. (2004, 2005b, 2006); Zhang et al. (2007)), we know of only one other contribution describing discrete phenomena occuring at different moments and periods by Terry (2010).

Remark: *In the present chapter, we will focus our discussion around partial harvest effects. Pesticide usage is discussed in the Chapter 7.* ■

6.2 The model

The processes involved in this system are represented schematically in Figure 6.1. Partial harvesting is considered to be instantaneous, hence the discontinuous arrows.

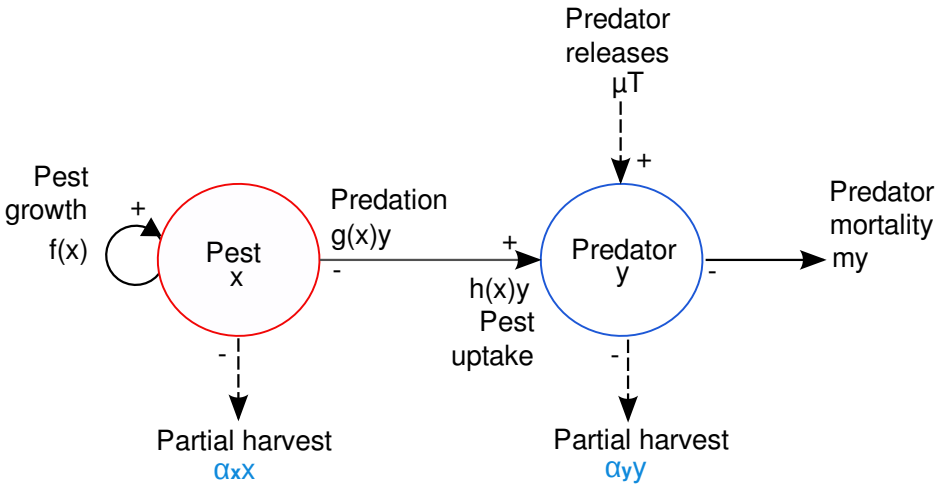

Figure 6.1: System schematic including partial harvesting on the predator and prey populations. The process is impulsive hence depicted in broken arrow.

Following the modelling framework detailed in the previous chapters, we get:

$$\left\{\begin{array}{ll} \dot{x} = f(x) - g(x)y & \\ \dot{y} = h(x)y - my & \\ x(vT_h^+) = (1-\alpha_x)x(vT_h) & \forall v \in \mathbb{N} \\ y(vT_h^+) = (1-\alpha_y)y(vT_h) + \delta\left(vT_h \bmod T_r\right)\mu T_r & \forall v \in \mathbb{N} \\ y(nT_r^+) = \left(1-\delta\left(nT_r \bmod T_h\right)\alpha_y\right)y(nT_r) + \mu T_r & \forall n \in \mathbb{N} \end{array}\right. \tag{$\mathcal{M}_v$}$$

Partial crop harvests and predator releases occur respectively every T_h and T_r time units. α_x and α_y represent the respective proportions of the prey and predator populations affected at each harvest. These parameters are allowed be different since in reality, it might be the case that each species occupies preferentially different parts of the plant. We also assume that the insects are uniformly distributed throughout our plantation so that the effect of partial harvesting is directly correlated with the number of plants harvested. We assume that the crop maturates at a constant velocity so the proportion of crops harvested each time and hence insects removed is considered as fixed. The δ-function, such that

$$\delta(0) = 1 \text{ and } \delta(z) = 0, \forall z \neq 0$$

is used thus to identify instants of simultaneous partial harvest and predator release. We note that while in our analysis we consider only the case when either period is the integer of the other, this formulation extends to many more possibilities (including irrational ratios between the periods).

As in the basic model, the functions $f(x)$, $g(x)$ and $h(x)$ are all locally Lipschitz continuous for $x, y \geq 0$. They are qualitatively defined according to Hypotheses (H1)-(H4) below. The prime indicates their derivative with respect to their argument, x.

General hypotheses

Pest growth at zero pest:

$$f(0) = 0 \quad \text{and} \quad f'(0) > 0 \tag{H1}$$

Functional response:

$$g(0) = 0, \quad g'(0) > 0, \quad \text{and} \quad \forall x > 0,\ g(x) > 0; \tag{H2}$$

Numerical response:

$$h(0) = 0 \quad \text{and} \quad \forall x > 0,\ h(x) \geq 0 \tag{H3}$$

Here, the ratio of the pest growth to functional response is upper bounded for all x.

Specific hypothesis
Ratio of pest growth to functional response:

$$\frac{f(x)}{g(x)} \text{ is upper bounded for } x \geq 0 \text{ such that } S_g = \sup_{x \geq 0} \left(\frac{f(x)}{g(x)} \right). \tag{H4}$$

Specific notation
Because of its Lipschitz continuity, the slope of the functional response – also the functional response per pest – can be upper bounded. We denote this upper bound as:

$$r = \sup_{x \geq 0} \left(\frac{g(x)}{x} \right) \tag{N1}$$

Period of reference and point of reference

We demonstrate that impulsive forcing by releases and harvests will provoke the predator population to reach a periodic pattern of period equal to $\max(T_r, T_h)$, which we shall refer to as the *period of reference*. The instant following a coinciding partial harvest and release is taken as the *point of reference*.

6.3 Existence of the zero-pest solution

In the absence of pest, the predator population will vary according to the number of predators manually injected into the system and also according to the partial harvest effect. The absence of their source of food will cause an exponential decay of the population.

We consider in this section first the case when the period of reference is the harvesting period, divided into subintervals between two releases, and then when the period of reference is the release period, divided into subintervals between two partial harvests.

6.3.1 Releases more frequent than harvests

When releases occur more frequently than partial harvests, the pest-free solution of System ($\mathcal{M}_v$) is calculated as follows. First we state the result.

Proposition 9 (Existence, releases more frequent) *Let $T_h = kT_r$ where $k \in \mathbb{N}^*$ and Hypotheses (H1)-(H3) be satisfied. Then, in the absence of pests, Model ($\mathcal{M}_v$) possesses a globally stable periodic solution*

$$\begin{aligned} &(x_{ph}(t), y_{ph}(t)) \\ &= \left(0, y^* e^{-m(t \mod T_h)} + \mu T_r e^{-m(t \mod T_r)} \sum_{j=0}^{\lfloor \frac{t \mod T_h}{T_r} \rfloor - 1} e^{-jmT_r} \right) \end{aligned} \tag{6.4a}$$

where

$$y^* = \frac{\left((1 - e^{-mT_h})(1 - \alpha_y) + (1 - e^{-mT_r})\alpha_y \right) \mu T_r}{(1 - (1 - \alpha_y)e^{-mT_h})(1 - e^{-mT_r})} \tag{6.4b}$$

Proof: When $T_h = kT_r$, in the absence of pests and using Hypotheses (H1)-(H3), the system is simplified to

$$\begin{cases} \dot{x} = 0 \\ \dot{y} = -my \\ x(nT_r^+) = (1 - \delta\,(n \bmod k)\,\alpha_x)x(nT_r) \\ y(nT_r^+) = (1 - \delta\,(n \bmod k)\,\alpha_y)y(nT_r) + \mu T_r, \quad \forall n \in \mathbb{N} \end{cases} \tag{6.5}$$

The solution $x_{ph}(t) = 0$ is trivial. To calculate $y_{ph}(t)$, we first show by induction that the predator population right after a release can be expressed in terms of the point of reference as follows

$$y(vT_h + iT_r^+) = y(vT_h^+)e^{-imT_r} + \mu T_r \sum_{j=0}^{i-1} e^{-jmT_r} \tag{6.6}$$

where $i \in [0, 1, \ldots, (k-1)]$

It is seen that (6.6) is valid for $i = 0$ since it is equal to

$$y(vT_h^+) = y(vT_h^+)e^0 + \mu T_r \sum_{j=0}^{-1} e^{-jmT_r} = y(vT_h^+)$$

Now suppose that (6.6) holds for $i = i_\star$ where $i_\star \in [0, 1, \ldots, k-2]$, i.e.

$$y(vT_h + i_\star T_r^+) = y(vT_h^+)e^{-i_\star mT_r} + \mu T_r \sum_{j=0}^{i_\star - 1} e^{-jmT_r}$$

We will now show that (6.6) is valid for $i = i_\star + 1$. We calculate $y(vT_h + (i_\star + 1)T_r^+)$ from $y(vT_h + i_\star T_r^+)$ using (6.5), and get

$$\begin{aligned} y(vT_h + (i_\star + 1)T_r^+) &= y(vT_h + i_\star T_r^+)e^{-mT_r} + \mu T_r \\ &= \left(y(nT_h^+)e^{-i_\star mT_r} + \mu T_r \sum_{j=0}^{i_\star - 1} e^{-jmT_r} \right) e^{-mT_r} + \mu T_r \\ &= y(nT_h^+)e^{-(i_\star+1)mT_r} + \mu T_r \sum_{j=1}^{i_\star} e^{-jmT_r} + \mu T_r \\ &= y(vT_h^+)e^{-(i_\star+1)mT_r} + \mu T_r \sum_{j=0}^{i_\star} e^{-jmT_r} \end{aligned}$$

so that (6.6) holds true for $i \in [0, 1, \ldots, k-1]$.

To evaluate the evolution of y according to the period of reference T_h, we need to calculate the value of $y((v+1)T_h^+)$, which is equivalent to $y(vT_h + kT_r^+)$, in terms of $y(vT_h^+)$. At this point however, partial harvesting takes place before predator release; so we first express it in terms of $y(vT_h + (k-1)T_r^+)$ then expand the expression using (6.6) as follows

$$\begin{aligned} y\left((v+1)T_h^+\right) &= y\left(vT_h + (k-1)T_r^+\right) e^{-mT_r}(1 - \alpha_y) + \mu T_r \\ &= \left(y(vT_h^+)e^{-m(k-1)T_r} + \mu T_r \sum_{j=0}^{k-2} e^{-jmT_r} \right) e^{-mT_r}(1 - \alpha_y) + \mu T_r \\ &= y(vT_h^+)e^{-mT_h}(1 - \alpha_y) + \mu T_r(1 - \alpha_y) \sum_{j=1}^{k-1} e^{-jmT_r} + \mu T_r \\ &= y(vT_h^+)e^{-mT_h}(1 - \alpha_y) + \mu T_r \left((1 - \alpha_y) \sum_{j=0}^{k-1} e^{-jmT_r} + \alpha_y \right) \end{aligned}$$

Note that the summation term can also be evaluated so the sequence can be expressed as

$$y\left((v+1)T_h^+\right) = (1-\alpha_y)y(vT_h^+)e^{-mT_h} + \mu T_r\left((1-\alpha_y)\frac{1-e^{-mT_h}}{1-e^{-mT_r}} + \alpha_y\right) \tag{6.7}$$

In this linear dynamical system, the coefficient of $y(vT_h^+)$, $(1-\alpha_y)e^{-mT_h}$ is less than one in magnitude, so the sequence will converge to a limit, the equilibrium of (6.7). This equilibrium yields (6.4b) and the convergence of $y(t)$ to a periodic solution $y_{ph}(t)$ based on y^*.

Now that we have established the existence of the periodic solution $y_{ph}(t)$, we seek to formulate it. We focus on a reference period over $vT_h < t \leq (v+1)T_h$ during which $y_{ph}(t)$ is piecewise continuous, with the continuous components separated by predator releases. The continuous intervals are defined over $vT_h + iT_r < t \leq vT_h + (i+1)T_r$ where $i \in [0, 1, \ldots, k-1]$. For a given value of t, the value of i is easily identified as being $i = \lfloor \frac{t \mod T_h}{T_r} \rfloor$. The value of $y_{ph}(t)$ is then of the form

$$y_{ph}(t) = y_{ph}(nT_h + iT_r^+)e^{-d(t \mod T_r)}$$

and, from (6.6) with $y(nT_h^+) = y^*$, we have that

$$y_{ph}(vT_h + iT_r^+) = y^*e^{-imT_r} + \mu T_r \sum_{j=0}^{i-1} e^{-jmT_r}$$

so that

$$\begin{aligned} y_{ph}(t) &= \left(y^*e^{-imT_r} + \mu T_r \sum_{j=0}^{i-1} e^{-jmT_r}\right) e^{-d(t \mod T_r)} \\ &= y^*e^{-m(t \mod T_h)} + \mu T_r e^{-m(t \mod T_r)} \sum_{j=0}^{i-1} e^{-jmT_r} \\ &= y^*e^{-m(t \mod T_h)} + \mu T_r e^{-m(t \mod T_r)} \sum_{j=0}^{\lfloor \frac{t \mod T_h}{T_r} \rfloor - 1} e^{-jmT_r} \end{aligned}$$

This is the form proposed in (6.4a), thereby completing our proof. □

Figure 6.2 illustrates $y_{ph}(t)$. The piecewise continuity is exploited in the calculation of its integral over a period $(0, T)$ (see Section 6.4.2).

6.3.2 Releases less frequent than harvests

When harvesting is more frequent than the release of predators, we have a similar result about the existence of a periodic solution.

Proposition 10 (Existence, releases less frequent) *Let $T_r = kT_h$ where $k \in \mathbb{N}^*$ and Hypotheses (H1)-(H3) be satisfied. Then, in the absence of pests, Model ($\mathcal{M}_v$) possesses a globally stable periodic solution*

$$(x_{pr}(t), y_{pr}(t)) = \left(0, y^*e^{-m(t \mod T_r)}(1-\alpha_y)^{\lfloor \frac{t \mod T_r}{T_h} \rfloor}\right) \tag{6.8a}$$

where

$$y^* = \frac{\mu T_r}{1-(1-\alpha_y)^k e^{-mT_r}} \tag{6.8b}$$

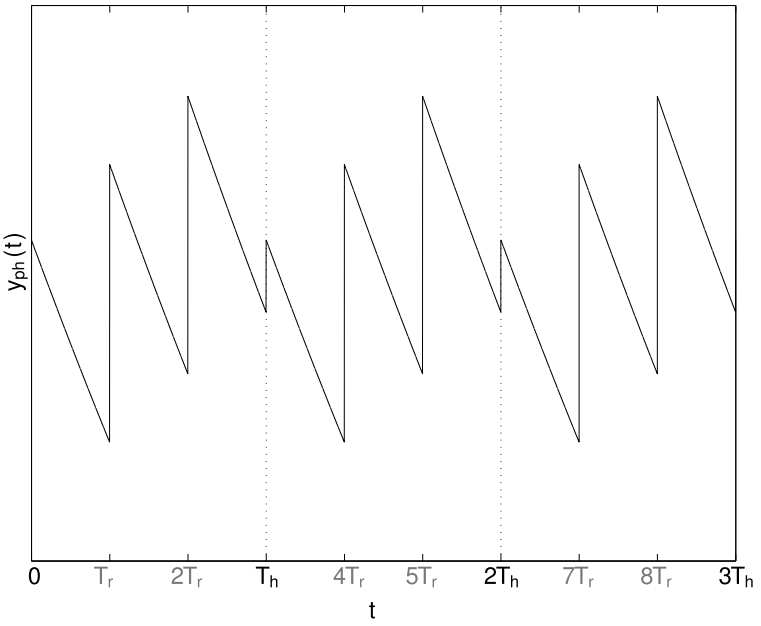

Figure 6.2: Form of the periodic solution $y_{ph}(t)$ in the case where $k = 3$. Harvests are apparent at every vT_h instant, while the release of predators dominates the harvest at every nT_r instant. Between those instants, the population decays exponentially since it has no prey to feed on.

Proof: When $T_r = kT_h$, in the absence of pests and using Hypotheses (H1)-(H3), the system is simplified to

$$\begin{cases} \dot{x} = 0 \\ \dot{y} = -my \\ x(vT_h^+) = (1-\alpha_x)x(vT_h) \\ y(vT_h^+) = (1-\alpha_y)y(vT_h) + \mu T_r \delta\left(v \bmod k\right), \quad \forall v \in \mathbb{N} \end{cases} \tag{6.9}$$

As previously explained, $x_{pr}(t)$ is solved for trivially as being

$$x_{pr}(t) = 0$$

We prove that the predator population again reaches a periodic solution. This time, however, the *period of reference* is T_r. The *point of reference* is the instant after a coinciding harvest and release, with the release occurring right after the partial harvest. We show by induction that the population after a harvest can be expressed as

$$y(nT_r + iT_h^+) = y(nT_r^+)e^{-imT_h}(1-\alpha_y)^i \tag{6.10}$$

where $i \in [0, 1, \ldots, (k-1)]$.

It is seen that (6.10) is valid for $i = 0$ since it resumes to

$$y(nT_r^+) \quad = \quad y(nT_r^+)e^0(1-\alpha_y)^0$$

Suppose (6.10) holds for $i = i_\star$ where $i_\star \in [0, 1, \ldots k-2]$, i.e.

$$y(nT_r + i_\star T_h^+) \quad = \quad y(nT_r^+)e^{-i_\star mT_h}(1-\alpha_y)^{i_\star} \tag{6.11}$$

We will now show that (6.11) is valid for $i = i_\star + 1$. We calculate the value of y when $i = i_\star + 1$ in terms of $y(nT_h + i_\star T_r^+)$, knowing from $\dot{y} = -my$ in System (6.9) it will be an exponential decay with the added component for the harvest. Using (6.11), we then get

$$\begin{aligned} y(nT_r + (i_\star + 1)T_h^+) &= y(nT_r + i_\star T_h^+)e^{-mT_h}(1-\alpha_y) \\ &= \left(y(nT_r^+)e^{-i_\star mT_h}(1-\alpha_y)^{i_\star}\right) e^{-mT_h}(1-\alpha_y) \\ &= y(nT_r^+)e^{-m(i_\star+1)T_h}(1-\alpha_y)^{i_\star+1} \end{aligned} \tag{6.12}$$

This is clearly the same form given from the expression in (6.10), thereby validating it.

y^* is given as the fixed point of the sequence representing post-release instants. Therefore, using (6.10) for $i = k$ and model (6.9), we next calculate $y((n+1)T_r^+)$ as

$$\begin{aligned} y((n+1)T_r^+) &= y(nT_r + kT_h^+) \\ &= y(nT_r^+)e^{-kmT_h}(1-\alpha_y)^k + \mu T_r \\ &= y(nT_r^+)e^{-mT_r}(1-\alpha_y)^k + \mu T_r \end{aligned}$$

In this linear dynamical system, the coefficient of $y(nT_r^+)$, $e^{-mT_r}(1-\alpha_y)^k$ is less than one in magnitude, which confirms the existence of the fixed point y^* to which the sequence converges. This equilibrium (6.8b) yields the convergence of $y(t)$ to a periodic solution $y_{pr}(t)$.

Now that we have established the existence of the periodic solution $y_{pr}(t)$, we seek to formulate it. We focus on a reference period over $nT_r < t \leq (n+1)T_r$ during which $y_{pr}(t)$ is piecewise continuous, with the continuous components separated by harvests. The intervals of continuity span $nT_r + iT_h < t \leq nT_r + (i+1)T_h$ where $i \in [0, 1, \ldots, k-1]$. For a given value of t, the value of i is easily identified as being $i = \lfloor \frac{t \mod T_r}{T_h} \rfloor$. The value of $y_{pr}(t)$ is then of the form

$$y_{pr}(t) = y_{pr}(nT_r + iT_h^+)e^{-m(t \mod T_h)}$$

and, from (6.10) with $y(nT_r^+) = y^*$, we have that

$$y_{pr}(nT_r + iT_h^+) = y^* e^{-imT_h}(1-\alpha_y)^i$$

so that

$$\begin{aligned} y_{pr}(t) &= \left(y^* e^{-imT_h}(1-\alpha_y)^i\right) e^{-m(t \mod T_h)} \\ &= y^* e^{-m(t \mod T_r)}(1-\alpha_y)^{\lfloor \frac{t \mod T_r}{T_h} \rfloor} \end{aligned}$$

which is exactly the expression given in (6.8a) and completes the proof. □

The form of the y_{pr} function is illustrated on Figure 6.3.

6.4 Stability of the pest-free solution

6.4.1 The methodology

We start by explaining the method and bounds used for finding the stability conditions, before applying it specifically to the case when releases are more frequent (Section 6.4.2) then when they are less so (6.4.3). We first define the deviation coordinates, which represent the difference between the actual state pair and the reference periodic solution:

$$\begin{aligned} \tilde{x}(t) &= x(t) - x_p(t) \\ \tilde{y}(t) &= y(t) - y_p(t) \end{aligned}$$

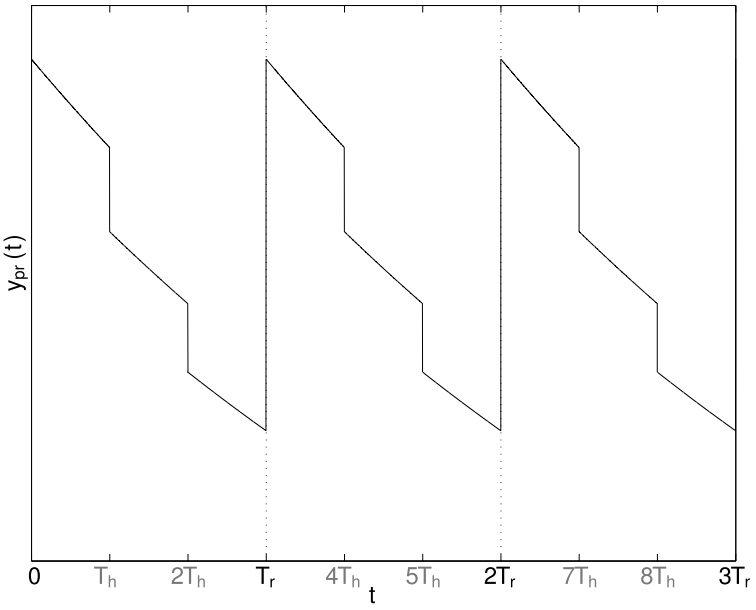

Figure 6.3: Form of the periodic solution $y_{pr}(t)$ in the case where $k = 3$. Harvests are apparent at every nT_h instant, while the release of predators dominates the harvest at every mT_r instant. Between those instants, the population decays exponentially since it has no prey to feed on.

In these new coordinates, the system becomes[1]

$$\begin{cases} \dot{\tilde{x}} = f(\tilde{x}) - g(\tilde{x})(\tilde{y} + y_p) \\ \dot{\tilde{y}} = h(\tilde{x})(\tilde{y} + y_p) - m\tilde{y} \\ \tilde{x}(vT_h^+) = (1 - \alpha_x)\tilde{x}(vT_h) \\ \tilde{y}(vT_h^+) = (1 - \alpha_y)\tilde{y}(vT_h) \end{cases} \tag{6.13}$$

We note that the partial harvesting effects are preserved, but that as in the previous sections, the releases disappear.

Then we bound $\tilde{y}$ as follows. Let the initial condition for system (6.13) be $(\tilde{x}_0, \tilde{y}_0)$ at time t_0, that is right after the harvest and the predator release. Note that since $\tilde{x} \geq 0$, $h(\tilde{x}) \geq 0$.

[1]The calculations are detailed here:

$$\begin{cases} \dot{\tilde{x}} &= f(x) - g(x)y - f(x_p) + g(x_p)y_p \\ &= f(\tilde{x}) - g(\tilde{x})(\tilde{y} + y_p) - f(0) + g(0)y_p = f(\tilde{x}) - g(\tilde{x})(\tilde{y} + y_p) \\ \dot{\tilde{y}} &= h(x)y - my - h(x_p)y_p + my_p \\ &= h(\tilde{x})(\tilde{y} + y_p) - m(\tilde{y} + y_p) - h(0)y_p + my_p = h(\tilde{x})(\tilde{y} + y_p) - m\tilde{y} \\ \tilde{x}(vT_h^+) &= x(vT_h^+) - x_p(vT_h^+) \\ &= (1 - \alpha_x)x(vT_h) = (1 - \alpha_x)\tilde{x}(vT_h) \\ \tilde{y}(vT_h^+) &= y(vT_h^+) - y_p(vT_h^+) \\ &= (1 - \alpha_y)y(vT_h) - (1 - \alpha_y)y_p(vT_h) = (1 - \alpha_y)\tilde{y}(vT_h) \\ \tilde{y}(nT_r^+) &= y(nT_r^+) - y_p(nT_r^+) \\ &= y(nT_r) + \mu T_r - (y_p(nT_r) + \mu T_r) = \tilde{y}(nT_r) \end{cases}$$

Moreover, $(\tilde{y} + y_p) \geq 0$ and $0 \leq (1 - \alpha_y) \leq 1$. So the dynamics can be bounded as:

$$\begin{cases} \dot{\tilde{y}} \geq -m\tilde{y} \\ \left|\tilde{y}(vT_h^+)\right| \leq |\tilde{y}(vT_h)| \end{cases}$$

Next, we know that at any moment, because of the positivity of the system,

$$\tilde{y}_0 \geq -y_p \geq -S_{y_p}$$

where $S_{y_p} = \sup_{t \geq 0} y_p(t)$ over the reference period T. Because they involve proportional *removal*, thus a decrease in magnitude, the impulsive component of the dynamics can be ignored. We can bound the $\tilde{y}$ dynamics as

$$\tilde{y}(t) \geq \tilde{y}_0 e^{-m(t-t_0)} \geq -S_{y_p} e^{-m(t-t_0)} \tag{6.14}$$

The aim of out analysis is to identify the conditions for which $\tilde{x}$ goes to zero. We are able to deduce subsequently that $\tilde{y}$ will also converge to zero, following a reasoning similar to those proposed in the previous chapters. The convergence of (x, y) to $(0, y_p)$ is then guaranteed for the abovementioned conditions. Similarly to what we've done in Chapter 3, we consider the integral

$$\int_{\tilde{x}_0}^{\tilde{x}} \frac{1}{g(s)} ds \tag{6.15}$$

which, in the limit that $\tilde{x}$ tends to zero, is

$$\lim_{\tilde{x} \to 0^+} \int_{\tilde{x}_0}^{\tilde{x}} \frac{1}{g(s)} ds = -\infty \tag{6.16}$$

This will thus provide the equivalent requirement for global convergence to the pest-free solution.

We point out here that if the integral starts and ends between two partial harvests, i.e. the initial and final times, t_0 and t, both lie within the same continuous interval $(vT_h^+, (v+1)T_h)$, we may use the $\dot{\tilde{x}}$-dynamics in (6.13) to express the integral (6.15) over the (continuous) time domain as

$$\int_{\tilde{x}_0}^{\tilde{x}} \frac{1}{g(s)} ds = \int_{t_0}^{t} \frac{\dot{\tilde{x}}}{g(\tilde{x})} d\tau = \int_{t_0}^{t} \left[\frac{f(\tilde{x})}{g(\tilde{x})} - (y_p + \tilde{y}) \right] d\tau \tag{6.17}$$

Over a harvest period, starting right after a partial harvest up to and including the subsequent one, we need to take care in computing (6.15) because, contrarily to what was done in the previous chapters, the x-dynamics is discontinuous: we need to take into account the discontinuity that occurs upon the partial harvest. The integral can be split into a continuous and a discrete component as shown below.

$$\begin{aligned} \int_{\tilde{x}(vT_h^+)}^{\tilde{x}((v+1)T_h^+)} \frac{1}{g(s)} ds &= \left(\int_{\tilde{x}(vT_h^+)}^{\tilde{x}((v+1)T_h)} + \int_{\tilde{x}(v+1)T_h}^{\tilde{x}(v+1)T_h^+} \right) \frac{1}{g(s)} ds \\ &= \int_{vT_h^+}^{(v+1)T_h} \left[\frac{f(\tilde{x})}{g(\tilde{x})} - (y_p + \tilde{y}) \right] d\tau + \int_{\tilde{x}((v+1)T_h)}^{\tilde{x}((v+1)T_h^+)} \frac{1}{g(s)} ds \end{aligned} \tag{6.18}$$

From Hypotheses (H2)-(H3), and the lower bound on $\tilde{y}$ established in (6.14), and further

noting that $\tilde{x}((v+1)T_h^+) = (1-\alpha_x)\tilde{x}((v+1)T_h)$, the integral (6.18) can be upper bounded as:

$$\begin{aligned}
&\int_{\tilde{x}(vT_h^+)}^{\tilde{x}((v+1)T_h^+)} \frac{1}{g(s)} ds \\
&\leq \int_{vT_h^+}^{(v+1)T_h} \left[S_g - (y_p - S_{y_p} e^{-m(\tau - t_0)})\right] d\tau + \int_{\tilde{x}(v+1)T_h}^{(1-\alpha_x)\tilde{x}(v+1)T_h} \frac{1}{r_g s} ds \qquad (6.19) \\
&= S_g T_h - \int_{vT_h^+}^{(v+1)T_h} y_p d\tau + \frac{S_{y_p}}{m}(1 - e^{-mT_h}) e^{-m(vT_h - t_0)} + \frac{\ln(1-\alpha_x)}{r_g}
\end{aligned}$$

where S_g and r_g are defined in Hypotheses (H4)-(N1) respectively.

Finally, the linear approximation of the deviation system around the periodic solution $(0, y_p(t))$ is given as

$$\begin{cases}
\dot{\tilde{x}} = (f'(0) - g'(0)y_p(t))\tilde{x} \\
\dot{\tilde{y}} = h'(0)y_p(t)\tilde{x} - m\tilde{y} \\
\tilde{x}(vT_h^+) = (1-\alpha_x)\tilde{x}(vT_h) \\
\tilde{y}(vT_h^+) = (1-\alpha_y)\tilde{y}(vT_h)
\end{cases} \qquad (6.20)$$

The stability analysis is carried out separately, first when T_h as the period of reference, then when T_r is so.

6.4.2 Releases more frequent than harvests

In order to state the results for the case when releases are more frequent than harvests, we first need to define the function

$$\underline{\mu}_{vh}(S, r) = m\left(S + \frac{\ln(1-\alpha_x)}{rT_h}\right) \frac{1}{1 - \left(\frac{\alpha_y\left(1-e^{-mT_h}\right)}{1-(1-\alpha_y)e^{-mT_h}}\right)\left(\frac{e^{-mT_h/k}}{k\left(1-e^{-mT_h/k}\right)}\right)}$$

This function is increasing in S and r because the sign of the partial derivatives is determined by the sign of the last factor, which can be shown to be positive. Indeed, this factor is positive when

$$\left(\frac{\alpha_y\left(1-e^{-mT_h}\right)}{1-(1-\alpha_y)\,e^{-mT_h}}\right)\left(\frac{e^{-mT_h/k}}{k\left(1-e^{-mT_h/k}\right)}\right) < 1$$

and we have $\alpha_y e^{-mT_h/k} \leq \alpha_y$ and $1-(1-\alpha_y)\,e^{-mT_h} > \alpha_y$, so that

$$\frac{\alpha_y e^{-mT_h/k}}{1-(1-\alpha_y)\,e^{-mT_h}} < 1$$

Also, $k\left(1-e^{-mT_h/k}\right) \geq \left(1-e^{-mT_h}\right)$ since both sides of the inequality have the same value in $T_h = 0$, and

$$\frac{\mathrm{d}}{\mathrm{d}T_h}\left[k\left(1-e^{-\frac{mT_h}{k}}\right)\right] = me^{-mT_h/k} \geq me^{-mT_h} = \frac{\mathrm{d}}{\mathrm{d}T_h}\left[1-e^{-mT_h}\right]$$

which shows that $\underline{\mu}_{vh}(S, r)$ is increasing in S and r. Theorem 11 summarises the results concerning the stability conditions.

Theorem 11 (Stability, releases more frequent) *When $T_h = kT_r$ with $k \in \mathbb{N}^*$, the solution $(x(t), y(t)) = (0, y_{ph}(t))$ of Model $\mathcal{M}_v$ is LAS iff*

$$\mu > \underline{\mu}_{vh}\left(\frac{f'(0)}{g'(0)}, g'(0)\right) \qquad (6.21a)$$

and is GAS if

$$\mu > \underline{\mu}_{vh}\left(S_g, r_g\right) \tag{6.21b}$$

where S_g and r_g are defined in Hypothesis (H4) and (N1) respectively.

Proof: We start with the proof of global convergence under condition (6.21b). The reference period in this scenario is T_h, which means that we count one impulse on x (*i.e.* one harvest) per period. We split the integral of (6.15) from $\tilde{x}_0$ to $\tilde{x}$ into integrals over complete partial harvest periods and remaining intervals at their fringe as follows:

$$\int_{\tilde{x}_0}^{\tilde{x}} \frac{1}{g(s)} ds = \left(\int_{\tilde{x}_0}^{\tilde{x}\left(\left(\lfloor \frac{t_0}{T_h} \rfloor + 1\right) T_h^+\right)} + \sum_{v=\lfloor \frac{t_0}{T_h} \rfloor + 1}^{\lfloor \frac{t}{T_h} \rfloor - 1} \int_{\tilde{x}(vT_h^+)}^{\tilde{x}((v+1)T_h^+)} + \int_{\tilde{x}\left(\lfloor \frac{t}{T_h} \rfloor T_h^+\right)}^{\tilde{x}(t)} \right) \frac{1}{g(s)} ds$$

The first and third components are bounded. The second term tends to $+\infty$ in magnitude as $t \to +\infty$. From (6.19), the summation of periodic integrals can also be upper bounded as:

$$\begin{aligned} &\sum_{v=\lfloor \frac{t_0}{T_h} \rfloor + 1}^{\lfloor \frac{t}{T_h} \rfloor - 1} \int_{vT_h^+}^{(v+1)T_h^+} \frac{1}{g(s)} ds \\ &\qquad \leq \sum_{v=\lfloor \frac{t_0}{T_h} \rfloor + 1}^{\lfloor \frac{t}{T_h} \rfloor - 1} \left[S_g T_h - \int_0^{T_h} y_{ph} d\tau + \frac{S_{y_{ph}}}{m} (1 - e^{-mT_h}) e^{-m(vT_h - t_0)} + \frac{\ln(1-\alpha_x)}{r_g} \right] \end{aligned} \tag{6.22}$$

Note that we integrate the periodic variation y_{ph} over the interval $(0, T_h)$, which is equivalent to integrating it over any arbitrary period. The term to be summed is decreasing in v. Thus, to ensure that the series goes to $-\infty$, we require that at some point, this term becomes negative. It suffices to consider that its behaviour in the limit that $v \to +\infty$ is as

$$\begin{aligned} \lim_{v \to +\infty} \left[S_g T_h - \int_0^{T_h} y_{ph} d\tau + \frac{S_{y_p}}{m} (1 - e^{-mT_h}) e^{-m(vT_h - t_0)} + \frac{\ln(1-\alpha_x)}{r_g} \right] < 0 \\ \Leftrightarrow \int_0^{T_h} y_{ph} d\tau > S_g T_h + \frac{\ln(1-\alpha_x)}{r_g} \end{aligned} \tag{6.23}$$

We calculate the y_{ph} integral[2]:

$$\begin{aligned}\int_0^{T_h} y_{ph}(\tau)d\tau &= \sum_{i=0}^{k-1}\left[y_{ph}(iT_r^+)\int_{iT_r}^{(i+1)T_r} e^{-m(\tau-iT_r)}d\tau\right] \\ &= \frac{\mu T_h}{m}\left(1-\frac{(1-e^{-mT_h})\alpha_y e^{-mT_r}}{k(1-e^{-mT_r})(1-(1-\alpha_y)e^{-mT_h})}\right)\end{aligned} \tag{6.24}$$

Plugging (6.24) into the inequality (6.23) and rearranging, we obtain

$$\mu > m\left(S_g+\frac{\ln(1-\alpha_x)}{r_g T_h}\right)\frac{1}{1-\left(\frac{\alpha_y\left(1-e^{-mT_h}\right)}{1-(1-\alpha_y)e^{-mT_h}}\right)\left(\frac{e^{-mT_h/k}}{k\left(1-e^{-mT_h/k}\right)}\right)} \tag{6.25}$$

which is the inequality (6.21b).

In order to have the global asymptotic stability, we are only left with the local asymptotic stability to prove. Here, we only have to consider the discrete system that maps the state at time nT_h^+ onto the state at time $(n+1)T_h^+$ with respect to the linear equation in $\tilde{y}$ and the discrete component of Equation (6.20).

After a few computations, we obtain:

$$\begin{pmatrix}\tilde{x}\\ \tilde{y}\end{pmatrix}((v+1)T_h^+) = \mathbf{B}\begin{pmatrix}\tilde{x}\\ \tilde{y}\end{pmatrix}(vT_h^+) \tag{6.26}$$

where

$$\mathbf{B} = \begin{pmatrix}(1-\alpha_x)e^{\int_{vT_h}^{(v+1)T_h} f'(0)-g'(0)y_{ph}d\tau} & 0\\ \ddagger & (1-\alpha_y)e^{-m\int_{vT_h}^{(v+1)T_h}d\tau}\end{pmatrix}$$

Note that $\ddagger$ is a term that we do not use in our analysis, therefore it is not expressed. Since the matrix is triangular and $|B_{22}|<1$, it is stable iff $|B_{11}|<1$, i.e.

$$\int_{vT_h}^{(v+1)T_h} y_{ph}d\tau > \frac{f'(0)T_h+\ln(1-\alpha_x)}{g'(0)} \tag{6.27}$$

[2]The calculation is detailed as follows:

$$\begin{aligned}\int_0^{T_h} y_{ph}(\tau)d\tau &= \sum_{i=0}^{k-1}\left[y_{ph}(iT_r^+)\int_{iT_r}^{(i+1)T_r} e^{-m(\tau-iT_r)}d\tau\right] \\ &= \sum_{i=0}^{k-1}\left[\left(y^*e^{-imT_r}+\mu T_r\sum_{j=0}^{i-1}e^{-jmT_r}\right)\frac{(1-e^{-mT_r})}{m}\right] \\ &= \sum_{i=0}^{k-1}\left[\left(y^*e^{-imT_r}+\mu T_r\frac{1-e^{-imT_r}}{1-e^{-mT_r}}\right)\right]\frac{(1-e^{-mT_r})}{m} \\ &= \left(y^*\sum_{i=0}^{k-1}e^{-imT_r}+\frac{\mu T_r}{1-e^{-mT_r}}\sum_{i=0}^{k-1}[1-e^{-imT_r}]\right)\frac{(1-e^{-mT_r})}{m} \\ &= \left(y^*\frac{1-e^{-kmT_r}}{1-e^{-mT_r}}+\frac{\mu T_r}{1-e^{-mT_r}}\left(k-\frac{1-e^{-kmT_r}}{1-e^{-mT_r}}\right)\right)\frac{(1-e^{-mT_r})}{m} \\ &= \left(y^*(1-e^{-kmT_r})+\mu T_r\left(k-\frac{1-e^{-kmT_r}}{1-e^{-mT_r}}\right)\right)\frac{1}{m} \\ &= \left(\frac{((1-e^{-mT_h})(1-\alpha_y)+(1-e^{-mT_r})\alpha_y)\mu T_r(1-e^{-mT_h})}{(1-(1-\alpha_y)e^{-mT_h})(1-e^{-mT_r})}-\frac{\mu T_r(1-e^{-mT_h})}{1-e^{-mT_r}}+\mu T_r k\right)\frac{1}{m} \\ &= \frac{\mu T_r k}{m}\left(\frac{1-e^{-mT_h}}{k(1-e^{-mT_r})}\left(\frac{((1-e^{-mT_h})(1-\alpha_y)+(1-e^{-mT_r})\alpha_y)-1+(1-\alpha_y)e^{-mT_h}}{1-(1-\alpha_y)e^{-mT_h}}\right)+1\right) \\ &= \frac{\mu T_h}{m}\left(1-\frac{(1-e^{-mT_h})\alpha_y e^{-mT_r}}{k(1-e^{-mT_r})(1-(1-\alpha_y)e^{-mT_h})}\right)\end{aligned}$$

Similarly to what was done earlier, it can be shown that (6.27) is equivalent to (6.21a), so that the necessary and sufficient condition for local stability is proven.

It is directly seen that (6.27) is satisfied when (6.21b) is because $\underline{\mu}_h(S,r)$ is increasing in S and r and we have

$$\frac{f'(0)}{g'(0)} = \lim_{x \overset{>}{\to} 0} \frac{f(x)}{g(x)} \leq \sup_{x \geq 0} \frac{f(x)}{g(x)} \text{ and } g'(0) = \lim_{x \overset{>}{\to} 0} \frac{g(x)}{x} \leq \sup_{x \geq 0} \frac{g(x)}{x} \tag{6.28}$$

This completes the proof of global stability, since we have shown global convergence and local stability when (6.21b) is satisfied. □

The lower bound on the release rate required for the stability of the periodic pest-free solution is the minimal release rate that the biological control practitioner needs to meet if pests are to be eliminated.

6.4.3 Releases less frequent than harvests

If we now consider the case where predators releases take place less often than harvests, we also obtain global and local stability results based on the following function

$$\underline{\mu}_{vr}(S,r) = m\left(S + \frac{\ln(1-\alpha_x)}{rT_h}\right)\frac{1-(1-\alpha_y)e^{-mT_h}}{1-e^{-mT_h}}$$

which is non-decreasing in S and r since the last fraction is positive and $\alpha_x \in [0,1]$.

Theorem 12 (Stability, releases less frequent) *When $T_r = kT_h$ with $k \in \mathbb{N}^*$, the solution $(x(t), y(t)) = (0, y_{pr}(t))$ of Model ($\mathcal{M}_v$) is LAS iff*

$$\mu > \underline{\mu}_{vr}\left(\frac{f'(0)}{g'(0)}, g'(0)\right) \tag{6.29}$$

and is GAS if

$$\mu > \underline{\mu}_{vr}\left(\sup_{x\geq 0}\frac{f(x)}{g(x)}, \sup_{x\geq 0}\frac{g(x)}{x}\right) \tag{6.30}$$

Proof: This proof does not depart very much from the one of Theorem 11. We make use of the same integral

$$\int_{\tilde{x}_0}^{\tilde{x}} \frac{1}{g(s)} ds.$$

The only difference is that the reference period is now T_r, which consists of k partial harvests. As before, we first consider its evolution over the reference period: the latter is expressed as a sum of k integrals as defined in (6.18), then bounded using (6.19).

$$\begin{aligned}
\int_{\tilde{x}(nT_r^+)}^{\tilde{x}((n+1)T_r^+)} \frac{1}{g(s)} ds &= \sum_{i=0}^{k-1} \int_{\tilde{x}(nT_r+iT_h^+)}^{\tilde{x}(nT_r+(i+1)T_h^+)} \frac{1}{g(s)} ds \\
&= \sum_{i=0}^{k-1} \left[\left(\int_{\tilde{x}(nT_r+iT_h^+)}^{\tilde{x}(nT_r+(i+1)T_h)} + \int_{\tilde{x}(nT_r+(i+1)T_h)}^{\tilde{x}(nT_r+(i+1)T_h^+)} \right) \frac{1}{g(s)} ds \right] \\
&\leq \sum_{i=0}^{k-1} \left[S_g T_h - \int_{iT_h}^{(i+1)T_h} y_{pr} d\tau + \frac{S_{y_{pr}}}{m}(1-e^{-mT_h})e^{-m(vT_h-t_0)} + \frac{\ln(1-\alpha_x)}{r_g} \right] \\
&= kS_gT_h - \int_{nT_r}^{(n+1)T_r} y_{pr} d\tau + \frac{kS_{y_{pr}}}{m}(1-e^{-mT_h})e^{-m(vT_h-t_0)} + \frac{k\ln(1-\alpha_x)}{r_g}
\end{aligned}$$

Over an interval of time $\tau \in (t_0, t)$, this integral may be decomposed as

$$\int_{\tilde{x}_0}^{\tilde{x}(t)} \frac{1}{g(s)} ds = \left(\int_{\tilde{x}_0}^{\tilde{x}\left(\left(\lfloor \frac{t_0}{T_r} \rfloor + 1\right) T_r^+\right)} + \sum_{n=\lfloor \frac{t_0}{T_r} \rfloor + 1}^{\lfloor \frac{t}{T_r} \rfloor - 1} \int_{\tilde{x}(nT_r^+)}^{\tilde{x}((n+1)T_r^+)} + \int_{\tilde{x}\left(\lfloor \frac{t}{T_r} \rfloor T_r^+\right)}^{\tilde{x}(t)} \right) \frac{1}{g(s)} ds.$$

The first and third terms are bounded. We focus on the second one.

We proceed as in the previous section to deduce that upper bound on this term's integrand is a decreasing function of n. So for it to tend to $-\infty$, it suffices that

$$\begin{aligned} &\lim_{n \to +\infty} \left[S_g T_r - \int_0^{T_r} y_{pr} d\tau + \frac{S_{y_p}}{m} (1 - e^{-mT_h}) e^{-m(nT_r - t_0)} + \frac{k \ln(1 - \alpha_x)}{r_g} \right] < 0 \\ \Leftrightarrow \quad &\int_0^{T_r} y_{pr} d\tau > S_g T_r + \frac{k \ln(1 - \alpha_x)}{r_g}. \end{aligned} \tag{6.31}$$

The y_{pr} integral is calculated as a sum of subintegrals[3]:

$$\begin{aligned} \int_0^{T_r} y_{pr} d\tau &= \sum_{i=0}^{k-1} y_{pr}(iT_h^+) \int_{iT_h}^{(i+1)T_h} e^{-m(\tau - iT_h)} d\tau \\ &= \frac{\mu T_r (1 - e^{-mT_h})}{m(1 - (1 - \alpha_y) e^{-mTh})} \end{aligned}$$

Substituting it in (6.31) and noting that $\frac{k}{T_r} = \frac{1}{T_h}$, we obtain, upon rearranging the terms,

$$\mu > m \left(S_g + \frac{\ln(1 - \alpha_x)}{r_g T_h} \right) \left(\frac{1 - (1 - \alpha_y) e^{-mT_h}}{1 - e^{-mT_h}} \right),$$

which corresponds to the expression (6.30).

Global convergence of $(\tilde{x}, \tilde{y})$ to $(0, 0)$ is then concluded by using the same argument as in the proof of Theorem 11 to show the convergence of $\tilde{y}$ to 0.

Similarly, the local stability condition (6.29) results from the analysis of the discrete linearized system that maps $\tilde{y}(nT_r^+)$ onto $\tilde{y}((n+1)T_r^+)$.

$$\begin{pmatrix} \tilde{x} \\ \tilde{y} \end{pmatrix} ((n+1)T_r^+) = \mathbf{B} \begin{pmatrix} \tilde{x} \\ \tilde{y} \end{pmatrix} (nT_r^+) \tag{6.32}$$

where

$$\mathbf{B} = \begin{pmatrix} (1 - \alpha_x)^k e^{\int_{nT_r}^{(n+1)T_r} f'(0) - g'(0) y_{pr} d\tau} & 0 \\ \ddagger & (1 - \alpha_y)^k e^{-m \int_{nT_r}^{(n+1)T_r} d\tau} \end{pmatrix} \tag{6.33}$$

[3]The complete calculation is as follows:

$$\begin{aligned} \int_0^{T_r} y_{pr} d\tau &= \sum_{i=0}^{k-1} y_{pr}(iT_h^+) \int_{iT_h}^{(i+1)T_h} e^{-m(\tau - iT_h)} d\tau \\ &= \sum_{i=0}^{k-1} y^* e^{imT_h} (1 - \alpha_y)^i \int_0^{T_h} e^{-m\tau} d\tau \\ &= \frac{y^*(1 - e^{-mT_h})}{m} \sum_{i=0}^{k-1} e^{imT_h} (1 - \alpha_y)^i \\ &= \frac{\mu T_r (1 - e^{-mT_h})}{m(1 - (1 - \alpha_y)^k e^{-mT_r})} \left(\frac{1 - (1 - \alpha_y)^k e^{-kmT_h}}{1 - (1 - \alpha_y) e^{-mT_h}} \right) \\ &= \frac{\mu T_r (1 - e^{-mT_h})}{m(1 - (1 - \alpha_y) e^{-mTh})} \end{aligned}$$

Again, since the system matrix is lower triangular, for stability we simply require that $|B_{11}| < 1$ and $|B_{22}| < 1$. The latter yields a trivial condition, while the former:

$$\int_{nT_r}^{(n+1)T_r} y_{pr} d\tau > \frac{f'(0)T_r + k\ln(1-\alpha_x)}{g'(0)} \tag{6.34}$$

This expression has the same form as (6.31). After some obvious changes, we obtain condition (6.29) for local stability. Thus the proof of global stability is also complete because $\underline{\mu}_{vr}$ is increasing in S and r and (6.28) is still satisfied. □

Varying the release period has no effect on the minimal rate when they take place less often than harvests; however, it influences this minimal bound if it takes place more often than the partial harvests. In the next section, we investigate more closely the effect of varying this release period (for a fixed partial harvesting period). We also look at how the partial harvest parameters will affect the minimal release rate required for biological control. We highlight in particular when partial harvesting (or insecticide usage in an IPM framework) contributes to control pests and when it is likely to disrupt a control program.

6.5 The minimal release rate

6.5.1 Dependence in biological processes

Whether releases take place more or less often than the partial harvests, if the pest growth rate is higher and the functional response lower, a higher bound is required. Predator mortality also raises the minimal release value. Moreover, the minimal value can be lowered if the strength of partial harvests on the pest increases while that on the predator decreases.

6.5.2 Dependence in the period of release

From our stability analysis in Theorem 12, the minimal release rate $\underline{\mu}_{vr}$, corresponding to harvests more frequent than releases, is independent of the release period (actually it does not depend on k). Thus

$$\frac{\partial \underline{\mu}_{vr}}{\partial T_r} = 0$$

The influence of T_r on $\underline{\mu}_{vh}$ (releases are more frequent) calculated in Theorem 11 cannot be deduced directly from the expression for the minimal release rate. Instead we need to evaluate the sign of its derivative with respect to the release period: Theorem 13 states the result.

Theorem 13 (Dependence in release period, $\underline{\mu}_{vh}$) *Let $T_h = kT_r$ where $k \in \mathbb{N}^*$. The minimal budget is monotonically decreasing with respect to the release period T_r for non-negative values of $\underline{\mu}_{vh}$, i.e.*

$$\frac{\partial \underline{\mu}_{vh}}{\partial T_r} < 0.$$

.

Proof: Knowing that T_r is equal to $\frac{T_h}{k}$, we may write

$$\frac{\partial \underline{\mu}_{vh}}{\partial T_r} = \frac{\partial \underline{\mu}_{vh}}{\partial k}\frac{\partial k}{\partial T_r} = \frac{\partial \underline{\mu}_{vh}}{\partial k}\left(\frac{-k^2}{T_h}\right)$$

So

$$\operatorname{sgn}\left(\frac{\partial \underline{\mu}_{vh}}{\partial T_r}\right) = -\operatorname{sgn}\left(\frac{\partial \underline{\mu}_{vh}}{\partial k}\right) \tag{6.35}$$

We note that μ_{vh} is expressed as the product of two distinctive parts, one of which is independent of k and which, for the non-trivial stability condition, should be positive:

$$S + \frac{\ln(1-\alpha_x)}{rT_h} > 0$$

where S and r are the parameters required for the local or global conditions, as defined previously. If the previous coefficient were to be negative, it would simply mean that the harvests would by themselves be sufficient to suppress the pest population: the $\left(\frac{\ln(1-\alpha_x)}{r}\right)$ term corresponding to the harvesting then dominates pest-growth during one period represented by ST_h. So (6.35) can be evaluated simply in considering the second factor of μ_{vh}:

$$\operatorname{sgn}\left(\frac{\partial \underline{\mu}_{vh}}{\partial T_r}\right) = -\operatorname{sgn}\left(\frac{\partial}{\partial k}\left[\frac{1}{1-\left(\frac{\alpha_y(1-e^{-mT_h})}{1-(1-\alpha_y)e^{-mT_h}}\right)\sigma(k)}\right]\right)$$

where $\sigma(k) = \frac{e^{-mT_h/k}}{k\left(1-e^{-mT_h/k}\right)}$. We can compute $\frac{\partial \underline{\mu}_{vh}}{\partial \sigma} > 0$, therefore:

$$\begin{aligned}\operatorname{sgn}\left(\frac{\partial \underline{\mu}_{vh}}{\partial T_r}\right) = -\operatorname{sgn}\left(\frac{\partial \sigma}{\partial k}\right) &= -\operatorname{sgn}\left(\frac{e^{-mT_h/k}}{k^2(1-e^{-mT_h/k})^2}\left(\frac{mT_h}{k} - 1 + e^{-mT_h/k}\right)\right) \\ &= -\operatorname{sgn}\left(ke^{-mT_h/k} + mT_h - k\right).\end{aligned}$$

This function behaves thus: first,

$$\frac{\partial}{\partial k}[ke^{-mT_h/k} + mT_h - k] = \left(1 + \frac{mT_h}{k}\right)e^{-mT_h/k} - 1 \leq 0,$$

and then, applying twice l'Hospital's Rule

$$\begin{aligned}\lim_{k\to\infty}\left(ke^{-dT_h/k} + dT_h - k\right) &= dT_h + \lim_{k\to\infty}\left(\frac{e^{-dT_h/k} - 1}{\frac{1}{k}}\right) \\ &= dT_h + \lim_{k\to\infty}\left(\frac{\frac{dT_h}{k^2}e^{-dT_h/k}}{-\frac{1}{k^2}}\right) \\ &= 0\end{aligned}$$

which means that $\operatorname{sgn}\left(ke^{-mT_h/k} + mT_h - k\right) > 0$. Therefore, $\operatorname{sgn}\left(\frac{\partial \underline{\mu}_h}{\partial T_r}\right) < 0$, what completes the proof. □

Thus we hit the smallest minimal value for the largest possible T_r, in this case when $k = 1$. This happens when the release frequency equals the partial harvest frequency. Figure 6.4 illustrates this observation.

Interestingly, the various analyses presented in this thesis as well as the optimisation problem solved in Mailleret and Grognard (2006), then in Mailleret and Grognard (2009) suggest that higher release frequencies are to be favoured in order to obtain fastest convergence to the pest-free solution or minimise the worst case damage on a pest invasion. Merging this observation involving partial harvests and the cited works suggests that the compromise is to increase the release frequency up to the partial harvest one, beyond which biological control is more demanding.

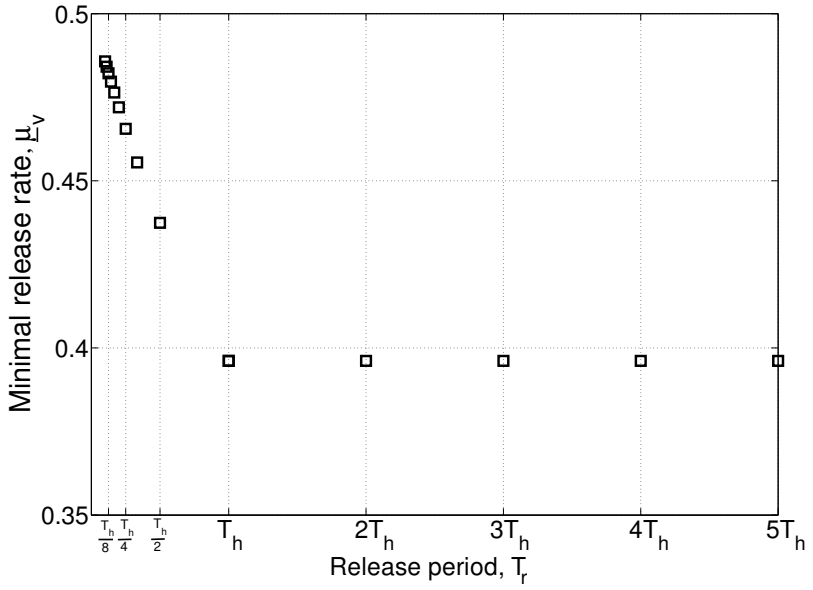

Figure 6.4: Variation of the minimal predator release rate as a function of release to harvest period ratio. In this simulation, parameters were given the values (in arbitrary units): $\alpha_x = \alpha_y = 0.5$, $m = 1$, the growth rate $f'(0)$, trophic responses $g'(0)$ and $h'(0)$ when the ecosystem is pest-free ($x_p = 0$) were all set to 1.

6.5.3 Dependence in partial harvesting parameters

The effects of partial harvest parameters are investigated by comparing the minimal release rate $\underline{\mu}_v$ (defined as the union of the functions $\underline{\mu}_{vh}$ and $\underline{\mu}_{vr}$) with $\underline{\mu}$ (the minimal release rate obtained from the analysis of the basic model we considered in Chapter 3, i.e. the same model but with no harvest.

We focus on the case with the most interest to us, *i.e.* when $\underline{\mu}_v$ is minimal, which is when releases take place less often or as often as the partial harvests. We set $k = 1$ without any loss of generality since it preserves the same form as $\underline{\mu}_{vr}$ when releases are less frequent.

There are four possibilities for the minimal release rate, depending on the partial harvest strengths and the frequency at which it takes place. We shall subdivide them into two categories: the first where biological control is unnecessary and the second where it is to be implemented in conjunction with the partial harvests. Figure 6.5 illustrates how $\underline{\mu}_v$ varies with T_h for different combinations of α_x and α_y.

Here as well, S and r refer to the parameters required for the local or global conditions, as defined previously.

Trivial $\underline{\mu}_v$: *Partial harvests provide complete pest control up to a harvest period.*

For a given α_x, provided that the partial harvest periods are small enough, the minimal release rate is a nonpositive value (it is trivial). Conversely, for a given partial harvest period, if α_x is large enough, partial harvests suffice to completely control pests **(scenario I)**.

The condition on α_x and T_h for $\underline{\mu}_v \leq 0$ is calculated as

$$S + \frac{\ln(1-\alpha_x)}{rT_h} \leq 0$$
$$\Leftrightarrow \alpha_x \geq 1 - e^{-SrT_h}$$

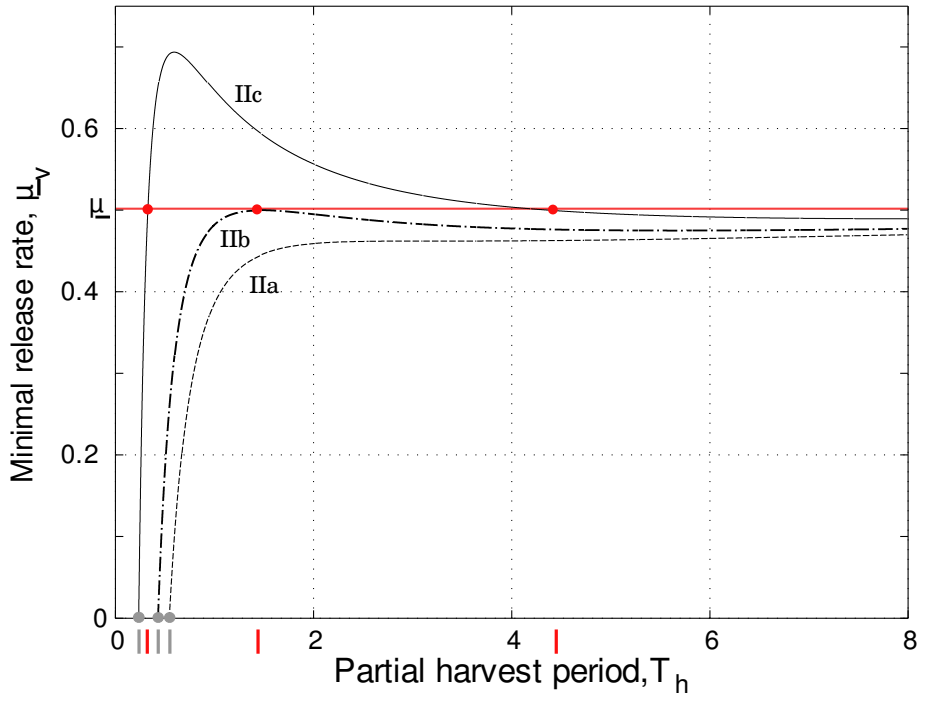

Figure 6.5: The minimal release rate as a function of the harvest period calculated for $\alpha_y = 0.45$ and three α_x values, which are: for curve, or scenario (IIa): 0.42; (IIb): 0.35 and (IIc): 0.21. As α_x is increased, we obtain increasing threshold values $T_{h(I)}$ which are referenced by the grey markers. $\underline{\mu}$, represented by a red line, is the minimal release rate for the basic model. The red markers are, in ascending order of their value $T^{\dagger}_{h_{(IIc)}}, T_{h_{(IIb)}}$ and $T^{\ddagger}_{h_{(IIc)}}$.

Alternatively, the intercept of $\underline{\mu}_v$ and the T_h-axis is an increasing function of α_x given as:

$$T_{h(I)} = -\frac{\ln(1-\alpha_x)}{rS}$$

This means that larger harvest effects on the prey can suppress the pest up to a larger period than with lower ones, which is quite natural.

Nontrivial $\underline{\mu}_v$: *As the harvesting period is increased, partial harvests aid towards pest control (i.e. $0 < \underline{\mu}_v < \underline{\mu}$), then may or may not disrupt it (i.e. $\underline{\mu}_v > \underline{\mu}$).*

The minimal release rate $\underline{\mu}_v$ converges to $\underline{\mu}$ (which is independent of the partial harvest period) as partial harvests become rarer and their effects attenuate.

$$\lim_{T_h\to+\infty} \underline{\mu}_v = \lim_{T_h\to+\infty} m\left(S + \frac{\ln(1-\alpha_x)}{rT_h}\right)\frac{1-(1-\alpha_y)e^{-mT_h}}{1-e^{-mT_h}} = mS \tag{6.36}$$

Moreover, we can show that this minimal release rate increases to that limit. Indeed

$$\begin{aligned}\frac{\partial \underline{\mu}_v}{\partial T_h} &= -\frac{m\ln(1-\alpha_x)}{rT_h^2}\frac{1-(1-\alpha_y)e^{-mT_h}}{1-e^{-mT_h}} \\ &\quad +m\left(S+\frac{\ln(1-\alpha_x)}{rT_h}\right)\frac{m(1-\alpha_y)e^{-mT_h}(1-e^{-mT_h}) - me^{-mT_h}(1-(1-\alpha_y)e^{-mT_h})}{(1-e^{-mT_h})^2} \\ &= -\frac{m\ln(1-\alpha_x)}{rT_h^2} - \frac{m\ln(1-\alpha_x)}{rT_h^2}\frac{\alpha_y e^{-mT_h}}{1-e^{-mT_h}} - m\left(S+\frac{\ln(1-\alpha_x)}{rT_h}\right)\frac{m\alpha_y e^{-mT_h}}{(1-e^{-mT_h})^2}\end{aligned}$$

so that $\lim_{T_h \to +\infty} \frac{\partial \underline{\mu}_v}{\partial T_h} = 0^+$. The plus superscript indicates that $\underline{\mu}_v$ approaches the asymptote $\underline{\mu}$ with a positive gradient. Indeed, the first term of the derivative goes to zero as the inverse of T_h^2, while the other two terms converge to zero exponentially. The first term, which is positive, then dominates the others for large values of T_h.

For a given α_x *and* α_y combination, we can identify three possibilities. The minimal release rate required is lower than when no partial harvest takes place (i.e. $0 < \underline{\mu}_{vh} < \underline{\mu}$):

- at *all* harvest periods $T_h > T_{h(I)}$ **(scenario IIa)**, or
- at all harvest periods except at *one critical period* where it is exactly $\underline{\mu}$, where partial harvesting or chemical control brings nothing to the control strategy **(scenario IIb)**, or
- at all harvest periods except over *a finite range of periods* where $\underline{\mu}_v > \underline{\mu}$, that is biological control is disrupted **(scenario IIc)**.

We are able to derive necessary or sufficient conditions which lead to either of (scenarios IIa-IIc) described above. Mathematically, the behaviour of $\underline{\mu}_v$ with respect to $\underline{\mu}$ is determined by the number of intersections between the two curves or the number of solutions satisfying the equation $\underline{\mu}_v = \underline{\mu}$, i.e.:

$$\left(S + \frac{\ln(1-\alpha_x)}{rT_h}\right) \frac{1-(1-\alpha_y)e^{-mT_h}}{1-e^{-mT_h}} = S. \tag{6.37}$$

Rearranging this equation yields

$$\frac{\ln(1-\alpha_x)}{rT_h}(1-(1-\alpha_y)e^{-mT_h}) = S((1-e^{-mT_h}) - (1-(1-\alpha_y)e^{-mT_h}))$$

so that finding the solutions of Equation (6.37) is equivalent to finding the solution of the following equation

$$-\frac{\ln(1-\alpha_x)}{rS\alpha_y} = \frac{e^{-mT_h}T_h}{1-(1-\alpha_y)e^{-mT_h}}. \tag{6.38}$$

The left-hand side of Equation (6.38) is constant with respect to T_h. The right-hand side is non-monotonic: we note that in the limit that $T_h \to 0^+$ and $T_h \to +\infty$, it is zero, and it is clearly positive in between. Thus the right-hand side curve should possess at least one local maximum. At this maximum we have,

$$\begin{aligned}
&\frac{\partial}{\partial T_h}\left[\frac{e^{-mT_h}T_h}{1-(1-\alpha_y)e^{-mT_h}}\right] = 0, \\
\Leftrightarrow \quad &\frac{e^{-mT_h}\left[(1-mT_h)\left(1-(1-\alpha_y)e^{-mT_h}\right) - mT_h(1-\alpha_y)e^{-mT_h}\right]}{(1-(1-\alpha_y)e^{-mT_h})^2} = 0, \\
\Leftrightarrow \quad &\frac{e^{-mT_h}(1-mT_h-(1-\alpha_y)e^{-mT_h})}{(1-(1-\alpha_y)e^{-mT_h})^2} = 0.
\end{aligned}$$

$e^{-mT_h} > 0$ for all non-negative T_h, so that at the maximum, we necessarily have

$$1 - mT_h^\star - (1-\alpha_y)e^{-mT_h^\star} = 0. \tag{6.39}$$

Solving this equation, we obtain[4] that the local maximum is actually single and thus global, and writes

$$T_h^\star = \frac{1}{m}\left(1 + W_0\left(\frac{\alpha_y - 1}{e^1}\right)\right),$$

which, we note, is a decreasing function of α_y.

Moreover, because $0 \leq \alpha_y \leq 1$, we deduce $-1 < W_0\left(\frac{\alpha_y - 1}{e^1}\right) < 0$, so that:

$$0 \leq T_h^\star \leq \frac{1}{m} \tag{6.40}$$

The conditions for the behaviour of the curves are then derived from this result. We note from (6.39) that $1 - (1 - \alpha_y)e^{-mT_h^\star} = mT_h^\star$; so, the maximum of the right hand side of equation (6.38) can be written as

$$\frac{e^{-mT_h^\star}T_h^\star}{1 - (1 - \alpha_y)e^{-mT_h^\star}} = \frac{e^{-mT_h^\star}}{m}.$$

For curves characterised by

- *no intercept*, i.e. the minimal release rate curve increases asymptotically to the no-harvest case without overshoot, we have using (6.38)

$$\begin{aligned} -\frac{\ln(1-\alpha_x)}{rS\alpha_y} &> \frac{e^{-mT_h^\star}}{m} \\ \Leftrightarrow \quad \ln(1-\alpha_x) &< -\frac{rS}{m}\alpha_y e^{-mT_h^\star} \end{aligned} \tag{6.41a}$$

  Because $0 \leq T_h^\star \leq \frac{1}{m}$, we can further specify this condition. In particular, a sufficient condition for (6.41a) to hold true, and thus to ensure that there is no intercept, is:

$$\ln(1-\alpha_x) < -\frac{rS}{m}\alpha_y \tag{6.41b}$$

  and a sufficient one for (6.41a) to be false, and thus to ensure that there is (at least) an intercept, is

$$\ln(1-\alpha_x) > -\frac{rS}{m}\alpha_y e^{-1} \tag{6.41c}$$

[4] The calculations are detailed as follows. Let $u = 1 - mT_h^\star$. Substituting in Equation (6.39), we obtain:

$$\begin{aligned} & u - (1-\alpha_y)e^{u-1} = 0 \\ \Leftrightarrow \quad & u = (1-\alpha_y)\frac{e^u}{e^1} \\ \Leftrightarrow \quad & -ue^{-u} = \frac{\alpha_y - 1}{e^1} \\ \Leftrightarrow \quad & -u = W_0\left(\frac{\alpha_y - 1}{e^1}\right), \end{aligned}$$

W_0 is linked to the Lambert's W (or Omega) function. The W function is the inverse of the function $f(z) = ze^z$ on the complex numbers, and is multivalued. The branch of W restricted to the real axis, and with an argument larger than $(\frac{-1}{e})$, is denoted W_0. W_0 is single valued and monotonically increasing from -1 at $(\frac{-1}{e})$; moreover $W_0(0) = 0$. A major reference on Lambert's W function is (Corless et al., 1996).

Reintroducing our original notations instead of u and rearranging yields

$$T_h^\star = \frac{1}{m}\left(1 + W_0\left(\frac{\alpha_y - 1}{e^1}\right)\right).$$

- *only one intercept*, i.e. partial harvests aid biological control up to a critical period of harvest,

$$T_{h(IIb)} = T_h^\star$$

at which it has no effect. This equation must be satisfied:

$$\ln(1-\alpha_x) = -\frac{rS}{m}\alpha_y e^{-mT_h^\star} \tag{6.42}$$

Once T_h is increased however, that is the partial harvests are carried out less often, we are able to lower the minimal release rate.

- *two intercepts*: here, $\underline{\mu}_v$ overshoots $\underline{\mu}$ at a threshold period $T^\dagger_{h(IIc)} \neq T_{h(IIb)}$, before decreasing back to lower values than $\underline{\mu}$ beyond a second threshold $T^\ddagger_{h(IIc)}$. In other words, over the range $(T^\dagger_{h(IIc)}, T^\ddagger_{h(IIc)})$, partial harvesting is detrimental to pest control. The condition for this scenario is given as:

$$\ln(1-\alpha_x) > -\frac{rS}{m}\alpha_y e^{-mT_h^\star} \tag{6.43}$$

As for the no-intercept case, due to the bounds on $T_h^\star$ we can derive sufficient conditions for the existence or not of the two intercepts scenario, which turn out to be the same than for the non-existence or existence of the no intercept scenario, respectively (6.41c) and (6.41b).

The conditions established are influenced by three intrinsic processes: the predator mortality m, the per prey capita functional response $\frac{g(x)}{x}$ and the ratio of the pest growth versus functional response $\frac{f(x)}{g(x)}$. Given the size of these processes, in order to ensure that the partial harvest always contributes towards pest control (scenario (IIa)), the pest removal α_x needs to be larger than a value calculated as a function of the predator removal α_y. If α_x is smaller than this value, the partial harvests will be disruptive over a range of periods. Figure 6.6 depicts the parameter space for the (α_x, α_y)-pairs which would result in either of scenarios (IIa)-(IIc).

We note that the size of parameter regions will be modified by the intrinsic processes. Thus a high per capita functional response and low pest growth rate to predation ratio reduce the space for overshoot, as would a high predator mortality. This is not to be confused with their impact on the actual minimal release rate required. Indeed, while a high m increases the range of (α_x, α_y) giving no overshoot, it does effectively increase the actual minimal release rate, both in the basic model and in the partial harvest one and is thus disadvantageous to the pest control strategy.

6.6 The pest control strategy

What is the recommended release period when partial harvesting (or removal) takes place?

When the partial harvest (or removal via other instantaneous methods) take place at periods where one is the integer multiple of the other (with the two events coinciding over the longer period), we found that the harvest frequency provided a threshold for the release frequency. We have calculated that a lower minimal release rate is required when releases are rarer than partial harvests. This threshold period does not depend on the absolute value of the harvest period in itself. Combined with the findings of Mailleret and Grognard (2006, 2009) and the previous chapters which point towards using as high a release frequency as possible, we would recommend as the current 'best' strategy to release predators at the same frequency as partial harvests take place.

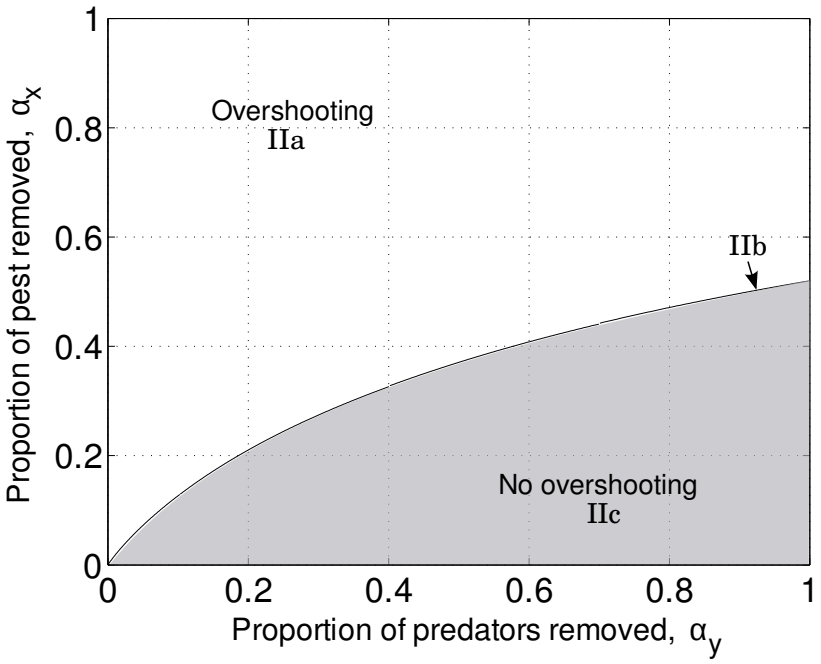

Figure 6.6: Parameter space for scenarios (IIa)-(IIc). The curve represents the minimal partial harvest strength required on the pest population, α_x, for a given partial harvest strength on the predators, α_y, so that partial harvests contribute to pest control. The values for the parameters fixing the regions are $f'(0) = 1$ and $g'(0) = 1$ and $m = 0.5$. The (α_x, α_y) pairs lying in 'no overshooting' region result in scenario (IIa), while the 'overshooting' region result in scenario (IIc). The curve represents scenario (IIb)

What is the recommended partial harvesting period?

It depends on the partial harvesting strength parameters. For a given harvest strength on the pest population, sufficiently frequent harvests can provide complete pest control. Alternatively, for a given harvest period, provided the removal of pests at each harvest is high enough, biological control is not needed. However, while our calculations show that a high frequency of high impact partial harvests is desirable, the latter is not always achievable (say if for instance the pest removal is too low or conversely the crop grows slowly so that it can not be harvested frequently). Depending on the strength of harvest on the predator population, there are three possible scenarios: partial harvests may either aid towards pest control by lowering the minimal release rate required, or have no effect, or disrupt pest control. Figures 6.5 and 6.6 give an indication of how combinations of harvest period and harvest strengths determine the release rate to use.

What if neither release period nor partial harvest period is an integer multiples of the other?

This is where one of the shortcomings of our approach lies. In the case where the ratio is rational, we are able to calculate y^* as follows, hence prove the existence of a pest-free solution. This result is expressed in Theorem 14 (the proof is not presented here).

Theorem 14 (Existence, neither period is integer multiple of the other) *Let T be the least common multiple of the release and partial harvest periods such that $T = k_r T_r = k_h T_h$,*

where $k_r, k_h \in \mathbb{N}^*$

$$y^* = \frac{\underline{\mu}_v T_r \sum_{j=1}^{k_r} e^{-m(T-jT_r)}(1-\alpha_y)^{\lceil \frac{T-jT_r}{T_h} \rceil}}{1-(1-\alpha_y)^{k_h} e^{-mT}} \tag{6.44}$$

The integral $\int_0^T y_p d\tau$, hence the minimal release rate, is more difficult to calculate. The case when the harvest and release periods are completely unrelated has not been investigated yet and would probably require another approach.

It is also possible that these intermediate ratios may induce other dynamics in the system. Whether they may stabilise it giving even lower minimal budget values or bring chaos is to be seen.

Terry (2010) does not consider any relation between the periods of the different impulsive components of his system and might provide cues. Nevertheless, our results already have a practical economic advantage: coinciding periods imply little or no additional costs incurred in terms of labour since the task of predator release can be assigned to workers in charge of partial harvesting.

See later: *We discuss in more detail the combined effects of partial removals and predator releases in an Integrated Pest Management context in Chapter 7. In the case that partial removals are due to pesticide usage, the parameters α_x and α_y can evolve. This will impact the choice of the pest control strategy to implement.* ■

7

Practical guidelines and experimental results

Overview

The modelling approach in this thesis is qualitative, that is the predator-prey processes are characterized by a few basic properties. This allows us to focus on the general trends or properties of a set of data points obtained from field and/or lab observations. In **Section 7.1**, we formulate practical guidelines based on the analyses we have made in Chapters 4-6. We illustrate how the qualitative nature of our models can be used to obtain estimates for minimal release rates and identify pest control strategies. In **Section 7.2**, we then give the results on the predatory mite *Neoseiulus cucumeris* (Oudemans) and its prey, the thrips *Frankliniella occidentalis* (Pergande) obtained from preliminary experiments carried out at URIH - INRA Sophia Antipolis (France).

Keywords: release rate, release frequency, pesticide resistance, *N. cucumeris*, *F. occidentalis*.

7.1 Guidelines

We formulate the control guidelines as follows: First, we assemble data sets related to the ecological processes (pest growth, predation and predator mortality). Second, we look at the general trends in some of these processes to identify a model with matching premises. Third, once the model has been identified, we measure or estimate some specific characteristics of the data sets that were collected (namely gradients, suprema and some intercepts); these values are relevant to the calculation of the minimal release rate predicted by the model that has been selected for use.

7.1.1 Matching data with the model

To be able to formulate a pest release strategy, we require data on the different processes that take place in the biological control system, namely on the pest growth, the predation and the mortality of the predators. In particular, we assemble data sets on the variation of:

1. the *pest growth* with respect to the pest population size;
2. the *predation on the pest per average predator (the functional response)* with respect to the *pest* population size;
3. the *functional response* with respect to the *predator* population size;
4. the *predator mortality* with respect to the predator population size.

From this data, it is useful to calculate:

5. the *per capita pest growth*, which is the pest growth rate at a given pest population size divided by that pest population size;
6. the *functional response per pest*, which is the functional response at a given pest population size divided by that pest population size;
7. the *pest growth divided by the functional response* for a given pest population size;
8. the *predator mortality rate*, which is the predator mortality divided by the predator population size.

We will refer to the collection of graphs representing the data as **the data grid**.

In this thesis we considered different situations and adapted a model accordingly. The process is now reversed as we go from data to model: given a set of data on a system, we need to identify the model that would provide the best recommendations for the pest control strategy and notably, the best estimate of the minimal predator release rate. To do so, we look at:

i. how the functional response varies with the predator population (whether it is constant or decreasing), then

ii. how the predator mortality rate varies with the predator population (whether it is constant or increasing), and

finally:

iii. whether we want to use an integrated pest management program or harvest the crop partially on a regular basis.

Based on these three characteristics, we match the data with the model that best describes it. A decision tree is given in Figure 7.1.

Remark: *In this chapter, we use artificially generated data for didactic purposes*[1]. ■

7.1.2 Simple augmentative control program

Data acquisition

The data grid given in Figure 7.2 is characterised by:

i. a functional response that is independent of the predator density *(top right graph)*;

[1]For an explanation of how the data can be collected, see e.g. references given in Begon et al. (1996)

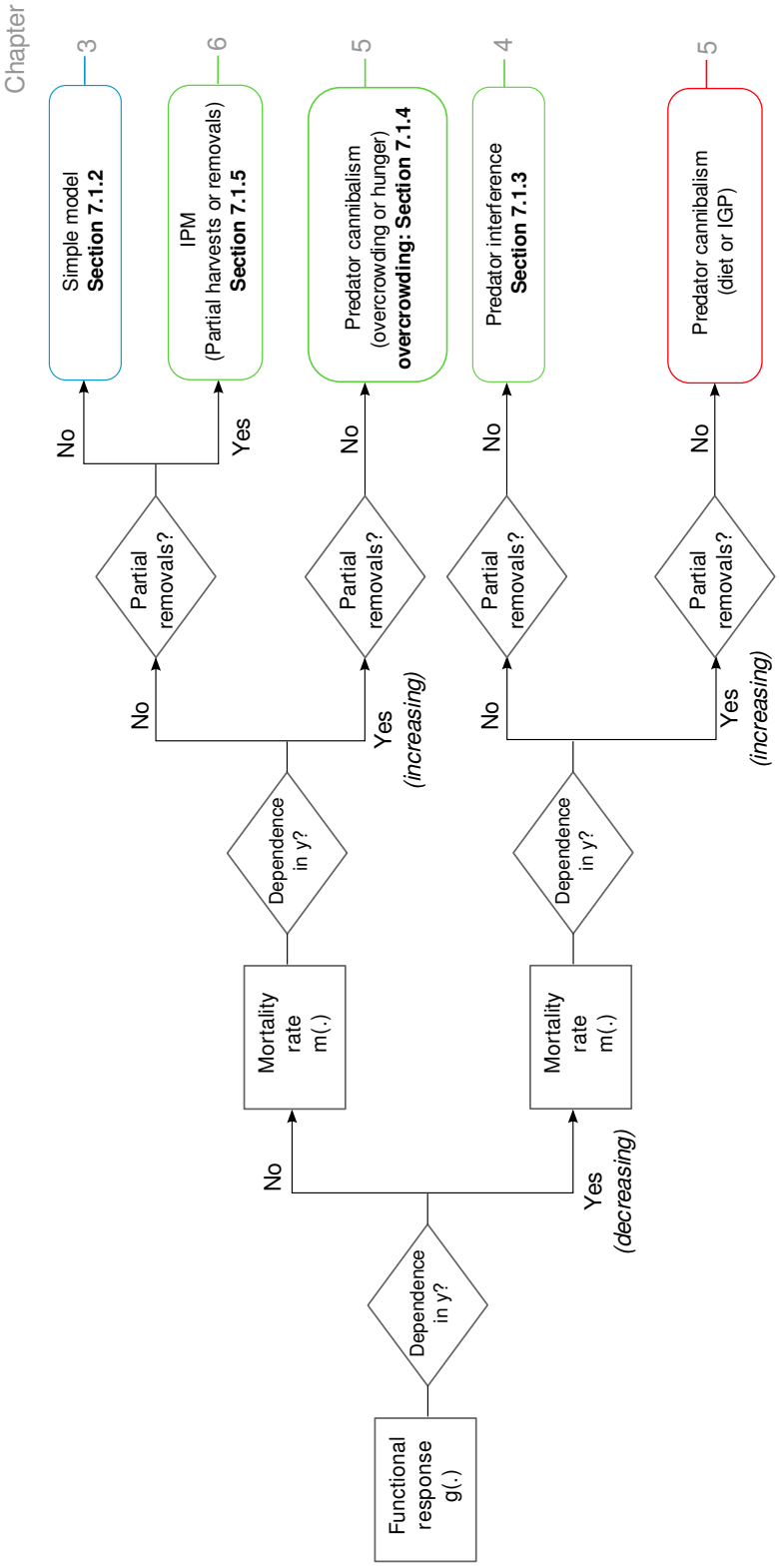

Figure 7.1: This decision tree describes how to match the data with a model. We look at the dependence of the functional response on the predator density (whether it is decreasing or constant); then at the predator mortality rate (increasing with predator density or constant); then whether we stay within a pure biological control vs. an IPM framework. The red box indicates that this model is not covered in the present chapter

ii. a mortality rate that is independent of the predator density *(bottom centre)*.

When only biological control is to be implemented, the **simple model** (Chapter 3) should provide a satisfactory description of the system dynamics.

Consequently, with reference to the Figure 7.2 and the choice of the model, the properties that need to be measured on the different data sets are:

1. *Top left.* the marginal pest growth at zero pest (the gradient of the pest growth at zero pest): $f'(0)$;
2. *Middle left.* the marginal functional response with respect to the pest at zero pest (the gradient of the functional response with respect to the pest at zero pest): $g'(0)$;
3. *Middle right.* the superiormost value of the ratio of the pest growth to the functional response over the range of pest population considered: S_g ;
4. *Bottom centre.* the mortality rate of the predator (the equation of this function with respect to the predator density): m.

Remark: *With respect to discrete data points, there is often some controversy that can arise in the number of points to consider in the calculation of marginal values, or gradients, at the origin. In our example, we chose to look at three consecutive points in order to calculate these gradients.* ■

Predator release strategy

The minimal release rate $\underline{\mu}$, that is the size of predators release **per unit time**, is then calculated as:

$$\begin{aligned} &\textit{For small invasions:} \quad \underline{\mu} = \frac{mf'(0)}{g'(0)}; \\ &\textit{For large invasions:} \quad \underline{\mu} = mS_g. \end{aligned} \tag{7.1}$$

For pest eradication, use a release rate above the $\underline{\mu}$.

If a lower release rate is used, the pest population is expected to persist. Figure 7.3 illustrates our model prediction. The left column shows pest eradication as the release rate is larger than $\underline{\mu}$ while the right column shows the pest population persisting as the release rate is insufficient.

Extra tip 1: To minimise the risk of pest damage [2], we recommend to use frequent small releases rather than rare large ones as it ensures the continued presence of predators in the crop. For example, if a 28-day calendar month $\underline{\mu}$ is calculated as 1200 predators per month, then a release strategy of 40 predators a day is more advisable than releasing 280 predators at the beginning of a week (which is in itself more advisable than releasing 1200 predators at the start of the month).

Extra tip 2: On detecting a pest invasion, the predators can be released in a large massive number satisfying the release rate for large invasions. Provided this release reduces the pest population quickly enough to small enough levels, a smaller predator release rate can be used - satisfying the release rate for small invasions - and spread over more frequent releases.

[2] This was recommended by Mailleret and Grognard (2009).

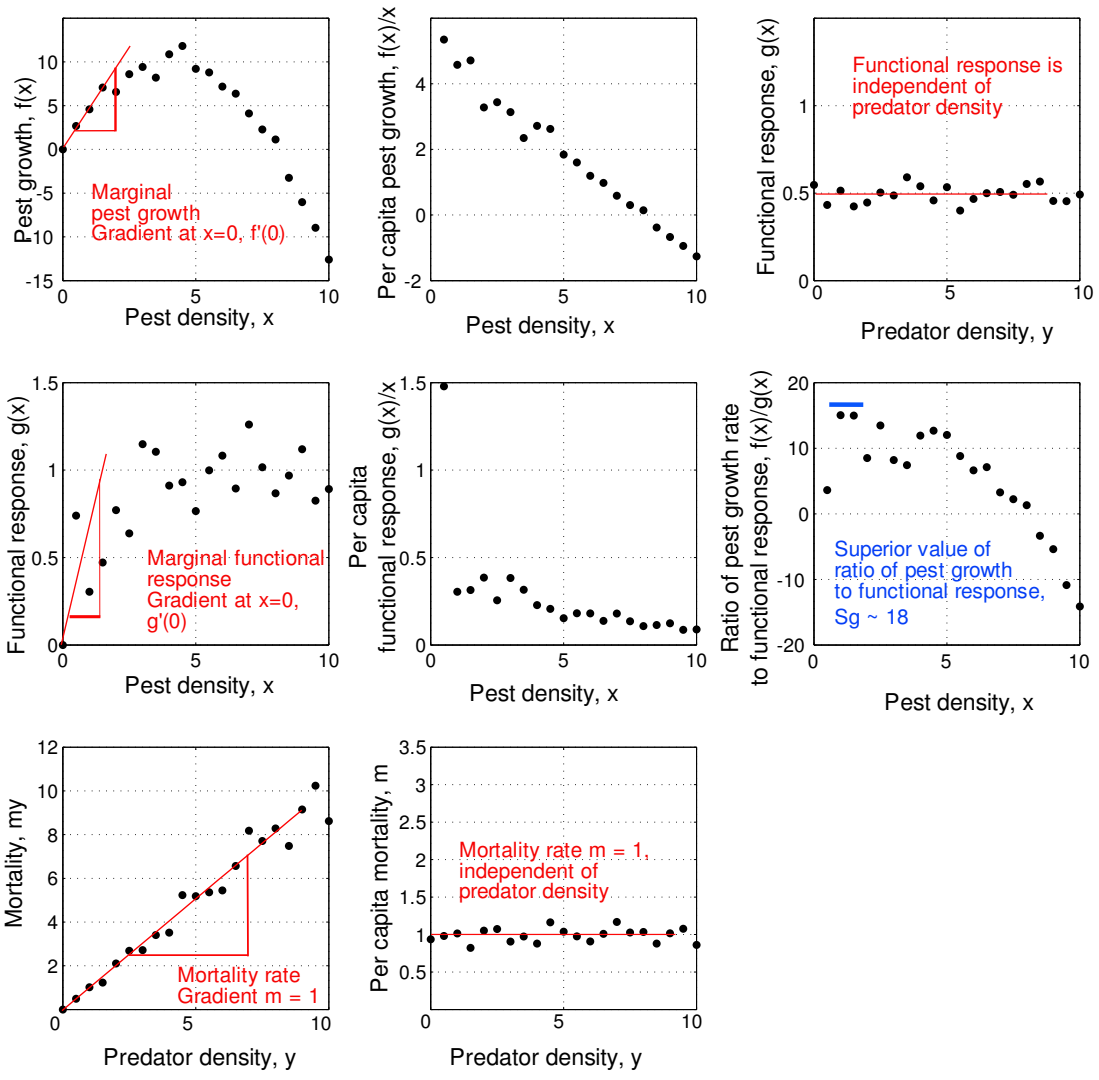

Figure 7.2: This data grid illustrates the values that need to be measured in order to calculate the minimal predator release rate for predator eradication predicted by the **simple model**.

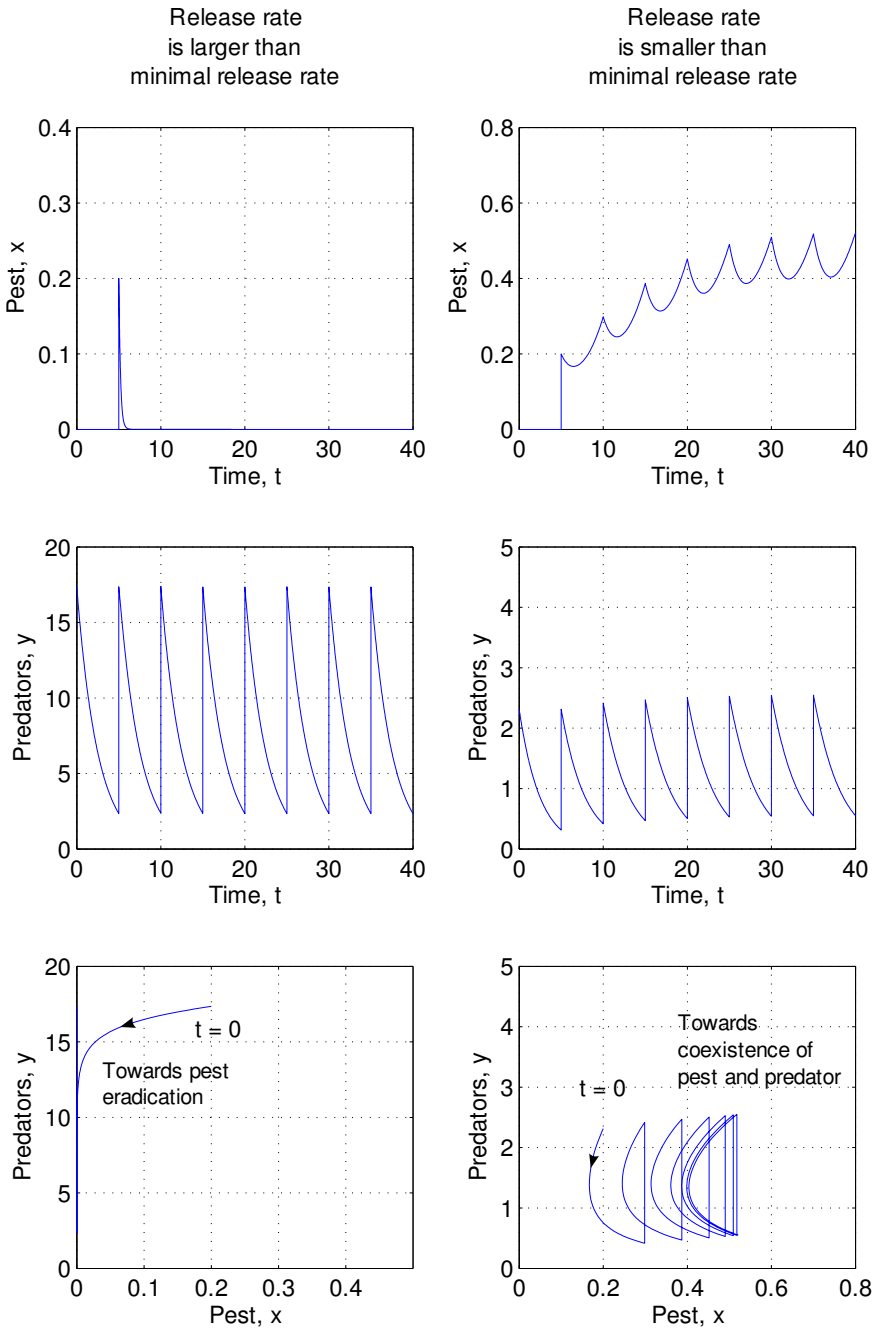

Figure 7.3: Simple model predictions: *Left.* The release rate is larger than the minimal release rate recommended at small invasions: for a small invasion, pests are eradicated. *Right.* The release rate is smaller than the minimal release rate: pests persist.

Remark: *Note that the units for the 'population size' are actually unknown; until experimental results allow us to callibrate the time and population scales, it is the* shape *of these curves as well as the ratio of one population to the other that the model predicts.* ■

7.1.3 Interfering predators

Data acquisition

The second data grid given in Figure 7.4 consists of a functional response that is decreasing with the predator density (but increasing with the prey). This implies interfering predators. When all other processes follow similar trends as in the 'simple model' case, the data is matched to the 'interference model' studied in Chapter 4 of this thesis.

The processes that need to be measured are given in Figure 7.4 – the figure is self-explanatory.

Here, note that a lower bound on the functional response is required *(Middle left)*. It is parameterised as:

$$\text{Lower bound} = g'(0)\frac{qax}{ax+qby+q}, \tag{7.2}$$

where a, b and q are constants:

a : index of pest vulnerability
b : index of intrapredator interference
q : parameter for lower bound

Predator release strategy

For pest eradication to be possible, first select the predator species appropriately: use predators with **interference index lower than a maximal interference threshold**, denoted $\bar{b}$ and calculated as

$$\begin{aligned} &\textit{For small invasions:} \quad \bar{b} = \frac{ag'(0)}{f'(0)}; \\ &\textit{For large invasions:} \quad \bar{b} = \frac{ag'(0)}{S}. \end{aligned} \tag{7.3}$$

Then, use a **predator release rate higher than the minimal release rate** $\underline{\mu}$ calculated as:

For small invasions:

$$\underline{\mu} = \frac{1}{b}\left(\frac{1-e^{-m\frac{bf'(0)}{ag'(0)}T}}{e^{-m\frac{bf'(0)}{ag'(0)}T}-e^{-mT}}\right)\left(\frac{1-e^{-mT}}{T}\right),$$

For large invasions:

$$\underline{\mu} = \frac{q+a\bar{x}}{qb}\left(\frac{1-e^{-m\frac{bS}{ag'(0)}T}}{e^{-m\frac{bS}{ag'(0)}T}-e^{-mT}}\right)\left(\frac{1-e^{-mT}}{T}\right).$$

where

T : release period
$\bar{x}$: estimate of maximal value the pest population can take

Figure 7.5 illustrates our model prediction when the predator interference is below $\bar{b}$ for the case that the release rate is large enough (the pest is eradicated), then too small (the pest persists).

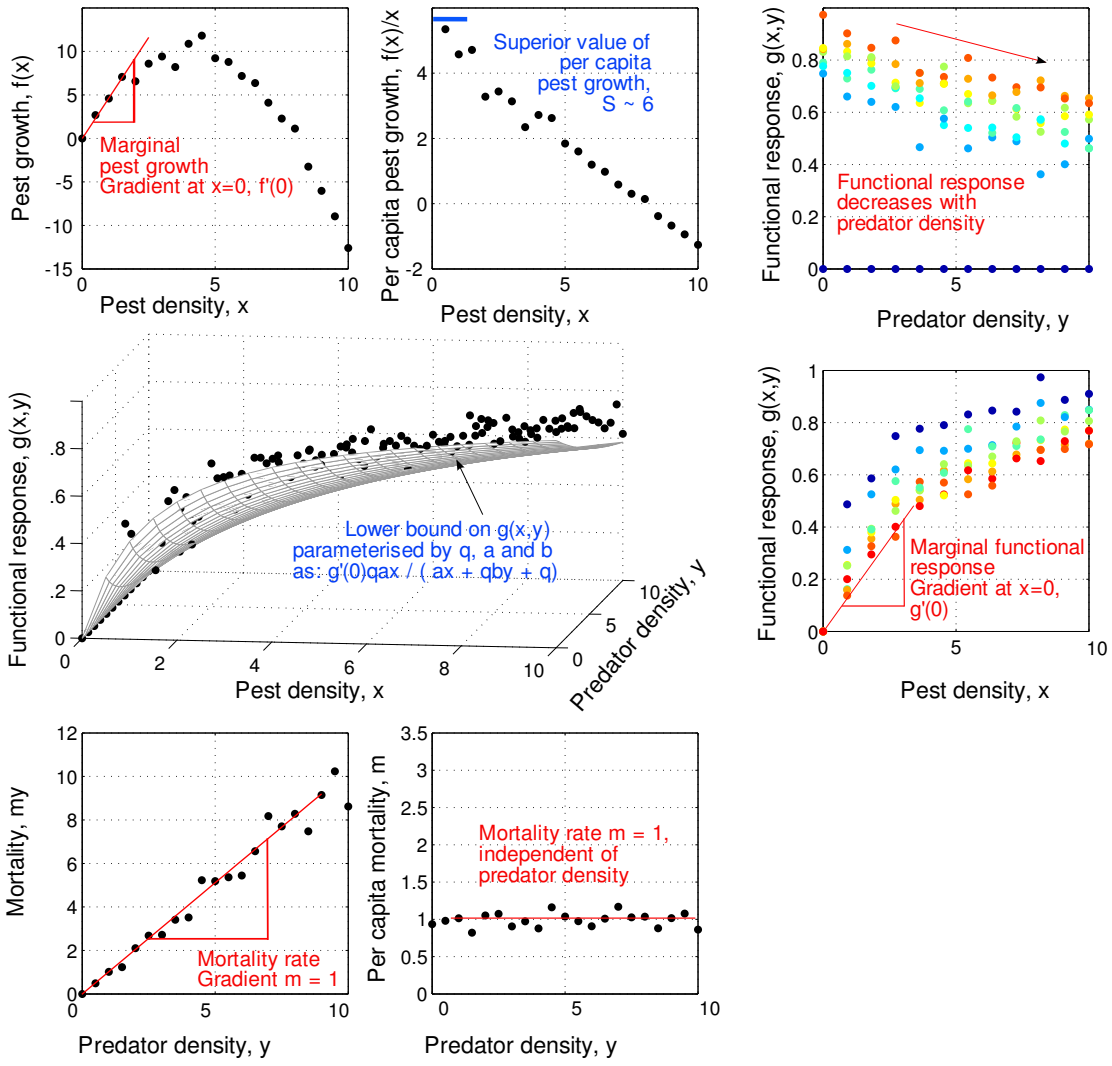

Figure 7.4: Data grid best matched by intrapredator interference model: *Top right.* The projection of the functional response data onto the $g(x, y)$ - y plane shows that the response decreases with predator density (dots with matching colour imply equal x values). *Middle right.* In the projection of the functional response data points onto the $g(x, y)$ - x plane , matching colours correspond to equal y values. *Middle left.* A key measurement for this model is the calculation of a lower bound on the functional response parameterised by q, a and b

Caution: If the predator species exhibits higher interference than $\bar{b}$, no release rate exists that can eradicate the pests.

Extra tips: Increase the speed of pest eradication by using as high a predator release rate as possible and as frequent releases as possible. Using as frequent releases as possible also contributes to lower the minimal release rate $\underline{\mu}$.

7.1.4 Overcrowding

Data acquisition

The third data grid given in Figure 7.6 consists of a functional response that is independent of predator density *(Top right)* but with a mortality rate that is affine in the predator density *(Bottom centre)*. This indicates overcrowding.

In a pure biogical control program, this system is best matched by the 'Cannibalism by overcrowing' model given in Chapter 5.

The processes that need to be measured are given in Figure 7.6 (the figure is self-explanatory).

Here, note that:

1. the natural predator mortality rate m is calculated as the intercept of the mortality rate with the vertical axis
2. the overcrowding parameter q calculated from the gradient of the mortality rate.

Predator release strategy

Use a release rate larger than the minimal release rate $\underline{\mu}$.

Here, $\underline{\mu}$ is calculated **numerically** such that:

$$y^* = \frac{1}{2}\left(\underline{\mu}T - \frac{m}{q} + \sqrt{\left(\underline{\mu}T - \frac{m}{q}\right)^2 + \frac{4\underline{\mu}mT}{q(1-e^{-mT})}}\right) \tag{7.4}$$

gives:

$$\begin{aligned} &\textit{For small invasions:} \quad \ln\left(1+\tfrac{q}{m}(1-e^{-mT})y^*\right) - \tfrac{f'(0)}{g'(0)}T = 0, \\ &\textit{For large invasions:} \quad \ln\left(1+\tfrac{q}{m}(1-e^{-mT})y^*\right) - ST = 0. \end{aligned} \tag{7.5}$$

Figure 7.7 illustrates our model prediction when the predator interference is below $\bar{b}$ for the case that the release rate is large enough (the pest is eradicated), then too small (the pest persists).

Extra tip: Use high frequency of releases, or as small T as possible, in order to lower the minimal release rate and guarantee pest eradication.

Remark: *We do not discuss the two other forms of cannibalism studied in Chapter 5: the data measurements are carried out in a similar manner as described above. Curve fittings for upper bounds on the mortality rates are required: for an example, see Section 7.1.3.* ■

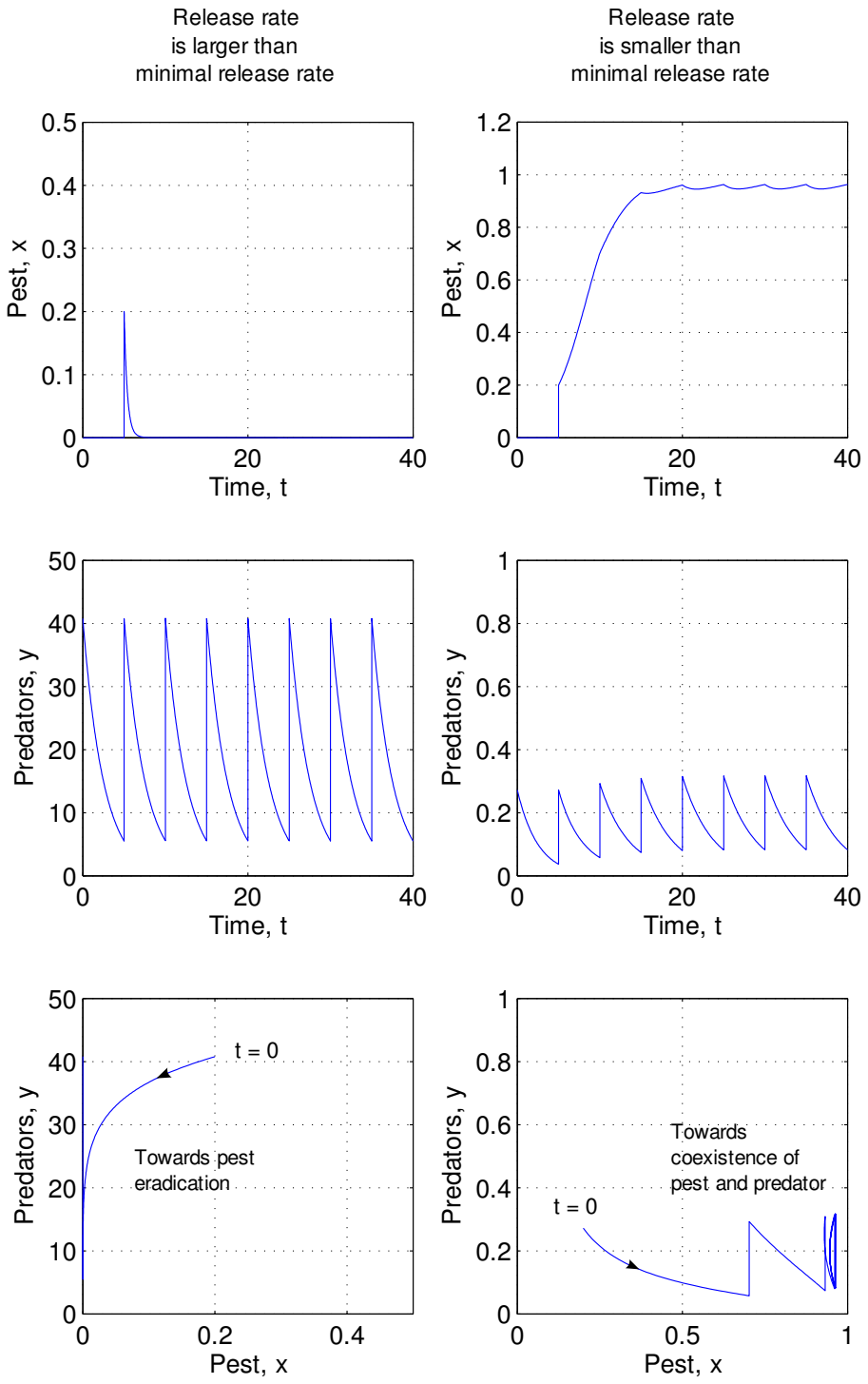

Figure 7.5: Intrapredator interference model predictions. *Left.* The release rate is larger than the minimal release rate recommended at small invasions: for a small invasion, pests are eradicated. *Right.* The release rate is smaller than the minimal release rate: pests persist. In both cases, the intrapredator interference is below the threshold $\bar{b}$.

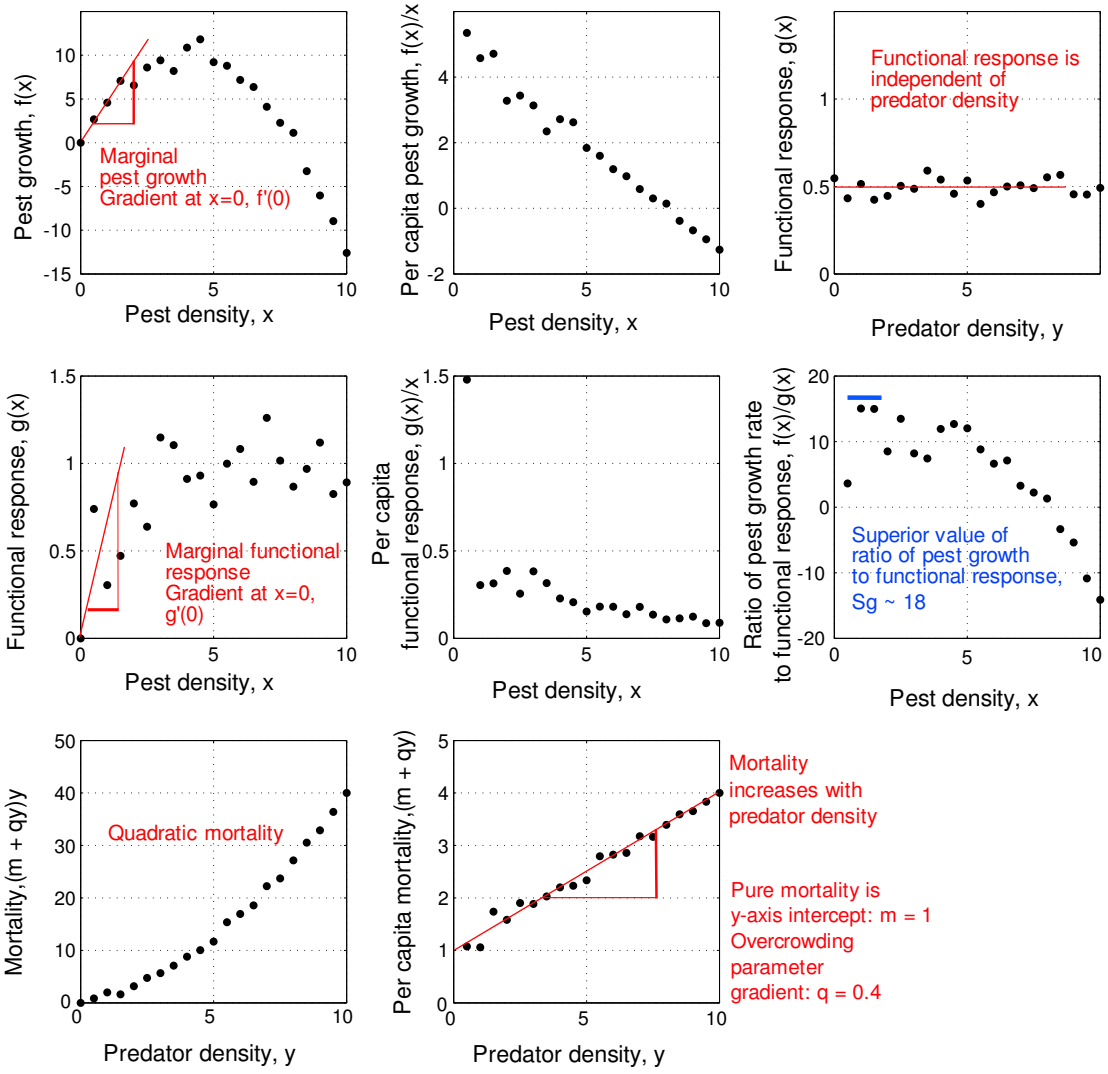

Figure 7.6: Data grid best matched by overcrowding model. *Bottom right.* When the mortality rate (or per capita mortality) increases with predator density, this indicates overcrowding effects. Among the key features that need to be measured is the intercept of the per capita mortality with the vertical axis for the natural mortality m; the overcrowding parameter is calculated as the gradient of this function.

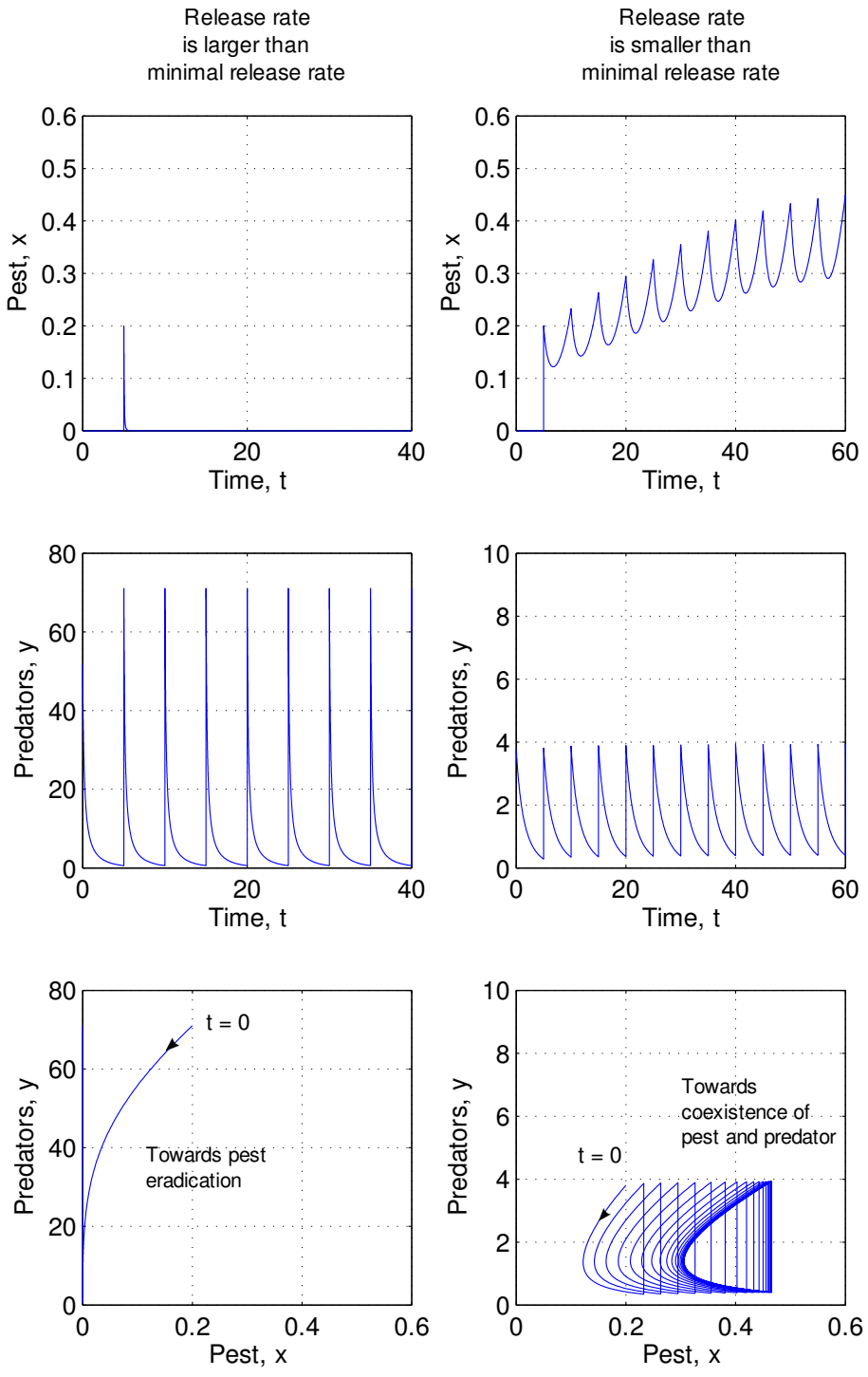

Figure 7.7: Overcrowding model predictions. *Left.* The release rate is larger than the minimal release rate recommended at small invasions: for a small invasion, pests are eradicated. *Right.* The release rate is smaller than the minimal release rate: pests persist.

7.1.5 Integrated Pest Management (or partial harvests)

Data acquisition

If we want to implement an Integrated Pest Management (IPM) program on a system where biological processes display similar general properties as in the data grid in Figure 7.2, the model to use is the 'partial harvest' model studied in Chapter 6 of this thesis.

This model caters for partial removals of either the pest or predator populations due to:

- partial harvests of the plant (e.g. ornamentals), pruning;
- pesticide usage within an IPM program;
- other physical removals (e.g. heat treatments, manual control, etc) available in an IPM program.

The model applies to the case when the predator release period is a complete integer multiple of the partial removal period ('**less frequent releases**'), and vice-versa ('**more frequent releases**').

The data sets and properties that need to be measured match those of the simple model (see Figure 7.2). In addition, we require the values of:

T_h : partial removal period (harvest period or period of pesticide spraying
α_x : fraction of pest *pop.* removed at harvest or pesticide application (that is pesticide strength on pest)
α_y : fraction of predator *pop.* removed at harvest or pesticide application (that is pesticide strength on predator)

Also,

$(1-\alpha_x)$: pest resistance to pesticide application
$(1-\alpha_y)$: predator resistance to pesticide application

Predator release strategy

Use a predator release rate **larger** than the minimal release rate $\underline{\mu}$ calculated as:

For less frequent releases:

$$\underline{\mu} = m\left(C_1 + \frac{\ln(1-\alpha_x)}{C_2\, T_h}\right)\frac{1-(1-\alpha_y)e^{-mT_h}}{1-e^{-mT_h}},$$

For more frequent releases:

$$\underline{\mu} = m\left(C_1 + \frac{\ln(1-\alpha_x)}{C_2\, T_h}\right)\frac{1}{1-(\frac{\alpha_y(1-e^{-mT_h})}{1-(1-\alpha_y)e^{-mTh}})\frac{e^{-mT}}{k(1-e^{-mT})}}.$$

where C_1 and C_2 values to take depend on the pest invasion size:

$$\begin{aligned} &\textit{For small invasions:} \quad C_1 = \frac{f'(0)}{g'(0)} \quad \text{and} \quad C_2 = g'(0) \\ &\textit{For large invasions:} \qquad\quad C_1 = S \quad \text{and} \quad C_2 = r \end{aligned} \tag{7.6}$$

Figure 7.8 illustrates our model prediction for the 'releases less frequent' case: when the release rate is above $\underline{\mu}$ (the pest is eradicated), then too small (the pest persists). Figure 7.9 illustrates our model prediction for the 'releases more frequent' case.

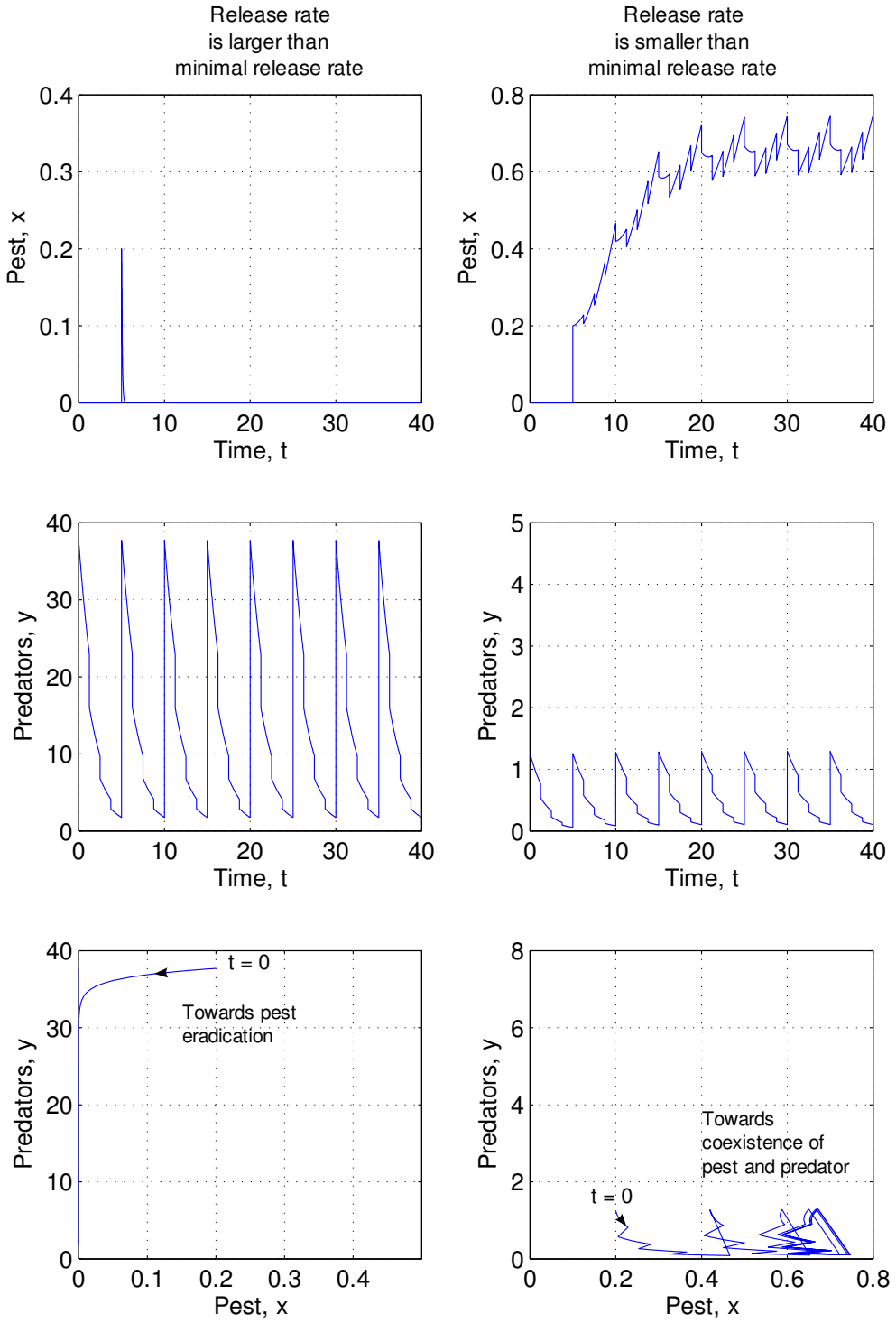

Figure 7.8: Partial harvest model predictions (releases less frequent: $T = 5$, $T_h = T/4 = 1.2$). On the left, the release rate is larger than the minimal release rate recommended at small invasions: for a small invasion, pests are eradicated. On the right, the release rate is smaller than the minimal release rate: pests persist.

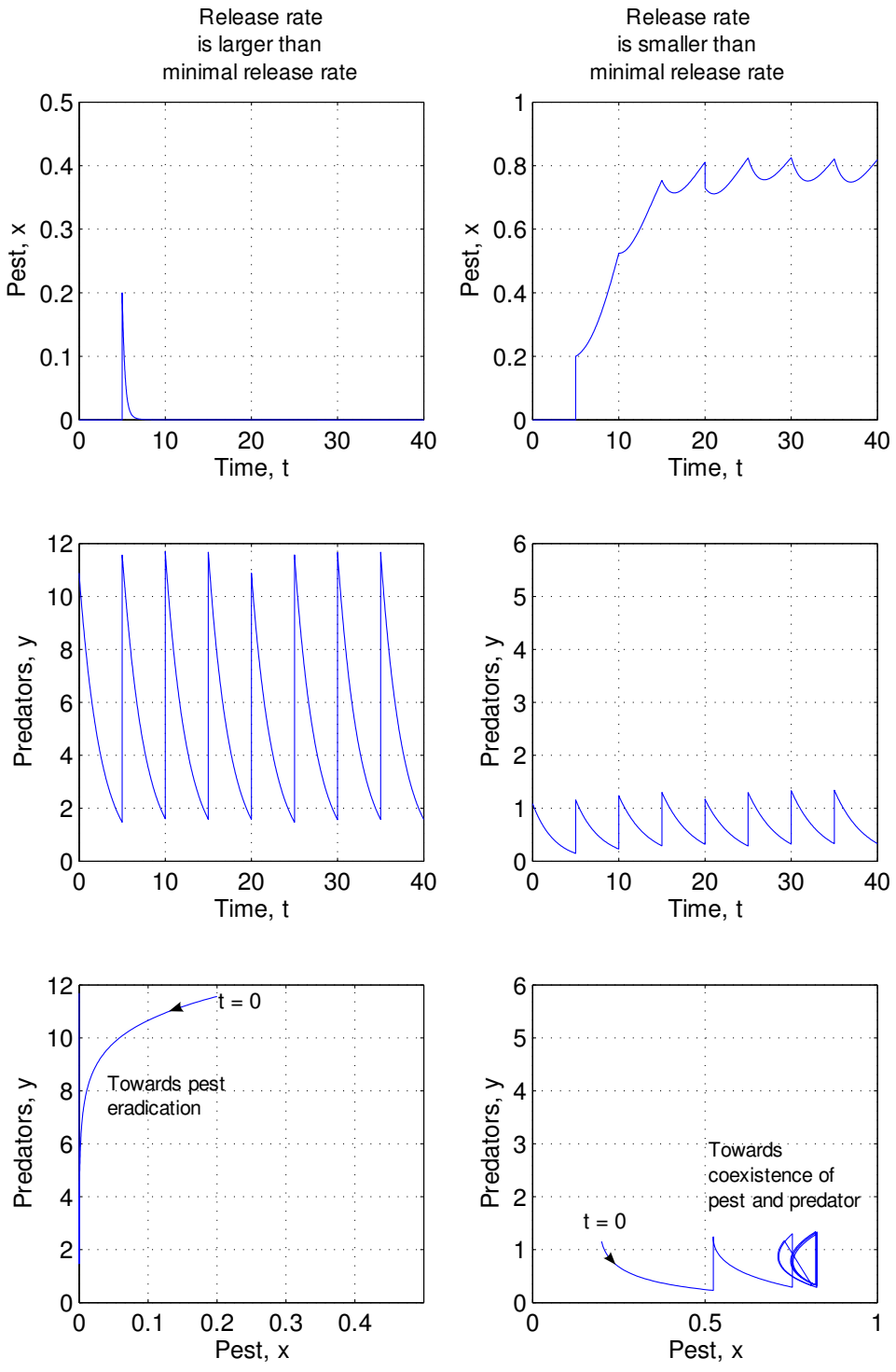

Figure 7.9: Partial harvest model predictions (releases more frequent: $T = 5$, $T_h = 4T = 20$). On the left, the release rate is larger than the minimal release rate recommended at small invasions: for a small invasion, pests are eradicated. On the right, the release rate is smaller than the minimal release rate: pests persist.

Extra tip: Release as frequently as partial harvests to lower the minimal release rate and to keep risks of damage on a pest invasion as low as possible.

Caution: The predictions do not apply when one interval is not a complete integer multiple of the other.

Dealing with pesticide resistance

In Chapter 6, we found that partial removals can be complementary to, have no effect on, or be disruptive to a biological control program. In an IPM program, the minimal release rate is[3]*:*

$$\underline{\mu}(\text{IPM}) = m\left(C_1 + \frac{\ln(1-\alpha_x)}{C_2\, T_h}\right)\frac{1-(1-\alpha_y)e^{-mT_h}}{1-e^{-mT_h}},$$

whereas in a purely biological control (only predator releases), the minimal release rate is:

$$\underline{\mu} = mC_1.$$

We define disruption', 'complementarity' and 'no effect' of pesticide applications as in Table 7.1. Graphical plots can be used to achieve the desired complementarity, and identify short-term and long-term pesticide application strategies as resistance to a pesticide increases.

	Sign[‡]	Effect	Action
$\underline{\mu}(\text{IPM}) < \underline{\mu}$:	$(+)$	Complementary	Maintain
$\underline{\mu}(\text{IPM}) = \underline{\mu}$:	0	None	Reassess
$\underline{\mu}(\text{IPM}) > \underline{\mu}$:	$(-)$	Disruptive	Reassess and change[†]

Table 7.1: Identifying the effects of the partial removals on a biological control program carried out by predator releases only, and determining the actions. [‡]Sign of $(\underline{\mu} - \underline{\mu}(\text{IPM}))$ indicates whether the IPM program requires a higher predator release rate than a pure biological control program. [†]Change of pesticide application strategy implies changing the frequency of application or switching to a purely biological control program if necessary.

Dealing with pesticide resistance requires a three-stepped strategy which we describe below:

1. **Initial strategy:** For a given pesticide strength, plot a graph of $\underline{\mu}(\text{IPM})$ versus application periods. Identify a value of T_h values for which $\underline{\mu}(\text{IPM}) < \underline{\mu} = mC_1$.

   The first graph is the green curve on Figure 7.10 (*top*). The chosen application period is T^*.

2. **Monitoring:** For the chosen application period, plot a map of $(\underline{\mu}(\text{IPM}) - \underline{\mu})$ with respect to the pesticide strength parameters α_x and α_y. As predator and pest resistance changes, refer to the map to identify whether pesticides aid towards the biological control program or not.

[3]Release as frequently as the partial pesticide usage as advised in the last 'Extra tip'.

Point 1 (also in green) in Figure 7.10 (*bottom*) corresponds to the pesticide strength on prey and predator at the chosen T^* value. The pest resistance evolves over Point 1 (green) - Point 2 (blue) - Point 3 (red) (decreasing pesticide strength). Here predator resistance is fixed.

3. **Reassessing strategy:** When pesticide strength has decreased so that $\underline{\mu}_{(\text{IPM})} \geq \underline{\mu}$, plot a graph of $\underline{\mu}_{(\text{IPM})}$ (for new and expected or a future pesticide strengths) versus application periods (e.g. Figure 7.10 (*bottom*)). Devise the new strategy based on this second graph.

   At Point 2 (blue - current pesticide strength, (bottom)), pesticide usage has no effect on the pest control program. This combination yields the blue curve for minimal release rate $\underline{\mu}_{(\text{IPM})}$ versus application period (*top*). Higher pesticide resistance from the pest (Point 3 - red, *bottom*) gives the red curve (*top*). From Point 2, the farmer has four choices.

   - Case (i) **(long-term cost: medium)**: maintain the pesticide application program as it is. The minimal release rate jumps to Point 3.
   - Case (ii) **(short-term cost: lowest, long-term cost: highest)**: apply pesticides more frequently. The minimal release rate is lowered (Point 2a, bottom plot), but a further increase in pest resistance requires the minimal release rate to jump to a much higher value (Point 3a, bottom plot).
   - Case (iii) **(low cost at all times)**: apply pesticides less frequently, there is only a slight short-term gain for the biological control program (Point 2b), but in the longer-term, the minimal release rate also is maintained below the minimal rate of a pure biological control program (Point 3b).
   - Case (iv) (not shown): stop pesticide applications. The minimal release rate stays fixed (Point 2).

Remark: *In this example, applying pesticides at low frequency gives a more 'robust' predator release program and has the added benefit that resistance is also developed more slowly.* ■

Caution: Pesticide applications can provide complete pest control provided they take place at periods smaller than some threshold value of T_h (given as the T_h intercept of the curves). This threshold period becomes smaller as α_x decreases. It becomes physically impossible to apply chemical pesticides at extremely high frequency; a more frequent application of chemicals only worsens the problem of resistance.

7.2 Experiments with the predator *Neoseiulus cucumeris* to control the pest *Frankliniella occidentalis*

The Unité de Recherches Intégrées en Horticulture (URIH) is a research team based at INRA - Sophia Antipolis (France) that is currently in charge of 4800 m^2 of experimental greenhouse-grown rose crops. These roses are prone to attacks by the western flower thrips *Frankliniella occidentalis* (Pergande), a major pest in the Mediterranean region. Females of the predatory

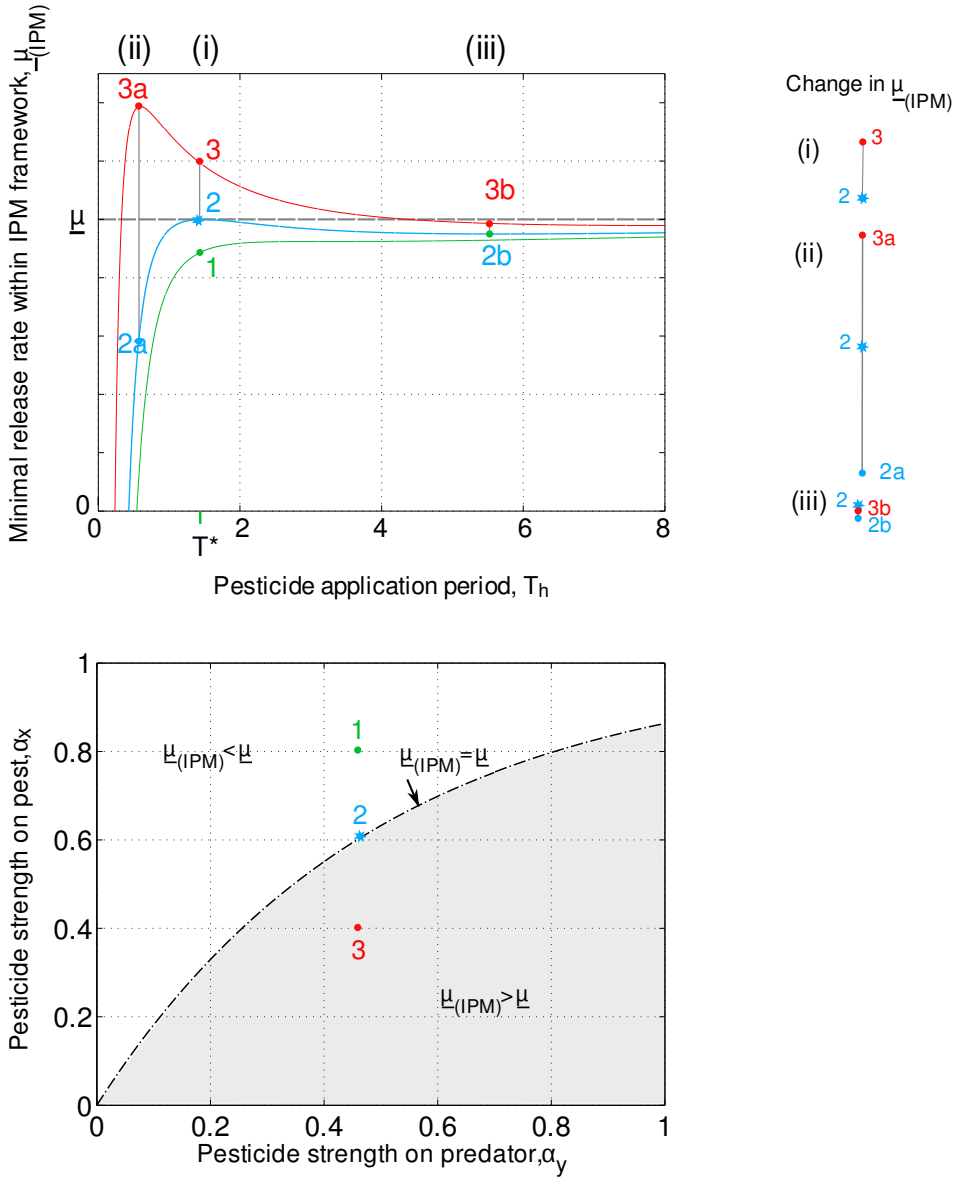

Figure 7.10: These two graphs provide a decision tool for the pesticide application strategy to put into place when pesticide resistance evolves; matching colours imply the same combination of predator-prey pesticide resistances. The evolution is as: green = initial (smallest) resistance $\rightarrow$ blue = current (medium) resistance $\rightarrow$ red = predicted (highest) resistance. A pesticide application strategy at a given period T^* is reassessed to Strategy (i), (ii) or (iii) when resistance reaches Point 2. The effect on $\underline{\mu}_{\text{IPM}}$ is visualised on the *top right* sticks. Strategy (iii) gives the best short-term benefits of the three, while Strategy (ii) gives the best long-term benefits. Note that this analysis is by no means exhaustive but has a didactic purpose.

mite *Neoseiulus cucumeris* (Oudemans) are well-known predators of the larvae of this pest: their success in biologically controlling *F. occidentalis* has been reported on potted roses (see Linnamäki et al. (1998) in Gerson (2007)). Following our proposals, the URIH set up two experiments to investigate intrapredator interference and compare predator release strategies.

7.2.1 Experiment 1: intrapredator interference

Interfering behaviour among N. cucumeris *in preying on* F. occidentalis *was reported by Skirvin et al. (2005) on chrysanthemums plants. The purpose of this experiment was to investigate if the predator exhibited similar interference on rose plants. It was motivated by the results in Nundloll et al. (2010a)*[4] *which demonstrated that the interference strength influenced a biological control program and that the selection of a potential biological control agent had to take into account such interference. So, in this experiment, we compared the per capita predation rates*[5] *of a single predator and a group of five predators. In particular we aimed to answer the question: is the per capita predation rate of this predator penalised by higher predator population sizes?*

Experiments were carried out by Guillaume Vitte during his internship at URIH in 2008, under the supervision of Alexandre Bout, and reported in detail in Vitte (2008).

Short description

This experiment consisted of a series of 24-hour trials in **controlled laboratory chambers**. Using a similar experimental protocol to Madadi et al. (2007); Zilahi-Balogh et al. (2007), the temperature was maintained at a constant level at 25°C and humidity at 60%. The light regime was set to 16 hours of light versus 8 hours of darkness.

Under this protocol, homogenoeous cohorts of the pest were required: to ensure a continuous supply of the prey (stage 1 larvae 'L1' thrips), thrips were reared on bean plants *Phaseolus vulgaris* that were grown in two phytotrons[6] following the protocol by Steiner (1998). The L1 thrips were selected by cohorts of 60 individuals, twice a week (30-60 L1 larvae were obtained per phytotron per harvest).

To identify female predators, the predatory mite population was isolated during 24 hours at the end of which only individuals that had laid eggs were selected. These selected females were then starved for a further 24 hours to control for satiation level.

To simulate leafscape effects (that is, related to the surface area, the shape and structure of the leaf), two sets of trials were carried out: one on circular leaf discs[7] with diameter equal to 4 cm ('disc') and the other on a whole leaf consisting of five leaflets ('leaf')). We collected data on the total surface area of the leaves used for each trial. The leaf discs were placed on humid flower foam soaked in a petri dish half-filled with water. Similarly, the leaves (with five leaflets) were pinned at their petiole in a humid flower foam. Figure 7.11 shows the experimental set-up.

Trials were carried out in batches of three in three separate petri dishes. At the start of the experiment, 10 thrips were placed in each of the petri dishes on the leaf disc or complex leaf. Predator numbers differed from one dish to the other: the dishes contained none (Dish 1: 'control'), 1 predator only (Dish 2: 'single'), and 5 predators (Dish 3: 'many') respectively.

After 24 h, a count of the number of dead (carcasses on the leaf only, that is excluding thrips in water) or disappeared thrips and predatory mite was taken (disappeared individuals

[4] See also Chapter 4 of this thesis

[5] The per capita predation rate corresponds to the functional response.

[6] A phytotron is a completely closed usually indoors greenhouse that can be used for the study of environmental plant growth and in which the production (and consumption) of gases can be monitored.

[7] The leaf disc is obtained by punching a 4 cm hole into a leaflet.

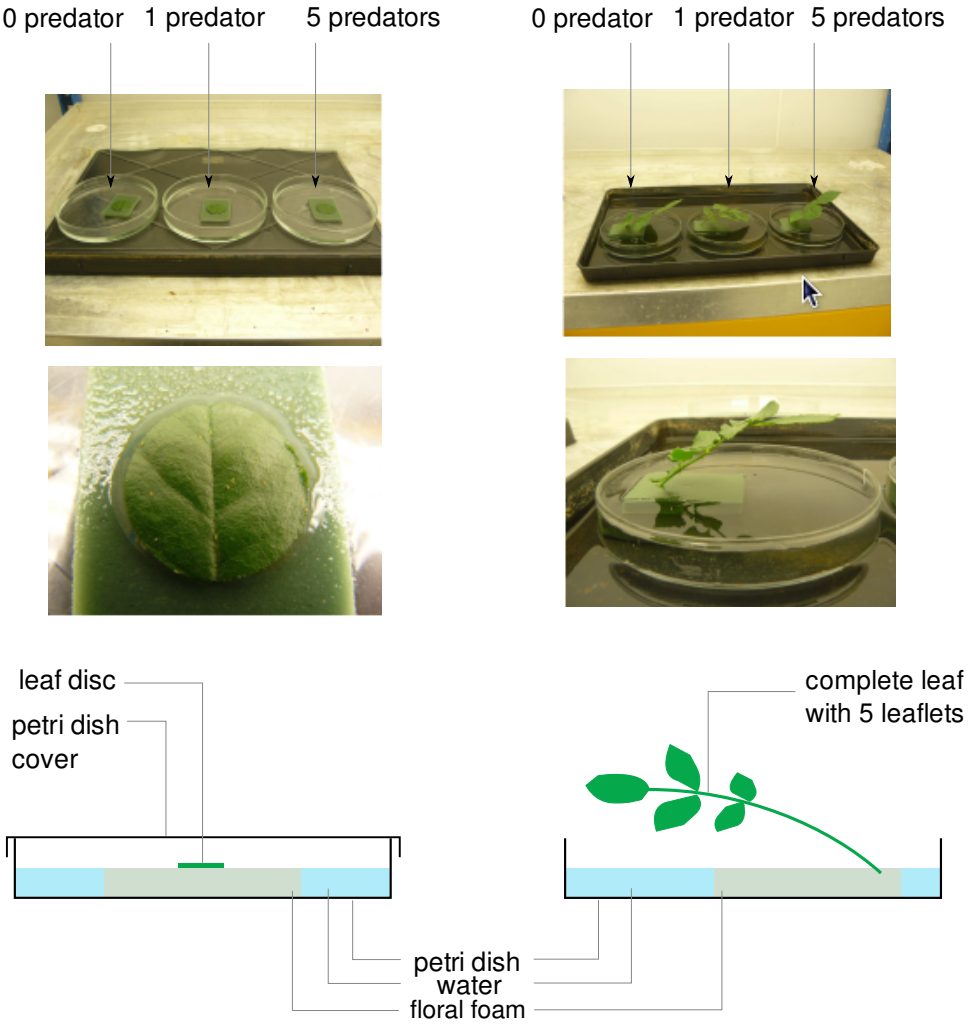

Figure 7.11: During this experiment, trials were carried out in batches of three in three separate petri dishes, all of which contained 10 thrips at the beginning of the experiment. Predator numbers differed from one dish to the other: the dishes contained none ('control'), 1 predator only ('single'), and 5 predators ('many') respectively. *Left:* leaf discs with diameter equal to 4 cm were used as the arena for the predatory mites and thrips interactions ('disc')). The yellowish spots on the leaf disc in middle figure are thrips and predatory mites. *Right:* complete leaf with five leaflets provided a more complex landscape feature ('leaf'). The experiment consisted of 20 trials.

were counted as dead). Thrips and predatory mites have lengths of the order of $0.1mm$, and being of the same colour, cannot easily be distinguished by the naked eye. The identification was carried out using a stereomicroscope at 20x magnification. If no thrips or no predatory mite (in Dishes 2 and 3) were recovered alive, the observation was not included in the data analysis.

We assume that there is no death of larval thrips due to natural causes at the end of the 24 hour period. Deaths of thrips due to predation was calculated as explained in Table 7.2 below. Note in particular that we use the average number of thrips killed by cannibalism over the number of valid trials.

Dish ID:	**Cause of thrips death**	**No. of dead thrips**
1:	Cannibalism only	N_1
2 (or 3):	Predation and/or cannibalism	N_2 (or N_3)
$\therefore$ 2 (or 3):	Predation only	$(N_2 - < N_1 >)^{\dagger}$ (or $(N_3 - < N_1 >)^{\dagger}$)

Table 7.2: At each trial, calculating the thrips death by predation involves subtracting the number of dead thrips in the 'control' dish (Dish 1) which corresponds to cannibalism. †We use the mean number of cannibalised thrips $< N_1 >$ averaged over the number of valid trials. The number of dead thrips is set to zero if the calculation yields a negative result.

We consider that the number of predators that have been found at the end of the experiment equals the number of predators which consumed the thrips. We calculate the mean number of predators at the end of a trial for Dish 3 over the whole series, and use this value to calculate the average predation on Dish 3 per trial.

Results

The mean predation were calculated for each modality ('single' versus 'many' predators) for each set-up ('disc' versus 'leaf'): this value was lower in the 'many' modality than in the 'single' one. Predation was also lower on the complete leaf than on the leaf disc. This suggests intrapredator interference that penalises predation. Results are given in Figure 7.12

The predation data from this experiment were subjected to an Analysis of Variance (ANOVA) test with respect to predator numbers ('single' versus 'many') and area ('disc' versus 'leaf'). The p-value indicates how similar the data sets being compared are. For the data to be statistically significant (that is, for data sets to be effectively different), p must be smaller than 0.05.

The results are summarised in Table 7.3. The first line confirms that the mean predation per predator at one predator being higher than that of at five predators (given in Figure 7.12 above) is statistically significant. Note however that surface area contributes significantly to predation (second and third lines).

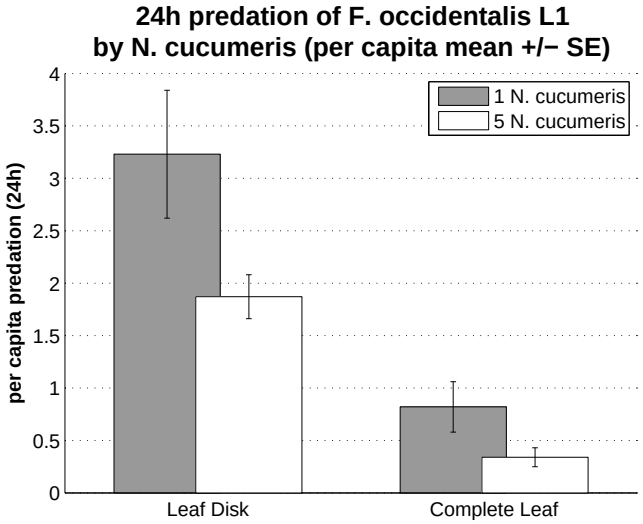

Figure 7.12: Per capita predation was lower in the 5-predator ('many') modality than in the 1-predator case ('single') in both the leaf disc and complete leaf set-ups. This suggests intrapredator interference penalising predation. The predation for the 1-predator modality was calculated over 12 trials; predation in the 5-predator modality was calculated over 17 trials.

Between 'single' and 'many' predators treatment:	$p = 0.00110$
Between 'leaf' and 'disc' set-up:	$p = 1.54$ x 10^{-8}

Table 7.3: Summary of statistical tests comparing predation. Tests are statistically significant.

Summary

In this experiment, predatory mites N. cucumeris *were less effective predators of the thrips* F. occidentalis *in the presence of conspecifics. Results were statistically significant. This indicates that some form of intrapredator interference which affects predation. Further investigations can be carried out for larger populations of thrips and predatory mites and to calculate the interference parameter strength.*

7.2.2 Experiment 2: release frequencies and pest control

Given the intrapredator interference observed in Experiment 1, we wanted to test the prediction by Nundloll et al. (2010a) that, for a fixed overall number of predators, more frequent and smaller releases were more effective than rare larger ones. In Experiment 2, two release treatments were therefore used to control an artificial pest invasion: 'T_{small}' (predators were released every week) and 'T_{large}' (predators were released every four weeks). To assess and compare these two treatments, the aim of the experiment was to answer the question: which one kept it at a lower level during the whole or most of the time?

The experiment can be read in more detail in Pizzol et al. (2009).

Short description

This experiment took place from April (**Week 17**) to September 2006 (**Week 37**) in a **greenhouse**. Predatory mites *N. cucumeris* were bought from Syngenta Bioline (UK) in packets of 1000. Thrips *F. occidentalis* were reared on bean plants in a phytotron (as described in Experiment 1).

The greenhouse consisted of three benches (denoted Bench A, Bench B and Bench C), each with three compartments of surface area $2\ m^2$. On the 28th of April 2006, in **Week 17**, two rows of roses (Aubade®cultivars) were planted in each bench. Humidity, temperature, light shades openings were controlled automatically, as well as irrigation with agricultural effluents rich in mineral fertilisers[8].

In **Week 24**, the plants were chemically treated with lufenuron[9], a pesticide, to eliminate any thrips *F. occidentalis* present in the greenhouse before the start of the experiment. The aim was to ensure that the population of thrips that is artificially released into the greenhouse is uniformly spread. A first count of thrips shortly after the pesticide release confirmed that the thrips population was zero.

Starting in **Week 25** until the end of the experiment in **Week 37**, predators were released in Benches B and C. No predator was released in Bench A ('Control'). In each of the compartments of Bench B ('T large' treatment), predators were released at a density of one packet per m^2, that is two packets were released in total every four weeks. In each of the compartments of Bench C ('T small' treatment), predators were released at a density of 0.25 packet per m^2, that is 0.5 packet was released every week . Overall, the same number of predators were released during Treatments 2 and 3 (that is two packets per month).

From **Week 30 to Week 34**, 20 thrips were released at a rate of five thrips per week in each of the nine compartments (over Benches A-C).

The thrip population on the roses was tracked weekly without destroying any plant by the following method: in each compartment six roses or rose plants under which a sheet of

[8] a process known as fertigation or fertirrigation in French

[9] Incidentally, lufenuron usage has been banned by the European Parliament in January 2009.

white paper was placed were tapped gently. Thrips that fell off onto the paper support were counted. The fallen thrips and predatory mites were put back onto the roses. This method minimised disruptions on the plant-thrips-predatory mite system. A separate count was kept for the larvae and the adult thrips at various stages of the plant growth.

Results

For our analyses, only the thrips population data from Week 30 (at the first release of thrips), then from Week 32 up to Week 37 were used. The data for the thrips population count was log-transformed (see Figure 7.13), then analysed. Results of the statistical analysis are summarised in Table 7.4.

In the absence of either form of treatment, the pest population grows unchecked ('Control'). Small-sized more frequent releases ('T_{small} treatment') keeps pests at lower levels than a larger-sized less frequent release ('T_{large} treatment'): this is validated by p-value of 0.1. Note that, as a side result, we obtain a statistically significant difference between the control and either release treatment which confirmed that *N. cucumeris* was an effective biological control agent against *F. occidentalis*.

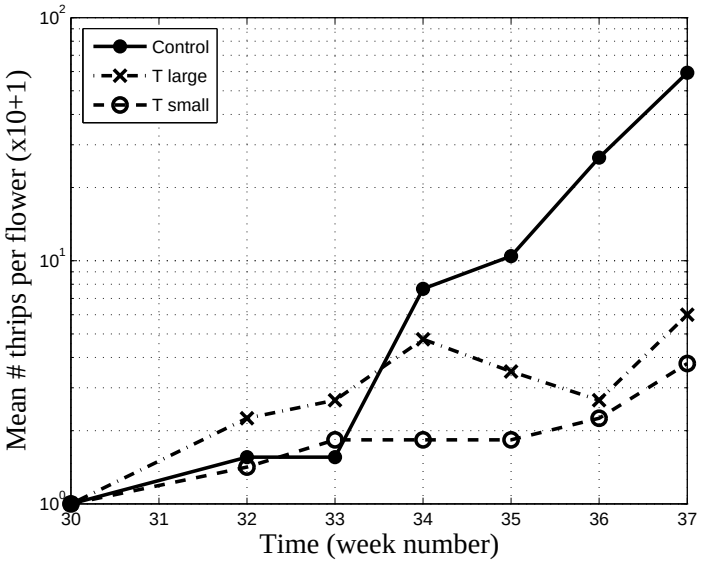

Figure 7.13: Artificial pest invasions or pest releases were carried out at Weeks 30-34. The pest population increased significantly when there were no predator releases ('Control'). A treatment of 500 predators every week ('T_{small}') kept the pest population at a lower level than 2000 predators every month ('T_{large}').

Remark: *The data for each compartment are repeated measures on the same population and so are subjected to what is called "pseudo-temporal replication". In any given compartment, the thrips population in one week depends on the population value in the preceding week. We applied the "repeated measures analysis" method proposed by the University of California in Los Angeles on its web site UCLA: Academic Tech-*

Between control and T_{small} treatment:	$p = 5.31$ x $10^{10} < 0.0001$
Between control and T_{large} treatment:	$p = 3.7$ x $10^{-7} < 0.0001$
Between T_{small} and T_{large} treatments:	$p = 0.101$

Table 7.4: Summary of statistical tests comparing predator release rates

nology Services (2010). This involves fitting under the software R[10] a linear model with, as factors, 'Treatment' (here 'T_{small}' or T_{large}), 'Time' (here week number) and the interaction between 'Treatment' and 'Time'. The significativity of the treatment effect over time is then given by the p-value associated with the interaction between time and effect.[11] ■

Summary

In this experiment, we were able to show that pest control is improved when smaller and more frequent predator releases are used as opposed to larger and rarer ones. This agrees with the prediction by Nundloll et al. (2010a). Results are however not significant enough (the statistical significance threshold being at 0.05): this implies that more experiments are required to validate it. In addition to replicate the experiment, we could, to improve it, also test the impact of other release periods and predator release sizes.

[10] See R Development Core Team (2009).

[11] Interestingly, this method also reduces to computing the significativity of the differences between the *slopes* of the counts in the different treatments with respect to time (Wonnacott and Wonnacott, 1990).

8

Conclusion and prospects

"A farm is not just a clever crop: it is an ecosystem managed with intelligence."
– The Economist, 2010.

Overview

In this chapter, we give a general summary of our work (in **Section 8.1**). We also specify the publications that arose from it. Then (in **Section 8.2**), based on our investigations, we give a critical analysis of our results. We highlight the limitations of our work, then describe how our models can be improved and what potential avenues exist for further research.

8.1 Summary

Generic models

We studied generic impulsive control models describing an augmentative biological control program. This genericity presents three major advantages: first, it lowers risks of modelling artefacts. Second, the results of our analysis apply to a wide class of models, consequently, for a given model with generic characteristics matching that of the generic model, specific functions can be simply plugged into the generic results in order to obtain specific conditions (Chapters 4–6). Third, it also provides us with a preliminary way to tackle uncertainty in 'data clouds' (Chapter 7). By focusing on some general trends in these data, we are already able to provide initial estimates for biological control programs.

Minimal predator release rate

Throughout our analyses, we defined a *minimal predator release rate*, which is the smallest number of predators released per unit time that can drive a pest population to zero. It is obtained from the stability analysis of the zero-pest solution of the model. Because it pertains to the predator release strategy, we say that this condition is managerial.

We can also identify how 'best' to spread the release rate by studying its dependence in the release period T and/or the partial harvest period T_h. In our thesis, 'best' is defined in terms of the lowest minimal release rate (which minimises the amount of money a planter invests in purchasing predators) and fastest pest eradication (which minimises pest damage on the crop and the planter's loss).

As is apparent in the works of e.g. Sharov and Liebhold (1998); Johnson et al. (2006) and discussed in the review by Mailleret and Lemesle (2009), this dependence in T (or the partial harvest period, T_h) also indicates that the releases and harvests must be modelled as impulsive processes rather than continuous ones.

Interfering predators penalising predation

After reviewing the effect of varying the components of a simple impulsive predator-prey model (given in Chapter 3), we investigated what happens when the functional response is penalised by predator interference.

We therefore have generalised Buffoni et al. (2005)'s formulation of the predator-dependent functional response through the use of sector conditions (Vidyasagar, 1993) – and analyse the effect of intrapredator interference within an impulsive framework. In this thesis, we presented the results only for the generalisation of the predator *density-dependent* class of models. We provided a small discussion on the *ratio-dependent* class of models which is covered fully in the article Nundloll et al. (2010a).

The analysis yields two conditions for pest eradication. First, the strength of the interference is required to be smaller than a ratio of the predation rate to the pest growth. Satisfying this first condition is critical to the existence of the minimal predator release rate (which is the second condition): when this condition is not satisfied, no release rate can eradicate the pest.

We found that releasing the predators as frequently as possible, lowers the minimal release rate. When a given release rate is higher than the minimal release rate, this accelerates (for small pest invasions) pest eradication; in fact when the given rate is lower, using higher frequency releases decelerates the speed at which the pest population settles at a positive value. When the pest invasion is lower (rather than higher) than this value, this implies that pest damage is reduced.

Our analysis of the zero-pest solution provides a generic version of that presented in Zhang and Chen (2006), specifying both local and global asymptotic stability conditions for the pest-free solution.

Peer-reviewed journal:
Nundloll, S., L. Mailleret, and F. Grognard: 2010, 'Influence of intrapredatory inter-

ferences on impulsive biological control efficiency.' *Bulletin of Mathematical Biology* **(accepted)**.

Cannibal predators enhancing mortality

In Chapter 5, we investigated the effect of cannibalism, which enhances the mortality of predators. We analysed three models of predator cannibalism.

We started by considering unlimited cannibalism (referred to as an elementary description of overcrowding by Goh (1976)). The minimal release rate was calculated in the limit that the release period is small enough ($T = 0$). We observed that the stability condition would not be satisfied beyond a large release period value. This pointed to more frequent smaller releases. The analysis extends the results of e.g. Rosenzweig (1972); Kuang et al. (2003); Deng et al. (2007) to the impulsive case, both local and global conditions being calculated.

We then considered the case when cannibalism was 'moderate' or limited by the predator population, arising from hunger. We calculated, using a sector approach, two thresholds for the minimal release rate - one above which pest eradication is guaranteed, and one below which pest eradication is not possible. This analysis covered a generic formulation of mortality, augmented with an impulsive component.

Finally, we analysed the case when cannibalism formed an integral part of the diet: we used sector conditions and considered a generic version of Kohlmeier and Ebenhoh (1995)'s model augmented by the impulsive component due to predator releases. This model accounted for a simplified description of intraguild predation. The analysis provided a first insight into the influence of intraguild predation in an augmentative biological control program. Results are similar to Chapter 4, with two conditions: one related to the competition strength and the other to the minimal release rate. Only local asymptotic stability and instability conditions were calculated.

The analyses of the last two forms of cannibalism extend the analysis of Kohlmeier and Ebenhoh (1995) to the impulsive case, specifically around the zero-pest solution. They have, however, not been published yet (we feel there is scope to improve them further). Results for the first (overcrowding) cannibalism model were published in the following:

Peer-reviewed journal:
Nundloll, S., L. Mailleret, and F. Grognard: 2010, 'Two models of interfering predators in impulsive biological control.' *Journal of Biological Dynamics* **4**(1), 102–114.

Impulsive partial removals

Finally, in Chapter 6, we looked at what happens when partial pest and predator populations removals take place during the partial harvesting of a crop or through other means within an Integrated Pest Management (IPM) program (these removals are impulsive processes).

We provided a complete analysis of the effects of partial harvesting or impulsive removals within an IPM program when either of the partial removal or predator release period is an integer multiple of the other. Both local and global conditions were

calculated. We found that the partial harvest period provided a threshold for the frequency of releases: for releases occurring more frequently than harvests, the minimal release rate was an increasing function of the frequency, while it was constant and smaller for less frequent releases. These results were published in two articles – the complete version in a peer-reviewed journal in applied mathematics; a shortened version is available in the IFAC 2008 proceedings (see below).

In this thesis, we also analysed in more detail the effect of the strengths of partial removals. Depending on the strength of the harvests, removals can either disrupt, have no effect on, or help towards pest control. In Chapter 7, we shifted the analysis to pesticide usage where resistance to pesticides would result in changes in the removal strengths.

Within an IPM context, our contribution generalises the results of Liu et al. (2005a,b) by being applicable to a wider class of models and allowing two distinct values for the harvest and release periods.

Peer-reviewed journal:
Nundloll, S., L. Mailleret, and F. Grognard: 2008, 'The effect of partial crop harvest on biological pest control.' *Rocky Mountain Journal of Mathematics* **38**, 1633-1662.

Conference proceedings:
Nundloll, S., L. Mailleret, and F. Grognard: 2008, 'Global stability in a general impulsive biological control model with harvest' *17th IFAC World Congress* Seoul, South Korea.

8.2 Discussion

Using feedback

In this thesis, the size and timing of releases are also fixed; the release rate (whether to use the local or global stability estimate) is based on a 'rough' estimate ('small' versus 'large') of pest presence. Predator releases are usually carried out as such to protect crops largely for logistic reasons: pest detection is usually a labour intensive process. The main problem with this approach (especially for large invasions) is that global asymptotic stability ensures convergence only in the limit that t tends to infinity. What happens during that time? The pest population converges to zero of course, but there is a risk it might do so too slowly (for e.g. during supertransients (Tang et al., 2005)) and already cause a lot of damage to the crop.

If unfixed sizes and timings of predator releases are deemed realistic, through adequate support for detection, a reactive approach can be adopted. In particular, pest control can be 'adapted' for the detected pest population to accelerate eradication. This detection can be achieved eg through a pest video surveillance (see e.g. Boissard et al. (2008)).

This also complicates things mathematically. The Floquet analysis technique as used in this thesis might not work as there is no release (or harvest) period that is set from the beginning. Tang et al. (2005), for instance, calculated that periodicity is maintained when only one impulse (one control action) is applied per period for a specific Lotka-Volterra types of models. What if using two or more actions (at other thresholds within the period) improves pest eradication further? What about predator-prey models with other forms of interactions?

Perhaps a solution would be to resort to a higher level of mathematical abstraction? One approach, that is currently dominating the hybrid control engineering literature, consists of using set theory (see e.g. Goebel et al. (2009) for a review).

Refining 'best'

As we said earlier, in this thesis, 'best' is defined in terms of the lowest minimal release rate (which minimises the amount of money a planter invests in purchasing predators) and fastest pest eradication (which minimises pest damage on the crop and the planter's loss). Of course, this is a highly simplistic definition. We can do better by incorporating other economic constraints into our model.

▼

There are a number of ways to improve on the definition, namely to include the cost of labour, other inputs such as pesticide usage, and pest damage: to do so, we could perhaps define a ***cost function*** *parameterised by the minimal release rate (to minimise), the cost of labour involved at predator releases (to minimise), as well as for partial harvesting, and finally – as an indicator for pest damage – the speed of pest eradication (to maximise). For pesticide applications, the cost of pesticides will have to be taken into account (to minimise).*

The formulation of a well-posed problem taking into account all these economic forces is not, however, trivial. A major difficulty here is that the control is impulsive (see e.g. the analysis by Xiao et al. (2006) on optimising harvests for the Maximum Sustainable Yield (MSY), and Mailleret and Grognard (2009) on minimising the risk of pest damage for examples of the difficulties). Perhaps, considering our model as a ***component of a larger, fuller description of the farming system*** *as above, we might use the numerically calculated multi-objective approach proposed by Cardoso et al. (2009), with other components of the farming program? Alternatively, for an analytical study, using the methodology presented by Chatterjee (1973) (who analyses the trade-offs between the damage to crops by pests and the damage to the environment by pesticides) might yield some insights on how to tackle this problem.*

More experimental validation

Ultimately, we also want to know how well our model fits within a more complete mathematical description of the system. However, our discussions and results have

no basis unless we test them in the field. Also, we can improve our model only if we can test it.

Unless we are able to obtain field data, some simple questions, if left unanswered, will undermine the progress in modelling biological control: were our premises correct and sufficient? Can we really ignore **spatial effects**? Plants for instance release chemicals when attacked by pests, that attract predators: can this be rightfully integrated within the functional response or would a spatial component provide a better prediction of the predator behaviours as they move along the 'chemical gradient' to the source of their food?

To which extent is it necessary to complexify our model? Do we need to consider **higher dimensions**? We analysed a simplified intraguild predation (IGP) system as given by Kohlmeier and Ebenhoh (1995), where the predator species were grouped together within one state value. The resultant minimal release rate provided the overall predator release rate, but not the specific mix of the species[1].

Data on agricultural predator-prey systems is hard to collect, which also explains their rarity in the literature: first, there is the problem of scale (see Section 3.3 in Chapter 3); second, invertebrate species involved have life-cycles that are of the order of week(s) – this is a relatively long life-cycle; These two problems are logistic and render the data collection a labour intensive process. The problem of scale also implies a high risk for the spread of the pest to other sites (it is difficult and very costly to monitor invertebrates without this risk). So the idea of reproducing a biological control scenario by artificially rearing pest species and releasing them on a test site is often met with objections. This makes for a third, equally significant, obstacle.

▼

A solution to the third obstacle would perhaps be to include farmers themselves in efforts for data collection. Naturally, this requires that an ***adequate data collection protocol*** *is set up by biologists, modellers, and farmers. This does not seem to be an easy task, but is probably the best way forward given the nature and diversity of the predator and prey species in agricultural systems.*

Let's think about it a little: chemostat experimenters have the advantage of controlled data collection, but it is also to their disadvantage that such experiments are technologically intensive! Our proposal is humanly or labour intensive but not impossible if technological means are lacking. The successes encountered by Van Lenteren (2007a) and Van Mele et al. (2007) lend support to our proposal. In fact, involving farmers could help in maintaining the critical mass of biological control professionals required for a large-scale implementation of such programs (see for instance the discussion in Van Driesche and Bellows Jr. (1996) which underlines that ad hoc *attempts at biological control solutions are rarely successful because they usually require sustained efforts of scientists with expertise in systematics, ecology and the rearing of natural enemies.").*

[1]This is not surprising: we had not in our model parameterised this mix. But this remains a valid question from a practitioner who now has a large choice of predators, many of which can be used at the same time.

A word of care though: the turnover (in terms of valid and refined data) of such large-scale experiments is not for the immediate future. The data collection protocol can only be improved through adequate feedback back and forth the farming to the biologist and modelling communities. This process takes time.

Shifting the focus: Conservation biological control?

In our introduction, we discussed the problem of pest resistance to pesticide usage, and how biological control circumvented this problem. Augmentative control, as modelled in this thesis has however a couple of drawbacks. First, though to a lesser extent than classical control, the introduction of an 'exotic' predator can pose a direct threat to 'indigenous' ones which are less competitive in exploiting their own niche than a migrant, and may even predate on it. A particularly successful predator may even be able to mate with a local variety and persist (an initially augmentative control program can thus become classical). The local species is wiped out in the process. In Europe, for instance, the invasive ladybird *Harmonia axyridis* outcompetes and preys on the eggs of the native *Coccinella septempunctata* Hironori and Katsuhiro (1997) and most other indigeneous coccinelidae; it tends to progressively spread to the whole continent Roy and Wajnberg (2008). There are plenty of other examples of such biological control programs gone wrong, where a predator becomes a new pest that needs to be controlled (see e.g. Cox (2004))[2].

Secondly, aside from the resistance issues, an augmentative control program does not really solve the other problems associated with pesticides, namely that of pest resurgence and secondary pest outbreaks (Metcalf, 1994). As a given pest is completely eliminated, its natural enemy populations which might have buffered previous invasions also dwindle; a resurgence of the same pest is likely to be more violent. Likewise, there often exist other species that are potentially harmful to a crop but are kept at bay by their position in their related food web: supposing that it shared a similar natural enemy guild as the pest, the absence of its enemies can result in an uncontrolled increase in its numbers to harmful levels.

▼

Lewis et al. (1997) frame this problem within the question: When is a pest a pest? They then argue that an ecosystem is self-regulatory so that instead of focusing on pest control techniques that are 'therapeutic', we must improve our understanding of the feedback mechanisms within an ecosystem that maintain its balance. We must favour first conservation control methods, then use augmentative control to drive back an ecosystem to its equilibrium state only when necessary.

Mathematically, this might indicate looking at higher dimensional systems. Would a greater overlap between niche theory (see e.g. Cohen (1978)) and dynamical systems hold the key to solving this problem?

[2] Sfar and Trondheim (2001) exploit this catastrophic feature for comical purposes in an unusual surgical operation (see Figure 8.2).

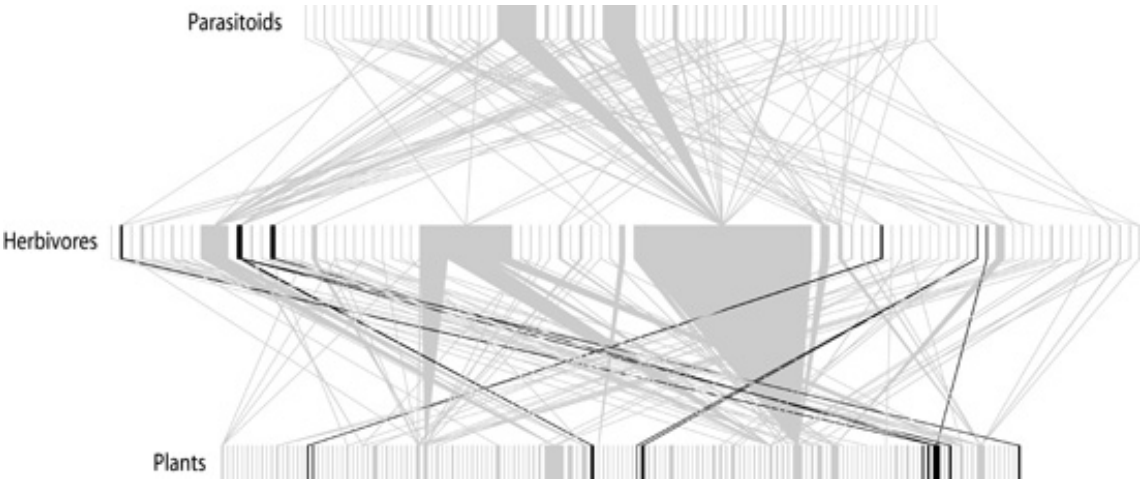

Figure 8.1: From Macfadyen et al. (2009), this web represents a whole-farm network. The bottom level shows all the plant species sampled on the farm, the middle level the insect herbivores, and the top level the parasitoid wasps. Each bar indicates a different species and the width of the bars represents their abundance on the farm. The lines between trophic levels indicate that these two species are interacting and the width of the lines indicates the frequency of this interaction. Arable plant species and their herbivores have been shaded black. These plants make up only a small proportion of the vegetation on each farm as they are short-lived in comparison to natural or semi-natural vegetation, being harvested as soon as they reach maturity, and also ephemeral, in comparison to, for example, field margins and rough ground.

Also how realistic is this in practice? Intensive agriculture, as it is practised nowadays, has changed substantially ecological landscapes: in particular, it has greatly diminished species diversity and probably the resilience of ecological webs (see Figure 8.1 on the difference in species diversity between a converntional and an organic farming system). Moreover, the world human population has grown to five times its size within the past century; can we go back to a lower impact agricultural system and sustain the population? Perhaps, in which case it is likely that transportation routes of food will have to be reorganised, mentalities will have to change, long-term policies and perseverence will be required. Can we rise to that challenge?

Comic removed
for copyright reasons

Figure 8.2: In Sfar and Trondheim (2001)'s 'Donjon : Coeur de Canard', the biological control operation on the dungeon keeper to remove a mysterious creature (known as a'berbouche') from his stomach goes horribly wrong. The minuscule men, predators of this 'berbouche', stay in the stomach after the operation and need to be treated by men-eating ants. The ants proliferate so the surgeon requires a giant anteater to remove them. For the giant anteater to reach the ants, the surgeon needs to flip the dungeon keeper inside out!... How will this chain of events stop?!

Appendix

A

Why reduce pesticide usage

"The dose makes the poison."[1]
– Paracelsus (1495–1541)

"Biocides are, by definition, designed to kill living organisms, however, and the presumption must be of some damage where they are detected."[2]
– Brian Moss, 2008

A little bit of history

The large-scale use of chemical pesticides as we know it today dates back to the post-World War period. In the 1950s, an intensive agriculture program - referred to as the Green Revolution - was developed to deal with a growing world population and mass famine threats in the developing countriesThis program was based on three inputs: first, more resistant cultivars or strains for various crops - especially cereals such as wheat. Second, fertilisers to enrich the soil with nutrients to stimulate plant growth. Third, chemical pesticides to fight pest invasions. It was largely accredited to Norman Borlaug (1914–2009), whose work on wheat cultivars and intensive plantation farming made Mexico self-sufficient in food. The success of the program led it to be applied in India, Pakistan and other developing countries which were undergoing very sharp population increases. Norman Borlaug was awarded the Nobel Peace Prize in 1970 for his efforts to fight world famine(The Economist, 2009). It is in fact the expertise developed in chemical processes (namely the Haber process) dur-

[1] *"Alle Ding sind Gift, und nichts ohn Gift; allein die Dosis macht, das ein Ding kein Gift ist."* Which translates literally as: *"All things are poison and nothing is without poison, only the dose permits something not to be poisonous."*

[2] Quoted from Moss (2008).

ing the World Wars which led to the industrial synthesis of fertilisers and pesticides (Szöllösi-Janze, 2001).

Harmless doses or time bombs?

Public perception of pesticide risks has been and still is largely swayed by the press. As I understand it, the media coverage on health and environmental issues related to pesticides gathered momentum around works such as Rachel Carson's 'Silent Spring' (Carson, 1962) and Van den Bosch's 'The Pesticide Conspiracy' (Van den Bosch, 1978).

To date, the situation in Europe and the US can be summarised as follows:

Contamination of the air, soil and water has been reported widely and in detail (see recent export reports such as Berenbaum et al. (2000); Voltz et al. (2005); RCEP (2005); Moss (2008)). However, all these reports acknowledge that, apart from a few organisms, the actual pesticide effects on biodiversity are too fragmentary to be conclusive.

Likewise, pesticides are currently developed to have shorter half-lives and more specific to organisms that they target to reduce undesired side-effects on natural enemies. Regulations on the availability of pesticides have also been tightened: new pesticides undergo a vigorous screening process by the European Commission before being put up on the market for sale. As a consequence, policy makers have responded by saying that current regulations sufficed for the control of pesticide usage (see, for instance, DEFRA (2006) which supports the use of pesticides in agriculture as being 'harmless'.).

The few attempts to measure the effects of pesticides on exposed agricultural workers and farmers have pointed to a higher incidence of some cancers specifically to these occupations (see Blair et al. (1992); Acquavella et al. (1998) in Baldi and Lebailly (2007)). Indirect effects on consumers due to persistence of pesticides in the environment seem however to be unknown.

That is, any threat that pesticides *potentially* represent is negligible under the current situation. They merely represent a form of environmental and health risk, which are kept at bay (satisfactorily, according to institutions).

In the developing countries however - where food security is an issue - the absence of legislation and lack of information on pesticide resistance often leads to their misuse. (Dhar et al. (2004), for instance, reports of leaching; Mathur et al. (2005) reports of an above average percentage of pesticides in the blood samples of a villagers' cohort in Punjab, the cotton belt of India). Furthermore, pesticides with shorter half-lives and or narrower spectrum of action are not used because they tend to be more costly. Toxicity is then not a mere risk but a real threat to the environment and human health.

With respect to the question of dosing pesticides appropriately, one may still quote Moss (2008):

> *"In an ideal world, it would be possible to take an individual agricultural activity, the spraying of a particular pesticide at a known dose rate, [...] for example, and measure precisely the effects of these on, respectively, the fecundity of a particular fish species,*

the growth of aquatic plants in a receiving lake or the extent of silting of a river stretch. The effects can be measured in a general way and modelled with varying degrees of uncertainty [...], but the sort of precision demanded by legislators and lobbies will never be attainable and this has been a major weapon used to delay regulation of agricultural activities.

[...] Although ecotoxicology is a precise science in simple systems in the laboratory, its accuracy in determining ecological consequences is highly questionable. A particular substance may be shown in laboratory systems to have a particular threshold at which a test organism survives, or at which there is no observable effect. These thresholds are determined on a very restricted array of test species that are extremely tough, otherwise they would not be flexible enough to be used in laboratory systems. Nor are such organisms exposed to the risks of competition, predation and environmental fluctuation that they, and far more sensitive species, will be exposed to in nature and which may reduce thresholds enormously. Nor are they exposed to the much more complex chemical environment of nature where an array of potentially damaging substances may be simultaneously present."

In other words, we might simply be sitting on an environmental and health time bomb.

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