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ORIGINAL PAPER

The importance of primary inoculum and area-wide disease management to crop health and food security

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Abstract In some epidemics that have devastating consequences, the primary inoculum plays an important role in both epidemic onset and intensification. This article documents the dynamics of such epidemics, and illustrates their importance using two examples: Huanglongbing of citrus and begomoviruses of tomato. The latter disease is a major constraint to tomato production in Brazil, while the former has become a threat to global citrus production and farmers' livelihoods. In spite of their importance little is known of the characteristics of these diseases and their management. This is because classical botanical epidemiology considers two types of diseases: polycyclic diseases, where the inoculum that causes infections is produced during the epidemic in or on individual plants that had been previously infected in the course of that epidemic; or monocyclic diseases, where inoculum that causes infection is not produced in or on individual plants that had been infected in the course of the epidemic, but in the soil, on secondary hosts, or in infected crop plants of the same host in other fields. Diseases of the first type typically present a logistic disease progress curve and management is based on reducing the rate of infection, whereas diseases of the second type present a monomolecular disease progress curve and management is based on reducing the initial inoculum. This article deals with plant diseases that depart in their structure and behaviour from these two archetypes, because they borrow elements from both. We address polycyclic diseases in which the primary inoculum has a continuous and dynamic role, and in which the secondary inoculum contributes to epidemic build-up, i.e., polycyclic diseases with continuous primary spread. This epidemiological structure generates less clear-cut disease progress curves, but usually follows a monomolecular dynamic. Our focus on this type of disease is multifold because (1) this more complex, combined, pattern is actually quite common, often leading to grave plant diseases

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epidemics, with impacts at the farm, community, and country scales, and (2) such epidemics are among the most difficult to manage. Our analysis leads us to assess past errors and current courses of action. It allows us to recognize, in addition to the conventional tools for management with local effects, the critical importance of collective action. Collective management action – at the farm, community, or national scales – is congruent with the characteristics of many epidemics, because they also entail properties at successive and nested scales. The management of such epidemics needs to address both the primary and secondary inoculum. More importantly, these actions have to be performed in an area-wide, regional basis in order to be effective.

Keywords Primary and secondary inoculum · Begomovirus · Huanglongbing · Botanical epidemiology · Disease simulation · Citrus · Tomato · Landscape · Polycyclic diseases · Monocyclic diseases · Plant disease epidemiology · Integrated disease management

Introduction

Plant diseases are primarily studied because of their impacts on societies, communities, and individuals (Zadoks and Schein 1979; Strange and Scott 2005). These impacts may affect the components of food security (FAO 2006): losses to disease impact not only food provisioning (food availability), but also food access (reduced incomes of producers, reduced employment on the farm and in the processing industry), food utilization (reduced produce quality, or toxins), and stability (negative effects on the economy and infrastructure). Conversely, disease management mobilizes resources at the same levels of social hierarchy: societies (through, e.g., policies), communities (through many kinds of collective action), and individuals (disease management at the farm or field scales). Yet, while the literature is replete with reports pertaining to individual actions (such as the application of a pesticide), comparatively very little research provides grounds for collective action at various scales. As a result, ironically, while farmers take individual, often isolated, action to manage plant diseases, entire communities are exposed to the possible impacts of plant diseases on several components of food security. The hypothesis then arises of the possibility of greatly improving disease management, and thus enhancing food security, when disease is managed at the collective rather than at the individual scale.

The purpose of botanical epidemiology is to address plant disease epidemics as ecological phenomena, and to derive knowledge that can be applied for disease management (Van der Plank 1963; Zadoks and Schein 1979; Bergamin Filho and Amorim 1996). The ultimate goal of plant disease epidemiology is therefore to solve disease problems.

This article especially addresses a class of plant disease epidemics that has received comparatively little attention, despite being quite common, and sometimes devastating. We also provide perspectives showing why this class of epidemics is hard to manage, but that solutions may be found. These solutions involve management at the landscape and farmers' community scales: these scales differ from the scales commonly considered i.e., the field and the individual farmer's scales. At times when food security is such a pressing global issue, it is important to underline the role that farmers' communities may play at the landscape scale.

Classical plant disease epidemiology considers two broad types of epidemiological patterns. The first involves recurrent overlapping disease cycles underpinning an epidemic; the second involves a source of infection that drives the course of an epidemic. These patterns have shaped both the theory and applications of botanical epidemiology to plant disease management. However, intermediate, more complex, patterns exist, where several cycles and continuous inflow of inoculum from a source occur simultaneously. Although it has not been investigated to the same degree of detail, this intermediate, dual, epidemiological pattern actually applies to numerous plant disease epidemics. Because of the dual source of inoculum – inoculum being both mobilized from a source, and also building up as the epidemic progresses – diseases associated with this intermediate pattern can be very hard to control.

The difficulty of achieving disease control in epidemics of this intermediate pattern lies first in the difficulty of determining the respective importance of the two sources of inoculum on the course of epidemics. Simulation modelling is a powerful approach to quantitatively assessing the importance of drivers in the behaviour of a system, as here, different sources of inoculum defining the dynamics of an epidemic. In this article, we show that different outcomes are to be expected depending on the importance of these sources, which have non-linear effects on the system's behaviour.

Because many technological advances have been made in the 20th century to identify, synthesize, and apply pesticides, a large fraction of the management options have leaned towards the chemical control of inoculum produced in the course of these epidemics. Such a choice conforms to the tools, equipment, and mind-sets that prevail in many agricultural contexts; this choice matches short-term economic interests; and it fits within one scale of management, the individual farmer's field. It has, however, led to environmental and human health externalities, and to failures in managing disease. We describe here situations where such a strategy cannot provide efficient disease control, that is, when incoming inoculum is an important determinant in epidemic build-up.

This article uses two plant diseases as examples: Citrus huanglongbing (HLB) and tomato begomovirus diseases. HLB has become a threat to citrus production worldwide, perhaps marking "the end of orange juice" (Kuchment

2013), with impacts on individual farms, farmers' communities, local and national economies, and global trade, therefore impacting food security. HLB, also known as citrus greening or citrus yellow shoot disease, has been responsible for the destruction of almost one hundred million citrus trees in the 20th century. As a result, the citrus industry has declined in many countries of southeast Asia, regions bordering the Indian Ocean, Arabian Peninsula, and south and east Africa (da Graça 1991; Aubert 1992). HLB is the cause of massive crop losses and fruit quality reduction as the disease symptoms progress throughout the canopy of affected citrus trees (Bassanezi et al. 2011). At the beginning of the 21st century, HLB became the most serious citrus disease and the single main threat to the future of the world citrus industry after its report in the two largest sweet orange and juice producers worldwide, in 2004 in São Paulo, Brazil (Coletta-Filho et al. 2004; Teixeira et al. 2005) and in Florida, USA (Halbert 2005). One decade after its first detection in the Western Hemisphere, HLB is already present in almost all major citrus regions in the Americas, challenging the sustainability of the citrus industry (Bové 2006; da Graça and Korsten 2004; Gottwald et al. 2007; Gottwald 2010). In Florida, where it is estimated that more than 60 % of trees are already infected, the disease has been associated with the progressive citrus yield decrease in 2011–2014 (from 146.7 million boxes in 2011/2012, to 133.6 million in 2012/2013, and to 104.4 million in 2013/2014, the lowest yield in the last 30 years). In São Paulo, more than 34 million citrus trees were eliminated from 2005 to 2013, while about 18 % of the standing trees in 2015 are infected. HLB has not yet been reported from the Mediterranean Basin and Australian citrus producing areas (Bové 2014).

In 1997, Polston and Anderson (1997) wrote: "Since the late 1980s, most of the tomato-producing areas of Florida, the Caribbean, Mexico, Central America, Venezuela, and Brazil have suffered from high incidences of whitefly-borne geminiviruses, with devastating economic consequences for their respective tomato industries". In their review, Polston and Anderson (1997) indicated crop (quality and quantity) losses ranging from 30 to over 50 % throughout the New World. Begomoviruses (whitefly-transmitted geminiviruses), became by the end of the 20th century a major constraint to the most common vegetable crop. Nowadays, the most frequently observed virus disease in tomato crops in Brazil is caused by begomoviruses (Albuquerque et al. 2012). The first begomovirus diseases in tomatoes were reported in the 60s (Flores et al. 1960). They were associated with *Bemisia tabaci* biotype A. However, the begomoviruses became important after the introduction of whitefly biotype B (MEAM-1) in the country. Biotype B is more polyphagous and aggressive (Lourenção and Nagai 1994). Since its introduction, which is assumed to have occurred in the early 90s, the incidence of the disease has progressively increased in Brazil, and today *Tomato severe rugose virus* is the virus disease with the highest occurrence in tomatoes (Faria et al. 2000; Inoue-Nagata 2013). Fields with 100 % incidence are commonly seen in the central part of the country.

Both diseases addressed in this article represent major threats to the production of key agricultural commodities at the eco-regional or global scales. Worldwide, tomato is the most important vegetable crop, with a production of 160 million tons, and citrus represents an important fruit source, with a production of 120 million tons. Both products are associated with (1) sources of food in terms of vitamins, minerals, and fibre, and (2) sources of income for very large numbers of labourers involved in production, harvest, and processing. As a result, two components of food security are rapidly affected: food availability (through production) and economic access to food (FAO, IFAD and WFP 2013). Diseases reducing the production of such crops may therefore not only reduce the availability of these commodities, but also threaten livelihoods of people depending on these crops for their income. Such consequences have been observed in coffee, another important agricultural commodity in Southern countries. The recent coffee rust epidemics in Latin America contributed to food insecurity in Honduras and Guatemala (Avelino et al. 2015; McCook and Vandermeer 2015).

We show that management of complex, dual, epidemiological dynamics, and avoidance of major losses to communities, national economies, and even world trade, may be based on the concerted development of strategies which may involve conventional management tools, but first and foremost, depend on shared understanding and collective action. The implementation of such strategies could contribute much in reducing food insecurity incurred by these types of diseases. We further show that taking into account the space-dependent properties (plant, field, farm, and landscape) of these diseases enables their management. New epidemiological understanding at these nested scales matches the scales of social and economic impact and action for disease management.

Some epidemiological concepts

Plant disease epidemics are traditionally classified into two broad groups, depending on the source of inoculum that encounters the host over the course of disease development. In the first group, inoculum that causes infections is produced during the epidemic by individuals that had been previously infected in the course of that epidemic. Epidemics of this group are polycyclic in structure, and diseases that cause them are called **polycyclic diseases** or compound interest diseases (Van der Plank 1963; Madden et al. 2007). In the second group, inoculum that causes infections is not produced by individuals that had been infected in the course of the epidemic in the considered cropping season, but by inoculum generated by other sources: in the soil, on secondary hosts, or in

infected crop plants of the same host in other field. Epidemics of this group are monocyclic in structure, and diseases that cause them are called **monocyclic diseases** or simple interest diseases (Van der Plank 1963; Madden et al. 2007; Savary 2007).

For both groups, the inoculum that initiates the epidemic is called **primary inoculum** and the infection caused by it is called **primary infection** (Butt and Royle 1980). This process is called **primary spread**. On the other hand, **secondary inoculum** and **secondary infection** occur only in polycyclic diseases. Secondary inoculum results from primary or secondary infections taking place during the current epidemic. Secondary infections originate from secondary inoculum. This process is called **secondary spread**. As noted by Madden et al. (2007), the primary infection process that initiates a polycyclic epidemic is analogous to the process that occurs throughout a monocyclic epidemic, and thus one can think of monocyclic epidemics as consisting of only primary infections.

Plant pathology textbooks generally consider that the epidemiological role of primary spread is to introduce the pathogen in areas where it is absent; the subsequent development of the epidemic is governed by the secondary spread (Gäumann 1950; Agrios 2005; Bergamin Filho et al. 1995; Amorim et al. 2011). The same view is predominant in modelling plant disease epidemics, in which most epidemics start by introducing few lesions or infected individuals (primary inoculum) instantaneously at time $t=0$ (Van der Plank 1963, 1965; Zadoks 1971; Zadoks and Schein 1979; Bergamin Filho and Amorim 1996; Madden et al. 2007). The reasoning is that, with sufficient time, secondary infections overshadow initial events. In this view, primary spread is relegated to a subservient role of the carryover of inoculum from the previous crop to initiate the first infections and is assumed to be of negligible importance relative to the dominating influence of secondary, plant to plant, or host unit to host unit, spread (Gilligan 1994).

As discussed by Madden et al. (2007), however, "it may not be realistic in some cases to assume an instantaneous start of the epidemic. It is possible, for instance, that the primary infections occur over an extended period of time, possibly concurrently with the new (secondary) infections occurring due to spread from individual to individual." A theoretical approach for epidemics in which primary spread occurs over an extended period of time has been proposed by Brassett and Gilligan (1988), Gilligan and Kleczkowski (1997), Gilligan (2002), and Madden et al. (2007). Disease progress curves in these cases are less clear-cut compared to the results obtained for strictly polycyclic or monocyclic epidemics, but usually follow monomolecular dynamics (Gilligan 2002; Madden et al. 2007; Savary 2014).

In this paper we address this third kind of disease, i.e., **polycyclic diseases with continuous primary spread**, using as examples the huanglongbing disease of citrus and begomovirus diseases in tomato. We will show that management of this group of diseases presents very specific aspects that usually have not been recognized in the plant pathology and botanical epidemiology literature.

Simulation models for dual patterns: polycyclic diseases with continuous primary spread

Epidemiological modelling is a very large and diverse field of investigation. In this section, we present an example of one of the many possible approaches, which we cannot review here. The interested reader will find entry points to this field in several sources (e.g., Bergamin Filho and Amorim 1996; Madden et al. 2007; Savary and Willocquet 2014).

A first approach is to consider that the rate of disease increase is dependent only on primary inoculum and on the amount of healthy tissues that are available at any given time. This corresponds to a monomolecular increase of disease, and to the flowchart of Fig. 1a. Under this hypothesis, the rate of (primary) disease increase RPI is proportional to a relative rate of disease increase RRPI, and the running fraction of healthy individuals (with dimension: [Nhost]) per unit time (with dimension: [T]):

$$\mathrm{RPI} = \mathrm{RRPI}*\mathrm{P}*(1-(\mathrm{Ndis}/(\mathrm{Ndis}+\mathrm{Nhealthy}))) \quad (1)$$

Note that this equation concerns the rate of increase of diseased individuals, i.e., diseased trees, plants, or individual sites in one plant. The dimension of RPI is that of speed: $[\mathrm{Nhost}.\mathrm{T}^{-1}]$. A monomolecular increase of disease (Eq. 1) assumes that the primary inoculum P is the "engine" of disease growth. Running a simulation model with Eq. 1 yields a typical inverted "J curve" described in Van der Plank (1963), i.e., a monotonal increasing disease increase, with a monotonal decreasing slope over time.

Another approach (Fig. 1b) is to consider the rate of disease increase as a function of the amount of disease that is already present (Ndis [Nhost]), and of the fraction of still healthy tissue:

$$\mathrm{RSI} = \mathrm{RRSI}*\mathrm{Ndis}*(1-(\mathrm{Ndis}/(\mathrm{Ndis}+\mathrm{Nhealthy}))) \quad (2)$$

In this case, a logistic increase of disease is hypothesized, where the "engine" of disease progress is not a fixed initial value (P) as in Eq. 1, but a variable, dynamic amount of (increasing) disease, Ndis. Running a simulation model with Eq. 2 yields a typical "S curve" described in Van der Plank (1963), i.e., a monotonal increasing disease increase, with an initially increasing slope, an inflexion point, and a decreasing slope as time elapses.

In many cases, however, one may assume that both growth engine terms – the primary inoculum, P, and the running level of disease, Ndis – may have roles to play. One thus considers Eq. 1 and Eq. 2 simultaneously, considering two rates of

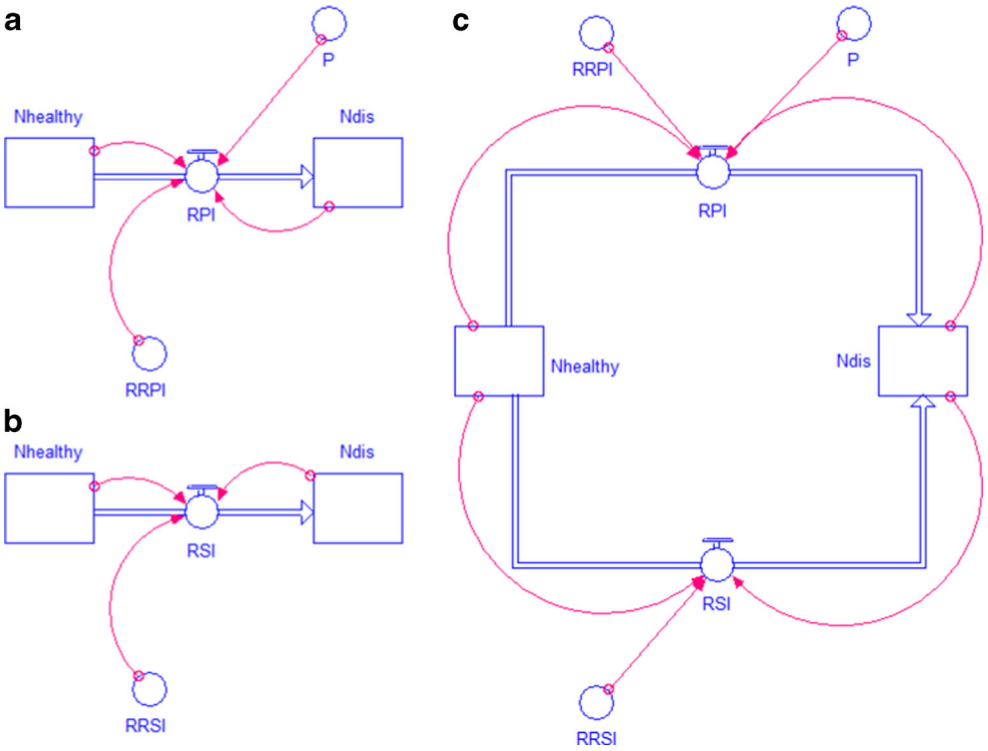

Fig. 1 Flowcharts of epidemiological simulation models. **a** Monomolecular (monocyclic) epidemic: the rate of disease increase (RPI) is proportional to the relative rate of primary infection (RRPI) and to the amount of primary inoculum (P; see equation 1); **b** Logistic (polycyclic) epidemic: the rate of disease increase (RSI) is proportional to the relative rate of secondary infection (RRSI) and to the running amount of disease (Ndis; see equation 2); **c** Combined model with primary and secondary infections occurring simultaneously. Nhealthy refers to the amount of healthy tissue available for infection

infection: a rate of primary infection (RPI, Eq. 1) associated with primary inoculum, and a rate of secondary infection (RSI, Eq. 2) which depends on the current amount of disease.

It may for instance be that one assumes that the primary inoculum, P, plays a particularly important role while the epidemic is still in its early phase, to be progressively relayed by an increasing role of the building-up disease amount. Berger (1981) noted that the logistic disease increase discussed by Van der Plank (1963) very often failed to represent the observed very rapid initial disease increase. Berger thus introduced the Gompertz model in botanical epidemiology, which is one of several ways to imply that initial disease progress owes much to the initial epidemiological conditions. Brassett and Gilligan (1988) later-on framed the hypothesis of a dual role of the primary and the secondary inoculum in a simple model structure, which can be represented as the flowchart of Fig. 1c. While such a modelling structure has been used to explore the behaviour of soil-borne disease epidemics, it has also been used to analyse rice sheath blight, which is caused by a soil-borne pathogen (*Rhizoctonia solani*), but in which secondary spread is canopy-borne (Savary et al. 1997). This structure can in general be used to explore the epidemiological behaviour of any disease that involves a strong contribution of the primary inoculum in the early stages of epidemics.

The structure of Fig. 1c corresponds to a very flexible model: when both RRPI and RRSI are similar (Fig. 2, curve a), disease progress is initially very rapid, with however an inflection point indicating the relay between primary (RRPI) and secondary (RRSI) inoculum in disease increase; with decreasing values of RRPI relative to RRSI (Fig. 2, curves b and

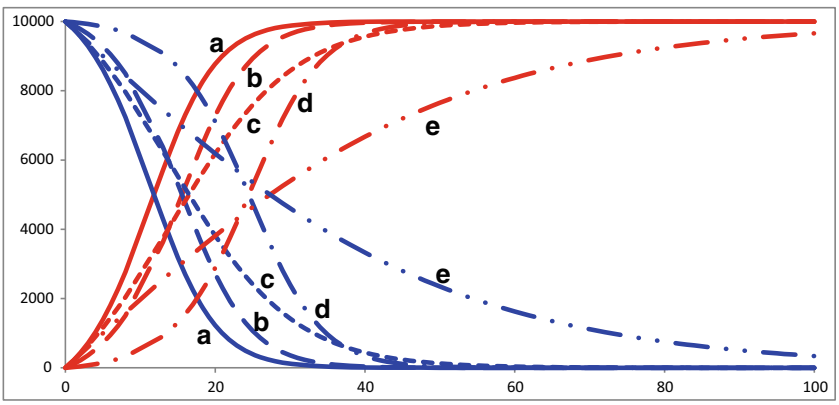

Fig. 2 Numerical simulation of a mixed model (Fig 1C) with a dual infection process involving the primary inoculum and the accumulated amount of disease. Blue: healthy individuals; Red: diseased individuals. **a** RRPI = 0.2 and RRSI = 0.2; **b** RRPI = 0.1 and RRSI = 0.2; **c** RRPI = 0.2 and RRSI = 0.1; **d** RRPI = 0.02 and RRSI = 0.2; **e** RRPI = 0.2 and RRSI = 0.02. Ordinates: Number of individuals; abcissa: Time

d), initial disease increase is reduced, inflection is more apparent, and the disease progress curve is more sigmoid; when RRPI is made increasingly larger compared to RRSI (Fig. 2, curves c and e), the disease progress curve increasingly appears monomolecular.

Citrus Huanglongbing: epidemiology and current management

HLB is mainly caused by three phloem restricted α-proteobacteria species: *Candidatus* Liberibacter asiaticus (CLas), *Ca*. Liberibacter africanus (CLaf), and *Ca*. Liberibacter americanus (Clam) (Bové 2006). The Asian Liberibacter species is responsible for the vast majority of HLB-infected trees worldwide, being found in all HLB-affected countries except in southern Africa. The African and the American species are so far restricted to Africa and Brazil, respectively (Bové 2006, 2014). The global spread of HLB is associated with the movement of infected citrus propagation material, and to the regional and local spread of two species of psyllid (Hemiptera: Liviidae) which are vectors of the pathogen. The Asian citrus psyllid (ACP) *Diaphorina citri* Kuwayama is the vector of CLas and CLam (Capoor et al. 1967; Yamamoto et al. 2006), and *Trioza erytrea* Del Guercio is the vector of CLaf (McClean and Oberholzer 1965).

HLB symptoms are observed on different tree organs, including: blotchy mottle and yellow veins on expanded leaves, yellow shoots on young flushes (Fig. 3a-d), and lopsided fruits (Fig. 3e) remaining green at the stylar end while the peduncular end colours first as the fruit, which contains brownish seeds, ripens (Bové 2006). Yield is reduced by premature drop of affected fruits and by gradual tree decline and death. Juice from lopsided fruits is of low quality, similar to juice from immature fruit, while fruits from asymptomatic branches of affected trees produce juice with similar quality to fruits from healthy trees (Bassanezi et al. 2009; Dagulo et al. 2010).

So far, no resistant or tolerant commercial citrus varieties are available, nor are there feasible effective and curative, economical methods to treat HLB-affected trees. Therefore the only possible management of this disease is based on the prevention of tree infection. Based on the experience of Asia's and South Africa's citrus growers and agricultural agencies, disease management can be achieved by (i) planting healthy citrus plants produced under insect-proof nurseries, (ii) eliminating pathogen inoculum by frequent removal of HLB-infected trees, and (iii) keeping populations as low as possible by chemical or biological insecticides (Aubert 1990; da Graça 1991; Bové 2006; Gottwald et al. 2007; Belasque et al. 2010a, b).

Among the recommended measures, the systematic elimination of symptomatic trees is the most debated and difficult to be accomplished by citrus growers, even in Brazil where it has been compulsory by law since 2005 (Belasque et al. 2010b). Growers argue that there is a direct and immediate loss when a symptomatic but productive tree is eliminated, especially in the case of adult trees with initial symptoms in a single or few branches that could remain relatively productive for a few more years. Moreover, due to the long incubation and latent periods of HLB, results of roguing would be observed only in the long term, and therefore do not provide an immediate return to growers (Bassanezi et al. 2013a). Chemical applications against the HLB psyllid vector are common because of the routine pesticide use against other citrus pests and pathogens (Gottwald et al. 2007). However, the additional insecticide applications are costly, and may have an impact on the environment. Despite the implementation of recommended measures by the growers in their own farm (local management), disease progress continues

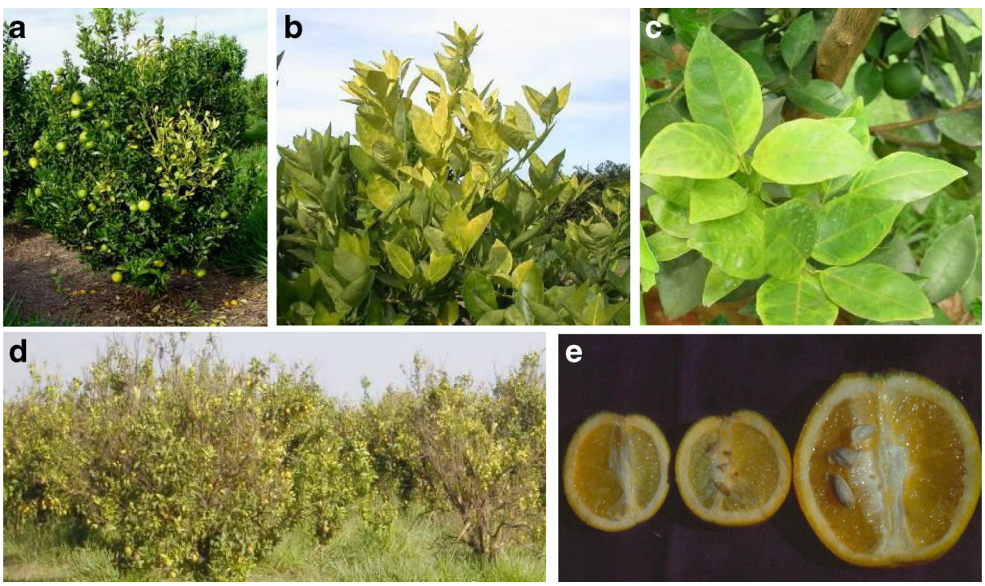

Fig. 3 Huanglongbing symptoms: yellow-shoot in young sweet orange (**a**, **b**); blotchy mottle in young leaves (**c**); defoliation in infected trees (**d**); asymetrical "lopsided" sweet orange fruits compared to a healthy one (**e**)

unabated, therefore reinforcing HLB management with more frequent roguing and more frequent insecticide spraying in the farm.

The ineffectiveness of conventional, yet recommended, practices to control HLB when disease inoculum sources and ACP populations are controlled only at local and small scales has been demonstrated in two production regions of Vietnam and Brazil (Gatineau et al. 2010; Bassanezi et al. 2013a, b). Although the frequent insecticide applications resulted in efficient reduction of psyllid populations in both cases (>80 %), the local vector control program had a limited effect on the disease progress rate and did not decrease the final disease incidence in the same proportion as the vector population. This occurs because immigrant psyllids that are infectious can, in many cases, transmit the bacteria to trees before being killed by insecticides. In this situation the primary infection is not efficiently avoided.

Adults of *D. citri* are constantly moving among citrus groves and other alternative hosts over short and long distances, searching for new shoots to feed on (Boina et al. 2009; Gottwald 2010; Gottwald et al. 2007, 2010). Therefore, even citrus plantings with intensive ACP control programs are constantly re-infested and subject to continuous primary infections, especially in the borders of the groves (Fig. 4). Thus, the higher the psyllid density in surrounding plantings, the higher the migratory flux of adult psyllids to the healthy plantings under good management, and the higher the chances for new infections to occur, especially when psyllids are bacterialiferous as in areas without HLB-infected trees removal.

A case study on HLB management practices adopted in São Paulo State by Brazilian citrus growers pointed out that good results on HLB control can be achieved if (i) inoculum removal and insect vector control are initiated at low HLB-incidence, (ii) the trees are adults (>5 years-old), (iii) the farm is large (several hundred hectares), (iv) HLB-management is practiced on all citrus farms within 4 km, and (v) the farm is in a region with an overall low HLB-incidence (Belasque et al. 2010a). The last four essential characteristics for the success of HLB control are related to the regional management of this disease, i.e., an area-wide management.

The effectiveness of area-wide reduction of inoculum sources and ACP population in reducing disease epidemics was experimentally demonstrated by Bassanezi et al. (2013b). This study compared the size of the vector populations and disease progress in two new planting areas, one close to groves without HLB management (i.e., with only local disease management) and the other surrounded by groves with psyllid control and inoculum removal (i.e., with area-wide disease management). In both cases the secondary spread of HLB (i.e., disease spread from diseased to healthy trees within a given plantation) was minimized by HLB symptomatic trees removal and frequent ACP control inside the experimental area. However, the epidemic began later and was slower to develop in the planting with area-wide disease management; the incidence of HLB 4 years after planting was 91 % lower compared to the planting with only local disease management (Fig. 5). The difference between the two newly established groves is attributable to the fact that the area-wide HLB management with frequent application of insecticides and regular roguing on the groves surrounding the experimental area reduced the surrounding ACP population responsible for the

Fig. 4 Orange field showing a border effect due to the influx of bacterialiferous psyllids from external sources

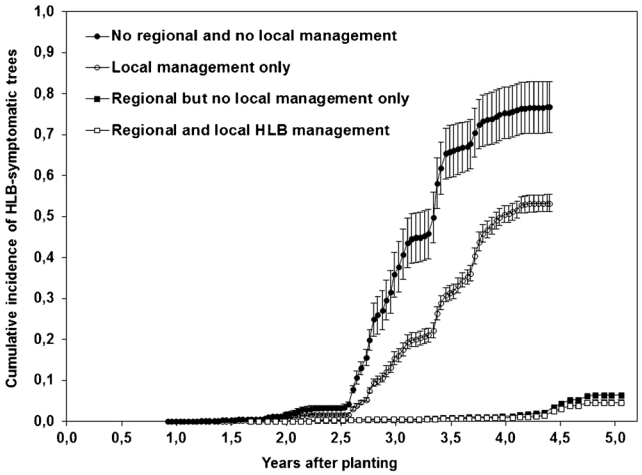

Fig. 5 Cumulative incidence of HLB-symptomatic trees in young sweet orange groves in different situations of area-wide and local HLB management. Regional HLB management (*black squares*) consisted of an annual program of 3 to 4 inspections and removal of symptomatic trees, and 4 to 14 foliar insecticide applications in a 2-km radius from the experimental grove. Local HLB management (*white circles*) consisted of an annual program of fortnightly to monthly inspections and a minimum of 4 removals of symptomatic trees, 18 foliar insecticide sprays from April to November at 2 weeks interval and two drench applications from November/December to February at 56-day intervals in the experimental grove

experimental area re-infestation at every flush-growth cycle (primary spread). By contrast, in the experimental area with only local disease management, the population of immigrant psyllids was not affected.

Box 1 summarizes the main features of HLB epidemiology:

Box 1: Main epidemiological features of HLB

- HLB spread occurs by two different ways: from outside (primary spread) and from inside (secondary spread) grove sources by bacterialiferous psyllids;
- in unmanaged groves, primary spread and secondary spread occur, although not necessarily simultaneously; thus, an epidemic results from the combination of monocyclic (primary) and polycyclic (secondary) processes (Gottwald et al. 2010);
- in well managed groves (with eradication of symptomatic trees and chemical vector control), secondary spread is not relevant; the epidemic results from a monocyclic (or primary) process;
- in managed and unmanaged groves, citrus trees are continuously subject to infestation by immigrating bacterialiferous adults of *D. citri*: dispersal may occur anytime in the year (Hall et al. 2013); primary inoculum usually does not decay as is the case with soil-borne pathogens; the main epidemiological role of primary spread is not related to introducing the pathogen in a field, but to sustain the rate of epidemic progress;
- even in well managed groves, primary spread from unmanaged groves is enough to cause infection in almost 100 % of trees in 2 to 5 years (Belasque et al. 2010a; Gatineau et al. 2010; Bassanezi et al. 2013b; Hall et al. 2013);
- *D. citri* moves bi-directionally between managed and unmanaged groves with a greater number of adult insects moving from unmanaged into managed groves than from managed into unmanaged groves (Boina et al. 2009); in most cases, the immigration of bacterialiferous vectors (in excess of those required for disease saturation) makes disease incidence almost insensitive to the mortality of vectors within the managed citrus groves (Belasque et al. 2010a; Bassanezi et al. 2013a);
- local measures (eradication of symptomatic trees and chemical vector control, aimed mainly against secondary spread) are not sufficient to effectively manage the disease; area-wide management (aimed mainly against primary spread) is, at present, the best way to keep disease incidence at an acceptable level (Bassanezi et al. 2013b);
- the monomolecular model fits well to the annual disease progress curves for HLB (Fig. 6).

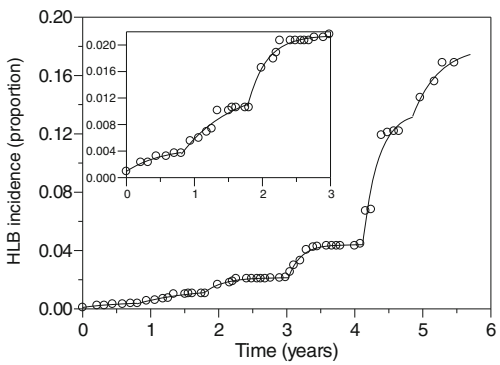

Fig. 6 Polyetic HLB progress curve showing waves of infection in each year (from 2008 to 2013) in São Paulo, Brazil. The inner graph shows a magnified view of the first 3 years of assessment

The concept of area-wide management has become the mainstay of HLB control programs in the USA, Brazil, and Mexico. In the USA, citrus growers are encouraged to join and form citrus health management areas (CHMAs) to control this disease more effectively on a regional basis. In Mexico, the government has established the ARCO (regional areas of control) program to control ACP in a coordinated manner based on regional monitoring of ACP organized by SIMDIA (*Diaphorina* monitoring system). In Brazil, voluntary groups of citrus growers control ACP population with three to four coordinated area-wide insecticide sprays in defined short periods based on an ACP Alert System developed by Fundecitrus. Other Brazilian growers with the agreement of their neighbours are supporting the control of ACP population and infected trees in their neighbouring groves to prevent HLB primary infections in their own groves.

Citrus Huanglongbing: a preliminary epidemiological model

The structure of HLB epidemics may be described using the architecture of Fig. 1. As earlier, the HLB model is centred on two equations that account for both the primary and secondary infections:

$$RP = RRP*P*(1-(DIS/(H+DIS))) \quad (3)$$

$$RS = RRS*DIS*(1-(DIS/(H+DIS))) \quad (4)$$

where RP and RS are the rates of primary and secondary infection, RRP and RRS are the relative rate of primary and secondary infection, and P, DIS, and H are the amount of (effective) inoculum, of diseased individuals (trees), and of healthy individuals, respectively.

Note that in this formulation, the amount of effective inoculum embeds the population of infectious vectors (and its epidemiological attributes, such as bacterial acquisition). The relative rates of epidemic speed, RRP and RRS, incorporate epidemiological characteristics of the insect vector as well (such as transmission to the host plants). The simplified model described in equations 3 and 4, and Fig. 7, takes into account the vector population, its dynamic, and its transmission characteristics in an implicit manner.

Box 2 summarizes the main hypotheses of this preliminary HLB model:

Box 2: HLB model main hypotheses for epidemics in Brazil

- System considered: an orchard of 50000 citrus trees
- Numerical simulation time step: 1 month
- Run duration: 10 years
- Dynamics addressed: number of trees that can be healthy (H), diseased, infectious, and asymptomatic (A), visibly diseased (symptomatic) and infectious (S)
- Infection has two origins: primary infection from infected vectors incoming into the system, and secondary infections that originate from

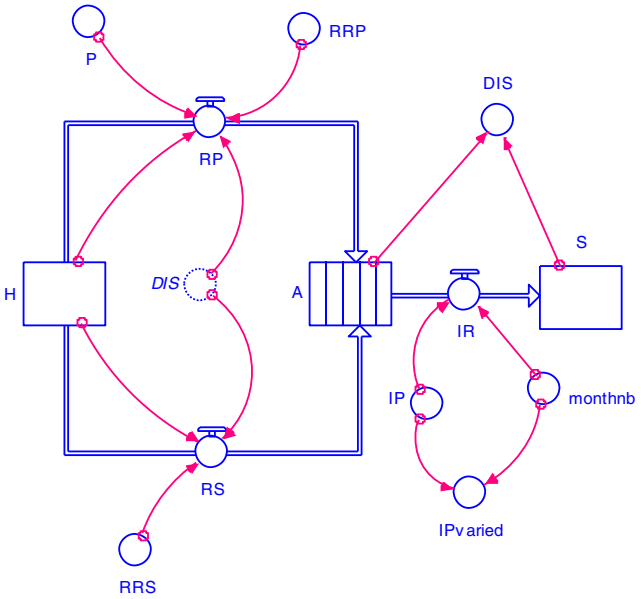

Fig. 7 Flowchart of an initial simulation model for Huanglongbing of citrus. Two flows of infections are considered, primary (RP) and secondary (RS), each with their relative rates (RRP, RRS). RP is proportional to P, the amount of primary inoculum (i.e., of infectious effective vectors), while RS is proportional to DIS, the accumulated number of infected asymptomatic (A) and symptomatic (S) trees. The incubation period (IP, i.e., residence time in A) is made variable (IPvaried) as a function of the date of infection in the year (month nb, i.e., 6 months in the summer and twice this duration in the winter, see text). H refers to healthy trees and IR refers to the infection rate. The diagram was built using Stella software ® (Richmond 2013)

diseased trees within the system (through vectors which are within the system considered)

- After infection, infected sites are infectious and asymptomatic
- Incubation period is 6 months (when infection occurs from Sept to Feb) or 12 months (when infection occurs from March to August)
- The primary inoculum, P [N_{vector}], is the pool of available infectious/infected insects that may enter the system at any time
- The rate of primary infection (RP) depends on a relative rate of primary infection (RRP) and on the quantity of primary inoculum (P), and is limited by the fraction of healthy trees remaining available to infection:

$RP = RRP * P * [1 - (DIS/(DIS + H))]$ [$N_{tree}.T^{-1}$]

Where: $DIS = A + S$ [N_{tree}], and RRP [$N_{tree}.N_{vector}^{-1}.T^{-1}$] is the relative rate of primary infection, i.e., the number of trees infected by incoming infected insects per month

- The rate of secondary infection (RS) is a function of the relative rate of secondary infection (RRS; [$N_{tree}.N_{tree}^{-1}.T^{-1}$]), of the amount of disease present (number of infected trees: DIS), and is limited in the same way as the rate of primary infection by the fraction of trees still healthy:

$RS = RRS * DIS * [1 - (DIS/(DIS + H))]$ [$N_{tree}.T^{-1}$]

- The model further assumes that P, RRS, and RRP are constant over time.
- Note on dimensions: because trees become infected by vectors, and vectors become infectious from trees: [N_{tree}] ≡ [N_{vector}]

In order to run the model of Fig. 7, additional hypotheses were made, relative to parameter values. The pool of potentially immigrating infectious insects was made: $P = 10^5$, to reflect the potentially very large number of effective propagules monthly reaching the system. RRP was, on the other hand, made much smaller than RRS: $RRP = 10^{-3}$ infected tree per infectious insect immigrating per month, vs. RRS = 0.05 infected tree per infectious tree per month, with the assumption that newly infectious vectors are much more effective within the citrus grove than immigrant propagules. The duration of the incubation period was made 6 months in the summer (from August to February), corresponding to a young orchard (age below 7 years), and was made 12 months in winter (March to July). Lastly, as in the theoretical models discussed above, no spatial aggregation of disease and disease spread was incorporated in the model.

The dynamics of the system are shown in Fig. 8. While the accumulated disease progresses (DIS = A + S, upper dashed curve) following a sigmoid pattern, the number of healthy trees (H) declines in a symmetric curve. On the other hand, the increase of symptomatic trees (S), that is, the amount of visible disease, does not increase in a smooth way, but instead in successive steps accounting for the succession of seasons in the simulation. The steps of increasing visible disease are initially small, then increase until the inflection point of the DIS curve, and then decrease towards the end of the run, when DIS (and S) approach the total size of the tree population. The first phase of the simulated visible disease (S), that is, the initial small first steps of disease, and their progressive increase resemble the shape of observed HLB progress curves.

Increasing values of RRS (Fig. 9a) lead to much steeper steps in S increase, whereas this increase is not as strong with increasing values of RRP (Fig. 9b). The system is very responsive to reduction of secondary spread (Fig. 9a), which could be achieved in HLB management by regular insecticide sprays. The background disease progress caused by the influx of infectious vectors is very hard to suppress – even very small values of RRP are enough for providing a sufficient basis for strong further disease increase (Fig. 9b). Both parameters, RRP and RRS, are in effect interacting on disease dynamics, and an optimum (not shown) may be found where RRP is maintained at a level low enough so that the constraint on RRS may be relaxed, that is, primary inoculum that is small

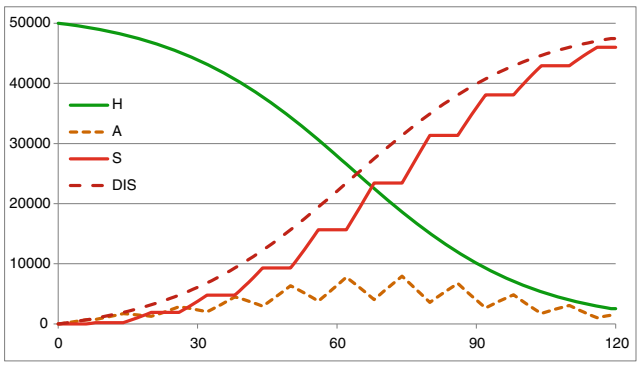

Fig. 8 Simulated HLB epidemic outputs. Ordinates: H: healthy trees, A: infected asymptomatic trees, S: infected symptomatic trees, DIS: accumulated (A + S) disease progress curve. Abscissa: time in months. Simulated outputs are for: IP = 6 or 12 (see text); $P = 10^5$; RRP = 0.001; RRS = 0.05

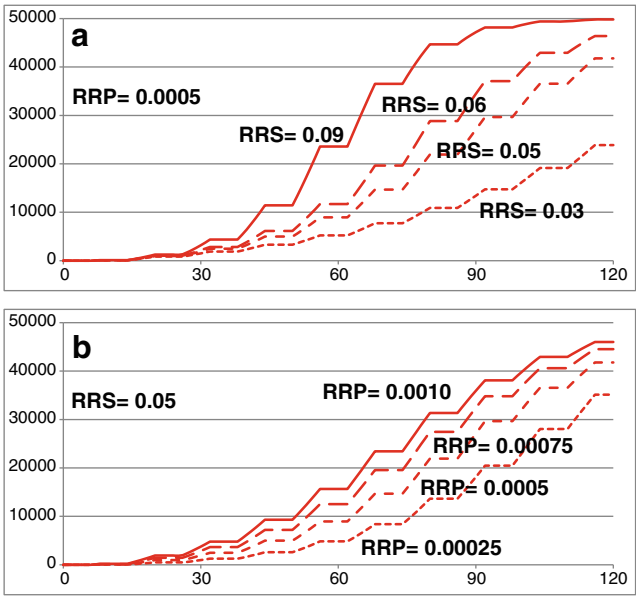

Fig. 9 Simulated dynamics of visibly diseased trees in a preliminary HLB model at variable relative rates of primary (RRP) and secondary (RRS) infection. **a** simulations at several values of RRS with RRP = 0.0005. **b** simulations at several values of RRP with RRS = 0.05. Ordinates: visibly diseased trees (S). Abscissa: time

enough to enable reducing the frequency of insecticide applications. From a disease management point of view, further information derived from this modelling analysis is that it is the variable delay in symptom incubation that causes the step-wise increase of visible disease, not variable delays for infected trees to become infectious (i.e., variation of the latency period). Similar simulation outputs (not shown) were obtained when only the visibly infected trees (S) are supposed to be infectious. This, and the simulated behaviour of the disease (Fig. 8, curves A and S), which indicates that the number of asymptomatic infected trees (A) is commensurate, and generally smaller than the number of visibly diseased trees (S), suggests that roguing is an efficient component of management, as long as it is combined with the management of inoculum outside the system (Fig. 7, P) and that of the infectious vectors inside the system (Fig. 7, RS).

Begomoviruses in tomato: epidemiology and current management

Begomoviruses are transmitted by whiteflies (*Bemisia tabaci*) in a circulative non-propagative manner. Very high levels of whitefly infestation (Fig. 10) in Brazil is the major cause of begomovirus outbreaks in recent years. Begomoviruses cause a range of different symptoms in tomatoes, from interveinal chlorosis, rugosity, leaf distortion, mosaic, and stunting (Fig. 10). Many virus species are reported in tomato plants in Brazil causing similar symptoms, and *Tomato severe rugose virus* (ToSRV) seems to predominate in the main growing regions (Fernandes et al. 2008).

Hybrid tomato varieties with moderate host plant resistance (HPR) for begomoviruses are available (Boiteux et al. 2007). The use of these moderately resistant materials is compulsory

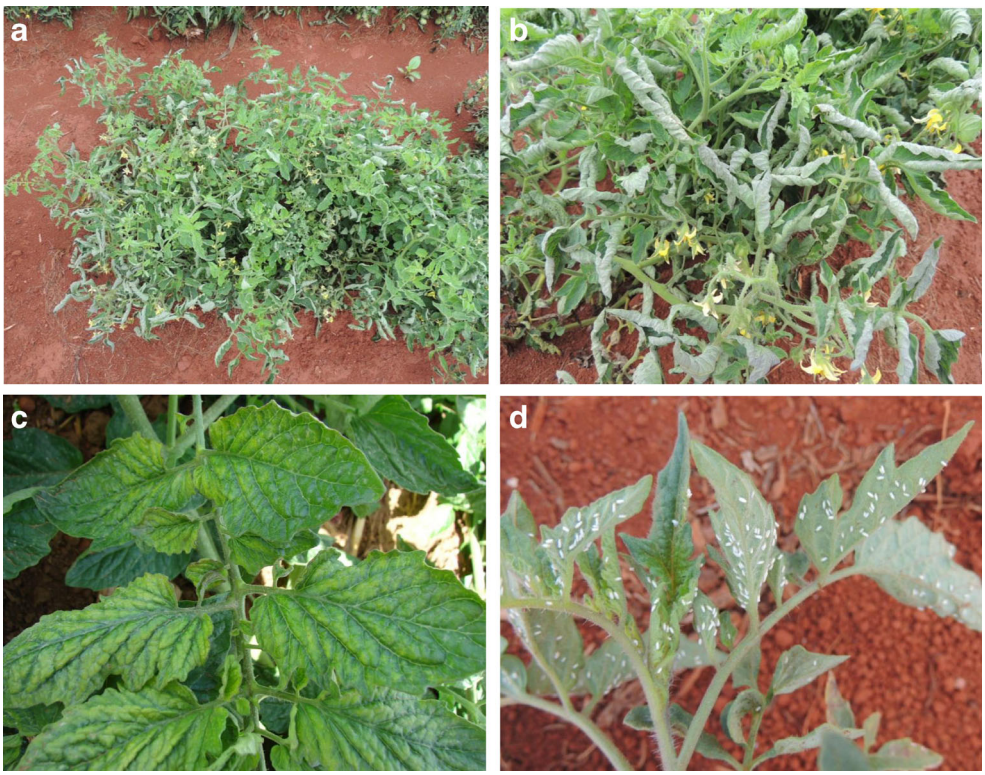

Fig. 10 Begomovirus infected tomato plants with strong leafrolling, interveinal chlorosis and stunting (**a**, **b**), interveinal chlorosis symptoms (**c**), and a young tomato plant infested with whiteflies (**d**)

in regions with high disease incidence. Both indeterminate and determinate tomatoes are cultivated, leading to distinct cultivation systems. Indeterminate-type growth tomatoes are grown for fresh market consumption with a large diversity of commercial hybrids with HPR in very diverse production systems, ranging from small growers of ca. 500 plants to large ones with more than 500,000 plants. These production systems can be very diverse, involving many cultivated vegetables. When inoculum is low, HPR and genetic diversity probably suppress disease efficiently; however, high inoculum load may lead to severe epidemics. By contrast, bushy-type tomatoes are mainly grown for processing in large, central-pivot irrigated fields (Fig. 11a) that are concentrated close to the industry. Fields of 1,000,000 to 3,300,000 plants (20 to 100 ha) are common. In these areas, tomatoes are planted next to soybean, bean, cotton, corn (Fig. 11b), sorghum, or sugarcane. Few resistant materials are currently commercially available. Our focus is on this system, where epidemics are the worst.

A tomato-free period was established in Brazil in 2003 to control the epidemics, first for processing tomatoes, then for fresh market tomatoes in regions where processing tomato cultivation is important. Implementation of the tomato-free period is actually highly recommended to all regions and for any growing system to increase the efficacy of this method, but inspections can be difficult in fresh market fields. Since 2007, the tomato-free period legislation was implemented only in the state of Goiás, the largest processing tomato state of Brazil. This was possible because of the pressure from the tomato paste industry on the Ministry of Agriculture, requesting a solution to the begomovirus/whitefly problem. In other states, the legislation was not implemented, although growers usually follow the transplanting calendar of the state of Goiás.

For processing tomatoes, transplanting starts on February 1st and ends on June 30th. Tomato crops are therefore present from February to the end of October or early November. Thus, the disease-free period takes place in December and January (Fig. 12), i.e., in the middle of the rainy season. During this period, large acreages of soybean, bean and cotton fields are distributed over the major agricultural area of central Brazil, where processing tomatoes are most cultivated. However, the soybean-free period (against Asiatic rust), the cotton-free period (against cotton boll weevil), and the dry bean-free period (against golden mosaic and whitefly) do not coincide with the tomato-free period. As a result, no whitefly host free period occurs in Brazil. Huge whiteflies clouds are seen from December to May, migrating out of the senescent crops. In this region, there are two major climate conditions, the rainy (October to April) and the dry (May to September) seasons (Fig. 12). The population of whiteflies is lowest in the cold season (May-July) and most numerous from September to May. Explosions of whiteflies are observed in the 'veranico' period, characterized by periods of at least four consecutive hot and dry days (and which lasts for 2–4 weeks) during the summer.

The best transplanting period for processing tomatoes is April to May. Although whitefly clouds are usually observed between December and February, they may appear in March, April and May, migrating from senescent cotton, soybean, and bean plants. Most of these whiteflies, however, are possibly non-transmitters of tomato begomoviruses. As during the growing season contiguous tomato areas with different aged plants are frequent, tomatoes are also a whitefly source (Fig. 13). In the begomovirus and tomato interaction, experimental results suggest the importance of primary infections (originating from external sources of inoculum) to the overall epidemics rather than secondary infections (within field disease spread). Border and gradient effects – earlier disease

Fig. 11 View of an agricultural area of central Brazil within the region of the highest density of central-pivot irrigated fields (**a**); a tomato field beside a corn field, both irrigated by the central-pivot system (**b**)

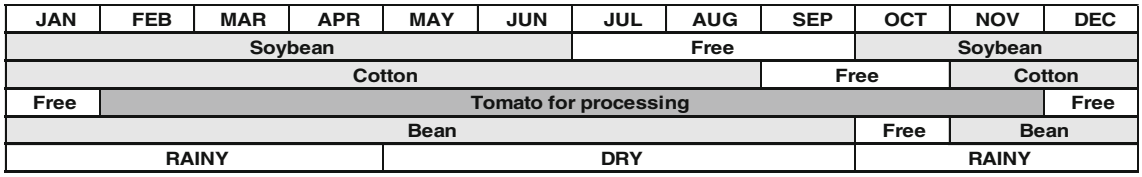

Fig. 12 Schematic view of host-free periods of pathogens of tomato and other crops implemented in Brazil

onset, higher whitefly population, and higher disease incidence in the periphery than within a field – was documented in a spatio-temporal analysis of a begomovirus disease epidemic in fresh market tomatoes in São Paulo state (Della Vecchia et al. 2007). Similar patterns were observed from a survey carried in 2011–2014 in irrigated fields intended for processing tomatoes (A.K. Inoue Nagata, unpublished data). A recent study carried out on ToSRV in a tomato field in Brazil indicated that the progress of disease incidence was well described by the monomolecular model, thus corresponding to a primary spread of disease (Barbosa et al. 2015).

A regular demand of fruits to supply the industry requires contiguous planting in a relatively close area surrounding the industry. In this case, older plants are sources of virus to the younger ones; the viruliferous whiteflies migrate in newly established crops, and infect younger plants. The insecticide spray scheme for young transplants does not prevent infection, probably because the whiteflies are able to transmit the virus before they are killed by insectides. On the other hand, the control of whiteflies by spraying the whitefly source is of low efficacy in mature plants, as the foliage cover protects the insects against direct contact with the insecticides, and systemic insecticides require time to circulate in the plant and affect the insect. We believe that this migration effect is directly related to the age of the plants. It was recently observed that a new tomato field transplanted near a 2 to 3 weeks older tomato field with high begomovirus infection incidence did not result in a high infection of the younger tomatoes. This was observed in two central pivot fields in 2012. While 30–96 % (in different places in the central pivot) older plants were infected with a begomovirus, 33–57 % younger plants were infected. The whitefly population was higher in older plants. It was speculated that the infection was kept low in the younger plants because of the aggressive insecticide spraying regime in the new transplants, and of the reduced migration from the older to the younger tomato field when the transplants differ by only 2 to 3 weeks. This suggests that contiguous transplanting may be done if the difference in plant age between contiguous fields is small.

Fig. 13 Younger tomato plants planted close to older ones

A question still to be addressed is whether whiteflies that propagate in cotton, bean or soybean can be viruliferous to tomato begomoviruses. While this question remains to be answered, it was shown that weeds can play an important role as a source of virus. This is the case for the begomovirus ToSRV and apple-of-Peru (*Nicandra physaloides*; Fig. 15a). Evidence was shown that this plant is an excellent host for ToSRV (Barbosa et al. 2009; Barreto et al. 2013) and it is widespread in the agricultural areas of Brazil. Other plants, such as *Euphorbia heterophylla* and *Sida santaremnensis*, which are important weeds in Brazil, are also sources of virus (Barreto et al. 2013). Furthermore, volunteer tomato plants are frequently found in fields, even years after the last planting of tomatoes. They are often seen in bean, soybean, and corn fields (Fig. 15b), but not in cotton fields, because they are not irrigated. Weeds and volunteer tomatoes are thus considered as major sources of viruliferous whiteflies. Therefore, it is highly recommended that weeds and volunteer tomatoes are removed from tomato-producing areas, and that the whiteflies are managed in these areas.

The complexity of controlling whiteflies becomes apparent when the agricultural systems involving processing tomato are considered as a whole. Tomato fields are surrounded by corn, sugarcane, sorghum, millet, dry-bean, soybean, and cotton fields, either irrigated or non-irrigated. Although the first four crops are not important as whitefly hosts (Fig. 15c), the other three are, with important consequences. In these crops, however, losses are not considered high enough to justify control costs. As a result, during the hot and dry season, whitefly populations dramatically increase unchecked in soybean and cotton crops in many agricultural areas, spreading as clouds to other crops and even invading cities. Most of these insects are disease non-transmitters. However, an IPM strategy incorporating all crops is necessary in order to reduce the insect

population size, and ultimately virus incidence (Gilbertson et al. 2011). Tomato crops are a primary target for management, especially when plants are old and host large insect populations. The situation in tomato may also worsen with the recent introduction of *Tomato chlorosis virus* (Barbosa et al. 2008), a crinivirus transmitted in a semi-persistent manner by whiteflies (Wintermantel et al. 2005). Next are the soybean and cotton crops, despite the fact that growers are far from being convinced to control the whiteflies in these crops. A public policy advocating support to tomato and bean growers today may prevent whitefly-transmitted virus outbreaks tomorrow in soybean and cotton.

Begomoviruses in tomato: epidemiological modelling

The above description reflects a very complex pathosystem, involving: (1) several virus species; (2) a single host with several biotypes; and (3) a wide range of alternate hosts, some cultivated, and others wild. It is not possible to develop here a preliminary simulation model as in the above example of citrus huanglongbing. However, we can retain the qualitative elements of this model: (1) the existence of a large reservoir of inoculum; (2) the ability of the insect vector(s) to transmit disease fairly rapidly (e.g., transmission may be partly hampered, but not fully prevented, by pesticides); (3) the existence of two processes of infection: one which enables disease spread within a given crop stand (secondary infection), and another enabling spread from sources outside the considered crop stand (primary infection).

Figure 14 presents the shape of a typical epidemic of ToSRV on tomato, where incidence and vector population sizes are plotted against time. A first observation is that the rate of disease increase is not necessarily proportional to the observed population of vectors. A second observation is that the overall progress of disease incidence, from onset (ca. day 28) till harvest (ca. day 105), roughly follows a monomolecular shape. A third observation is that this overall shape incorporates successive waves, apparently of decreasing height as disease progresses. The curve in Fig. 14 therefore resembles curves of

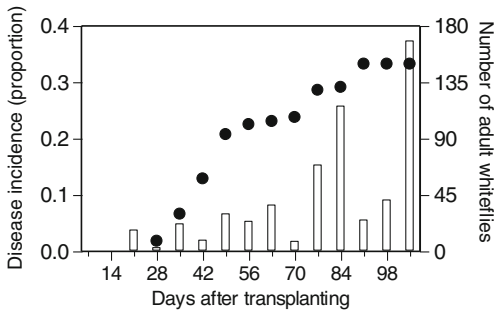

Fig. 14 Disease progress curve (*closed circles*) and number of adult whiteflies (*bars*) collected weekly in a tomato field from August to November, 2006, in São Paulo, Brazil. Source: modified from Barbosa et al. (2015)

Figs. 5 and 6 (progress of citrus huanglongbing over time) and also Fig. 8 (simulated output of a dual epidemic model). Because of the similarity of hypotheses concerning the epidemiology of begomovirus diseases in tomato and the epidemiology of huanglongbing in citrus, we hypothesis that this similarity in shapes is due to the same underpinning mechanisms. If this hypothesis is correct, the successive waves of the curve in Fig. 14 represent successive inflows of viruliferous whiteflies, each resulting in a monomolecular-type increase in disease intensity. The pattern of increase of Fig. 14 thus reflects the importance of incoming primary inoculum, as has been found in the interaction tomato - tospovirus (Puche et al. 1995), or in other begomovirus diseases (Cohen et al. 1988; Polston et al. 1996; Holt et al. 1999). If primary infections plays a key role for such epidemics, the best disease control strategy involves the control of incoming viruliferous whiteflies originating from external inoculum sources – other tomato fields, other crops, or infected wild hosts.

Discussion and conclusions

Aside from the two main types of epidemiological patterns that have classically been addressed in botanical epidemiology, this article considers a third, intermediate one, where the

Fig. 15 *Nicandra physaloides* with interveinal chlorosis and rugosity (**a**); volunteer tomato plant in a corn field (**b**); soybean infested with whitefly eggs and nymphs of *Bemisia tabaci* (**c**)

primary inoculum plays an important role throughout epidemics, and where secondary infections occur simultaneously. Diseases that follow this pattern are called polycyclic diseases with continuous primary spread.

The two diseases addressed in this article highlight the key commonalty between diseases exhibiting this type of epidemiological pattern: the importance of inoculum flow from outside the field or orchard to epidemic build-up calls for observations, measurement, understanding, and management that accounts for processes occurring outside the field under consideration. In other words, this corresponds geographically to linking field and landscape, and to linking farmer to community in terms of social and decision making processes. Such considerations have been outlined for livestock epidemics, where both the individual (individual farm) and the community (neighbouring farms) scales need to be considered to estimate optimum strategies to control epidemics (Matthews et al. 2003).

The two diseases, by contrast, highlight also differences which have consequences for the identification of management strategies. First, HLB pathogen and its vector have a comparatively narrower range of hosts than begomoviruses and their vectors. Second, perennial (citrus) and annual (tomato) crops, inherently confer strong differences from their lifespan: the development of resistant materials is obviously more difficult in the case of perennials, and management options associated with changes in host population density over time (e.g., consideration of a crop free period) and space (e.g., elimination of plants or plots) clearly correspond to contrasted situations in both cases. While the former feature (host range) raises difficulties in achieving efficient area wide management strategies for tomato virus diseases, the second feature corresponds to a more manoeuvrable landscape management for tomato.

In this article, we used two important diseases as working examples which correspond to major threats to current production systems. There are, we believe, many more examples of this third epidemiological pattern. One of them is rice tungro disease (RTD), a major constraint to rice production in Asia (Azzam and Chancellor 2002). The epidemiology and management of this disease have been very well documented and are therefore not discussed in detail here. The interested reader is referred to articles cited below, while highlights from research findings are briefly outlined here. This disease, simultaneously caused by two virus species, is also dependent on vectors (in this case, mostly, the green leafhopper, *Nephotettix virescens*), for its spread. In the case of RTD, the main reservoir of the viruses is the cultivated host plant, rice. A major feature of the epidemiology of RTD is its dependence on the presence, biology, and dispersal activity of the vector. For instance, severe epidemics of RTD are associated with large viruliferous fractions of the vector population – not with the number or density of vectors (Savary et al. 1993), a result that led to seriously question the usefulness of regular, systematic pesticide campaigns of the 60s and 70s. Further, careful research both in the field and in the laboratory, in research stations as well as with farmers' communities, demonstrated that (i) synchrony of crop establishment is a powerful means to reduce disease risk (Cabunagan et al. 2001), and (ii) partial HPR in rice may efficiently be deployed (Azzam and Chancellor 2002). This led to a major success in managing the disease, based on a strategy combining HPR and synchronization of crop establishment – pesticide use being essentially removed from the components for disease control, except, perhaps, in extreme and localized cases.

This additional example further shows that the third epidemiological pattern discussed in this article requires different levels of decision making, aimed to address both the community (management of the primary inoculum) and the farm (management of the secondary inoculum) levels. To act at farm level poses no particular problem to the farmer, but acting at community level involves aspects that transcend plant pathology due to the necessity of cooperation among farmers at a larger scale, where common resources are shared. In large area crops such as citrus, the shared environment is the diversity and size of the cultivated area; in the case of tomato begomoviruses, the shared resource is the irrigated area as well as the neighbouring crops; and in the case of rice tungro, the shared environment is largely determined by human and water resources. In all three cases, area-wide management needs to take place where not only material (e.g., water) resources must be shared, but also information, risks, and costs, to achieve collective benefit.

This collective area-wide management seems at first quite difficult to implement, even in well-structured production systems (as the citrus and HLB, for example, which benefits from a strong advisory and research system), and where the nature of the biological problem is well understood (e.g., in citrus and HLB, where the external inoculum sources are well known). Difficulties are to be expected in less-structured production systems (such as the tomato and begomoviruses, for example, where the external inoculum sources are poorly understood). Yet, the key example of RTD and rice in Asia is here to show that, even for a crop which literally means life for the farmers, solutions can be achieved.

Due to both the key role of primary infection and the lack of effective measures to suppress it, HPR represents an important element for the management of **polycyclic diseases with continuous primary spread**. Availability of HPR and

possibilities of rapid deployment are very variable according to the pathosystem considered. HPR prospects are good in the case of the tomato-begomoviruses pathosystem; but they are still quite remote in the case of citrus and HLB. In their 1997 review, Polston and Anderson highlighted the importance of HPR, but insisted on the need for a systems approach to manage tomato begomoviruses. The example of rice tungro in Asia shows that management of polycyclic diseases with continuous primary spread requires the consideration of several control components operating at different scales, the field and the landscape. Progress made in the case of citrus huanglongbing shows this approach to be valid. Much will depend on the ability of farmers, communities, and support organizations to develop collective, area-wide management strategies, while research explores yet new avenues.

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References

Agrios, G. N. (2005). *Plant pathology.* Burlington: Elsevier Academic Press.

Albuquerque, L. C., Varsani, A., Fernandes, F. R., Pinheiro, B., Martin, D. P., Ferreira, P. T. O., Lemos, T. O., & Inoue-Nagata, A. K. (2012). Further characterization of tomato-infecting begomoviruses in Brazil. *Archives of Virology, 157*, 747–752.

Amorim, L., Rezende, J. A. M., & Bergamin Filho, A. (2011). *Manual de Fitopatologia. Princípios e Conceitos*. São Paulo: Ceres.

Aubert, B. (1990). Integrated activities for the control of huanglongbin-greening and its vector *Diaphorina citri* Kuwayama in Asia. In: B. Aubert, S. Tontyaporn, D. Buangsuwon (Eds.), *Rehabilitation of Citrus Industry in the Asia Pacific Region*, (pp.133-144). Proceedings of Asia Pacific International Conference on Citriculture, Chiang Mai, Thailand, 4–10 Febr. 1990. Rome: UNDP-FAO.

Aubert, B. (1992). Citrus greening disease, a serious limiting factor for citriculture in Asia and Africa. *Proceedings of the International Society of Citriculture*, 817–820.

Avelino, J., Cristancho, M., Georgiou, S., Imbach, P., Aguilar, L., Bornemann, G., & Morales, C. (2015). The coffee rust crises in Colombia and Central America (2008–2013): impacts, plausible causes and proposed solutions. *Food Security, 7*, 303–321.

Azzam, O., & Chancellor, T. C. B. (2002). The biology, epidemiology, and management of rice tungro disease in Asia. *Plant Disease, 86*, 88–100.

Barbosa, J. C., Teixeira, A. P. M., Moreira, A. G., Camargo, L. E. A., Bergamin, A., Kitajima, E. W., & Rezende, J. A. M. (2008). First report of *Tomato chlorosis virus* infecting tomato crops in Brazil. *Plant Disease, 92*, 1709–1709.

Barbosa, J. C., Firmino, A. C., Reis, M. S., Barreto, S. S., Inoue-Nagata, A. K., Bergamin Filho, A., & Rezende, J. A. M. (2009). Natural infection of *Nicandra physaloides* by *Tomato severe rugose virus* in Brazil. *Journal of General Plant Pathology, 75*, 440–443.

Barbosa, J. C., Rezende, J. A. M., Amorim, L., & Bergamin Filho, A. (2015). Temporal dynamics of *Tomato severe rugose virus* and *Bemisia tabaci* in tomato fields in São Paulo, Brazil. *Journal of Phytopathology*. doi:10.1111/jph.12402. Online first.

Barreto, S. S., Hallwass, M., Aquino, O. M., & Inoue-Nagata, A. K. (2013). A study of weeds as potential inoculum sources for a tomato-infecting begomovirus in central Brazil. *Phytopathology, 103*, 436–444.

Bassanezi, R. B., Montesino, L. H., & Stuchi, E. S. (2009). Effects of huanglongbing on fruit quality of sweet orange cultivars in Brazil. *European Journal of Plant Pathololgy, 125*, 565–572.

Bassanezi, R. B., Montesino, L. H., Gasparoto, M. C. G., Bergamin Filho, A., & Amorim, L. (2011). Yield loss caused by huanglongbing in different sweet orange cultivars in São Paulo, Brazil. *European Journal of Plant Pathololgy, 130*, 577–586.

Bassanezi, R. B., Belasque, J., Jr., & Montesino, L. H. (2013a). Frequency of symptomatic trees removal in small citrus blocks on citrus huanglongbing epidemics. *Crop Protection, 52*, 72–77.

Bassanezi, R. B., Montesino, L. H., Gimenes-Fernandes, N., Yamamoto, P. T., Gottwald, T. R., Amorim, L., & Bergamin Filho, A. (2013b). Efficacy of area-wide inoculum reduction and vector control on temporal progress of huanglongbing in young sweet orange plantings. *Plant Disease, 97*, 789–796.

Belasque, J., Jr., Bassanezi, R. B., Yamamoto, P. T., Ayres, A. J., Tachibana, A., Violante, A. R., Tank, A., Jr., Di Giorgi, F., Tersi, F. E. A., Menezes, G. M., Dragone, J., Jank, R. H., Jr., & Bové, J. M. (2010a). Lessons from huanglongbing management in São Paulo State, Brazil. *Journal of Plant Pathology, 92*, 285–302.

Belasque, J., Jr., Yamamoto, P. T., Miranda, M. P., Bassanezi, R. B., Ayres, A. J., & Bové, J. M. (2010b). Controle do *huanglongbing* no estado de São Paulo, Brasil. *Citrus Research and Technology, 31*, 53–64.

Bergamin Filho, A., & Amorim, L. (1996). *Doenças de Plantas Tropicais: Epidemiologia e Controle Econômico*. São Paulo: Ceres.

Bergamin Filho, A., Kimati, H., & Amorim, L. (1995). *Manual de Fitopatologia. Princípios e Conceitos*. São Paulo: Ceres.

Berger, R. D. (1981). Comparison of the Gompertz and logistic equations to describe plant disease progress. *Phytopathology, 71*, 716–719.

Boina, D. R., Meyer, W. L., Onagbola, E. O., & Stelinski, L. L. (2009). Quantifying dispersal of *Diaphorina citri* (Hemiptera: Psyllidae) by immunomarking and potential impact of unmanaged groves on commercial citrus management. *Environmental Entomology, 38*, 1250–1258.

Boiteux, L. S., Oliveira, V. R., Silva, C. H., Makishima, N., Inoue-Nagata, A. K., Fonseca, M. E. D., & Giordano, L. D. (2007). Reaction of tomato hybrids carrying the Ty-1 focus to Brazilian bipartite Begomovirus species. *Horticultura Brasileira, 25*, 20–23.

Bové, J. M. (2006). Huanglongbing: a destructive, newly-emerging, century-old disease of citrus. *Journal of Plant Pathology, 88*, 7–37.

Bové, J. M. (2014). Huanglongbing or yellow shoot, a disease of Gondwanan origin: Will it destroy citrus worldwide? *Phytoparasitica* published online 06 July 2014.

Brassett, P. R., & Gilligan, C. A. (1988). A model for primary and secondary infection in botanical epidemics. *Zeitschrift fur Pflanzenkrankheiten und Pflanzenschutz, 95*, 352–360.

Butt, D. J., & Royle, D. J. (1980). The importance of terms and definitions for a conceptually unified epidemiology. In J. Palti & J. Kranz (Eds.), *Comparative epidemiology. A tool for better disease management* (pp. 29–49). Wageningen: Pudoc.

Cabunagan, R. C., Castilla, N., Colloquio, E. L., Tiongco, E. R., Truong, X. H., Fernandez, J., Du, M. J., Zarargosa, B., Hozak, R. R., Savary, S., & Azzam, O. (2001). Synchrony of planting and proportions of susceptible varieties affect rice tungro disease epidemics in the Philippines. *Crop Protection, 20*, 499–510.

Capoor, S. P., Rao, D. G., & Viswanath, S. M. (1967). *Diaphorina citri*: a vector of the greening disease of citrus in India. *Indian Journal of Agricultural Sciences, 37*, 572–576.

Cohen, S., Kern, J., Harpaz, I., & Ben-Joseph, R. (1988). Epidemiological studies of the *Tomato yellow leaf curl virus* (TYLCV) in the Jordan valley, Israel. *Phytoparasitica, 16*, 259–270.

Coletta-Filho, H. D., Targon, M. L. P. N., Takita, M. A., De Negri, J. D., Pompeu, J., Jr., & Machado, M. A. (2004). First report of causal agent of Huanglongbing ("*Candidatus* Liberibacter asiaticus") in Brazil. *Plant Disease, 88*, 1382.

Da Graça, J. V. (1991). Citrus greening disease. *Annual Review of Phytopathology, 29*, 109–136.

Da Graça, J. V., & Korsten, L. (2004). Citrus huanglongbing: review, present status and future strategies. In S. A. M. H. Naqvi (Ed.), *Diseases of fruit and vegetables* (v.1, pp. 229–245). Dordrecht: Kluwer Academic Press.

Dagulo, L., Danyluk, M. D., Spann, T. M., Valim, M. F., Goodrich-Schneider, R., Sims, C., & Rouseff, R. (2010). Chemical characterization of orange juice from trees infected with citrus greening (huanglongbing). *Journal of Food Science, 75*, C199–C207.

Della Vecchia, M. G. S., Rosa, D. D., Bergamin Filho, A., Amorim, L., Rezende, J. A. M., & Ribeiro, A. (2007). Dinâmica temporal e espacial da begomovirose causada por *Tomato yellow vein streak virus* em tomateiro na região de Campinas, SP. *Summa Phytopathologica, 33*, 388–396.

FAO. (2006). Food security. Policy Brief June 2006, Issue 2, p 4.

FAO, IFAD and WFP. (2013). *The state of food insecurity in the world 2013. The multiple dimensions of food security*. Rome: FAO.

Faria, J. C., Bezerra, I. C., Zerbini, F. M., Ribeiro, S. G., & Lima, M. F. (2000). Situação atual das geminiviroses no Brasil. *Fitopatologia Brasileira, 25*, 125–137.

Fernandes, F. R., Albuquerque, L. C., Giordano, L. B., Boiteux, L. S., Avila, A. C., & Inoue-Nagata, A. K. (2008). Diversity and prevalence of Brazilian bipartite begomovirus species associated to tomatoes. *Virus Genes, 36*, 251–258.

Flores, E., Silbersmidt, K., & Kramer, M. (1960). Observações de "clorose infecciosa" das malváceas em tomateiros do campo. *O Biologico, 26*, 65–69.

Gatineau, F., Bonnot, F., Yen, T. T. H., Tuan, D. H., Tuyen, N. D., & Truc, N. T. N. (2010). Effects of imidacloprid and fenobucarb on the dynamics of the psyllid *Diaphorina citri* Kuwayama and on the incidence of *Candidatus* Liberibacter asiaticus. *Fruits, 65*, 209–220.

Gäumann, E. (1950). *Principles of plant infection*. London: Crosby Lockwood and Son.

Gilbertson, R., Rojas, M., & Natwick, E. (2011). Development of integrated pest management (IPM) strategies for whitefly (*Bemisia tabaci*)-transmissible Geminiviruses. In W. M. O. Thompson (Ed.), *The whitefly, Bemisia tabaci (Homoptera: Aleyrodidae) interaction with geminivirus-infected host plants* (pp. 323–356). Dordrecht: Springer.

Gilligan, C. A. (1994). Temporal aspects of the development of root disease epidemics. In C. L. Campbell & D. M. Benson (Eds.), *Epidemiology and management of root diseases* (pp. 148–194). Berlin: Springer.

Gilligan, C. A. (2002). An epidemiological framework for disease management. *Advances in Botanical Research, 38*, 1–64.

Gilligan, C. A., & Kleczkowski, A. (1997). Population dynamics of botanical epidemics involving primary and secondary infection. *Philosophical Transactions of the Royal Society London B, 352*, 591–608.

Gottwald, T. R. (2010). Current epidemiological understanding of citrus huanglongbing. *Annual Review of Phytopathology, 48*, 119–139.

Gottwald, T. R., da Graça, J. V., & Bassanezi, R. B. (2007). Citrus huanglongbing: the pathogen and its impact. *Plant Health Progress*. doi:10.1094/PHP-2007-0906-01. PubMed RV.

Gottwald, T. R., Irey, M., Gast, T., Parnell, S., Taylor, E., & Hilf, M. (2010). Spatio-temporal analysis of an HLB epidemic in Florida and implications for future spread. *Proceedings of the 17th Conference of the International Organization of Citrus Virologists*, Riverside, CA. 84–97. (http://www.ivia.es/iocv/).

Halbert, S. E. (2005). The discovery of huanglongbing in Florida, p. H-3. In: *Proceedings 2nd International Citrus Canker and Huanglongbing Research Workshop*. Florida Citrus Mutual, Orlando, FL.

Hall, D. G., Gottwald, T. R., Stover, E., & Beattie, G. A. (2013). Evaluation of management programs for protecting young citrus plantings from Huanglongbing. *HortScience, 48*, 330–337.

Holt, J., Colvin, J., & Muniyappa, V. (1999). Identifying control strategies for tomato leaf curl virus disease using an epidemiological model. *Journal of Applied Ecology, 36*, 625–633.

Inoue-Nagata, A. K. (2013). Doenças Viróticas. In M. A. R. Alvarenga (Ed.), *Tomate. Produção em campo, casa de vegetação e hidroponia* (pp. 327–344). Lavras: Editora Universitária de Lavras.

Kuchment, A. (2013). The end of orange juice. *Scientific American, 308*, 44–51.

Lourenção, A. L., & Nagai, H. (1994). Surtos populacionais de *Bemisia tabaci* no estado de São Paulo. *Bragantia, 53*, 53–59.

Madden, L. V., Hughes, G., & Van den Bosch, F. (2007). *The study of plant disease epidemics*. St Paul, Minnesota, USA: The American Phytopathology Press.

Matthews, L., Haydon, D. T., Shaw, D. J., Chase-Topping, M. E., Keeling, M. J., & Woolhouse, M. E. J. (2003). Neighbourhood control policies and the spread of infectious diseases. *Proceedings of the Royal Society B, 270*, 1659–66.

McClean, A. P. D., & Oberholzer, P. C. J. (1965). Greening disease of sweet orange: evidence that it is caused by a transmissible virus. *South African Journal of Agricultural Science, 8*, 253–276.

McCook, S., & Vandermeer, J. (2015). The big rust and the red queen: long-term perspectives on coffee rust research. *Phytopathology, 105*, 1164–1173.

Polston, J. E., & Anderson, P. K. (1997). The emergence of whitefly-transmitted geminiviruses in tomato in the western hemisphere. *Plant Disease, 81*, 1358–1369.

Polston, J. E., Chellemi, D. O., Schuster, D. J., McGovern, R. J., & Stansly, P. A. (1996). Spatial and temporal dynamics of tomato mottle geminivirus and *Bemisia tabaci* (Genn) in Florida tomato fields. *Plant Disease, 80*, 1022–1028.

Puche, H., Berger, R. D., & Funderburk, J. E. (1995). Population dynamics of *Frakliniella* species (Thysanoptera: Thripidae) thrips and progress of spotted wilt in tomato fields. *Crop Protection, 14*, 577–583.

Richmond, B. (2013). *An introduction to systems thinking. Stella software*. New Hampshire: Isee Sytems.

Savary, S. (2007). Epidemics of plant diseases: mechanisms, dynamics and management. Chapter 7. In M. Tibayrenc (Ed.), *Encyclopaedia of infectious diseases* (pp. 125–136). London: Wiley.

Savary, S. (2014). The roots of crop health: cropping practices and disease management. *Food Security* (Submitted).

Savary, S., & Willocquet, L. (2014). Simulation modeling in botanical epidemiology and crop loss analysis. APSnet education center. *The Plant Health Instructor*. doi:10.1094/PHI-A-2014-0314-01.

Savary, S., Fabellar, N., Tiongco, E. R., & Teng, P. S. (1993). A characterization of rice tungro epidemics in the Philippines from historical survey data. *Plant Disease, 77*, 376–382.

Savary, S., Willocquet, L., & Teng, P. S. (1997). Modelling sheath blight epidemics on rice tillers. *Agricultural Systems, 55*, 359–384.

Strange, R. N., & Scott, P. R. (2005). Plant disease: a threat to global food security. *Annual Review of Phytopathology, 43*, 83–116.

Teixeira, D. C., Danet, J. L., Eveillard, S., Martins, E. C., Jesus, W. C., Jr., Yamamoto, P. T., Lopes, S. A., Bassanezi, R. B., Ayres, A. J., Saillard, C., & Bové, J. M. (2005). Citrus huanglongbing in São Paulo State, Brazil: PCR detection of the *Candidatus* Liberibacter species associated with the disease. *Molecular and Cellular Probes, 19*, 173–179.

Van der Plank, J. E. (1963). *Plant diseases. Epidemics and control*. New York: Academic.

Van der Plank, J. E. (1965). Dynamics of epidemics of plant diseases. *Science, 147*, 120–124.

Wintermantel, W. M., Wisler, G. C., Anchieta, A. G., Liu, H.-Y., Karasev, A. V., & Tzanetakis, I. E. (2005). The complete nucleotide sequence and genome organization of tomato chlorosis virus. *Archives of Virology, 150*, 2287–98.

Yamamoto, P. T., Felippe, M. R., Garbim, L. F., Coelho, J. H. C., Ximenes, N. L., Martins, E. C., Leite, A. P. R., Sousa, M. C., Abrahão, D. P., & Braz, J. D. (2006). *Diaphorina citri* (Hemiptera: Psyllidae): vector of the bacterium *Candidatus* Liberibacter americanus. Proceedings of Huanglongbing – Greening International Workshop. P. 96. Fundecitrus: Araraquara.

Zadoks, J. C. (1971). Systems analysis and the dynamics of epidemics. *Phytopathology, 61*, 600–610.

Zadoks, J. C., & Schein, R. D. (1979). *Epidemiology and plant disease management*. New York: Oxford University Press.

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