Annex 1

Identifying control strategies for tomato leaf curl virus disease using an epidemiological model

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Summary

1. A number of insect vectors of plant-virus diseases make only transitory visits to the crop in which the economic effects of the disease are important. The incidence of disease in the crop depends primarily on the immigration of vectors from alternative hosts which act as a reservoir of both the virus and vector.

2. An epidemiological model was developed to represent this situation and parameters were estimated for the case of tomato leaf curl virus disease (TLCVD) (Geminiviridae, Subgroup III) in India. From an analysis of the model, the following possibilities for the management of TLCVD emerged.

3. It was clear that varietal resistance to infection could be an important component of disease management but whether, once infected, the tomato plants acted as a source of inoculum had little impact on disease incidence in the tomato crop.

4. A very low rate of simulated vector immigration into a tomato crop sufficed to cause almost total infection. Around Bangalore, vectors may migrate into tomato crops in numbers in excess of those required for disease ‘saturation’, explaining why, using conventional insecticides, very efficient and intensive vector control is currently required to reduce disease incidence.

6. Disease incidence was sensitive to vector mortality only when vector numbers were low. In most cases, the immigration of viruliferous vectors made disease incidence insensitive to the mortality of vectors within the tomato crop.

7. A strategy for disease management which targets more than one of the parameters to which the model proved most sensitive is likely to be necessary. In particular, the use of protective netting combined with the growing of resistant varieties has the potential to reduce both B. tabaci immigration to the crop and to reduce virus inoculation by those insects which do reach the crop.

Key-words: Bemisia tabaci, geminivirus, India, insect dispersal, pest management

Introduction

For some economically important virus diseases of agricultural crops, the insect vector makes little use of that crop species for oviposition and development. Instead, alternative and preferred host-plant species are utilized. These may be other crops or wild host plants or both. These can be abundant and provide a substantial reservoir of both the vector and the virus outside the affected crop. In such situations, the adult vector typically makes only transitory visits to the crop host, where activity is restricted to feeding and virus transmission. In some cases, the virus may best be considered as a pathogen of wild plants that has become important to man when crops are grown in their vicinity. Many viruses of wide distribution in annual crops, but which are not seed-borne, are of this character (Duffus 1971). Well known are maize streak virus disease in Africa (Rose 1978), Rio Cuarto virus disease of maize in Argentina (Trumper, Gorla & Grilli 1996) and tomato leaf curl virus disease in southern India (Sastry, Singh & Sastry 1978; Saikia & Muniyappa 1989). In this paper we focus on the last example.

Indian tomato leaf curl geminivirus (TLCV) is considered to be the most important viral pathogen of tomato in India (Vasudeva & Sam Raj 1948; Sastry & Singh 1973; Muniyappa & Saikia 1983), causing yield losses, when infection takes place early in plant development, of 47 - 95% (Saikia & Muniyappa 1989). Infected plants show a variety of symptoms, including leaf curling, vein clearing, stunting and partial or complete sterility (Sastry & Singh 1973; Saikia & Muniyappa 1989; Ramappa 1993).

The whitefly, *Bemisia tabaci* (Gennadius) is the only known vector of TLCV (Vasudeva & Sam Raj 1948; Butter & Rataul 1977; Saikia & Muniyappa 1989). The virus is transmitted in a persistent manner and a single *B. tabaci* adult can transmit the virus to tomato plants of all ages after a single acquisition access period of 10 - 30 minutes (Butter & Rataul 1977; Seetharama Reddy, Yaraguntaiah & Sastry 1981; Ramappa 1993).

*B. tabaci* has a very wide host range in south India and has been found on 173 species, representing 31 plant families in the Bangalore area (Saikia & Muniyappa 1989). More than 22 of these species, including both annuals and perennials, are also
known hosts of TLCV. Species in the latter group, such as *Hibiscus rosasinensis* L., can therefore act as a reservoir of both the vector and the virus throughout the year (Sastry, Singh & Sastry 1978). Due to their relative abundance, however, the most important group of alternative host plants are probably weed species such as *Acanthospermum hispidum* DC., *Ageratum conyzoides* L., *Euphorbia geniculata* Ort., *Parthenium hysterophorus* L., *Malvastrum coromandelianum* (L.) and *Phylanthus asperulatus* Hutch. (= *P. niruri* Auct.), which are extremely common throughout south India, and frequently cover the banks and open ground surrounding areas of tomato cultivation (Ramappa, Muniyappa & Colvin 1998).

Both recent and earlier field experiments on the management of TLCV have shown that significant numbers of *B. tabaci* adults arrive and feed on tomato plants throughout the life of the crop, although the number of nymphs found developing on tomato, in comparison with those on the weeds in the surrounding area, is extremely low (Ramappa, Muniyappa & Colvin 1998; A. Cherian unpublished data; H.M. Ventatesh unpublished data).

In this paper, we use the above biological and epidemiological data to develop a mathematical model of the dynamics of Indian tomato leaf curl disease (TLCVD) in southern India, where irrigated tomato production takes place continuously and alternative host plants of both virus and vector are abundant. To allow a more targeted search for effective and sustainable disease management strategies, our purpose is to examine the implications of the epidemiology of this disease for its control.

**Materials and methods**

**THE MODEL**

In previous dynamic models of the epidemiology of plant virus diseases, the plant population has been divided into convenient categories, reflecting their disease status: healthy, latently infected, infectious, and post-infectious (e.g. Chan & Jeger 1994). Similarly, it is possible to define analogous categories for the vector population: non-infective, viruliferous but not yet infective, and infective, and to devise a model which links the epidemiology of the disease with the population dynamics of the vector.
Such models have been developed for rice dwarf virus disease (Nakasuji et al. 1985), and cassava mosaic virus disease (Holt et al. 1997). In both of these, certain details were incorporated to reflect important features of the particular pathosystems. In Holt et al. (1997), relatively complicated terms were needed to specify the planting rate of the crop and the reproduction of the vector. A general framework for this type of model was given in Jeger et al. (1998), in which a minimum of non-essential detail was included so, for example, the population size of both the host and the vector were regarded as constant.

In the TLCVD pathosystem, a model with the following components was required. Only three categories of crop host need be defined: healthy \( (H) \), latently infected with the virus \( (L) \), and infectious \( (S) \). Once infectious, a crop-host plant was assumed to remain so until harvest, so no post-infectious class was included. All classes were assumed to be removed at a constant rate, \( \beta \), in accordance with the period of the crop cycle. Replanting took place at a rate which exactly balanced those plants removed. In a year-round vegetable production system, these assumptions are considered reasonable and, to specify disease dynamics in the host, equations similar to those of Jeger et al. (1998) were appropriate.

An infection parameter, \( a \), determined the rate at which healthy crop-host plants became latently infected and this also depended on the availability of healthy hosts, \( H \), and the abundance of infective vectors, \( Z \). Latently infected plants, \( L \), passed to the infectious class at a rate, \( b \), which is inversely proportional to the mean latent period. An acquisition parameter, \( \lambda \), determined the rate at which non-infective vectors, \( X \), became infective which also depended on the availability of non-infective vectors and the abundance of infectious hosts, \( S \). The latent period in the vector between acquisition of the virus and the ability to transmit the virus is c. 30 min. (Butter & Rataul 1977). It was assumed, therefore, to be negligible and no latent category was defined for the vector.

Because we consider the special case in which vectors make only a transitory visit to the crop and do not reproduce, the equations for vector population dynamics in tomato could be simplified. All reproduction was assumed to take place on alternative host species, which were also hosts for the virus. A constant rate of immigration, \( \mu \), was assumed to take place from these alternative hosts, with a proportion, \( \theta \), being infective, so arrivals of infective and non-infective vectors were
given by $\theta \mu$ and $(1 - \theta)\mu$, respectively. Of the vectors present at any one time, a proportion was assumed to die or depart per day, given by a total vector loss rate, $g$.

These assumptions lead to a model specified by a system of rate equations:

\[
\begin{align*}
\frac{dH}{dt} &= \beta (L + S) - aHZ \\
\frac{dL}{dt} &= aHZ - bL - \beta L \\
\frac{dS}{dt} &= bL - \beta S \\
\frac{dX}{dt} &= -\lambda SX - gX + (1 - \theta)\mu \\
\frac{dZ}{dt} &= \lambda SX - gZ + \theta \mu
\end{align*}
\]

where $\beta = 1 / \text{crop period (day}^{-1})$, 
$a = \text{host plant infection rate (vector}^{-1}\text{day}^{-1})$, 
$b = 1 / \text{latent period (day}^{-1})$, 
$\lambda = \text{vector acquisition rate (plant}^{-1}\text{day}^{-1})$, 
$g = \text{vector loss rate (day}^{-1})$, 
$\theta = \text{infectivity of arriving vectors (proportion)}$, 
$\mu = \text{vector arrival rate (number plant}^{-1}\text{day}^{-1})$.

As can been seen from equations 1, $(dH + dL + dS) / dt = 0$, so the total abundance of hosts is constant, and depends on the starting values used for $H$, $L$ and $S$. Equations 1 can be simplified by omitting an equation for one of the host categories and including a parameter, $K$, the total host abundance. $K$ was arbitrarily scaled to unity so that vector number is expressed per plant.
\[
\begin{align*}
\frac{dH}{dt} &= \beta(K - H) - aHZ \\
\frac{dL}{dt} &= aHZ - bL - \beta L \\
\frac{dX}{dt} &= -\lambda SX - gX(1 - \theta)\mu \\
\frac{dZ}{dt} &= \lambda SX - gZ + \theta \mu
\end{align*}
\]
eqns 2

Partial solutions for the equilibria of equations 2 were derived, as a function of the number of infectious hosts, \(S\). By replacing \(S\) with \(K - (H + L)\) in equations 2, a full solution was possible but which had a relatively complicated quadratic form. The partial solution was sufficient to make numerical analysis of the equilibria straightforward. The partial solutions were:

\[
\begin{align*}
X^* &= \frac{\mu(1 - \theta)}{\gamma}, \quad Z^* = \frac{\mu \omega}{g \gamma}, \quad H^* = \frac{\beta g K \gamma}{\alpha \mu \omega + \beta g \gamma}, \quad L^* = H^* \frac{a \mu \omega}{(\beta + b)g \gamma}
\end{align*}
\]
eqns 3

where \(\gamma = S^* \lambda + g\), and \(\omega = S^* \lambda + g \theta\). The equilibrium points of the variables \((H^*, \text{etc.})\) provided a convenient measure of the potential long-term impact of parameter changes on disease incidence and vector abundance. For example, the magnitude of the increase in \(H^*\) associated with a decrease in inoculation rate caused by the growing of a resistant variety gives a measure of the potential effect of such a variety on TLCVD incidence.

Two quantities, \(\gamma\) and \(\omega\) simplify the expressions for the critical points. The quantity \(\gamma\) is the total loss rate of non-infective vectors and so the expression for \(X^*\) can be interpreted simply as gains / losses. The quantity \(\omega\) has a less clear biological interpretation. It is difficult to draw conclusions directly from the expression for \(Z^*\) because the vector departure rate \(g\) and virus acquisition rate \(\lambda\) appear in both numerator and denominator. As with \(X^*\), however, \(\mu\) appears only in the numerator indicating that the numbers of both the infective and the non-infective vectors to be found in the crop are directly proportional to total immigration rate. In the expression for \(H^*\), \(K\) appears only in the numerator and \(a\) and \(\theta\) only in the denominator. Other
parameters occur in both. Obviously, inferences drawn from the position of parameters are made in the context that $S'$ is also present in the solutions.

ESTIMATION OF THE PARAMETERS

Parameter values were estimated from the literature (Table 1). Two parameters can be estimated relatively accurately, the crop turnover rate, $1/\beta$, and the latent period of the disease in the plant, $1/b$, which are approximately three months and 10 - 14 days respectively (Nateshan et al. 1996; Ramappa 1993). From laboratory studies, a good estimate can also be obtained of the maximum potential for virus transmission between plants, but values of the inoculation rate ($a$) and acquisition rate ($\lambda$) which are appropriate under field conditions are less easy to estimate. After an acquisition access feed of 24 h, in which vectors were confined on infectious tomato plants, individual adult female $B. tabaci$ transmitted TCV to 30% of test plants. Shorter acquisition access feeds, however, resulted in much lower percentages of transmission (Butter & Rataul 1977). The number of tomato plants visited by a vector, and the extent to which probes of sufficient duration occur to inoculate the host with the virus will also be critical determinants of the inoculation rate in the field. When individual viruliferous female $B. tabaci$ were transferred every two days to new healthy seedlings, an average infection rate of 0.086 new infections vector$^{-1}$ day$^{-1}$ was obtained (Butter & Rataul 1977). This experimentally-controlled rate of movement between plants probably approximates to the maximum rate in the field, where females may probe several plants, over a period of several days. In developing models with appropriate parameter values for field conditions, estimates of the inoculation rates for plant viruses ranged from 0.0005 to 0.01 vector$^{-1}$ day$^{-1}$ (Jeger et al. 1998). For TCV, values in the range 0.001 to 0.1 have been investigated in the analysis.

Data also exist for the number and infectivity of vectors which can be found within the tomato crop. $B. tabaci$ adults in southern India have been sampled from randomly selected plants in tomato fields and tested with molecular methods for the presence of TCV. The data indicated that the percentage of viruliferous adult $B. tabaci$ at the time of sampling was between 8 - 60% (Ramappa 1993; Ramappa, Muniyappa & Colvin 1998). An estimate was made of the virus acquisition rate $\lambda$, by
selecting a value, which resulted in a proportion of viruliferous \textit{B. tabaci} similar to that observed. A value of $\lambda = 0.003 \text{ plant}^{-1} \text{ day}^{-1}$ gave a proportion viruliferous of c. 30\% though, of course, the infectivity of the immigrant vectors also affects this value. In the analysis, $\lambda$ was varied between 0 and 0.2.

The number and infectivity of immigrant vectors and the rate at which those present in the crop die or depart were of particular interest in the analysis. Sensitivity to these parameters ($\mu$, $\theta$ and $g$, respectively) was determined by varying them over the ranges shown in Table 1. It was possible to judge to some extent whether the ranges used for $\mu$ and $g$ were reasonable by comparing predicted values of $X^* + Z^*$ with vector abundance in field observations (Table 1).

**Results and Discussion**

**GENERAL COMPARISON OF SIMULATIONS WITH FIELD DATA**

A general model of the epidemiology of TLCVD has been formulated and parametrised from known epidemiological and ecological information. The purpose is not to fit the model to individual cases but to model the general features of the pathosystem and gain insights into its dynamics. The model considers the long-term incidence of TLCVD within a locality containing many tomato fields (Nateshan et al. 1996). A comparison with data from individual tomato fields was nevertheless useful to provide some assessment of whether model parameters estimated from the literature were reasonable. Observed disease progress on TLCV-susceptible and resistant tomato varieties were compared with disease progress curves simulated by the model (Figs 1a and b). The field data were collected during the hot Kharif season (February to May) when whiteflies are the most abundant and disease progress most rapid (Saikia & Muniyappa 1989). Assuming a high number and infectivity of immigrants the model predictions compared well with independent field observations. The inoculation rate was assumed to be ten times greater on susceptible varieties (Fig. 1a) than on resistant ones (Fig. 1b) and the vector departure rate five times less, but no data are available to support these assumptions. In the field experiments, total infection occurred in the susceptible varieties (Fig. 1a) but this was not reflected in model output due to the assumption of a turn-over of tomato crops within a locality.
PARAMETER SENSITIVITY AND ITS IMPLICATION FOR CONTROL MEASURES

An assessment was made of the sensitivity of the equilibrium values of the variables to changes in the model’s parameter values. Different disease management options can be represented by changes in certain parameter values (Table 2). For example, the deployment of a tomato variety resistant to both the virus and the vector might reduce infection and acquisition rates, increase the latent period of the disease in the crop and increase the vector departure rate from the crop. The sensitivity of the model to the parameters concerned gives an indication of the relative impact that the different management options might have.

The predicted abundance of healthy plants at the minimum and maximum parameter estimates (Table 3) illustrates the sensitivity of the model to each of its parameters. The model was insensitive to $b$, suggesting that any changes in the disease latent period associated with a resistant variety would not be important in reducing TLCV incidence. Almost no change in mean incidence occurred when the latent period was doubled from 10 days ($b = 0.1$) to 20 days ($b = 0.05$). The model was also fairly insensitive to $\beta$, as a reduction in crop turn-over rate from 4 months ($\beta = 0.005$) to 2.5 months ($\beta = 0.015$) reduced mean disease incidence from about 50% to 30% (with other parameters at their default values). Increasing crop turn-over rate therefore had a small beneficial effect by decreasing the period of exposure to infection.

Varietal resistance might also be reflected in both lower infection ($a$) and acquisition ($\lambda$) rates. TLCVD incidence was very sensitive to the infection rate but rather less sensitive to the acquisition rate. The acquisition rate might be expected to be important only when a significant proportion of disease transmission is due to vectors which have acquired the virus within the crop. Despite the fact that the latent period in the vector was considered to be negligible, it appeared that immigration of infective vectors was the principal driving force in host infection. Whether or not vectors acquire the virus within the crop, therefore, was not important for disease progress in the crop. The model did however become increasingly sensitive to acquisition rate ($\lambda$) when both infectivity of the immigrant vectors ($\theta$) and the
departure rate of the vectors from the crop \((g)\) were reduced (Fig. 2). These factors, in combination, provided the conditions for virus acquisition rate to be important in disease dynamics and appear to be met in the contrasting case of cassava mosaic virus disease (CMD) in Africa, where \(B. tabaci\) multiplies on cassava and increases in CMD incidence correlate well with the size of the \(B. tabaci\) population on the crop six weeks earlier (Fargette et al. 1990; Colvin et al. 1998). In a model of CMD dynamics (Holt et al. 1997), disease incidence in cassava proved equally sensitive to the inoculation and acquisition rates.

For TLCVD in southern India, it is likely that infectivity of whitefly immigrants from alternative hosts is high because disease is widespread in such hosts (Sastry, Singh and Sastry 1978; Saikia & Muniyappa 1989). Consequently, there is likely to be no great advantage in varieties from which it is difficult for vectors to acquire the virus, if they are not also resistant to infection. Where the crop is an important source of inoculum, however, resistance mediated by a low acquisition rate may have a role. For TLCV in Bangalore, the tomato crop sources are greatly outnumbered by alternative hosts, both wild and cultivated, so the contribution of tomato as an inoculum source is probably small. This is evident from the results of field trials where tomato plots were planted in an area new to tomato production and the final incidence of TLCVD exceeded 80%. It was also found, however, that the disease progress curves did increase slightly more rapidly when new tomato fields were planted adjacent to older infected ones (Ramappa 1993; Ramappa, Muniyappa & Colvin 1998).

The model was very sensitive to both the number \((\mu)\) and infectivity \((\theta)\) of immigrant vectors. Thus, isolation of tomato fields would be expected to have a substantial effect if it was also possible to isolate the crop from alternative hosts of both virus and vector. The results of the model, however, predict that disease ‘saturation’ can occur at relatively low immigration rates (Fig. 3). As the value of \(\mu\) was increased, the marginal impact of changes in \(\mu\) on disease incidence became less and less. Ultimately, addition of more and more vectors made very little difference to incidence due to disease saturation. Where incidence is high, as commonly occurs in the Bangalore area, vectors may be very much in excess of what is required to cause disease saturation, and immigration would have to be reduced greatly for a significant reduction in incidence to be seen. With resistant varieties, infection rate is lower and
incidence below saturation, (Fig. 3), and in this case the relationship with immigrant number is more linear.

When vector number is not excessive, disease incidence was very sensitive to the vector loss rate ($g$), but this sensitivity was affected by the number ($\mu$) and infectivity ($\theta$) of the immigrants (Fig. 4). An increase in loss rate from 0.03 to 0.1 day$^{-1}$ reduced incidence by two or three-fold, provided that both immigrant number and infectivity were not too high. When the crop was subject to high immigration of infective vectors, however, then even high loss rates were insufficient to have much impact. In the examples shown in Fig. 4, the difference in sensitivity to loss rate at selected values of immigrant number and infectivity can be seen (with other parameters at default settings). A control method which both reduces $\mu$ and increases $g$, therefore, has the potential for much greater impact than one which only increases $g$. Current management practices which possibly work in this way include the use of boundary trap crops that are relatively more attractive to the vector, repellents such as neem oil, conventional insecticides with any irritant/repellent effect and the application of wood ash to tomato foliage (N. Nagaraju unpublished data).

A novel approach, with a potentially big impact on both $\mu$ and $g$, currently being tested in southern India, is to position a nylon-net screen, designed to make use of recognised $B.\ tabaci$ adult behaviour, around a tomato field. The screen is treated with a persistent, knock-down insecticide and is also dyed bright yellow on the side facing the tomato crop. Yellow is highly attractive to $B.\ tabaci$ adults (Husein & Trehan 1940; Mound 1962) and we suggest that such nets could reduce immigrant numbers significantly, because most $B.\ tabaci$ adults appeared to fly close to the ground (Colvin et al. 1998; H.M. Venkatesh et al. unpublished data) and so should be intercepted by the nets. In addition, any adults that fly over the netting may be attracted out of the crop again by the colour of the material.

By increasing $g$ alone, there may be considerable potential for a useful effect in reducing disease incidence, provided that the arrival rate of infective immigrants is not too high. It is important to remember, however, that this effect must be sustained. An instantaneous reduction in $g$ brought about by a single insecticide or mycopesticide application would have little effect. The transitory nature of the whitefly population in the crop would mean that $B.\ tabaci$ losses would soon be replaced by further vector immigration. This conclusion probably explains the very
intensive use of all classes of conventional insecticides by tomato farmers in southern India, particularly during the summer season when the whitefly vector is most abundant (Sastry, Sastry & Singh 1974; Rataul & Butter 1976).

Although, in theory, natural enemies have the potential to impose a sustained mortality on $B. tabaci$ numbers (i.e. a sustained increase in $g$), this appears not to happen in practice as the $B. tabaci$ present in the tomato crop are mostly adults rather than nymphs (Ramappa, Muniyappa & Colvin 1998). These adults are also widely dispersed and transitory and appear to suffer little mortality from predators and parasites whilst on tomato (H.M. Ventatesh et al. unpublished data).

In summary, parameters $a$, $\theta$ and $\mu$ were the parameters to which the model was most sensitive. A 12.5, 5.6 and 5-fold change occurred, respectively, in the abundance of healthy plants when these parameters where varied between their estimated minimum and maximum values (Table 3). Thus any control measure which significantly reduces these parameters (Table 2) is likely to reduce TLCVD incidence. Any strategy directed against the vector must have a large impact on the number of immigrants and/or their infectivity. If this can be combined with the deployment of virus-resistant varieties in which the infection rate is also reduced, then there exists the basis for an integrated management strategy targeted at those parameters to which TLCVD incidence was most sensitive in this model system.

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References


Table 1. Ranges of parameters and variables used in the model with data sources

<table>
<thead>
<tr>
<th>Default value</th>
<th>Estimated range</th>
<th>Reference sources</th>
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</thead>
<tbody>
<tr>
<td>( \beta )</td>
<td>0.01 day(^{-1} )</td>
<td>0.0083 - 0.013</td>
</tr>
<tr>
<td>( a )</td>
<td>0.01 vector(^{-1} ) day(^{-1} )</td>
<td>0 - 0.1</td>
</tr>
<tr>
<td>( b )</td>
<td>0.075 day(^{-1} )</td>
<td>0.05 - 0.1</td>
</tr>
<tr>
<td>( \lambda )</td>
<td>0.003 plant(^{-1} ) day(^{-1} )</td>
<td>0 - 0.2</td>
</tr>
<tr>
<td>( g )</td>
<td>0.06 day(^{-1} )</td>
<td>0.03 - 1</td>
</tr>
<tr>
<td>( \theta )</td>
<td>0.2 proportion</td>
<td>0 - 1</td>
</tr>
<tr>
<td>( \mu )</td>
<td>0.3 number plant(^{-1} ) day(^{-1} )</td>
<td>0 - 1</td>
</tr>
<tr>
<td>( K )</td>
<td>1 unit area(^{-1} )</td>
<td>arbitrary constant</td>
</tr>
<tr>
<td>( X^<em>+Z^</em> )</td>
<td>vector</td>
<td>0 - 5</td>
</tr>
<tr>
<td>( Z^<em>/X^</em> )</td>
<td>proportion</td>
<td>0.08 - 0.6</td>
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Table 2. Disease management options and potential effect on model parameters

<table>
<thead>
<tr>
<th>Option</th>
<th>Potential effect</th>
<th>Parameter concerned</th>
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<tbody>
<tr>
<td>nursery net</td>
<td>decrease in vector immigration</td>
<td>$\mu$</td>
</tr>
<tr>
<td></td>
<td>decrease in vector emigration</td>
<td>$g$</td>
</tr>
<tr>
<td>resistant variety</td>
<td>reduction in infection rate</td>
<td>$a$</td>
</tr>
<tr>
<td></td>
<td>reduction in acquisition rate</td>
<td>$\lambda$</td>
</tr>
<tr>
<td></td>
<td>increase in vector departure rate</td>
<td>$g$</td>
</tr>
<tr>
<td></td>
<td>increase in latent period</td>
<td>$b$</td>
</tr>
<tr>
<td>short-duration variety</td>
<td>increase in crop turn over</td>
<td>$\beta$</td>
</tr>
<tr>
<td>insecticide</td>
<td>increase in vector death rate</td>
<td>$g$</td>
</tr>
<tr>
<td>mycopesticide</td>
<td>increase in vector death rate</td>
<td>$g$</td>
</tr>
<tr>
<td>natural enemies</td>
<td>increase in vector death rate</td>
<td>$g$</td>
</tr>
<tr>
<td>isolated planting</td>
<td>decrease in vector immigration</td>
<td>$\mu$</td>
</tr>
<tr>
<td></td>
<td>decrease in immigrant infectivity</td>
<td>$\theta$</td>
</tr>
<tr>
<td>repellent</td>
<td>increase in vector departure rate</td>
<td>$g$</td>
</tr>
<tr>
<td>yellow-screen net &amp;</td>
<td>decrease in vector immigration</td>
<td>$\mu$</td>
</tr>
<tr>
<td>attractive boundary trap crop</td>
<td>increase in vector departure rate</td>
<td>$g$</td>
</tr>
<tr>
<td>wood ash on crop foliage</td>
<td>possible decrease in vector immigration</td>
<td>$\mu$</td>
</tr>
<tr>
<td></td>
<td>possible increase in vector departure</td>
<td>$g$</td>
</tr>
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</table>
**Table 3.** The abundance, at equilibrium, of healthy plants, $H^*$, obtained using the minimum and maximum estimates of each parameter given in Table 1. Each parameter was varied individually whilst the others were held constant at the default value.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Values of $H^*$ for the minimum and maximum estimates of each parameter</th>
<th>Multiples of $H^*$ between the minimum and maximum estimates of each parameter</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Min.</td>
<td>Max.</td>
</tr>
<tr>
<td>Crop turn-over rate $\beta$</td>
<td>0.45</td>
<td>0.55</td>
</tr>
<tr>
<td>Infection rate $a$</td>
<td>1</td>
<td>0.08</td>
</tr>
<tr>
<td>Latent period $b$</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>Acquisition rate $\lambda$</td>
<td>0.5</td>
<td>0.21</td>
</tr>
<tr>
<td>Departure/loss rate $g$</td>
<td>0.29</td>
<td>0.94</td>
</tr>
<tr>
<td>Immigrant infectivity $\theta$</td>
<td>1</td>
<td>0.18</td>
</tr>
<tr>
<td>Immigration rate $\mu$</td>
<td>1</td>
<td>0.2</td>
</tr>
</tbody>
</table>
Figure legends

Fig. 1. TLCVD progress in (a) susceptible tomato varieties, Rashmi ( ■) and PSCL-4 ( ●), and (b) resistant tomato varieties, LA 1582 ( ■) and F1 Tyking ( ●), in Bangalore, summer 1993 (after Nateshan et al. 1996). Lines indicate simulated progress curves, (a) to represent a susceptible variety with \( a = 0.1 \) and \( g = 0.06 \), and (b) a resistant variety with \( a = 0.01 \) and \( g = 0.3 \). High immigration (\( \mu = 1 \)) and immigrant infectivity (\( \theta = 0.4 \)) were assumed in both cases to reflect summer season conditions. All other parameters as Table 1.

Fig. 2. Relationship between disease incidence, and the rate of virus acquisition by the vector, \( \lambda \). Examples shown at two values of immigrant infectivity \( \theta \), and two values of vector turn-over rate, \( g \); \( \theta = 0.05, 0.5, 0.05 \) and 0.5, and \( g = 0.06, 0.06, 0.2 \) and 0.2 in cases 1, 2, 3 and 4, respectively. Acquisition of the virus by vectors within the crop was important in determining disease incidence only when both vector turn-over was slow and the infectivity of immigrant vectors was low (case 1).

Fig. 3. Relationship between disease incidence and vector immigration rate. Examples shown at three values of infection rate, \( a = 0.001, 0.01 \) and 0.1, in cases 1, 2 and 3, respectively. When infection rate was high, as in susceptible tomato varieties (case 3), disease incidence reached near maximum even at a relatively low vector immigration rate. In the situation of high disease incidence frequently seen near Bangalore, vectors may be present in far greater numbers than required for disease saturation.

Fig. 4. Relationship between disease incidence and the turn-over or loss rate of vectors from the crop, \( g \). Examples shown at two values of immigration rate \( \mu \), and two values of immigrant infectivity, \( \theta \); \( \mu = 0.05, 0.05, 0.5 \) and 0.5, and \( \theta = 0.05, 0.5, 0.05 \) and 0.5 in cases 1, 2, 3 and 4, respectively. When the numbers of infective vectors were not in excess, changes in vector loss rate had an important impact on incidence, especially for changes in the range 0.02 to 0.1 day\(^{-1} \).
Figure 2. The effect of beneficial insect augmentation on the incidence of ToLCV and the *B. tabaci* population.
Figure 1

Spread of ToLCV in AVRDC tomato genotypes and yield

% ToLCV incidence

0 20 40 60 80 100 120

0 1 2 3 4 5 6 7 8 9 10 11 12

Weeks after transplanting

ATY-1
ATY-2
ATY-5
ATY-13
AVINASH-2
Local sus. var.

Yield (tonnes/ha)

0 5 10 15 20 25 30 35

ATY-1
ATY-2
ATY-5
ATY-13
AVINASH-2
Local sus. var.

Genotypes
Figure 3

Proportion of host plants diseased vs. Loss rate of vectors from the crop (day$^{-1}$)

- Line 1
- Line 2
- Line 3
- Line 4