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An age-structured population model of citrus rust mite: a fruit–mite–fungal pathogen system

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Abstract

An age-structured model of a fruit–mite–fungal pathogen system was developed to study interactions between the 'Hamlin' orange fruit, the citrus rust mite (CRM) *Phyllocoptruta oleivora*, and its fungal pathogen *Hirsutella thompsonii*. The model consists of a set of difference equations incorporating the age and stage change of CRM and its fungal pathogen. The abiotic factors included in the model were daily mean temperature and daily dew period the biotic factors were mite density (egg, protonymph, deutonymph, and adult), pathogen density (latent pathogen, and infectious pathogen), and undamaged fruit surface area. One data set for the fruit–mite–pathogen dynamics was used to estimate parameters related to density-dependent CRM egg production and pathogen transmission rate. Parameters were estimated by choosing the parameter combinations which gave the least combined error sum of squares between observed and simulated populations for CRM and pathogen. For the data set used for parameter estimation, the model captured 92.2% of variation in CRM population dynamics, and 61.9% of variation in the density curve pattern of the pathogen, over a period of 5 months. Three additional data sets were used to partially test model predictability. For one data set, the model captured 87.1% of variation in CRM population dynamics, and 94.4% of variation in the density curve pattern of the pathogen, over a period of 5 months. The model, however, did not make accurate predictions for low CRM density level-pathogen interactions. The corresponding cumulative mite days and fruit surface damage were also generated by the model for each data set. With further modification and refinement, the model could be developed into a useful tool for CRM management. © 1997 Elsevier Science B.V.

Keywords: Population model; Age structure; Stage structure; *Phyllocoptruta oleivora*; *Hirsutella thompsonii*

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Table 1
Temperature-dependent cumulative frequency distribution for CRM and pathogen development, and for CRM egg production (modified from Allen et al., 1995)

Stage	a_0	a_1	a_2	b_0	b_1	R^2
Egg[a]	−59.8296	1.2130	809.5260	−0.2184	20.2139	0.708
Nymph 1[a]	−15.8103	0.3422	229.9321	0.1160	3.9289	0.911
Nymph 2[a]	−14.6519	0.2809	229.3402	0.1770	2.3891	0.867
Adult[b]	−4.5137	256.9135	NA	−0.0893	22.4732	0.971
Lat path.[a]	−14.6519	0.2809	229.3402	0.1770	2.3891	0.867
Inf. path.[b]	−4.5137	256.9135	NA	−0.0893	22.4732	0.971
Egg prod.[b]	−4.5659	202.5222	NA	−0.1260	32.6422	0.951

[a]The cumulative distribution function is $F(t,T) = 1/1+\exp(-t-a(T)/b(T))$, where, $a(T) = a_0+a_1\,T+a_2/T$, $b(T) = b_0+b_1/T$, t = cohort age (day).
[b]Same as above except that $a(T) = a_0+a_1/T$.

1. Introduction

The citrus rust mite (CRM), *Phyllocoptruta oleivora*, is a major pest of citrus in most humid regions of the world where citrus is grown (Commonwealth Institute of Entomology, 1970; Davidson and Lyon, 1987). It infests fruit, leaves, and young twigs of all varieties of citrus. Heavy infestation on fruit causes reduced fruit grade, increased fruit drop, and retarded fruit growth (Allen, 1976, 1978, 1979, 1981; Allen et al., 1994; Yang et al., 1994). It takes only 3–5 weeks, under favorable weather conditions, for mite populations to reach high levels, causing severe damage. High mite populations often crash within just 2–3 weeks, as a result of the combined effect of weather, the fungal pathogen *Hirsutella thompsonii*, and food availability (Swirski, 1962; McCoy, 1981). Although extensive studies have been conducted on the population ecology of the CRM (Swirski, 1962; Van Brussel, 1975; Hobza and Jeppson, 1974; McCoy, 1978, 1979; Peña and Baranowski, 1990; Hall et al., 1991; Allen, 1978, 1979; Allen et al., 1995; Yang et al., 1994, 1995a,b), no simulation models are currently available for mite population prediction or study. The objective of this study was to build a simulation model of the fruit-mite-pathogen system which could make short-term (1–2 months) prediction of mite populations and the resulting fruit damage, and subsequently aid in mite control.

2. Materials and methods

2.1. The fruit–mite–pathogen system

2.1.1. The fruit

The CRM feeds on the fruit surface. The fruit as a food source and living environment for the CRM could affect its population dynamics in two ways: (1) fruit surface area growth will dilute mite population density; (2) extensive feeding by CRM will cause fruit surface damage, resulting in a decline in local food availability for CRM, which will limit or reduce mite population growth. Fruit surface area growth as a function of calendar time was developed by Yang et al. (1994). Percent fruit surface damage as a function of cumulative mite days was developed by Yang et al. (1995b).

2.1.2. The mite

There are four developmental stages for the citrus rust mite: egg, protonymph, deutonymph, and adult. The nymph and adult stages feed on fruit, leaves, and young twigs, with most mites being found on the fruit surface. In this study, the net migration of CRM between fruit and leaves and young twigs was assumed to be zero. Mite development time, fecundity, and survival rate were factors considered most important for its population growth. Mite development time and fecundity as functions of temperature were established by Allen et al. (1995) (Table 1). Mite survival was mainly related to temperature,

humidity, and the activity of the fungal pathogen, *H. thompsonii*.

2.1.3. The pathogen

The fungal pathogen, *H. thompsonii*, is one of the most important biological factors regulating mite population dynamics. It attacks the nymph and adult stages of CRM (C.W. McCoy, University of Florida, personal communication), causing regular epizootics under natural conditions in Florida (McCoy, 1981). Conidia are produced by conidiophores of the mycelia outside the infected mite bodies on the plant substrate. Once inside the mite body, the hyphae form a ramifying growth within the hemocoel, and after host death, erupt through the host cuticle onto the plant surface to repeat the process. It takes a minimum of 4 h for a spore to penetrate the mite cuticle and about 2 days from infection to sporulation at 25–30°C (McCoy, 1979; Gerson et al., 1979; Kenneth et al., 1979). Full sporulation at 27°C takes place within 12 h after death of the host (Gerson et al., 1979). Mite cadavers can sustain the fungus for a few days after which the fungus itself dies, whether or not conidia have been produced. Pathogen survival is mainly a function of humidity, solar radiation, and temperature (Fuxa, 1987). Infectivity is dependent on the presence of free water and high humidity (McCoy, 1978). In this study, the pathogen development was divided into two stages: (1) latent stage or incubation period, referring to the period from initial infection to the beginning of sporulation; (2) infectious stage, referring to the period from sporulation to the complete loss of infectivity of the mite cadaver. For convenience, each Hirsutella-infected mite (dead or alive) is considered a pathogen unit. The life time of this pathogen unit was assumed to begin with the initial infection and end with its complete loss of infectivity. For lack of detailed information, temperature was assumed to be the only factor affecting the life span of the pathogen, and only temperature and leaf wetness duration were assumed to affect pathogen infectivity. The two CRM nymph stages and the adult stage were assumed to have equal susceptibility to the fungal pathogen.

2.2. Model development

Matrix models have been widely used to study population dynamics (Lewis, 1942; Leslie, 1945, 1948; Pielou, 1977; Liu and Cohen, 1987; Caswell, 1989; Manly, 1990; Renshaw, 1991; Keen and Spain, 1992; Sandberg et al., 1992; Shimada and Tuda, 1996). The matrix method developed by Chi and Liu (1985) was used in this study, but with modifications to allow simulation under varying temperature conditions (Yang and Huang, 1991). Chi and Liu (1985) used a multiple column matrix to express the age-stage-structure of animal populations with metamorphosis. In the following sections, we use growth in terms of age change within a stage, and development in terms of stage change through metamorphosis. The process of animal population growth can be simulated through a set of difference equations. Five basic matrices are needed for the simulation: (1) age-stage-structure matrix (**N**); (2) age-stage-specific growth rate matrix (**G**); (3) age-stage-specific development rate matrix (**D**); (4) age-stage-specific fecundity matrix (**F**); and (5) age-stage-specific mortality matrix (**M**) (Chi and Liu, 1985).

2.2.1. The age-stage-structure matrix

The population structure is given in matrix **N** with k rows and m columns, where k is the number of age classes, and m is the number of stages. In the case of citrus rust mite, six columns were used:

Matrix **N**

$$\begin{pmatrix} \text{Egg} & \text{N}_1 & \text{N}_2 & \text{Adult} & \text{P}_1 & \text{P}_2 \\ n_{11} & n_{12} & n_{13} & n_{14} & n_{15} & n_{16} \\ n_{21} & n_{22} & n_{23} & n_{24} & n_{25} & n_{26} \\ \cdot & \cdot & \cdot & \cdot & \cdot & \cdot \\ n_{i1} & n_{i2} & n_{i3} & n_{i4} & n_{i5} & n_{i6} \end{pmatrix}$$

representing egg, protonymph (N_1), deutonymph (N_2), adult, latent pathogen (P_1), and infectious pathogen stage (P_2), respectively. The continuous age variable for a stage was divided into discrete age classes of the same duration (Chi and Liu, 1985). Element n_{ij} of matrix **N** is the number of

individuals in age i and stage j. After one age interval, individuals in age i and stage j may grow to age $i+1$ but are still in stage j, or may develop to the first age class of stage $j+1$, or may die without further growth or development, or may develop to the first age class of the latent pathogen stage if they are infected by the fungal pathogen.

2.2.2. Age-stage-specific growth rate, developmental rate, mortality rate, and fecundity

There are four attributes related to all individuals (Chi and Liu, 1985): age-stage-specific growth rate, developmental rate, mortality rate, and fecundity. These four attributes were described with four matrices of the same dimension:

Matrix **G**

$$\begin{pmatrix} \text{Egg} & \text{N}_1 & \text{N}_2 & \text{Adult} & \text{P}_1 & \text{P}_2 \\ g_{11} & g_{12} & g_{13} & g_{14} & g_{15} & g_{16} \\ g_{21} & g_{22} & g_{23} & g_{24} & g_{25} & g_{26} \\ . & . & . & . & . & . \\ g_{i1} & g_{i2} & g_{i3} & g_{i4} & g_{i5} & g_{i6} \end{pmatrix}$$

Matrix **D**

$$\begin{pmatrix} \text{Egg} & \text{N}_1 & \text{N}_2 & \text{Adult} & \text{P}_1 & \text{P}_2 \\ d_{11} & d_{12} & d_{13} & d_{14} & d_{15} & d_{16} \\ d_{21} & d_{22} & d_{23} & d_{24} & d_{25} & d_{26} \\ . & . & . & . & . & . \\ d_{i1} & d_{i2} & d_{i3} & d_{i4} & d_{i5} & d_{i6} \end{pmatrix}$$

Matrix **F**

$$\begin{pmatrix} \text{Egg} & \text{N}_1 & \text{N}_2 & \text{Adult} & \text{P}_1 & \text{P}_2 \\ 0 & 0 & 0 & f_{14} & 0 & 0 \\ 0 & 0 & 0 & f_{24} & 0 & 0 \\ . & . & . & . & . & . \\ 0 & 0 & 0 & f_{i4} & 0 & 0 \end{pmatrix}$$

Matrix **M**

$$\begin{pmatrix} \text{Egg} & \text{N}_1 & \text{N}_2 & \text{Adult} & \text{P}_1 & \text{P}_2 \\ m_{11} & m_{12} & m_{13} & m_{14} & m_{15} & m_{16} \\ m_{21} & m_{22} & m_{23} & m_{24} & m_{25} & m_{26} \\ . & . & . & . & . & . \\ m_{i1} & m_{i2} & m_{i3} & m_{i4} & m_{i5} & m_{i6} \end{pmatrix}$$

In the growth rate matrix **G**, element g_{ij}, the age-stage-specific growth rate, is the probability that an individual in age i and stage j will grow to age $i+1$ but still be in stage j after one age interval. In the developmental rate matrix **D**, element d_{ij}, the age-stage-specific developmental rate, is the probability that an individual in age i and stage j will develop to stage $j+1$ after one age interval. Because adult mites and the infectious pathogen will not develop to further stages (i.e. they will die), d_{ij} for them is the probability that an individual in age i and stage j will die after one age interval. In the fecundity matrix **F**, element f_{ij}, the age-stage-specific fecundity, is the number of offspring that will be produced by every individual of n_{ij} within one age interval. The CRM females have values of $f_{ij}>0$, while the other f_{ij} have a value of zero. In the mortality matrix **M**, element m_{ij}, the age-stage-specific mortality rate, is the probability that an individual in age i and stage j will die after one age interval. For the adult mite and the infectious pathogen stages, m_{ij} should be the mortality rate excluding d_{ij}.

2.2.3. Mite and pathogen population growth

When the age-stage structure of mite and pathogen populations at time, $\mathbf{N}(t)$, is known, the age-stage structure for time $t+1$, $\mathbf{N}(t+1)$, can be obtained through the operation of the following difference equations:

$$n_{11}(t+1) = \sum_{i=1}^{i\max(4)} n_{i4}(t) f_{i4}(t) \quad \text{new eggs} \tag{1a}$$

$$n_{1j}(t+1) = \sum_{i=1}^{i\max(j)} n_{i(j-1)}(t) d_{i(j-1)}(t)(1-m_{i(j-1)}(t)) e^{-\gamma N_6 P_j} \quad \text{to next stage } (1<j\le 4) \tag{1b}$$

$$n_{ij}(t+1) = n_{(i-1)j}(t)(1-d_{(i-1)j}(t))(1-m_{(i-1)j}(t)) e^{-\gamma N_6 P_j} \quad \text{in same stage } (i>1;\ 1\le j\le 4) \tag{1c}$$

$$n_{1j}(t+1) = \sum_{j=2}^{4} \sum_{i=1}^{i\max(j)} n_{ij}(t)(1-e^{-\gamma N_6 P_j}) \quad \text{newly infected } (j=5) \tag{1d}$$

$$n_{1j}(t+1) = \sum_{i=1}^{i\max(j)} n_{i(j-1)}(t)d_{i(j-1)}(t)(1-m_{i(j-1)}(t)) \quad \text{to next stage } (j=6) \tag{1e}$$

$$n_{ij}(t+1) = n_{(i-1)j}(t)(1-d_{(i-1)j}(t))(1-m_{(i-1)j}(t)) \quad \text{in same stage } (i>1; j=5,6) \tag{1f}$$

where n_{ij}, f_{ij}, d_{ij} are matrix elements; $i\max(j)$ is the number of age classes for stage j (see Eq. (3c)); $e^{-\gamma N_6 P_j}$ is the time step survival rate after pathogen attack (Nicholson and Bailey, 1935) (see Section 2.3.5 Pathogen-induced mortality for details); γ is the time step pathogen transmission rate; P_j is the proportion of infectious pathogen attacking mite stage j $(2 \le j \le 4)$; N_6 is the total infectious pathogen.

This simulation procedure assumes that the simulation time step equals the length of age class interval, that mortality occurs before growth and development but after egg production, and that pathogen-induced mortality occurs before mortality incurred by other mortality factors. Eq. (1a) represents the first age class for CRM egg stage at time $t+1$, which equals the total number of new eggs produced by all age classes of CRM females within one time step. Eq. (1b) represents the first age class for any stage $1 < j \le 4$ at time $t+1$, which equals the total number of individuals which develop from all age classes of the previous stage after one time step. Eq. (1c) represents the number of individuals which grow to age i from the previous age class i-1. Eq. (1d) represents the first age class of the latent pathogen stage, which equals the total number of nymphs and adults infected by the pathogen within a time step. Eq. (1e) represents the first age class for the infectious pathogen, which equals the total number of infected mites which have become capable of sporulation after one time step. Eq. (1f) represents the number of individuals which grow to age i from the previous age class $i-1$ for latent or infectious pathogen stage.

2.3. Model parameter specification

2.3.1. Number of age classes

The continuous age variable for each stage was divided into discrete age classes of equal duration. The number of age classes for a stage was calculated as

$$\text{number of age classes} = \frac{\text{maximum development time (day)}}{\text{age class interval (day)}}$$

where the age class interval = time step Δt(day) = 1 day. The maximum development time is the time by which all individuals of a cohort of a stage have developed to the next stage. Allen et al. (1995) used the logistic distribution function to describe the cumulative emergence of different CRM stages in relation to temperature (Table 1)

$$F(t) = \frac{1}{1+\exp\left(-\frac{t-a}{b}\right)} \tag{2}$$

where $F(t)$ is the proportion of individuals of a cohort of a stage which have developed to next stage by time t, and the temperature dependence of parameters a and b has been dropped for simplicity (Table 1). Since Eq. (2) is continuous between -∞ and $+\infty$, there is no absolute maximum development time. We chose to terminate the emergence when $F(t) = 0.9999$, incurring a very small rounding error (α) of 10^{-4}. For stage j, we have $1-\alpha = F(t_{\alpha,j})$, and

$$1-\alpha = F(t_{\alpha,j}) = \frac{1}{1+\exp\left(-\frac{t_{\alpha,j}-a}{b}\right)} \tag{3a}$$

rearranging Eq. (3a)

$$t_{\alpha,j} = a - b\ \mathrm{Ln}\frac{\alpha}{1-\alpha} \tag{3b}$$

Dividing the maximum development time($t_{\alpha,j}$) by the age class interval, which equals the time step (Δt), yields the maximum number of age classes for stage j

$$i\max(j) = \frac{t_{\alpha,j}}{\Delta t} \tag{3c}$$

When $t_{\alpha,j}/\Delta t$ is not an integer, its integer part plus one was used as the number of age classes, $i\max(j)$. The highest age class number among all stages, based on the lowest daily mean temperature during a simulation, was used to determine the number of rows for all matrices. Elements which were out of the age class range of a stage were set to zero. All individuals in the last age class, $i\max(j)$, will not develop to further age classes, but will either die or develop to the first age class of the next stage.

2.3.2. Elements for the growth rate matrix **G** *and developmental rate matrix* **D**

Element values for matrices **G** and **D** were obtained based on cumulative emergence functions (Yang and Huang, 1991; Berry and Stinner, 1992)

$$d_{ij} = \frac{F_j(i) - F_j(i-1)}{1 - F_j(i-1)} \tag{4a}$$

$$g_{ij} = 1 - d_{ij} = \frac{F_j(i)}{1 - F_j(i-1)} \tag{4b}$$

Gerson et al. (1979) reported that it took about 2–3 days from infection of carmine spider mite (*Tetranychus cinnabarinus*) by *H. thompsonii* to sporulation. This period is close to the CRM deutonymph development time, therefore the latent pathogen was assumed to have the same cumulative emergence function as the CRM deutonymph stage, and the survival of the infectious pathogen was assumed to follow an exponential decay process similar to that of the adult rust mites. The functional forms and corresponding coefficients for the mite and pathogen development are summarized in Table 1.

2.3.3. Elements for the fecundity matrix **F**

A temperature-dependent (T) cumulative distribution function of CRM egg production over time t was developed by Allen et al. (1995), which has the same form as Eq. (2) (Table 1). Eggs produced by each female per time step was calculated as

$$f_{ij} = (F(t+\Delta t) - F(t))R_{\text{total}} \tag{5a}$$

where $F(t+\Delta t)$-$F(t)$ = proportion of total eggs produced by one CRM female of age t within one time step (Δt); R_{total} = total eggs produced by one female, which was described as a polynomial function of temperature (T) by Allen et al. (1995)

$$R_{\text{total}} = 11.5909 - 3.6225T + 0.2918T^2 - 0.0057T^3 \tag{5b}$$

A female to male sex ratio of 1:1 was assumed, so we have

$$f_{ij} = 0.5\,(F(t+\Delta t) - F(t))R_{\text{total}} \tag{5c}$$

The reproductive rate represented by Eq. (5c) may not be fully realized due to intraspecific competition for food and/or space under high densities, so we introduced Eq. (5d) as the density-limiting factor

$$P_{\text{D}} = 1 - \frac{1}{1 + \exp\left(-\dfrac{N_{1-4} - a}{b}\right)} \tag{5d}$$

where $N_{1\text{-}4}$ is the total mites per cm^2 of undamaged fruit surface; a and b are parameters.

Based on Yang et al. (1995b), the proportion of undamaged fruit surface (y) as a function of accumulated mite days (x) can be described as

$$y = 1 - \frac{e^{-13.9010}\,x^{2.0860}}{100} \tag{5e}$$

where accumulated mite days at time, τ, $x(\tau)$, was calculated using the following equation

$$x(\tau) = \sum_{t=t_0}^{t=\tau} \frac{N_{2-4}(t) + N_{2-4}(t+\Delta t)}{2}\,\Delta t \tag{5f}$$

where t_0 is the beginning time of observation or simulation; $N_{2\text{-}4}(t)$ is the sum density of the protonymph, deutonymph and adult stages at time t. $N_{2\text{-}4}(t+\Delta t)$ is the sum density of the protonymph, deutonymph and adult stages at time $t+\Delta t$. The beginning time (t_0) should start when mite populations are very low for accurate prediction because Eq. (5e) was obtained using field data starting with very low mite populations (Yang et al., 1995b). The reproductive rate with the density-limiting factor is therefore

$$f_{ij} = 0.5\left(1 - \frac{1}{1 + \exp\left(-\frac{N_{1-4} - a}{b}\right)}\right)$$
$$(F(t + \Delta t) - F(t))R_{\text{total}} \quad (5g)$$

Parameters a and b were estimated through model simulation (see Section 2.6. Model parameter estimation).

2.3.4. Elements for the mortality matrix **M**

In addition to pathogen mortality discussed below (Section 2.3.5), we assumed a 18% stage mortality for CRM eggs, protonymphs, and deutonymphs, respectively, based on laboratory data under constant temperature conditions (J.C. Allen, unpublished data). Because CRM adults develop to death, no extra mortality was assumed. The latent and infectious pathogen stages were assumed to have no extra mortality except for their gradual lose of infectivity. Mortality rate was assumed to be the same for any age class of a stage. The time step mortality, m_{ij}, was obtained by solving the following equation iteratively

$$1 - m_{\text{stage}} = (1 - m_{1j})d_{1j} + (1 - m_{1j})^2(1 - d_{1j})d_{2j} + \cdots + (1 - m)^{i\max(j)}(1 - d_{1j})(1 - d_{2j}) \cdots (1 - d_{i\max(j)-1})d_{i\max(j)} \quad (6)$$

where m_{stage} is the total mortality for stage j, d_{ij} is the probability that an individual in age i and stage j will develop to the first age class of next stage. The left side of the equality represents stage survival to the next stage for a cohort of 1 individual. The first term on the right side of the equality is the survival to the next stage for the first age class; the second term is the survival for the second age class; the last term is the survival for the last age class where $d_{i\max(j)}$was set to 1 so that all individuals would develop to the next stage. Eq. (6) means that starting with one individual at age class 1, the sum of survival to the next stage for all age classes should equal the stage survival $1(1 - m_{\text{stage}})$ which is $(1 - m_{\text{stage}})$.

2.3.5. Pathogen-induced mortality

The Nicholson–Bailey model (1935) was used to describe pathogen-induced mortality, $1 - e^{-\gamma N_6 P_j}$, where N_6 is the total number of infectious pathogen units, P_j is the proportion of infectious pathogen units (mite cadavers) attacking mite stage j ($2 \le j \le 4$). Pathogen transmission rate (γ) is mainly a function of humidity and temperature (Filajdic and Sutton, 1992; Mathieu and Kushalappa, 1993; Tamm and Fluckiger, 1993; Carisse et al., 1993). If maximum pathogen transmission rate is $\gamma_{\max}$, then the realized pathogen transmission rate (γ), in relation to temperature (T) and leaf wetness duration (W), is

$$\gamma = \gamma_{\max} P_T P_W \quad (7a)$$

Based on Fig. 2 in Gerson et al. (1979), the effect of temperature on pathogen transmission rate was quantified as a modified normal density function

$$P_T = \exp\left(-\frac{(T - 25.7792)^2}{2*7.1362^2}\right) \quad (7b)$$

where P_T is the proportion of maximum pathogen transmission rate; T is the temperature (°C); $R^2 = 0.923$ ($n = 7$). Eq. (7a) assumes a symmetrical temperature effect on pathogen transmission rate toward temperature extremes.

Previous studies suggested a sigmoid increase in pathogen transmission rate with increasing leaf wetness duration (Filajdic and Sutton, 1992; Mathieu and Kushalappa, 1993; Tamm and Fluckiger, 1993; Carisse et al., 1993). The following logistic distribution function was used to describe the effect of dew period on pathogen transmission rate

$$P_W = \frac{1}{1 + \exp\left(-\frac{W - a}{b}\right)} \quad (7c)$$

where P_W is the proportion of maximum pathogen transmission rate; W is the daily leaf wetness duration (h); a and b are parameters. Based on the incidence of infection in Table 2 in Gerson et al. (1979), parameter a was estimated as 11.0 h. Parameter b was estimated through model simulation (see Section 2.6. Model parameter estimation).

Since *H. thompsonii* is a facultative pathogen and can survive without a rust mite host (Gerson et al., 1979), a minimum background density of 0.0001 pathogen unit per cm^2 fruit surface was assumed throughout the simulation for the latent and infectious stages, respectively.

Table 2
Accuracy of model prediction

Data set	Population	Period[a]	n	Error SS[b]	R^2	Purpose
Polk County 1993	CRM	167 ~ 322	19	4247.2	0.922	Parameter estimate
	Pathogen	167 ~ 322	19	2157.5	0.619	Parameter estimate
Alachua Co. 1993	CRM	145 ~ 310	29	7276.5	0.871	Prediction
	Pathogen	145 ~ 310	29	304.5	0.944	Prediction
Alachua Co. 1992	CRM	143 ~ 330	51	26704.9	0.747	Prediction
	Pathogen	143 ~ 330	51	16111.5	0.038	Prediction
Collier Co. 1991	CRM	147 ~ 235	42	5706.0	0.008	Prediction
	Pathogen	147 ~ 235	42	15361.4	0.005	Prediction

[a]Simulation period in Julian days (1 = 1 January).
[b]Sum of squares for error which is $\Sigma_{i=1}^{n}(\hat{y}_i - y_i)^2$, where $\hat{y}_i$ is the simulated value; y_i = observed value. For the pathogen, the observed pathogen density was first proportionally adjusted to the same level as simulated density for infectious pathogen (i.e. with the same maximum value), and then used to compute the sum of squares for error.

2.4. Mite and pathogen population density adjustment due to fruit growth

Since fruit surface area growth, from time t to $t+1$, will dilute mite and pathogen populations on fruit, it is necessary to adjust mite and pathogen population density after every simulation time step. A fruit surface area growth function was developed by Yang et al. (1994), which is

$$y(t) = \frac{146.3346}{1 + \exp(4.3891 - 0.0230t)} \tag{8}$$

where $y(t)$ is the fruit surface area (cm^2); t = Julian day (1 = 1 Jan.). The age-stage-structure matrix **N** was adjusted by multiplying the matrix by $y(t)/y(t+1)$after every time step simulation.

2.5. Simulation under varying temperature

For simulation under varying temperature conditions, the number of age classes for a stage may vary due to different developmental durations. Because the development, survival, and reproduction of individuals in a specific age class change with temperature, elements of the age-stage-structure matrix **N** was adjusted as follows: When the number of age classes at time $t+1$, $i\max_{t+1}$, was different from that at time t, $i\max_t$, we first redistributed the individuals at time t to new age classes at time $t+1$ using the following formula:

$$n_i(t+1) = \sum_{ii=1}^{ii=k(i)} n_{ii}(t) + R_i n_{k(i)+1}(t) - \sum_{ii=1}^{ii=k(i-1)} n_{ii}(t) - R_{i-1} n_{k(i-1)+1}(t) \tag{9}$$

where $n_i(t+1)$ is the number of individuals in new age class i ($i = 1, 2, \ldots, i\max_t$) at time $t+1$; $n_{ii}(t)$ is the number of individuals in original age class ii ($ii = 1, 2, \ldots, i\max_t$) at time t; R_i is the decimal part of $i(i\max_t/i\max_{t+1})$; $k(i)$ is the integer part of $i(i\max_t/i\max_{t+1})$; R_{i-1} is the decimal part of $(i-1)\,i\max_t/i\max_{t+1}$; $k(i-1)$ = integer part of $(i-1)\,i\max_t/i\max_{t+1}$. Eq. (9) means that the number of individuals in new age class equals the cumulative number of individuals at time t by age i ($i\max_t/i\max_{t+1}$), i.e. $\Sigma_{ii=1}^{k(i)} R_i n_{k(i)+1}(t)$ minus the cumulative number of individuals at time t by age $(i-1)\,i\max_t/i\max_{t+1}$, i.e. $\Sigma_{ii=1}^{k(i-1)} n_{ii} + R_{i-1} n_{k(i-1)+1}(t)$. Eq. (9) assumes that individuals are evenly distributed within an age class. This method proportionally expands the column age vector when $i\max_t/i\max_{t+1}$ is less than one, and compresses the column age vector when $i\max_t/i\max_{t+1}$ is larger than one. After this adjustment, we can still use the general model for simulation under varying temperature conditions. Vector expansion and contraction methods are used in signal processing, and the Matlab commands 'Interp', 'Decimate', and 'Resample' could be

directly used for age class expansion or compression (MathWorks, 1992; Stearns and David, 1996).

2.6. Model parameter estimation

Four sets of fruit-mite-pathogen dynamics data were obtained by Yang (1994): one from Polk County (1993), two from Alachua County (1992 and 1993) and one from Collier County (1991). The data set from Polk County (1993) was used to estimate model parameters. The error sum of squares (ESS) was used as a criterion (Wallach and Goffinet, 1989)

$$\mathrm{ESS} = \sum_{i=1}^{n} (\hat{y}_i - y_i)^2 \tag{10}$$

where n is the number of observations; $\hat{y}_i$ is the predicted value; and y_i is the observed value. Because the observed pathogen population was very low compared to simulated data, the observed pathogen population data were first proportionally adjusted to the same level as simulated data (i.e. with the same maximum value), and then used to compute the error sum of squares. Parameters a and b in Eq. (5d), parameter $\gamma_{\max}$ in Eq. (7a), and parameter b in Eq. (7c) were estimated through direct model simulation based on the population data from Polk County grove (1993) (Yang, 1994). The error sum of squares between the observed and simulated data was calculated for the CRM and the pathogen populations separately for each parameter combination, and the parameter combinations which gave the smallest combined error sum of squares for the CRM and the pathogen populations were used. The major environmental factors included in the simulation were daily temperature, and dew period (Yang, 1994); the major biological factors were fruit age, surface damage, and CRM and pathogen population densities. The observed CRM and pathogen densities on an early season sampling date were used as the initial population densities in the simulation.

2.7. Model Predictability

The other three independent CRM population data sets (Yang, 1994: Alachua County 1992, County 1993, Collier County, 1991) were used to test model predictability (Welch et al., 1981). Since there was no recording of the leaf wetness duration for the 1992 and 1993 data sets from Alachua County, a daily mean of 10.5 h of leaf wetness duration was assumed throughout the simulation. The observed CRM and pathogen densities on an early season sampling date were used as the initial population densities in the simulation. R^2 values were calculated as (Kvalseth, 1985)

$$1 - \frac{\sum(\text{observed} - \text{predicted})^2}{\sum(\text{observed} - \text{sample mean})^2} \quad \text{or}$$

$$1 - \frac{\text{Residual SS}}{\text{Corrected Total SS}}.$$

3. Results

3.1. Parameter estimates

Parameters a and b of the density-dependent CRM egg production function (Eq. (5d)) were estimated as $a = 130$ mites cm^{-2} and $b = 30$ mites cm^{-2}. A larger value of b would indicate a slow reduction in mite reproductive rate. The maximum pathogen transmission rate ($\gamma_{\max}$) of Eq. (7a) was estimated at 0.08 $(\text{pathogen unit})^{-1}$ day^{-1}. Parameter b of dew period-dependent pathogen transmission rate (Eq. (7c)) was estimated at 1.5 h. Statistics related to model simulation are presented in Table 2.

3.2. Observed vs. predicted

The data set from Polk County (1993) was used to estimate model parameters (see Section 2.6. Model parameter estimation). R^2 values of 0.922 and 0.619 were obtained for the CRM and pathogen populations (Table 2). The simulated peak timing for both the CRM and the pathogen population was close to the observed peak timing (Fig. 1a,b). The model slightly underestimated CRM population in the growth phase and in the declining phase. As a result, the cumulative mite days was underestimated (Fig. 1d), and so was the fruit

surface damage (Fig. 1c). The observed fruit surface damage was about 35% (Fig. 1c); the simulated fruit surface damage was about 29% (Fig. 1c).

The data set from Alachua County 1993 was an independent data set used to test model predictability. Because no leaf wetness data were available, a daily average of 10.5 h of leaf wetness was assumed during the simulation. R^2 values of 0.871 and 0.944 were obtained for the CRM and pathogen populations (Table 2). The simulated peak timing for the CRM population was about 3 days later than the observed (Fig. 2a). A second small peak was observed in the simulated results (Fig. 2a) which was not obvious in the observed data, indicating a possible missing component in the model (see Section 4). The sharp decline around Julian day 200 (Fig. 2a) was due to pesticide application to control citrus aphids (Yang, 1994). The simulated peak timing for the pathogen population was close to the observed peak timing (Fig. 2b). The observed fruit surface damage was about 25% (Fig. 2c); the simulated fruit surface damage was about 31% (Fig. 2c). The overestimation of fruit surface damage was mainly due to the second small peak (Fig. 2a,c).

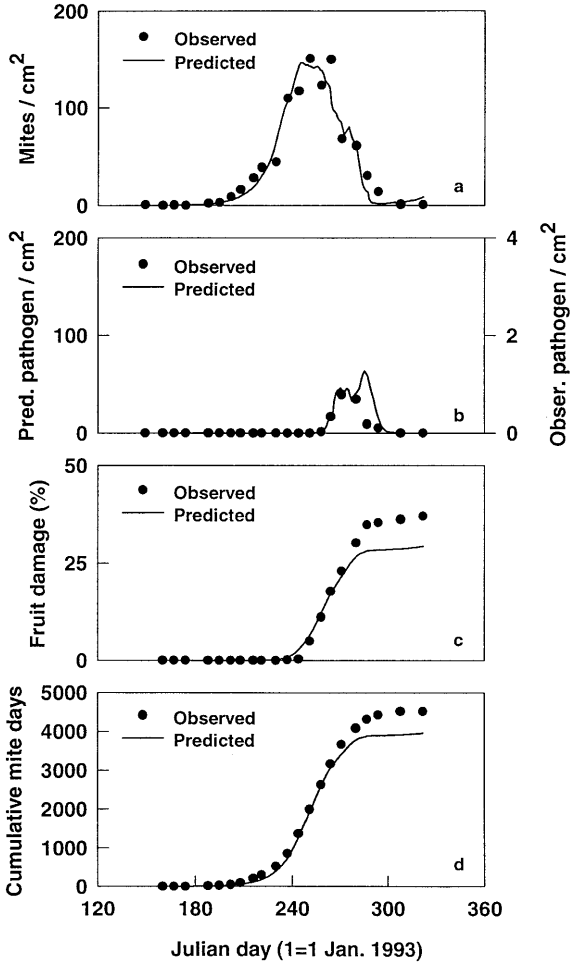

Fig. 1. Observed and predicted fruit-mite-pathogen system dynamics. (a) Mite population; (b) pathogen population; (c) fruit surface damage; (d) cumulative mite days (Lake Alfred, Polk County, Florida, 1993).

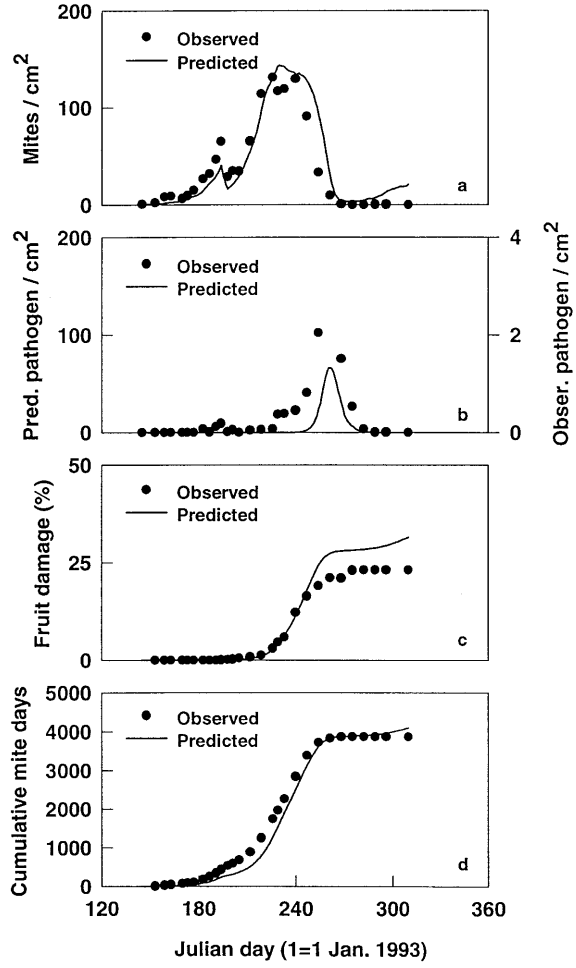

Fig. 2. Observed and predicted fruit-mite-pathogen system dynamics. (a) mite population; (b) pathogen population; (c) fruit surface damage; (d) cumulative mite days (Gainesville, Alachua County, Florida, 1993).

The data set from Alachua County 1992 was another independent data set used to test model predictability. Because no leaf wetness data were available, a daily average of 10.5 h of leaf wetness was assumed during the simulation. R^2 values of 0.747 and 0.038 were obtained for the CRM and pathogen populations (Table 2). The simulated peak timing for the CRM population was about 1 week later than observed (Fig. 3a). A second small peak was also observed in the simulated results (Fig. 3a) which was not obvious in the observed data. The simulated peak timing for the pathogen population was about 2 weeks later

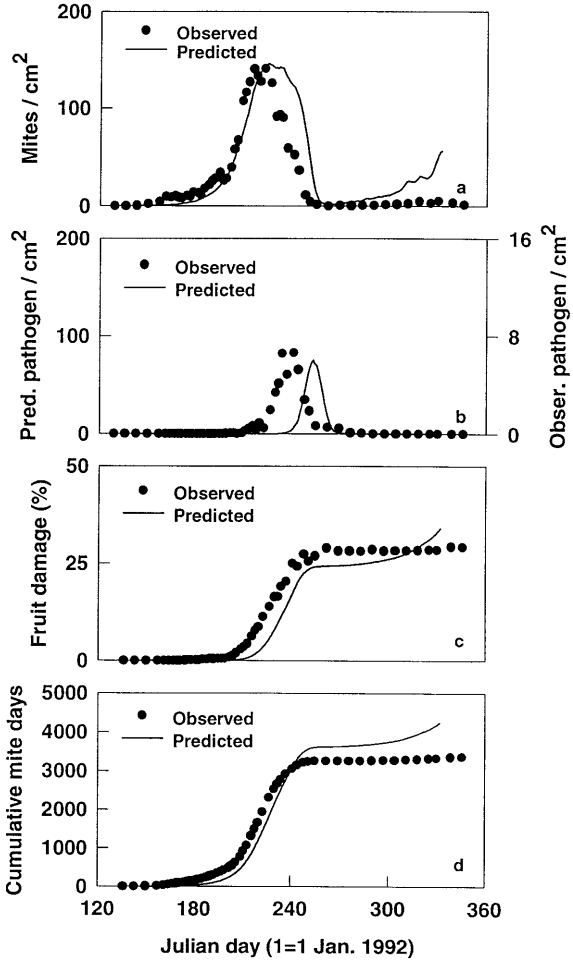

Fig. 3. Observed and predicted fruit-mite-pathogen system dynamics. (a) mite population; (b) pathogen population; (c) fruit surface damage; (d) cumulative mite days (Gainesville, Alachua County, Florida, 1992).

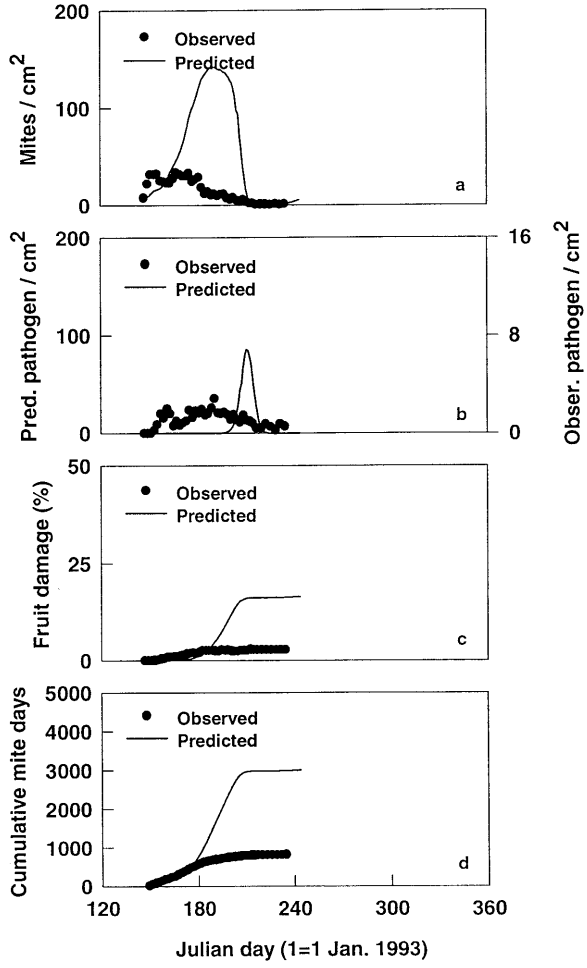

Fig. 4. Observed and predicted fruit-mite-pathogen system dynamics. (a) mite population; (b) pathogen population; (c) fruit surface damage; (d) cumulative mite days (Immokalee, Collier County, Florida, 1991).

than the observed peak timing (Fig. 3b), which resulted in a very low R^2 value. The observed fruit surface damage was about 28% (Fig. 3c); the simulated fruit surface damage was about 34% (Fig. 3c).

The data set from Collier County 1991 was another independent data set used to test model predictability. R^2 values of 0.008 and 0.005 were obtained for the CRM and pathogen populations (Table 2). The model failed to make accurate predictions in this case of low CRM density level-pathogen interactions. The fungal pathogen was very high and kept the mite population at a low

level (Fig. 4a,b), while the predicted populations were much higher than the observed (Fig. 4a). Factors unaccounted for in the model (such as solar radiation, relative humidity, and rainfall) may be partly responsible for the difference between the observed and the predicted.

4. Discussion

4.1. Need for CRM biology

The model underestimated early CRM population growth (Figs. 1–4) in each of the four cases studied, indicating that the actual development and reproduction of CRM may be higher than the data used by the model. The CRM development and fecundity data used in the simulation were obtained under constant temperature conditions (Allen et al., 1995). Although development rates under fluctuating-temperature regimes are comparable and frequently similar to rates under constant temperatures within the 'linear region' of a development-rate function (Campbell et al., 1974), there have been numerous reports suggesting that daily temperature cycles can play an important role in insect development (Hogg, 1985; Roltsch et al., 1990; Taylor and Shields, 1990; Worner, 1992). For instance, daily temperature cycles may decrease mortality or increase development rate beyond predictions based on constant temperature studies when some temperatures in the cycling regime exceed upper or lower thresholds (Beck, 1983). No information is currently available on the possible effects of fluctuating temperature on the development, reproduction, and survival of CRM.

In the simulation, a 18% non-pathogen-induced stage mortality was also assumed for the CRM eggs, protonymphs, and deutonymphs, respectively, based on laboratory observations. Under natural conditions, CRM survival may be a complicated function of environmental factors including temperature, humidity, rainfall, and wind. Quantifying the effect of these factors on CRM survival, though extremely difficult, would greatly improve model predictability.

4.2. Need for pathogen biology

As compared to the citrus rust mite, information on the biology of the fungal pathogen is very limited (McCoy, 1979; Gerson et al., 1979; Kenneth et al., 1979). During the simulation process, the development of latent pathogen was assumed to follow the same temperature-dependent curve as the CRM deutonymph stage, and the development of infectious pathogen to death was assumed to follow the same temperature-dependent curve as the CRM adult stage. Although these assumptions were partly based on laboratory observations (McCoy, 1979; Gerson et al., 1979), the actual curve of pathogen development as a function of temperature might be different from that for CRM, especially after the death of the infected mites. Furthermore, other factors, especially relative humidity, rainfall, and solar radiation, may have an important effect on the pathogen development after the death of the infected mites (Fuxa, 1987).

Very limited information is available on pathogen transmission rate. Temperature and dew period are probably the two most important factors affecting pathogen transmission rate. In this simulation, effect of temperature and dew period on pathogen transmission rate was quantified separately, their potential interaction was not considered. Furthermore, data used for such quantification were not very detailed (Kenneth et al., 1979).

4.3. Modelling pathogen-induced CRM mortality

The Nicholson–Bailey model (1935), which was used to describe pathogen-induced CRM mortality, assumes random contact between susceptible CRM stages and pathogen infectious stages (Hassell, 1978; Gutierrez, 1996). The effective area (proportion) exposed to infectious pathogen per time unit is expressed as $1 - e^{-\gamma N_6 P_j}$, and susceptible CRM in the effective area are to be infected by the pathogen (Gutierrez, 1996). It represents a type I functional response (Gutierrez, 1996), i.e. for a given pathogen density mortality increases linearly with susceptible CRM density. This approach requires a random spatial distribution of

CRM and pathogen (Onstad and Carruthers, 1990). Because citrus rust mite prefers areas of the fruit and tree where there is enough sunlight, and avoids direct sunlit and shaded parts (Van Brussel, 1975; Allen and McCoy, 1979), incorporation of space heterogeneity in CRM and infectious pathogen distribution should improve model predictability.

Many functional response models (Types I, II, III) have been proposed to study predator-prey (or host-parasitoid) interactions (Gutierrez, 1996). Because pathogen transmission (in terms of an insect-pathogen system) is mainly a passive process (Fuxa, 1987; Tanada and Kaya, 1993), a linear relationship between pathogen-induced mortality and host (CRM) density is a reasonable description. The possibility of using other functional response models (Types I, II, III) (Gutierrez, 1996) in a CRM-pathogen system deserves further study.

4.4. Food quality and space competition

In the simulation, the undamaged fruit surface was assumed to be equally suitable for CRM development, reproduction, and survival over the entire fruit growth season. Food quality for CRM might decline toward fruit maturity. This effect was not considered in our simulation model.

It has been observed that citrus rust mite prefers areas of the fruit where there is enough sunlight, but it avoids direct sunlit area, and shaded parts of the fruit surface (Yothers and Mason, 1930; Van Brussel, 1975; Allen and McCoy, 1979). With the progress of the season, increasing fruit weight would tend to bring the fruit inside the canopy, and the emergence of new flushes will further shade the fruit, resulting in a reduction in the proportion of the fruit surface suitable for mite feeding. The effect of increasing fruit surface shading over the season on CRM population growth was not included in our simulation. The small second peak for CRM (Figs. 2 and 3) generated by the model is probably due to lack of consideration for decreased food quality and increased fruit shading toward the end of the fruit growing season.

4.5. CRM migration between fruit and leaves

During the fruit growing season, CRM are found on fruit, leaves, and young twigs, though most mites are found on fruit (Yang, 1994). In our current model, net mite migration between fruit and leaves and twigs was assumed to be zero. Migration of CRM can be achieved by active crawling or by wind. At the beginning of the fruit growing season, mites on newly formed fruit will have to come from leaves. McCoy (1979) observed that mites were well-established on new flush before they were detected on new fruit. It is also possible that at extreme high densities, mites on fruit will tend to migrate to leaves or to disperse by wind. It would be more realistic to develop a coupled fruit–leaf CRM model with consideration for CRM migration or dispersal. Because food quality of leaves may be quite different from fruit quality, a different set of parameters may be needed for modelling mite dynamics on leaves.

4.6. Modelling pesticide-induced mortality

For the developed model to be used in practical CRM management, a pesticide-induced mortality model will have to be incorporated, with differential pesticide-induced mortality to mite and pathogen populations. Jones et al. (1977) developed a process-oriented model to simulate dynamic insect mortality through time after pesticide application. This process-oriented model has been used by Hardman (1989) for simulating pesticide-induced mortality for the European red mite, *Panonychus ulmi* (Kock), and could be incorporated into our system as a further development.

4.7. Practical application

Although the established model is in a preliminary form, the simulated results were close to observed data, and it can be further modified to improve accuracy for practical application. With further modifications and refinement, the model could be used to predict CRM trends and the resulting fruit surface damage, and to study the

effect of pesticides on CRM and pathogen populations. Coupled with a yield loss model from CRM damage (Allen, 1981; Allen et al., 1994), the current model could be developed into a useful CRM management tool.

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