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## Modelling fungal (*Neozygites cf. floridana*) epizootics in local populations of cassava green mites (*Mononychellus tanajoa*)

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### ABSTRACT

The fungus, *Neozygites cf. floridana* is parasitic on the cassava green mite, *Mononychellus tanajoa* (Bondar) (Acari: Tetranychidae) in South America and may be considered for classical biological control of cassava green mites in Africa, where cassava is an important subsistence crop, cassava green mites are an imported pest and specific natural enemies are lacking. Spider mites generally have a viscous structure of local populations, a trait that would normally hamper the spread of a fungus that is transmitted by the contact of susceptible hosts with the halo of capilliconidia surrounding an infectious host. However, if infected mites search and settle to produce capilliconidia on sites where they are surrounded by susceptible mites before becoming infectious, then the conditions for maximal transmission in a viscous host population are met. Because the ratio between spider mites and the leaf area they occupy is constant, parasite-induced host searching behaviour leads to a constant per capita transmission rate. Hence, the transmission rate only depends on the number of infectious hosts. These assumptions on parasite-induced host search and constant host density lead to a simple, analytically tractable model that can be used to estimate the maximal capacity of the fungus to decimate local populations of the cassava green mite. By estimating the parameters of this model (host density, per capita transmission rate and duration of infected and infectious state) it was shown that the fungal pathogen can reduce the population growth of *M. tanajoa*, but cannot drive local mite populations to extinction. Only when the initial ratio of infectious to susceptible mites exceeds unity or the effective growth rate of the mite population is sufficiently reduced by other factors than the fungus (e.g. lower food quality of the host plant, dislodgement and death by rain and wind and predation), will the fungal pathogen be capable of decimating the cassava green mite population. Under realistic field conditions, where all of these growth-reducing factors are likely to operate, there may well be room for effective control by the parasitic fungus.

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**Key words:** Acari, Tetranychidae, spider mites, *Mononychellus*, cassava, *Manihot*, parasitic fungus, *Neozygites*, epizootics, transmission, parasite-induced behaviour, parasite–host interaction, microparasite–host model.

## INTRODUCTION

Cassava, *Manihot esculenta* Crantz, is an important subsistence crop for more than 200 million people in tropical and subtropical Africa (Herren and Bennett, 1984). In the early 1970s, the cassava green mite, *Mononychellus tanajoa* (Bondar) (Acari: Tetranychidae), was accidentally introduced into Africa from South America (Lyon, 1974; Z.M. Nyiira, unpublished) and has since been the target of a classical biological control campaign (Girling *et al.*, 1977; Yaninek *et al.*, 1993). In addition to predatory arthropods, the fungal pathogen *Neozygites* sp. causes widespread mortality among green mites in cassava fields in South America (Agudelo-Silva, 1986; Delalibera *et al.*, 1992). A related fungus has been recorded in Kenya (Bartowski *et al.*, 1988) and Benin (Yaninek *et al.*, 1996), but this strain appears not to be capable of suppressing *M. tanajoa* to non-damage levels. Hence, it may be worthwhile searching for more effective strains originating from the area of origin of the pest.

Epizootics in South America become manifest in the rainy season (Alvarez-Afanador, 1990) with considerable regional variation (I. Delalibera, personal communication). Such variation may be explained by differences in host-related factors, such as population size and genetic variability and to pathogen-related factors such as host specificity, virulence and the response to climatic conditions (Hajek and Legers, 1994). Generally, there is agreement on the relative importance of climatic conditions that allow the pathogen to form infective stages, i.e. the capilliconidia (Benz, 1987). These climatic conditions include temperature, humidity and photoperiod. In the case of a strain of *Neozygites* cf. *floridana* Fisher, originating from Bahia in Brazil, the effect of these conditions on the time from host infection to the formation of capilliconidia was studied (Oduor, 1995; Oduor *et al.*, 1995a,b, 1996a,b). It may now be questioned to what extent the development of the epizootics can be attributed to climatic conditions alone.

An answer to this question is unlikely to be obtained when the spatial scale under consideration is too large, since there may well be spatial variation in the climatic conditions and in the genetic heterogeneity among the hosts and pathogens (Ebbert, 1994). It is therefore preferable to choose a small spatial scale in order to limit the genetic variability to a population and to increase the probability of detecting strains with properties deviating from the ones we studied in the laboratory. It is probably wise to select a spatial scale at which the host population is deterministically coupled to the dynamics of the fungal pathogens. As spider mites generally exhibit aggregated distributions with deterministic population growth within and low connectivity between popula-

tions inhabiting different infestation foci (Sabelis, 1991), it seems appropriate to study host–pathogen dynamics at the spatial scale of a single infestation focus, which may comprise a cluster of mite-infested leaves or plants. This is our operational definition of what will be further referred to as a local population.

In this paper we propose a modified version of the Kermack–McKendrick model for microparasite–host interactions and we provide estimates of the model parameters and functional relationships with climatic factors (temperature and humidity) as the only independent driving variables. This model is developed for two purposes: (1) to assess the climatic conditions for epizootics in local cassava green mite populations and (2) to determine the *maximal* capacity of the fungus to drive these local host populations to extinction. The latter purpose implies that the model is based on idealized assumptions regarding the transmission process, whereas all the growth and reproduction parameters represent the constraints set by the strains of fungus and host investigated. The predictions from this model can then serve as a template to evaluate the importance of other factors not included in the biological studies carried out so far, such as genetic variability between strains.

#### MODEL DESCRIPTION

As in the classic models for microparasitic infections, we classify hosts with respect to their state of infection, thereby ignoring the per host pathogen load (Kermack and McKendrick, 1927). We also ignore the age structure of the host population and assume identical host individuals within each host class. Susceptible hosts ( $S$ ), i.e. non-infected mites, may become infected upon walking through a ‘halo’ of infective capilliconidia surrounding a mummified host. These infected hosts are not immediately infectious, but take some time before they are mummified and give rise to a halo of capilliconidia. This class of infected hosts is represented by  $I$ , whereas infectious hosts are represented by  $C$  (contagious hosts). It should be emphasized that the state variables  $S$ ,  $I$  and  $C$  represent the numbers of (susceptible, infected and infectious, respectively) host individuals, not their densities.

In the absence of the pathogen, the host population increases exponentially with a constant  $r$ , the per capita net rate of population increase. In the presence of infectious hosts, the susceptible host population multiplies at a rate proportional to the number of susceptible hosts only ( $S$ ), because infected hosts do not reproduce. The overall number of healthy susceptible hosts decreases due to contacts with the halos of capilliconidia surrounding infectious dead hosts. This would normally imply that the infectious host transmission rate decreases with the overall decrease in the number of healthy hosts. However, for spider mites this may not hold for three reasons. First, their local populations on a plant are certainly not homogeneously mixed. What determines the rate of contact of an infectious host with susceptible hosts is not the overall number of

susceptible hosts within an infested area, but the density of susceptible hosts in the direct neighbourhood of the infectious host. Second, before dying and becoming infectious infected mites are still mobile and, if the parasite has control over its host's behaviour, then one would expect natural selection to favour parasites 'steering' their host to sites with high densities of susceptible hosts within the plant area colonized by the local population. Indeed, this may lead to the maximization of the per capita transmission rates. Third, spider mites create a rather constant number of offspring per unit of infested leaf area (the so-called characteristic prey density; Sabelis, 1991). Females deposit their offspring next to their feeding site and the juveniles stay very close to where they are born, a generalization confirmed for *M. tanajoa* (Yaninek, 1988). When mature and inseminated young females avoid settling on leaf areas already occupied with the web, faeces and eggs of other females. Rather, they move to uninfested leaf areas that are quite nearby. Once settled, they spend most of their time in feeding, move very little and, hence, do not mix randomly with the population at large. Contacts are therefore predominantly between individuals from nearby broods and the per capita contact rate is therefore bound to be constrained to a fixed value set by the characteristic prey density.

For these three reasons the per capita transmission rates may well be constant for a large part of an interaction cycle. For the sake of simplicity, we will assume that this holds throughout the period of local pathogen–host interaction. Under this assumption, transmission of the pathogen occurs at a rate proportional only to the number of infectious hosts. The proportionality constant then equals the 'neighbourhood' mean per capita contact rate of an infectious host with a mean neighbouring susceptible host ( $\beta$ ), multiplied by the characteristic local host density ( $s$ ).

This notion of the transmission process differs essentially from the classic Kermack and McKendrick (1927) version of the transmission rate. They modelled contact rates based on the law of mass action assuming homogeneous mixing within the local host population. Here the transmission rate is proportional to the product of the number of susceptible hosts and the number of infectious hosts ( $S C$ ), divided by the area occupied by the population ( $A$ ):

$$\text{Transmission rate} = \frac{\beta}{A} SC$$

This form of the transmission term has been applied to the *Neozygites–Tetranychus* model of Brown and Hasibuan (1995), which therefore assumes that the epidemic takes place in a homogeneously mixed population in an environment of fixed size.

When the area occupied by the population increases with population size (as in spider mites), the total host density is constant within this area  $A$ , i.e.

$s = N/A = (S + I + C)/A$ , = constant and we obtain (Diekmann *et al.*, 1994):

$$\text{Transmission rate} = \beta s \frac{SC}{N}$$

This version has been used by de Jong *et al.* (1995) to describe the transmission of a virus in animal populations of different sizes, but kept at a constant density.

In the case of small contact neighbourhoods and optimal parasite-induced searching behaviour of infected hosts for susceptible hosts the ratio  $S_{\text{local}}/N_{\text{local}}$  approximates unity (and  $I_{\text{local}} + C_{\text{local}}$  approximates zero), which reduces the formula to the simple linear form used in our model:

$$\text{Transmission rate} = \beta s C$$

Note that this form of the transmission rate is identical to the linear predation term for an optimally foraging predator in an area with constant prey density, as proposed by Sabelis (1992) and Janssen and Sabelis (1992).

Once infected, the hosts do not recover, but go through various stages of disease development, which for the sake of simplicity are lumped into one class. These stages include (1) within-host multiplication of the pathogen, (2) the production of primary conidia (sporulation) and (3) the germination of primary conidia into capilliconidia, the infectious stage. Thus, the class of infected hosts includes living infected mites which have just acquired the infection, those in which the fungal hyphal bodies are actively multiplying, dead mites from which the fungus has not started sporulating and those from which the fungus has sporulated but the liberated primary conidia have not yet produced the infective capilliconidia. The time it takes from infection to the formation of capilliconidia is referred to as the transition time, which is a function of climatic factors; its inverse is the per capita rate of transition to the infectious stage ( $\alpha$ ) in the differential equations for  $I$  and  $C$ . The transition period represents no more than a delay in the response of the pathogen population, but at unfavourable temperatures and humidities these delays can be of considerable length. We assume that the quality of the capilliconidia is not affected by the length of the preceding transition period and that infected hosts do not disappear other than by their transition to infectious hosts, since they are assumed not to disperse away from the local population.

Most of the primary conidia end up in a halo-like circle around the infected, dead host and most of the capilliconidia germinating from them stay put in the halo. The remainder end up in the air and may lead to long-distance dispersal, thereby incurring high risks of mortality. As we are focusing on local population dynamics the airborne conidia are ignored. Thus, one may consider the infectious units to be the halos rather than the individual conidia.

Halos lose their infectious capacity by ageing of the capilliconidia and through depletion of the capilliconidia taken up by the passing (healthy and infected) hosts or by external forces, such as rain and wind. With respect to the first cause little biological information is available, but the per capita loss of

halo infectivity is likely to be a constant,  $\mu$ . With respect to the second cause of halo infectivity loss it seems also reasonable to assume a constant per capita rate of loss, since the local host density is constant. However, wind and rain events are unpredictable and are ignored for reasons of simplicity.

This completes the description of the basic model (Appendix 1, model 1). For the case of exponentially distributed transition periods, we may now write the following three-compartment system of differential equations for the dynamical changes of the state variables of the hosts ( $S$ ,  $I$  and  $C$ ):

$$\frac{dS}{dt} = rS - \beta sC$$

$$\frac{dI}{dt} = \beta sC - \alpha I$$

$$\frac{dC}{dt} = \alpha I - \mu C$$

As the state variables are upper case characters, the parameters are shown as lower case characters. The host-related parameters are given by italic characters ( $r$  and  $s$ ), whereas the primarily pathogen-related parameters are given by Greek symbols ( $\alpha$ ,  $\beta$  and  $\mu$ ).

Alternatively, we can model the transition as a period of fixed duration, thereby obtaining the following two-compartment ( $S$  and  $C$ ) version (Appendix, model 2).

$$\frac{dS}{dt} = rS_t - \beta sC_t$$

$$\frac{dC}{dt} = \beta sC_{t-\tau} - \mu C_t$$

where the fixed time spent in transition from the infected state to the start of the infectious state, i.e.  $\tau$ , equals the inverse of  $\alpha$  ( $\tau = 1/\alpha$ ).

The derivation of the dynamical properties of both the two- and three-compartment models is presented in Appendix 1. It is shown that the equilibria are unstable in all of these versions. Thus, one may observe either of the following cases: (1) the pathogen population becomes extinct first and the host population increases exponentially, (2) the pathogen population increases, but does not drive the host population to extinction and (3) the pathogen population increases and the host population ultimately becomes extinct (followed by the pathogen population).

#### PARAMETER ESTIMATION

*Host-related parameters* ( $r$ ,  $s$ ): The intrinsic rate of population increase, usually referred to as the Malthusian parameter  $r_m$ , can be used as an estimate of the

host population growth rate  $r$  in the pathogen–host model. For the cassava green mite estimates of  $r_m$  are provided by Yaninek *et al.* (1989) for various temperatures in the range of 20–34°C. The shape of the relation within this temperature range is parabolic with a maximum of 0.281 females per female per day at approximately 31°C. How the relative humidity influences this relationship between  $r_m$  and the temperature is not known.

In principle, the characteristic host density,  $s$ , should represent the density of mites that can effectively contract the disease by passing a halo of capilliconidia surrounding an infectious host. Hence, it is important to note that not all developmental stages of the spider mites are susceptible to infection. In particular, the eggs are always born uninfected (no vertical transmission) (Alvarez-Afanador, 1990) and cannot become infected. Hence, the characteristic density of susceptible spider mites should be multiplied by the fraction of eggs to obtain the effective number of susceptibles (i.e. multiplication by 0.5, representing the share of eggs under conditions of a stable age distribution; Yaninek *et al.*, 1989). Here, the eggs were excluded from the counts of mite numbers to assess the density. The characteristic host density was assessed by counting all the mobile and moulting stages in a sample of 32 cassava leaves and by dividing this mite number by the total infested leaf area in the leaf sample. The infested leaf area was demarcated as the area within which leaf damage symptoms, moulting skins, faeces, silken threads and feeding mites can be observed. In this way we found a mean of 4.4 mites per cm<sup>2</sup> infested leaf area (SE = 0.31 and  $n = 32$ ). The densities may well be higher in the end phase of exploitation where the leaves are fully colonized; Elliot (personal communication, February 1997) found similar or lower densities in six samples, but 2–3-fold higher densities in another six samples and in yet one other case even 3.6 times higher densities. The mean characteristic host densities in this near-overexploitation phase were approximately seven mites per cm<sup>2</sup>.

It should be noted that the term tracking the population growth of susceptibles concerns the female part of the population only, because  $r_m$  has the dimension females per female per day (males do not reproduce; they only serve to guarantee all females are mated). However, the transmission term in the equation tracking the susceptibles ( $S$ ) concerns both sexes, since the characteristic host density( $s$ ) was based on counts of all the active stages. Hence, this term should be multiplied by the probability of the disease being transmitted to a female, whereas this correction should be omitted from the transmission term in the equation tracking the number of infecteds ( $I$ ), since both sexes are capable of transmitting the disease. However, the sex ratio of *M. tanajoa* is strongly female biased so that such a correction can be ignored for the sake of simplicity; the overall proportion of daughters in the offspring when mature (secondary sex ratio) is approximately 70% according to Gutierrez *et al.* (1988), whereas the sex ratio in the field (tertiary sex ratio) usually exhibits an even stronger female bias due to the sex-differential mortality (Sabelis, 1991).



An important assumption underlying the estimate of  $r_m$  is the existence of a stable age distribution in the host population. It may be questioned, however, whether the stable age distribution is maintained under pathogen pressure and, if not, whether it may still serve as a reasonable approximation. Including the age structure would render the model much more complex and the analysis of age-structured extensions is left as an open problem for future research.

*Per capita rate of transmission ( $\beta$ ):* An estimate of  $\beta$  was obtained from the relation between  $I$  and  $S$ , 6 days after introducing one infectious host to a susceptible host population of size  $S$  on a single leaf with an area of approximately  $46 \text{ cm}^2$  at  $25 \pm 2^\circ\text{C}$ . The susceptible host population consisted of mobile stages only, i.e. the larvae, protonymphs, deutonymphs and adults (males and females) (in equal proportions). They were introduced on the upper side of one leaf attached to an intact potted cassava plant placed on moist cotton wool, whereas a single mummy was placed on a drop of water on the underside of that leaf. By covering the plants with large dark jars, the humidity was increased and the fungus readily sporulated, giving rise to a halo of capilliconidia around the mummy. To allow favourable conditions for penetration into the host, the jar-covered plants were alternately removed and placed back at 12 h intervals, thereby changing the humidity and light conditions. After 6 days the mites were mounted in lactophenol-cotton blue and observed under a compound microscope for the presence of the fungus. During the 6 days none of the newly infected hosts sporulated. Thus, the newly infected mites all arose from contact with the single mummy introduced at the start. Since not all introduced susceptibles were retrieved at the end of the experiment, the number of infected mites was estimated from the product of the initial number of susceptibles and the fraction of infecteds among the retrieved mites. The results are presented in Table 1, as well as estimates of  $\beta$  for each initial host density. Although the estimate of  $\beta$  at the lowest density deviates ( $> 50\%$  lower), there is no obvious trend in the remainder of the estimates of  $\beta$ , i.e. at initial densities of 40, 80 and

TABLE 1

Numbers of infected cassava green mites 6 days after placing a single mummy on a leaf ( $46 \text{ cm}^2$ ) with different initial numbers of susceptible mites

Number of susceptibles	Mean number infected	Standard error	Estimate of $\beta$ ( $\text{cm}^2 \text{ day}^{-1}$ )
20	1.7a	0.8	0.014
40	8.3b	0.9	0.035
80	14.9b	1.6	0.031
120	31.4c	3.2	0.043

The mean and standard error of the mean are presented based on six replicates for each initial number of susceptibles. The percentage of infected mites is significantly affected by the initial number of susceptible mites (ANOVA;  $F = 47.57$ ,  $df = 3, 20$  and  $p < 0.05$ ). Values followed by the same letter are not significantly different at the 5% significance level (Student–Newman–Keuls test).

120 per arena. Because the characteristic density of cassava green mites would correspond to approximately 200 per arena and is thus higher than the range investigated, it seems reasonable to ignore the deviation at the lowest density and – in absence of a trend in the remainder of the data – use linear regression of the numbers of infected mites ( $I$ ) on the initial numbers of susceptibles ( $S$ ). The slope of the regression line is an estimate of  $\beta$  over the exposure period. Forcing the regression line through zero yielded

$$I = 0.234 S \quad (R^2 = 0.846)$$

Dividing the slope by the exposure period (6 days) gives  $\beta = 0.039 \text{ cm}^2 \text{ day}^{-1}$ .

*Per capita rate of transition to the halo stage,  $\alpha$* : The rate at which infected mites become infectious ( $\alpha$ ) is estimated as the inverse of the sum of the time spent in the three host infection phases together: (1) incubation (the time from host contact with the capilliconidia to host death), (2) sporulation (the time it takes for the primary conidia to be liberated from the mummies) and (3) germination (the time to formation of the capilliconidia from the liberated primary conidia). Each of the component time periods can be influenced by climatic conditions (Oduor, 1995; Oduor *et al.*, 1995a,b, 1996a,b).

The temperature but not humidity influenced the incubation period of *N. floridana* in adult female cassava green mites (Oduor *et al.*, 1995a,b). Regression of the mean time to 50% death of the infected mites ( $LT_{50}$  in days) against temperature ( $T$  in the range 18–33°C) yields:

$$LT_{50} = 10.319 - 5.189 \log(T) \quad (R^2 = 0.891)$$

The incubation period depends additionally on the developmental stage of the host ( $D$ ) (Oduor *et al.*, 1997). These experiments were carried out at one temperature only, i.e. 28°C. To extrapolate the effect of temperature measured for the adult female hosts to the other developmental stages the following relation was used:

$$\hat{\tau}_i(D,T) = \hat{\tau}_i(D, T=28) - a \log(28/T) \text{ with } a = -5.189$$

where  $\tau_i(D,T)$  is the incubation time at temperature  $T$  and stage  $D$ ,  $\tau_i(D,T=28)$  is the incubation time at 28°C,  $a$  is the regression coefficient of log temperature (estimated above exclusively for adult females at 28°C) and  $T$  is temperature as before.

The incubation time can now be estimated as a sum of all the stage-specific incubation times weighted for the fraction of each stage present under conditions of a stable age distribution ( $f_D$ ):

$$\tau_i(T) = \sum_{\text{over } D} \tau_i(D,T) f_D$$

The values of  $f_D$  (0.48, 0.28, 0.2 and 0.04 for the larvae, the protonymphs, deutonymphs and adults, respectively) were obtained from Yaninek *et al.* (1989).

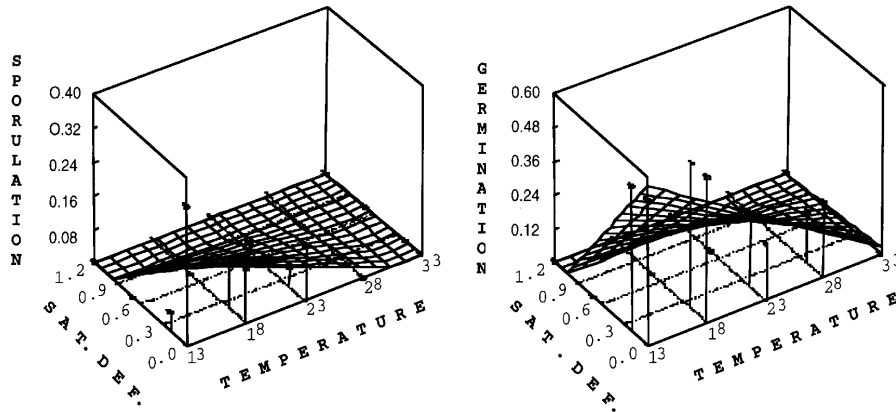


Fig. 1. Observed rates and fitted response surfaces for the rate ( $\text{h}^{-1}$ ) of initiation of sporulation (top) and germination (bottom) of primary conidia of *N. floridana* at different constant temperatures ( $^{\circ}\text{C}$ ) and saturation deficits (mmHg). The response surfaces were derived from  $\tau_s$  and  $\tau_g$ , respectively (see the text).

The time to sporulation ( $\tau_s$ ) depends on the temperature and humidity, as shown by Oduor *et al.* (1996a). This was taken as the time to 50% sporulation estimated by regressing the proportion of non-sporulated mummies (maintained at different temperatures (13–33 $^{\circ}\text{C}$ ) and saturation deficits (0–1.2 mmHg)) on the exposure time:

$$\hat{\tau}_s(T, SD) = 0.334 - 0.011T - 0.341SD + 0.011T SD \quad (R^2 = 0.654)$$

where again  $T$  is the temperature and  $SD$  is the saturation deficit. Figure 1 shows the actual data and the fitted  $T, SD$  plane.

The time to the formation of capilliconidia from primary conidia ( $\tau_g$ ) depends on the temperature and humidity, as shown by Oduor *et al.* (1996b). This was taken as the time to 50% germination estimated by regressing the proportion of non-germinated capilliconidia (in halos maintained at different temperatures (13–33 $^{\circ}\text{C}$ ) and saturation deficits (0–1.2 mmHg)) on the exposure time:

$$\hat{\tau}_g(T, SD) = 0.917 - 0.027T - 0.883SD + 0.026T SD \quad (R^2 = 0.824)$$

where again  $T$  is the temperature and  $SD$  is the saturation deficit. Figure 1 shows the actual data and the fitted  $T, SD$  plane.

The value of  $\alpha$  is now obtained as the inverse of the sum of the incubation, sporulation and germination times:

$$\hat{\alpha}(T, SD) = 1/\hat{\tau}(T, SD) \quad \text{where } \hat{\tau}(T, SD) = \hat{\tau}_i(T) + \hat{\tau}_s(T, SD) + \hat{\tau}_g(T, SD)$$

*Per capita rate of halo loss* ( $\mu$ ): The loss of halos may be caused by physical factors, such as wind and rain, by ageing of the capilliconidia and by depletion of capilliconidia due to attachment to passing mites. It is difficult to estimate the effect of the physical factors and we know very little about the effect of ageing.

Instead we concentrated on the effect of depletion (thereby implicitly assuming that this is a fast process relative to ageing and weather effects). This depletion effect can be estimated from the relationship between the contact rate with a given halo and the number of capilliconidia that become attached to the passing mites. Since the contact rates are known from the estimation of  $\beta_s$ , it suffices to assess the number of capilliconidia attaching to susceptible mites consecutively passing a given halo. Hence, an experiment was carried out in which healthy mites were made to walk through a halo area and the number of capilliconidia attached after each passage was assessed.

To do so, mummies were individually placed on glass coverslips ( $1.8 \times 1.8$  cm) and held at 100% RH, 18°C in the dark for 24h on moist cotton wool in 14 cm diameter covered glass Petri dishes. A coverslip, each with a mummy surrounded by a halo of capilliconidia, was then placed on moist cotton wool in another Petri dish at room conditions (light, approximately 24°C and  $50 \pm 5\%$  RH). An adult female cassava green mite was placed at one end of the halo and allowed to walk to the other end (hereafter termed a 'run').

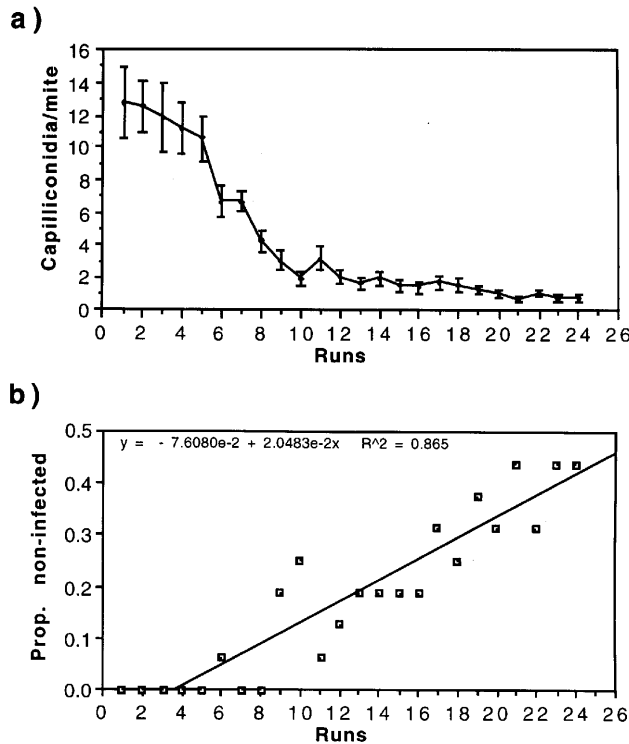


Fig. 2. (a) Mean number of capilliconidia ( $\pm$ SE bars) which attached to mites and (b) the proportion of non-infected mites at each run when successive adult female *M. tanajoa* were allowed to walk through a halo of capilliconidia of *N. floridana*.

Subsequently, the adult female was mounted in a drop of lactophenol-aniline blue and examined under a compound microscope for the number of attached capilliconidia. This procedure was repeated using 24 mites on each of 16 different halos. The mean number of capilliconidia attached to the mites following consecutive runs through the same halo is shown in Fig. 2a and the proportion of mites that remained without any conidia in Fig. 2b. The total number of capilliconidia picked by 24 mites from each halo varied from 29 to 164 (mean = 101.81 conidia). The number of conidia attached to mites decreased in subsequent runs in a more or less exponential fashion (Fig. 2a). Simultaneously, the proportion of mites that remained uninfected had increased to 45% by the last (twenty-fourth) run in the experiment. The 50% point was estimated to occur at the twenty-eighth run (from the linear regression shown in Fig. 2b).

To calculate the per capita rate of loss of halo infectivity it is necessary to estimate the time until 50% of the mites passing a halo remain uninfected. This is because the infected host will die with certainty, irrespective of the number capilliconidia attached (Oduor *et al.*, 1996). Since the per capita contact rate in the infested leaf area equals  $\beta s = 0.172 \text{ day}^{-1}$ , 28 contacts would be completed in approximately 163 days. The rate of halo loss,  $\mu$ , can then be estimated as  $\ln(2)/163 = 0.004 \text{ day}^{-1}$ . Clearly, it takes a long time (more than 5 months) for a halo to be depleted (under ideal conditions). Thus, the estimation of the survival time of a halo under a constant rate of depletion should be considered as an upper limit. It may well be that the time scale imposed by physical factors (wind and rain) is much shorter.

There was great variability in the number of capilliconidia attached to the mites passing the halo. The number of capilliconidia ranged from zero to 39 with a mean of 4.24 conidia per mite. However, what really matters to the survival of the host mite is whether they are infected or not; the mites will die anyway from the disease independent of the precise value of the conidial load (Oduor *et al.*, 1997). The most important information from these experiments is the high probability of contracting the disease upon passing a halo. Averaged over all the experiments, the probability of contracting an infection ( $p_{mh}$ ) was 0.815 per run, i.e. 18.5% escaped the disease per halo contact.

#### MODEL PREDICTIONS

##### *Conditions for epizootics ( $R_o$ )*

As follows from Appendix 1, the epidemic threshold,  $R_o$  (Anderson and May, 1981, 1991), is given by

$$R_o = \frac{\beta s}{\mu} > 1$$

provided that the latency period  $\tau(T,SD)$  is not too large.

Substituting the estimated values of the parameters,  $R_o$  equals 40.5. This leads

to the important conclusion that the conditions for an epizootic are always fulfilled, provided that the climatic conditions allow infected mites to become infectious.

However, this prediction depends critically on the accuracy of the parameter estimates. Most notably, our estimate of  $\mu$  seems rather cumbersome, as it represents a lower limit resulting from mites picking up capilliconidia from the halo. Clearly, the real value of  $\mu$  is probably higher due to ageing and the effect of rain and wind on halo loss and, thus, in particular in the rainy season when the temperature and humidity conditions are more likely to favour the active development of the fungus. For  $\mu$  to reach a value that brings the epizootic threshold,  $R_0$ , below unity, it should exceed 0.172; in other words the mean lifetime of a halo should be reduced to values below 5.8 days. As this requires a very strong influence of wind and rain on the lifetime of the halos, we conclude that the conditions for epizootics will usually be fulfilled provided the climatic conditions for sporulation (temperature and humidity) are suitable.

#### *Predictions of population dynamics*

To assess the capacity of *N. floridana* to drive local populations of *M. tanajoa* to extinction it is instructive to use a similar model of local predator-prey dynamics which was analysed by Sabelis (1992) and Janssen and Sabelis (1992). Adapted to the parameter setting used in this paper their model has the following form:

$$\frac{dS}{dt} = rS - \beta sC$$

$$\frac{dC}{dt} = r_f C$$

This model follows from the one discussed earlier when dropping the equation explicitly modelling the transition from infected to infectious host ( $\tau$ ). Instead  $\tau$  is now implicit in  $r_f$ , the effective rate of population growth of the fungal pathogen (expressed in halos per halo per day), together with the parameters determining the contact rate ( $\beta s$ ,  $\mu$ ). For the extreme scenario, where the infected mites become infectious after  $\tau$  and then infect new hosts at a rate  $\beta s$  for an infinitely long time (the so-called Methuselah schedule; see Sabelis and Janssen 1994), the relation between these parameters is as follows:

$$r_f = \beta s e^{-r_f \tau}$$

As this equation contains  $r_f$  in and outside the exponent of  $e$ , the value of  $r_f$  can only be obtained by iteration. Using  $\beta s = 0.172$  and  $\tau = 4.1$  days, representing the shortest possible time in transition from infection to infective state, the estimate of  $r_f$  is slightly less than 0.11 halos per day.

The above system of differential equations can be solved analytically for the

trajectories of  $S$  and  $C$  (Janssen and Sabelis, 1992). Starting from  $S_0 (= S_{t=0})$  susceptibles and  $C_0 = 1$  mummy or halo, the solution yields:

$$S_t = \{S_0 + S_{\text{crit}}\}e^{rt} - S_{\text{crit}}e^{\mu t}$$

$$S_{\text{crit}} = \frac{\beta s}{r_f - r}$$

$$C_t = e^{\mu t}$$

The time from the introduction of one halo ( $C_0$ ) to the extinction of the susceptible host population ( $t_{\text{ext}}$ , i.e. when  $S_t = 0$ ) is

$$t_{\text{ext}} = \frac{\ln(1 + S_{\text{crit}}S_0)}{r_f - r}$$

As the intrinsic rate of fungal population growth, i.e.  $r_f = 0.11$ , is smaller than the intrinsic rate of mite population growth, i.e.  $r = 0.281$ ,  $t_{\text{ext}}$  may become infinitely long for certain values of initial host population size:

$$\frac{S_0}{C_0} > -S_{\text{crit}} = \frac{\beta s}{r - r_f} \approx 1$$

In other words, the fungal pathogen will not be capable of driving the host population to extinction, unless the initial ratio of susceptible hosts to halos is less than unity, i.e. more than one halo for every susceptible host. Practically speaking, this means that the host population in the field will not be controlled by the fungal pathogen alone. Thus, under conditions where the cassava green mites may reach their maximal rate of population growth (high  $T$ , such as  $28^\circ\text{C}$  and RH approximately  $< 70\%$ ) the fungal pathogen may not drive local cassava green mite populations to extinction. Instead the cassava green mites will increase exponentially, albeit at a lower rate due to the fungal disease. Only when the effective rate of mite population increase is reduced by other factors such as inferior food quality of the host plant, dislodgement and death by rain and wind and/or predation by generalist predators, will the fungal pathogen be capable of decimating the cassava green mite population.

## DISCUSSION

The most salient feature of our model is that the transmission term includes a constant host density within the leaf area infested (or, more precisely, the leaf area occupied) by cassava green spider mites. This implies that the infested leaf area increases in proportion to the number of spider mites, which is in sharp contrast to the classic Kermack–McKendrick version where the overall number of hosts changes in an environment of fixed size and, consequently, the host density varies. Sabelis (1991) argued that the constant density assumption is a

better approximation of reality because when reaching maturity females move to uninfested parts of the leaves where they feed and lay eggs close to their feeding sites. In this way, the females create a sort of oviposition 'territories'. Thus, spider mite populations are viscous rather than homogeneously mixed (Sabelis, 1991). In fact, several life-history phenomena among the family of spider mites (Tetranychidae) can only be explained by natural selection theory when their populations are viscous. An example is the ability of spider mites to control the offspring sex ratio and produce female-biased sex ratios. Current theory on the evolution of sex allocation strategies predicts a female bias in the sex ratio when the number of local foundresses are small and male-male competition for mates tends to be among sibs (Charnov, 1982; Sabelis, 1991). This agreement between theoretical prediction and observations on the sex ratios produced may be taken as support for the assumption that spider mite populations are viscous.

The important consequence of viscosity in the host population is that transmission depends on the number of susceptible hosts in the direct neighbourhood of the infectious hosts. Thus, if spider mites die close to where they became infected, the spread of the fungal disease would be greatly hampered. In our model, however, we assumed 'maximal transmission' implying that infected hosts move to sites with a high density of susceptible hosts. Such behaviour may arise when parasites control the behaviour of their current host (parasite-induced host behaviour), but in parasite-mite interactions this aspect has never been studied and clearly needs scrutiny by experimental observations on the behaviour of infected mites in two-choice situations. (Do infected hosts settle near infectious or near healthy hosts?) Another possibility promoting the transmission of the fungus is male dispersal. When males have inseminated all their sisters, they will probably move to adjacent leaf areas or leaves. If these males are carriers of the fungus they may promote its spread by increasing the contact rate with healthy mites. Whether the spread of the fungus is promoted by the dispersal of infected females or males is not known, but it is a distinct possibility as shown by the observation of Brandenburg and Kennedy (1982) that some of the two-spotted spider mites (*Tetranychus urticae* Koch) migrating between corn plants were infected with *N. floridana*. Whether this leads to maximal transmission, remains to be investigated, but under this assumption our model reflects the most favourable conditions for a fungal epidemic and, hence, may overestimate the capacity of the fungus to suppress the local host population.

The assumption of a constant and maximal transmission rate per infectious host ( $\beta_s$ ) has major effects on the dynamics of the host-pathogen model; instead of sustained (diverging) oscillations (as would be expected for variable densities of the susceptible hosts) the host population will be driven to extinction after one interaction cycle, provided that the conditions for transition to the infectious stage apply. Field evidence shows that *Neozygites* sp. epizootics can indeed decimate local mite populations (Carner and Canerday, 1968; Smith and Furr,



1975), but data are lacking to assess whether these populations are virtually driven to extinction or resurge to give rise to a next cycle.

Our estimate of the per capita transmission rate,  $\hat{\beta} = 0.039 \text{ cm}^2/\text{day}^{-1}$ , is of the same order of magnitude as reported recently by Brown and Hasibuan (1995) for *N. floridana* attacking the two-spotted spider mite *T. urticae*. They found either  $\beta = 0.045$  or  $\hat{\beta} = 0.024 \text{ cm}^2/\text{day}^{-1}$  depending on whether the linear regression was forced through the origin or not. Brown and Hasibuan (1995) argued that the difference between the two regression slope estimates indicates non-linearity in the relation between the number of new infections and the product of *S* and *C*; the best fitting relation was close to a square-root function of *S* and *C*. Non-linearities did not show up when Brown and Hasibuan (1995) kept the density of susceptibles constant while varying the number of mummies (halos). This result lends support to the linear representation of the transmission term ( $\beta sC$ ) based on the assumption of a constant density of susceptibles (*s*).

From a biological point of view it is of interest to analyse the components of  $\beta$ . When the spider mites walk randomly with respect to the position of the halos, then  $\beta$  can be calculated as the product of the sum of the diameters of the mite and the halo ( $d_m + d_h$  in cm), the distance walked per day ( $w_m$  in cm day<sup>-1</sup>) and the probability of successful infection after passing a halo ( $P_{mh}$ ). In formula, this is

$$\beta = (d_m + d_h) w_m P_{mh}$$

The diameter of the halo is on average 0.9 cm (G.I. Oduor, unpublished data), whereas the effective diameter of a walking female mite is equal to its width and thus 0.015 cm according to Bondar (1938). Since  $P_{mh}$  is on average equal to 0.815, we obtain a value of  $\hat{\beta} = 0.039$  only when the mites walk 0.052 cm per day<sup>-1</sup> ( $w_m$ ); this corresponds to a displacement of 3.5 times their body width per day. This seems a very unrealistically low value since observations on *T. urticae* have shown a displacement of a few centimetres per day (Sabelis, 1981). This may be interpreted as an indication that the cassava green mites avoid contacting the halos. This avoidance can be achieved either by reducing walking activity in the presence of halos or by avoiding their location. It would be of great interest to assess the contact rates experimentally with an imaginary halo versus a real halo, in order to obtain direct evidence for the existence of avoidance responses.

One of the major conclusions of this study is that the effective rate of population increase of *N. floridana* (i.e.  $r_f = \beta s - \mu$ , expressed in halos per halo per day) is quite low relative to that of the cassava green spider mite. It is interesting to compare this with the model proposed by Janssen and Sabelis (1992) for the local dynamics of predatory mites and spider mites. As argued before, their model equations are exactly the same as the model proposed here for the local dynamics of fungus–mite interactions. Thus, we can compare directly the intrinsic rates of population increase of the fungus (expressed in halos per halo per day) and that of the predators (expressed in predator females

per female per day). This comparison shows that the maximum value of  $r_f$  calculated for the fungus (0.11 halos per halo per day) corresponds to the lower range of values found for predatory mites in the family Phytoseiidae (Sabelis and Janssen, 1994). As shown by Janssen and Sabelis (1992) in their studies on predator-prey dynamics, this relatively low value could be the reason why the fungus alone would not be able to control the spider mite populations under a wide range of initial mummy-host ratios. Only, when the spider mite populations are subject to other mortality factors, such as predation, wind and rain, will the effective population growth rate of the spider mite be sufficiently reduced to increase the halo:host ratios that lead to finite host extinction times.

For this reason the most critical question to ask is whether there are reasons to suspect that the fungal growth  $r_f$  is underestimated. This would require a lower value of  $\mu$  and a higher value of  $\beta s$ . However, our estimate of  $\mu = 1/\tau$  really represents a lower limit as our  $\tau$  equals 233 days based on conidia depletion only and thus excludes the effect of wind, rain and other factors (such as other arthropods). In addition, there is little reason to expect that the estimate of  $\beta$  is much too low. Two sources of factors that may lead to some underestimation of  $\beta$  should be mentioned, however. One is the effect of temperature on walking activity and speed. As our estimate was obtained at approximately 25°C, higher temperatures may give rise to a higher  $\beta s$ . However, temperatures higher than approximately 25°C occur during part of the day. Due to the lower temperatures at night, 25°C appears a fairly realistic estimate of the daily mean temperature. Another source of underestimation may arise because the infected mites were not replaced by healthy ones. However, this effect is probably of minor importance, because the fraction of infected mites in the experiments to estimate  $\beta$  did not reach high values (maximally 34%!).

Another source of underestimation of  $r_f$  is the characteristic density ( $s$ ). Not only does it show considerable site-to-site variation, but the densities can be much higher in the end phase of host plant exploitation (Elliot, personal communication). For example, for a characteristic density of seven active spider mites per cm<sup>2</sup> the condition on the initial ratio of susceptibles to halos is still very restrictive in that more than one halo is required for every two susceptible hosts in order to (ultimately) achieve host population decimation.

Yet another source of underestimation arises from the model assumption that transmission of the fungus takes place only via healthy mites that pass the halos of capilliconidia surrounding infectious hosts. This ignores the possibility that spores drifting in the air will reach other hosts within the same local population. The aerial dispersal of spores is assumed to be relevant only for long-distance dispersal (from population to population). Clearly, this assumption needs scrutiny by, e.g. analysing the spread of spores using molecular-genetic markers.

Perhaps, the most important reason for underestimating the capacity of *Neozygites* to control cassava green mites is that our biological studies relate to just one isolate of *Neozygites*. Clearly, one should be cautious in inferring

species properties from studying the properties of just one strain. For the fungal pathogen to be effective when acting as a single biological control agent, strains with larger values of  $\beta$  and  $\alpha$  (i.e. a shorter  $\tau$ ) than measured in the present study are needed.

However, in the absence of data on other strains, we have to conclude that under conditions where the cassava green mites reach their maximal rate of population growth the fungal pathogen under study cannot drive local cassava green mite populations to extinction. Instead, the cassava green mites will increase exponentially, albeit at a lower rate due to the fungal disease. Only when the effective rate of population growth of the cassava green mites is reduced by other factors such as lower temperature, lower food quality of the host plant, dislodgement and death by rain and wind or predation, will the fungal pathogen be capable of decimating the cassava green mite population. Thus, under realistic field conditions (where all of these growth reducing factors are likely to operate) there may well be more room for effective control by the parasitic fungus, as an additional mortality agent.

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#### APPENDIX 1

##### *Model 1: linear three-compartment system*

When pathogen transmission occurs at a rate proportional to the density of the susceptibles ( $s$ ) and number of infectious hosts, i.e.  $\beta sC$ , then the model is described by a system of linear differential equations:

$$\frac{dS}{dt} = rS - \beta sC$$

$$\frac{dI}{dt} = \beta sC - \alpha I$$

$$\frac{dC}{dt} = \alpha I - \mu C$$

This system can be solved analytically having only the trivial steady state

$$\{S, I, C\} = \{0, 0, 0\}$$

The epidemic threshold is determined by the condition

$$\beta sC - \mu C > 0$$

Thus, by the introduction of one infectious individual into a population of an arbitrary number of susceptibles we find the parameter condition

$$\beta s > \mu$$

or

$$\frac{\beta s}{\mu} > 1$$

Supposing particular solutions of the form  $e^{\lambda t}$  the characteristic equation is found by

$$\det \begin{vmatrix} r - \lambda & 0 & -\beta s \\ 0 & -\alpha - \lambda & \beta s \\ 0 & \alpha & -\mu - \lambda \end{vmatrix} = 0$$

i.e.

$$(r - \lambda)\{(-\alpha - \lambda)(-\mu - \lambda) - \alpha\beta s\} = 0$$

and we find the eigenvalues

$$\lambda_1 = r$$

$$\lambda_{2,3} = -\frac{\alpha + \mu}{2} \pm \frac{1}{2}\sqrt{(\alpha - \mu)^2 + 4\alpha\beta s}$$

For all positive parameter values the discriminant is positive so the eigenvalues are real. Since  $\lambda_1 > 0$  the steady state is unstable: the trajectories will go to infinity.

The exact solution can be found through calculation of the eigenvectors; we find

$$S(t) = c_1 e^{rt} + c_2 e^{\lambda_2 t} + c_3 e^{\lambda_3 t}$$

$$I(t) = c_2 \frac{r - \lambda_2}{\alpha + \lambda_2} e^{\lambda_2 t} + c_3 \frac{r - \lambda_3}{\alpha + \lambda_3} e^{\lambda_3 t}$$

$$C(t) = c_2 \frac{r - \lambda_2}{\beta s} e^{\lambda_2 t} + c_3 \frac{r - \lambda_3}{\beta s} e^{\lambda_3 t}$$

The constants  $c_i$  are determined by the initial values  $S_0$ ,  $I_0$  and  $C_0$ . We find

$$c_1 = S_0 - \frac{\alpha\beta s I_0 + (\alpha + r)\beta s C_0}{(r - \lambda_2)(r - \lambda_3)}$$

$$c_2 = \frac{\alpha\beta s I_0 + (\alpha + \lambda_2)\beta s C_0}{(r - \lambda_2)(\lambda_2 - \lambda_3)}$$

$$c_3 = \frac{\alpha\beta s I_0 + (\alpha + \lambda_3)\beta s C_0}{(r - \lambda_3)(\lambda_2 - \lambda_3)}$$

Since  $\lambda_3 > 0$ , the contribution of the third eigenvalue will vanish. The second eigenvalue will be negative if

$$\alpha + \mu > \sqrt{(\alpha - \mu)^2 + 4\alpha\beta s}$$

i.e. if

$$\mu > \beta s$$

Then both the  $I$  and the  $C$  populations will vanish and  $S$  will grow unlimited. This is in agreement with the findings on the epidemic threshold.

Depending on the initial conditions and parameter values the model may lead to a limited growth of the  $S$  population followed by a fast die off; the moment  $S$  reaches zero, the model assumptions do not hold. However, if the growth rate of the host  $r$  exceeds the positive eigenvalue  $\lambda_2$  the host population may increase unlimited and so will  $I$  and  $C$ .

#### *Model 2: linear two-compartment system with timelag*

This model differs from model 1 in the period of transition to the infectious stage. Here we assume a fixed time,  $\tau = \alpha^{-1}$ , from the infected to the infectious stages. Consequently, we do not consider the compartment of infecteds and the resulting equations are

$$\frac{dS}{dt} = rS_t - \beta s C_t$$

$$\frac{dC}{dt} = \beta s C_{t-\tau} - \mu C_t$$

The epidemic threshold is given by

$$\beta s > \mu$$

and the only steady state is  $\{0,0\}$ , with the same properties as in model 1.

#### LIST OF SYMBOLS

##### *Variables*

$S$  Number of susceptible mites.

$I$  Number of infected mites.

$C$  Number of infectious mites ( i.e. number of 'halos' with capilliconidia ).

##### *Parameters*

$r$  Intrinsic rate of increase of susceptible mites ( $\text{day}^{-1}$ ).

$\beta$  Per capita rate of transmission ( $\text{cm}^2 \text{day}^{-1}$ ).

- $\alpha$  capita rate of transition to the infectious (halo) stage ( $\text{day}^{-1}$ ).  
 $\mu$  Per capita rate of halo loss ( $\text{day}^{-1}$ ).  
 $\tau$  Time lag between being infected and becoming infectious (days).  
 $s$  Characteristic host density ( $\text{cm}^{-2}$ ).

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