

What is pathogen-mediated insect superabundance?

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When increasing abundance of insect vectors is manifest across multiple fields of a crop at the landscape scale, the phenomenon is sometimes referred to as insect superabundance. The phenomenon may reflect environmental factors (i.e., environmentally mediated insect superabundance, *EMiS*), including climatic change. A number of pathogens, however, are also known to modify the quality of infected plants as a resource for their insect vectors. In this paper, we term increasing vector abundance when associated with pathogen modification of plants: *pathogen-mediated insect superabundance* (henceforth *PMiS*). We investigate PMiS using a new epidemiological framework. We formalise a definition of PMiS and indicate the epidemiological mechanism by which it is most likely to arise. This study is motivated by the occurrence of a particularly destructive cassava virus epidemic that has been associated with super-abundant whitefly populations in sub-Saharan Africa. Our results have implications for how PMiS can be distinguished from EMiS in field data. Above all, they represent a timely foundation for further investigations into the association between insect superabundance and plant pathogens.

Key words: plant pathogen | manipulation | epidemiology | vector | phytophagous | superabundance.

INTRODUCTION

1 There is empirical evidence for increasing abundance of whitefly over large areas of Africa
2 [1, 2, 3, 4]. This has several important consequences for crop production. High densities of the
3 insect can cause damage to plants through their feeding activity, and, in addition, whitefly

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4 are vectors of important viral pathogens of major subsistence crops, such as cassava. When
5 increasing abundance of vectors is manifest across multiple fields of a crop at landscape scales,
6 the phenomenon is sometimes known as insect superabundance [1, 5, 2, 6]. The increase in
7 abundance may be associated with a range of factors including climatic change [3] (termed
8 here as *environmentally mediated insect superabundance*, EMiS). But there is evidence that
9 pathogen infection of plants can itself increase the abundance of vectors on infected plants
10 [7, 8]. In this report, we examine the epidemiological dynamics of pathogens that modify
11 plants as a resource for vectors. Based on epidemic dynamics, our goal is to identify the
12 epidemiological mechanism that is most favorable for the occurrence of *pathogen-mediated*
13 *insect superabundance* (i.e., PMiS, as distinct from EMiS). We motivate the problem using
14 whitefly-borne begomoviruses (which include, for instance, cassava mosaic virus and tomato
15 leaf curl virus) which are well-studied experimentally, and in which regional epidemic spread
16 has coincided with superabundance of the polyphagous tobacco whitefly, *Bemisia tabaci*, the
17 species complex that transmits these viruses [9].

18 When superabundance is pathogen mediated, the increased density of vectors on infected
19 plants leads to more successful transmission of infection, as increased numbers of insect vectors
20 disperse from infected to surrounding healthy plants. This, in turn, leads to a cycle of increased
21 vector density leading to more infected plants that give rise to more vectors. A range of
22 epidemiological mechanisms have been proposed whereby pathogen infection could modify
23 plants to support higher densities of vectors. The underlying biological mechanisms are usually
24 investigated using molecular and physiological tools. Typically, these analyses are supported
25 by experiments that demonstrate correlations between vector density and plant traits. For
26 example, high insect densities have been linked to high amino-acid concentrations in virus-
27 infected cassava phloem [10, 11]; high insect densities have been linked to altered plant defense
28 hormones in virus-infected tobacco and tomato plants [12, 13]; increased egg production has
29 been found on virus-infected tomato plants [14]. Here we focus on the consequences of such
30 pathogen-induced changes in plant traits (i.e., epidemiological mechanisms) on the population
31 dynamics of vector and pathogen. In particular we evaluate the ability of each epidemiological
32 mechanism to induce elevated insect abundance at the landscape scale (i.e., PMiS).

33 We approach the problem of establishing the epidemiological mechanisms that lead to
34 PMiS by first deriving the vector dynamics for a given incidence of the pathogen among plants
35 (Methods: Vector dynamics subsection); then deriving the pathogen dynamics for a given
36 abundance of the vector (Methods: Epidemiological dynamics subsection). Using the resulting
37 set of equations, we provide a quantitative definition of PMiS and use it to differentiate the
38 effects of the distinct epidemiological mechanisms. The proposed epidemiological mechanisms
39 that alter vector dynamics encompass changes to multiplication rate, carrying capacity and
40 preference of the insect vector for infected plants. With this approach it is possible to
41 distinguish the roles of vector and pathogen in accounting for superabundance and to evaluate
42 methods for detecting PMiS in empirical survey data. We discuss the implications of our results
43 for the unprecedented increase in abundance of the *B. tabaci* whitefly, vector of multiple
44 cassava viruses, that has occurred in *East and Central Africa* since the 1990s [1, 9, 5], where
45 one-hundred-fold increases in *B. tabaci* abundance together with the accompanying cassava
46 mosaic disease pandemic caused crops to be abandoned, leading to widespread food shortages
47 and famine-related deaths [15, 2].

48 MATERIALS AND METHODS

49 Vector dynamics

In order to investigate the ability of putative epidemiological mechanisms to lead to insect superabundance we model the joint population dynamics of insect colonies and pathogen epidemics. For simplicity, the complex life-stages of specific insect vectors are not incorporated here, we instead focus on vector dynamics of the adult insects. Phytophagous insect vectors of plant pathogens like whitefly, aphids and thrips, move between host plants assessing their acceptability through probing behaviour. The insect vectors settle and feed on the phloem tissue of a plant's vascular system if the plant is acceptable, and, when settled, reproduce (Fig. 1A-1B). We consider a general case in which pathogen modification of plants affects the population dynamics of vectors, leading to relatively high vector abundance on infected compared with healthy plants (Fig. 1B). Fundamentally, the insect population dynamics

involve reproduction, mortality and dispersal with density dependence constraining population growth of the vector at the level of individual plants (see Donnelly et al. [16]). The major limitation on phytophagous insect growth rates relates to the nutritional status of insect food. If PMiS occurs, leading to elevated insect abundance, it is therefore reasonable to assume that some aspect of growth or dispersal depends upon the infectious state of the host plant. To take account of these factors we considered a fixed population of H plants comprising healthy ($S(t)$) and infected ($I(t)$) individual plants (i.e., $S(t) = H - I(t)$). We formulated the following equations for V_S and V_I (vector density on the average healthy and infected plant respectively):

$$\text{S plant colony} \quad \frac{dV_S}{dt} = \overbrace{aV_S(1 - \frac{V_S}{\kappa})}^{\text{Reproduction}} - \overbrace{bV_S}^{\text{Death}} - \overbrace{\theta V_S}^{\text{Dispersal loss}} + \overbrace{\theta(V_S S + V_I I) \frac{S}{S + \epsilon_3 I} \frac{1}{S}}^{\text{Dispersal gain (scaled for indiv. plants)}}, \quad (1)$$

$$\text{I plant colony} \quad \frac{dV_I}{dt} = \epsilon_1 a V_I (1 - \frac{V_I}{\epsilon_2 \kappa}) - b V_I - \theta V_I + \theta (V_S S + V_I I) \frac{\epsilon_3 I}{S + \epsilon_3 I} \frac{1}{I}. \quad (2)$$

50 In Eq.s 1-2 a and κ denote low-density net reproduction rate and the maximum vector density
 51 per plant for vector multiplication to occur, b denotes the natural mortality rate of vectors, and
 52 θ denotes the rate of vector dispersal between plants. In addition, ϵ_j (for $j \in 1, 2, 3$), accounts
 53 for increase of the resource quality of infected host plants for vectors if $\epsilon_j > 1$. This may benefit
 54 vector dynamics through increased vector reproduction rate ($\epsilon_1 > 1$), increased plant carrying
 55 capacity for vectors ($\epsilon_2 > 1$) or increased vector acceptance of probed plants (termed here as
 56 *preference* for infected plants) ($\epsilon_3 > 1$) (note that for comprehensiveness $\epsilon_j < 1$, representing
 57 decreased plant quality, is also possible in our formulation).

58 Note that virus modifications may also alter insect preference with respect to feeding
 59 retention of infected insects for healthy plants, and of uninfected insects for infected plants.
 60 Such traits, which can involve pathogen modification of the insect vector, are not our focus
 61 here, and have been discussed elsewhere [17, 18]. Nevertheless, for completeness, see SI
 62 Appendix 2 for an outline of how this form of modification can be incorporated in our

63 framework, and, for an explanation of why they are not associated with pathogen mediated
64 insect superabundance.

65 The pathogen is carried between host plants by insect vectors as they disperse over
66 landscapes. When we consider *PMiS*, we are referring to elevated insect abundance at the
67 landscape scale that is associated with the incidence of infection among plants. Accordingly,
68 we define the degree of pathogen-mediated insect superabundance, denoted $M(I)$, in terms
69 of the total population size of the vector in the population of host plants, as the conditional
70 ratio,

Degree of PMiS :

$$M(I) = \frac{V_S^*(I)S(t) + V_I^*(I)I(t)}{V_S^*(0)H} \begin{cases} > 1 & \text{pathogen-mediated superabundance} \\ = 1 & \text{no effect of pathogen on abundance} \\ < 1 & \text{pathogen-mediated subabundance} \end{cases} \cdot (3)$$

71 The magnitude (degree) of PMiS is high when vector population size in the endemic landscape,
72 i.e. numerator in Eq. 3, is high, compared with its size in the infection-free landscape, i.e.,
73 denominator in Eq. 3. Note that in the above equations we take V_S (and V_I) at its dynamic
74 attractor, i.e., $V_S^*(I)$ (and $V_I^*(I)$), as the epidemic, $I(t)$, spreads. This assumption implies that
75 vector density on individual plants reaches a steady-state faster than the spread of infection
76 among plants. The assumption has been relaxed in representative simulations to confirm the
77 robustness of the main conclusions.

78 **Epidemiological dynamics**

For the majority of insect-borne plant pathogens, the overall transmission rate to plants is proportional to the number of infected vectors that are feeding on individual healthy plants denoted V_S^+ (Fig. 1A), i.e.,

$$\text{inoculation rate:} \quad r^{inoc} S V_S^+. \quad (4)$$

In Eq. 4, r^{inoc} is the per infected vector rate at which plants are inoculated during feeding. The total number of infected insects that are feeding on healthy plants (SV_S^+) can be expressed as Yp_S , where Y is the total number of infected vectors in the local population of host plants, and p_S denotes the probability that infected vectors are found on healthy plants. Conversely, the transmission rate to vectors (also referred to as the acquisition rate) is proportional to the number of uninfected vectors that are feeding on individual infected plants denoted V_I^- (Fig. 1A), i.e.,

$$\text{acquisition rate:} \quad r^{acq}IV_I^-, \quad (5)$$

79 In Eq. 5, r^{acq} denotes the per uninfected vector rate at which the pathogen is acquired during
 80 feeding on infected host plants. The total number of uninfected insects feeding on infected
 81 plants (IV_I^-) can be expressed as $IV_I - Yp_I$. In this work we use the expected proportion
 82 of the infected insect's life spent on a healthy plant denoted ρ_S (or alternatively on an
 83 infected plant denoted ρ_I) as a proxy for the probability that infected vectors are found
 84 on healthy (or infected) plants (i.e., we substitute $p_S = \rho_S$ and $p_I = \rho_I$ in Eq. 4 and 5; see
 85 SI Appendix 1 for derivation). Using expected lifespan proportions in this way (see e.g. [19]),
 86 greatly simplifies calculations without impacting on accuracy (as we have confirmed using
 87 complementary computer simulations).

Combining the terms for inoculation and acquisition rate, and taking account of the expected duration of insect and plant infections, the epidemic is described by equations for the number of pathogen-infected plants and for the number of pathogen-infected vectors at time t , i.e., $I(t)$ and $Y(t)$,

$$\text{Pathogen infected plants} \quad \frac{dI}{dt} = r^{inoc}Y\rho_S - \delta I \quad (6)$$

$$\text{Pathogen infected vectors} \quad \frac{dY}{dt} = r^{acq}(IV_I - Y\rho_I) - (\sigma + b)Y. \quad (7)$$

88 In Eq.s 6-7 epidemics are limited by the rate at which infected plants cease being infectious,
 89 denoted δ , through mortality or removal by growers (known as roguing). We assume dead
 90 plants are replaced with healthy plants so that the total population of plants remains constant.

91 In addition, the infectious period of the vector is limited by the rate that vectors cease being
 92 infectious (the sum of the constant rates that vectors lose the pathogen, σ , and natural
 93 mortality, b). All parameters are listed and defined in Table 1.

94 RESULTS

95 We now analyse the effects of the putative epidemiological mechanisms of PMiS, to identify
 96 those that, when present, are most consistent with PMiS at landscape scales. In Fig. 2 we show
 97 the shapes of response curves relating vector abundance per plant and disease incidence to
 98 changes in three critical parameters controlling the epidemiological mechanism of modification.
 99 The parameters are pathogen modification of vector reproduction rate (ϵ_1), of vector carrying
 100 capacity (ϵ_2) and of vector preference for infected plants (ϵ_3) (cf. Eq. 1-2).

101 The suppression of plant defences to insects by plant pathogen infection leads to more
 102 frequent acceptance of probed plants for sustained phloem feeding. Therefore, defence
 103 suppression can effectively increase vector preference for infected plants. We find that although
 104 insect preference for infected plants leads to higher abundances on infected plants than on
 105 healthy ones, it lowers the overall incidence of infection among plants. Therefore, increased
 106 preference for infected plants leads to a lower overall abundance at the landscape scale when
 107 the modifying pathogen is endemic than when no disease is present (i.e., $M < 1$, Fig. 2I).
 108 As a corollary, lower preference for infected plants can actually increase incidence as infected
 109 vectors encounter healthy plants more frequently (cf. non-monotonic curve in Fig. 2c). At
 110 first sight these results appear counter-intuitive, but they are a direct consequence of the
 111 effect of insect preference for infected plants. Though it increases the occurrence of pathogen
 112 acquisition, it also serves to decrease the overall rate of inoculation to susceptible plants (note
 113 the related point that system stability is lost for substantially lower preference because of
 114 reduced pathogen acquisition, cf. unstable region Fig. 2c).

115 For increased vector carrying capacity of infected plants ($\epsilon_2 > 1$) however, both abundance
 116 per infected plant (Fig. 2E) and incidence of pathogen infection among plants (Fig. 2B)
 117 are dramatically higher than when infected plants are not modified, leading to vector
 118 superabundance ($M > 1$, Fig. 2H). For increased vector reproduction rate on infected plants

119 ($\epsilon_1 > 1$) a similar pattern to that of increased carrying capacity is found, but the degree of
120 superabundance is very minor (Fig. 2G cf. Fig. 2H). Therefore, we find that PMiS is most
121 likely to occur for modifications of carrying capacity, and is not expected to arise at all through
122 the modification of insect preference.

123 What are the implications for testing PMiS in field data? We have shown that PMiS arises
124 through pathogen modification of plant traits that alter insect reproduction, most particularly
125 through the elevation of their insect carrying capacity. A key insight from Fig. 2 is that when
126 the pathogen modifies such traits then insect abundance per healthy and per infected plant
127 are positively correlated (Fig. 2D-E red vs green curves). The positive correlation occurs
128 for a simple reason: the presence of large insect colonies on infected plants are a source of
129 insects for neighbouring uninfected plants. In other words local insect dispersal from crowded
130 to less crowded plants tends to reduce insect aggregation on infected plants but increases
131 abundance on neighbouring uninfected plants. As a consequence, it may not be possible to
132 establish statistically significant differences between abundances on healthy and neighbouring
133 infected plants, even when a strongly modifying pathogen leads to a high degree of insect
134 superabundance (e.g., Fig. 2H).

135 DISCUSSION

136 For a number of arthropod-transmitted plant pathogens, infected plants support higher
137 densities of the insect vector than plants that are uninfected in controlled experiments. There
138 is substantial evidence that this synergistic interaction between plants and insect vector is
139 caused by pathogens that modify plant susceptibility to vector colonisation [7]. When taken
140 at the scale of fields and landscapes, this interaction may lead to pathogen-mediated insect
141 superabundance (PMiS); but insect superabundance may alternatively be a consequence
142 of environmental factors (i.e., EMiS) or of processes of insect invasion. We developed an
143 epidemiological model to analyse the role of pathogen modification mechanisms in elevated
144 insect vector abundance over landscapes i.e., ‘superabundance’ [1, 5, 2, 6]). Our modelling
145 showed that only modifications of the vector carrying capacity of infected plants are capable
146 of causing vector superabundance over landscapes. We also found that abundance per healthy

147 and per infected plants are positively correlated in conditions of pathogen-mediated insect
148 superabundance, with implications for the detection of PMiS.

149 In the case of cassava mosaic disease (CMD), which is caused by a *Begomovirus*, an
150 unprecedented increase in the abundance of the whitefly vector, *B. tabaci*, has occurred
151 throughout cassava-growing regions of East and Central Africa since the 1990s [1, 9, 5]. In some
152 regions *B. tabaci* abundances on cassava shoot tips changed from a few adults to hundreds
153 [20]. Two principal hypotheses have been advanced to explain this increased abundance,
154 namely: a synergistic interaction between CMD-infected cassava plants and *B. tabaci* [9], or
155 genetic changes in the *B. tabaci* population itself [21]. To date neither has been definitively
156 proven, although the two are not mutually exclusive [2]. Understanding the factors underlying
157 superabundant insect populations, like whitefly in East and Central Africa, is especially
158 important because of the secondary emergence of pathogens (for instance cassava brown streak
159 virus which now constitutes a major threat to regional food security) transmitted by shared,
160 vector populations [2]. Furthermore, for plant viruses in general, though there is substantial
161 empirical evidence that pathogen infections of plants can increase vector abundance, it is not
162 clear which aspects of the insect life-cycle are affected [14, 22, 13, 23, 12, 24]).

163 Using a framework based on the explicit modelling of a general insect vector, through the
164 population dynamics of insect colonies on individual infected and healthy plants, we showed
165 that modifications of vector reproduction but not insect preference can lead to the occurrence
166 of insect superabundance at landscape scales (cf. Fig. 2). The shape of the trends in Fig. 2
167 demonstrate the simple intuition underlying the result. For PMiS to arise over landscapes not
168 only does abundance per infected plant need to be high, but the incidence of infection among
169 plants also needs to be high. When insects prefer infected plants the abundance per infected
170 plant increases, but the incidence of infected plants decreases (precluding PMiS). When insect
171 reproduction is higher on infected plants, in particular through increased carrying capacity, the
172 abundance per infected plant, and, in addition, the incidence of infected plants are both higher
173 (enabling PMiS). Of the modifications that influence reproduction, increased insect carrying
174 capacity leads to very substantial superabundance while increased per capita reproduction
175 rate leads to only very minor PMiS.

176 A consequence of the analysis here is that a simplistic approach to detecting evidence
177 for PMiS in which comparisons are made between insect abundance on healthy and infected
178 plants is prone to error. The reason is that colonies on healthy vs. infected plants in a field
179 are positively correlated through dispersal (Fig. 2). In a subsequent paper, we will show how
180 observations of insect abundance over fields on a landscape, together with variation in the
181 incidence of infection among plants in the respective fields, can be used to test more robustly
182 for PMiS. The methods will be applied to field data for a CMD epidemic to shed new light
183 on the original factors underlying *B. tabaci* whitefly superabundance in sub-Saharan Africa.

184 Although we are motivated by the begomovirus-*B. tabaci* interaction, PMiS may be a more
185 widespread phenomenon among plant pathogens. Accordingly, the simplicity of the framework
186 introduced here, which is based upon pathogen transmission during insect feeding, allows broad
187 qualitative application. An exception to this, however, are the non-persistently transmitted
188 viruses that are acquired during probing by aphids rather than through feeding and hence
189 require a different modelling approach [16]. In addition, numerical predictions for a given
190 insect vector species may also be of interest. For this purpose the framework can be extended
191 to include specific reproductive and behavioural processes for the species of interest, and
192 may incorporate measured effects on insect reproduction and preference (see e.g. [14, 25]). A
193 further important consequence of PMiS, which is beyond the scope of this work, is its effect
194 on the host range of insect vectors. For instance, broad host ranges are known to occur in all
195 phytophagous insect orders, and, in particular, *B. tabaci*, the vector of cassava mosaic virus,
196 has a relatively wide host range. In future work, that expands upon the present framework,
197 the expected loss or gain in host breadth that constitutes the evolutionary response of insect
198 vectors to PMiS, will be analysed.

199 *Conclusions*

200 A common theme underscores the results on PMiS in this paper: superabundance is a
201 landscape measure and as such must be analysed at the scale of landscapes. Thus, we found
202 that evaluating potential epidemiological mechanisms underlying PMiS required a landscape
203 perspective, i.e., their viability depended on their effects on the incidence of infection among

204 plants in fields and not just abundance on infected plants. Likewise, appropriate methods for
205 testing field data for PMiS must be based on landscape measures. Suitable methods of this
206 kind will take account of variation in the incidence of infection among plants when analysing
207 abundance - assessing abundance on infected plants in relation to abundance on healthy plants
208 alone is insufficient.

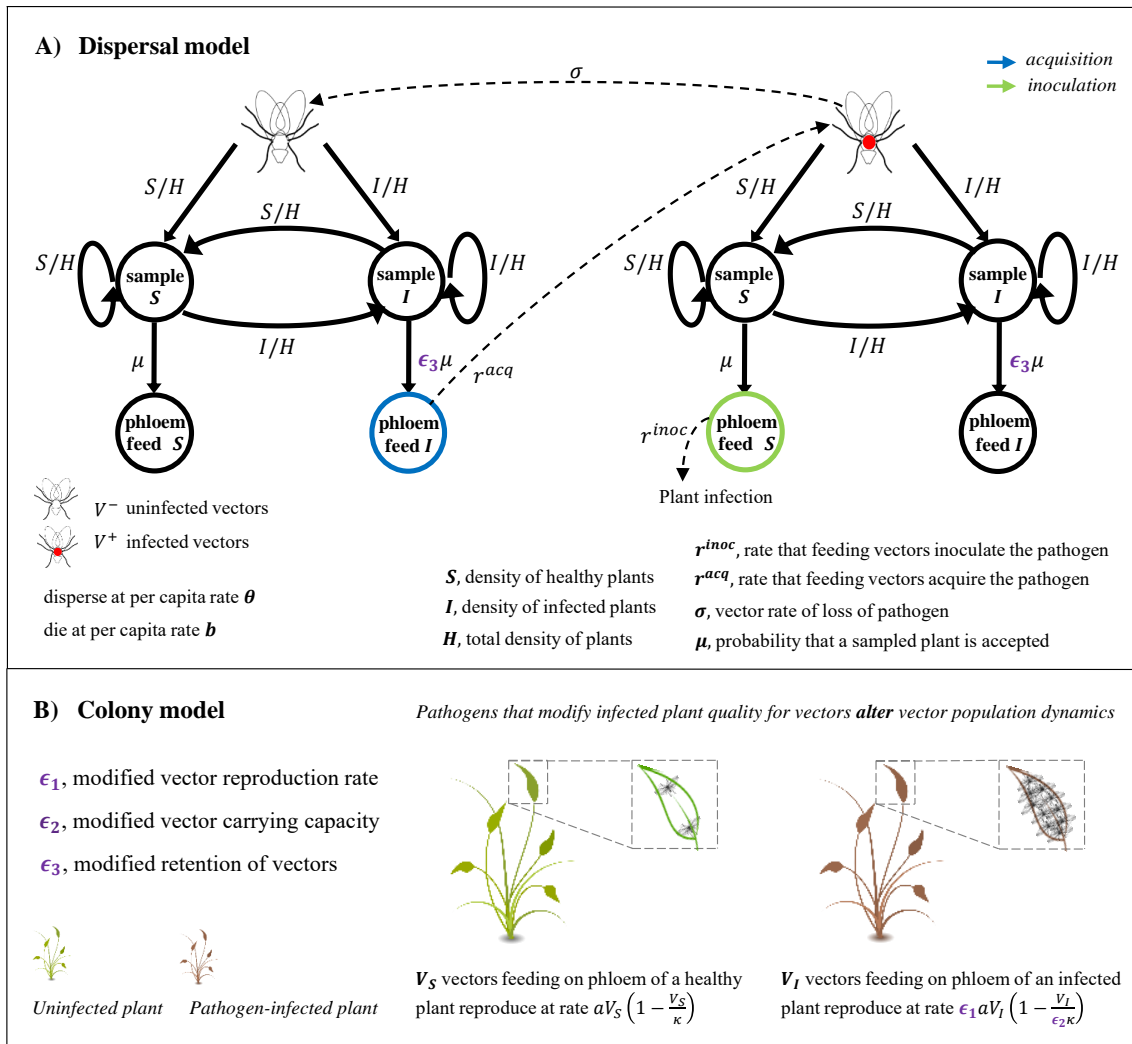


Figure 1. Pathogens that modify plants as a resource for vectors may influence dispersal or reproductive processes (A-B). The pathosystem model, which combines A and B, is comprised of A: a Markov chain model of vector feeding dispersals (with associated pathogen transmission), and B: vector reproduction when the insect vector is settled and feeding. Pathogen infection of plants determines vector abundance as a consequence of altered reproductive processes on infected plants (if $\epsilon_1 \neq 1$ or if $\epsilon_2 \neq 1$ in A), or, as a consequence of altered retention of vectors after they have sampled infected plants (if $\epsilon_3 \neq 1$ in B).

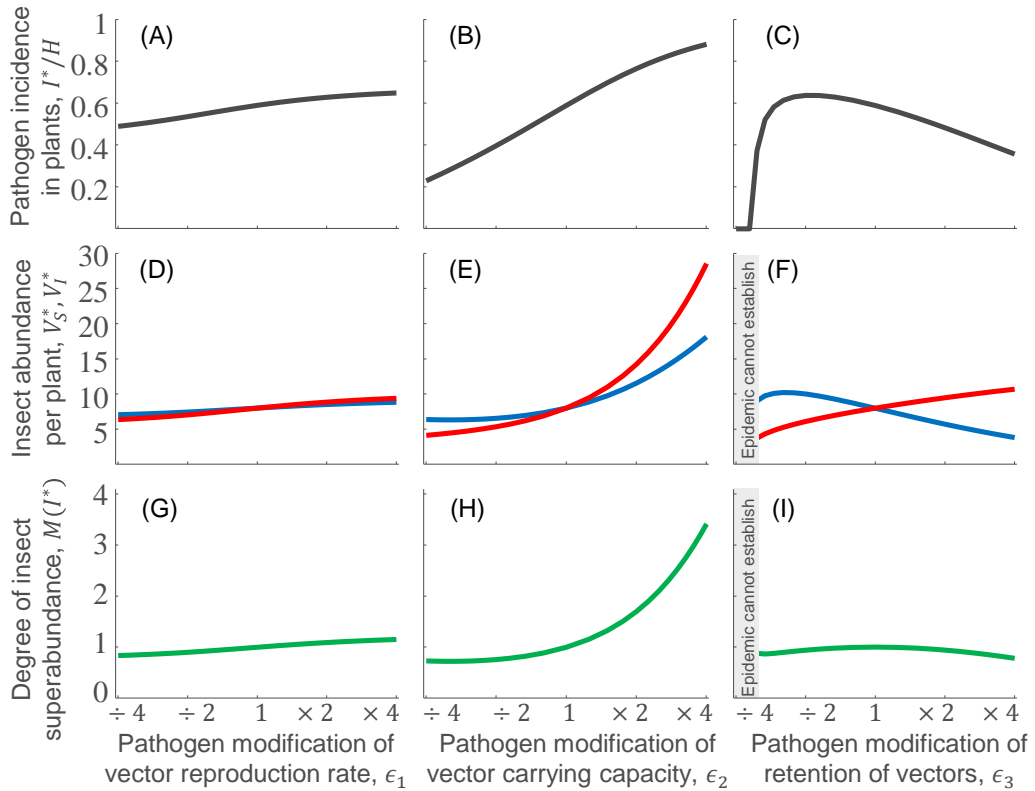


Figure 2. Pathosystem dynamics and insect superabundance, the consequences of pathogen modifications of plant resource quality for vector dynamics and pathogen epidemics (A-I). When the modifying pathogen is endemic, different levels of modifications (x-axis) lead to, **A-C**: different values for pathogen incidence among plants; **D-F**: different values of vector abundance per healthy (blue curves) and per infected (red curves) plants; **G-I**: different values for the degree of vector superabundance (green curves). A-I were generated with $K = 10$ over a host plant population size of $H = 1000$; rates per day were: $a = 1$, $\mu = 1/5$, $r_{acq}=1/2$, $r_{inoc} = 1$, $\delta = 0.3$, $\theta = 2$ and $\sigma = 2$.

Definition of notation used		
<i>(i) Population dynamics (plants, vectors)</i>		<i>Units</i>
Y	Density of infected vectors	<i>per field</i>
V_S	Vector abundance per <i>average</i> healthy plant	<i>per plant</i>
V_I	Vector abundance per <i>average</i> infected plant	<i>per plant</i>
V^+	Abundance of infected vectors	<i>per plant</i>
V^-	Abundance of uninfected vectors	<i>per plant</i>
M	Pathogen-mediated insect superabundance	<i>degree</i>
<i>(ii) Putative modification mechanisms</i>		
ϵ_1	Modification of reproduction rate	<i>scaling factor</i>
ϵ_2	Modification of carrying capacity	<i>scaling factor</i>
ϵ_3	Modification of vector retention	<i>scaling factor</i>
<i>(iii) Additional parameters</i>		
δ	Plant mortality rate	<i>per day</i>
b	Vector mortality rate	<i>per day</i>
θ	Vector dispersal rate	<i>per day</i>
a	Vector reproduction rate (for 0 vector abundance)	<i>per day, per capita</i>
κ	Vector reproduction limit (upper limit on density)	<i>max vectors per plant</i>
r^{acq}	Rate of acquisition of pathogen	<i>per day</i>
r^{inoc}	Rate of inoculation of pathogen	<i>per day</i>

Table 1. Summary of population variables and parameters. The mathematical model tracks changes in plant and vector population variables (i). Vector processes on infected plants are altered by epidemiological mechanisms of pathogen modification (ii) that may underly pathogen mediated superabundance. Pathosystems are characterised by vector and pathogen life history parameters (iii).

209 **ACKNOWLEDGEMENTS**210 **REFERENCES**

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