Plant Disease Epidemiology
Final Exam – 2006
2 hours (closed book, closed notes)

Name: _____

1. Explain in detail the meaning of the basic reproduction number (R_0). How can it be used to determine: *a)* whether or not an epidemic (of the polycyclic type) will occur; *b)* the final level of disease intensity when time is not limiting; and *c)* the exponential rate of increase early in an epidemic? What is the most straight-forward way of estimating R_0 from observed disease progress data?

2. Explain the difference between latent period ($\wp$) and latent disease density (L). Explain why latent period has no relation to R_0 (that is, explain why the latent period does not affect the basic reproduction number).

3. Explain the relationship between the binomial and the beta-binomial discrete distributions. How does one use these distributions to determine the degree of small-scale aggregation of disease incidence? (Answer in words: I am not looking for the equations here). What type of disease intensity should be analyzed with these two distributions? *BONUS*: explain (on the back) why spatial autocorrelation and semi-variograms do not provide any information on spatial patterns at scales smaller than the size of the sampling unit.

4. Define or explain the following terms.

    a. Primary dispersal gradient

    b. Exponential disease gradient model

    c. Intra-cluster correlation (ρ)

    d. Single-point crop loss model

e. Logistic-logistic spatio-temporal disease dynamics

f. $\partial s/\partial t = r_L/b_L$

g. Crop loss

h. Attainable yield

i. $\ln[y/(\boldsymbol{K}\text{-}y)]$

5. Suppose you measured the spatial front of an epidemic (e.g., at $y' = 0.01$) at three different months, and obtained:

t (mon)	*s* (meters)
0	3
2	5
4	9

Based on these values, what can you say about the rate of isopath movement (i.e., the rate of focus or disease expansion)? (I am not looking for an exact calculation here, only a qualitative description as to what is happening). Which disease-gradient model (or corresponding contact distribution) is most likely responsible for the above observations? Explain.

6. For polycyclic diseases, two valuable models are:

$$dH/dt = -\Upsilon YH = -(r_I/M)YH \qquad \text{(eq. 1)}$$

and

$$dH/dt = -\beta IH = -(\alpha\theta\psi)\, IH \qquad \text{(eq. 2)}$$

Explain the essential difference (or differences) between these two models in terms of disease dynamics. What names are given to the two models? Compare and contrast the meaning of β (eq. 2) and Υ (that is, r_I/M) (eq. 1). Give one advantage and one disadvantage of <u>each</u> model. (All terms are as defined or used in class).