Crop Loss Assessment (*last lecture topic*)

Review:

- Concepts of epidemics
- Measurement of disease intensity
- Modeling of disease intensity (general issues)
- Temporal analysis of epidemics
  - One-variable processes (***Y*** or ***y***), monocyclic and polycyclic ($dY/dt = \Upsilon YH$ or $dy/dt = r_L y(1-y)$,
    - or $r_M(1-y)$
  - Components (***M = H+L+I+R***) --> $dY/dt = \beta IH$

- Spatial analysis: disease **gradients, isopaths, ...**
- Spatial analysis: **patterns**
  - ***(Sampling issues: see chapters 10 and 11 in MHV)***
  -
- ***Now:*** **Crop losses**
  - Epidemics have many direct and indirect effects on crops, including the reduction in yield
  - That is, the yield of a crop is determined, in part, by the intensity of disease at various times

Although it can see the dying elms along the streets, society in general is only vaguely aware of how much damage it suffers from the depredations of other plant pathogens. That is because we have not told society about the losses, and that is because we do not know, and that is because we have not researched it very well.

--Horsfall and Cowling, 1977

Pioneers:
E. C. Large
Clive James
Paul Teng
Jan Zadoks
Richard Berger,
Forrest Nutter,
Serge Savary
(others)

We have researched it very well over the last quarter century (or longer), but we still do not necessarily know *exactly* the losses due to plant disease. This may now be more related to high cost of obtaining the raw data, not necessarily to our understanding of the **disease:loss** relationship.

Crop Loss Assessment

- Result of an epidemic is often a reduction in yield
- Final topic of course: crop loss due to disease
- Use crop loss information to:
  - Evaluate controls
  - Make optimal disease management decisions (cost-benefit)
  - Make yield predictions
  - Evaluate need for research on a particular disease
  -
- **CROP LOSS: reduction in either quantity or quality of yield**
  - **YIELD:** *measurable produce of a crop (seed, fruit, leaves, etc.)*

- In practice, with regard to plant diseases, an ***operational*** definition:
  - **Crop Loss:**

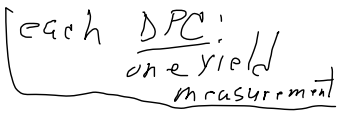
    difference between actual yield and yield obtained in absence of disease

Quantification of crop loss:

- Measure disease intensity (*y* or *Y*) and yield (**for multiple epidemics**)
  a. **Conventional field experiments**
     Impose treatments on experimental units
  b. **Survey of "natural" epidemics**
     (in commercial fields or in forests, etc.), with no imposition of treatments (no control of the variables of interest)
  c. **Expert opinion (panels)**
- What is needed for experiments or surveys?
  - **Several epidemics (i.e., different intensities of disease, different disease progress curves) and corresponding yield**
- Loss -- two possible ways of utilizing:

  L = **(yield in disease-free field) - (yield in field with given *Y*)**

  ℓ = **[(yield in disease free) - (yield with given *Y*)] /(yield in disease free)**



Conventional experiments (with replication, randomization, blocking) -- obtain different epidemics (i.e., different disease intensities and possibly different yields) using:

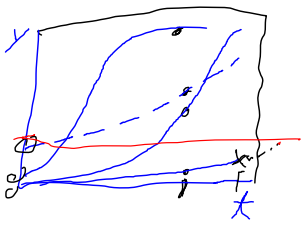
- **Inoculations**
  - Times, frequencies, densities of inoculum
- **Protection** (fungicides, insecticides)
  - Different number of fungicide applications, timing, etc. (lowering *Y* compared to when no control is used)
- **Genetically**
  - Isolines (crop cultivars varying only in susceptibility to pathogen) - *CAUTION*
- **Environmentally**
  - Irrigation, etc. - *CAUTION*
- **Geographically**
  - Growing the same crop in different locations - *CAUTION*

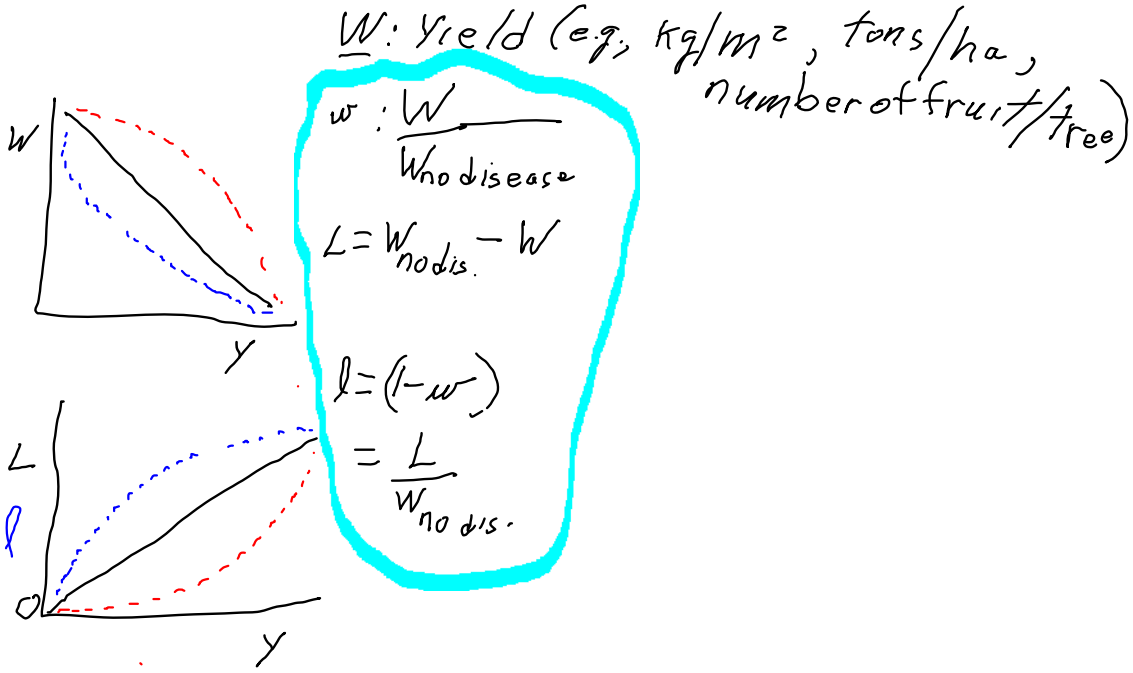
Advantages and disadvantages of each.

Relationships between loss and disease intensity -- Models

- ***L = f(*disease epidemic*)***
-
  - A yield for each epidemic
  - Need several epidemics (usually)
  - *L*: obtained from each yield relative to a disease-free (no epidemic) case
- ***f(*disease epidemic*)***:
  - ***y*** (or ***Y***) at particular time
    - y_{final} (not necessarily *K* or y_∞),
    - y_0,
    - *key time* in crop growth
  - r_*,
  - **AUDPC**,
  - time to reach a certain *y (y')*: t'
  - Others?

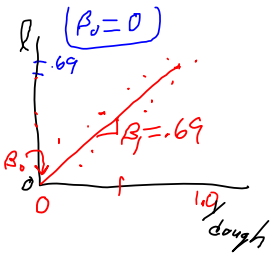
Inter-related



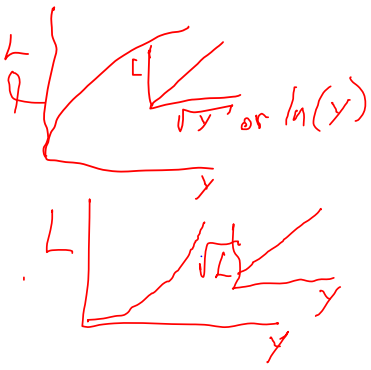


Models -- Single Predictors

1. **Single-point models** -- *y* at <u>one time</u> during an epidemic: $\ell = \beta_0 + \beta_1 y$ or $L = \beta_0 + \beta_1 y$
   - $\ell = 0.69 y_{dough}$ (southern corn leaf blight)
   - If y_{dough} = 1.0 (100%), ℓ=0.69 (69%) , i.e., max.
   - (reminder: need data from several epidemics)

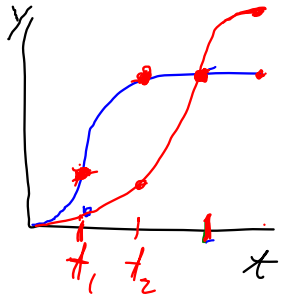


- Often, the *L:y* relationship is curved (but possibly still described by a linear model):
  - $L = \beta_0 + \beta_1 y^*$, where $y^* = ln(y)$ or $\sqrt{y}$, etc., a transformation of *y* at a given time



- Single-point models are sometimes known as **critical-point models**
  - When the single time is "**critical**" for crop development (e.g., a certain growth stage when a high percentage of products of photosynthesis are converted into seed or fruit)
- β_1: Slope, change in *L* with unit change in y^*
  - $dL/dy^* = \beta_1$
  - *Can* indicate **tolerance** to a given disease

Models with multiple predictors --



- More than one predictor variable, either disease intensity at each of several times, change in intensity, or multiple attributes of the epidemics
- **Multiple point models:**

  $$L = \beta_0 + \beta_1 y_1 + \beta_2 y_2 + \beta_3 y_3 + \ldots$$

  - where the ***y***s are disease intensities at select times (e.g., y_1: intensity at t_1, which could be at a specific time [30 days] or a specific growth stage [flowering])
  - and the βs are parameters
- Premise: **crop loss is a function, really a *weighted* function, of the entire epidemic**
  - The importance (weight) of *y* at each time is not the same
  - βs are the weights
- Caution: ***y***s are **intercorrelated** (because *y* increases over time)
  - Thus, estimated βs are not unambiguous indicators of the true weights (**estimates are correlated**)

Models with multiple predictors --

- More than one predictor variable, either disease intensity at each of several times, changes in intensity, or multiple properties of the epidemics

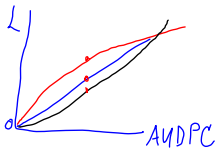
- **Other forms of models--example:**

  $$L = \beta_0 + \beta_1 y_1 + \beta_2 r_* + \beta_3 t_{y'}$$

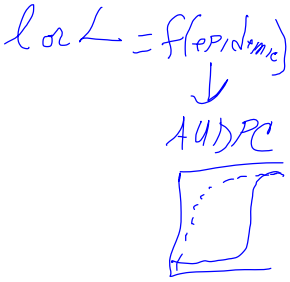
- Analysis requires many disease progress curves, to obtain many *L* values.
  - Finding an 'ideal' relationship is challenging, partly because of the inter-correlations of the predictors
- One uses, typically, multiple regression analysis to estimate parameters

Integral models -- Example: $L = \beta_0 + \beta_1 \cdot AUDPC$

- Integral models are very popular for crop loss assessment
  - From a modeling perspective, it is much easier to work with one predictor variable than many
  - It is easy to determine or estimate AUDPC from an observed disease progress curve (just using the mid-point rule [*making several rectangles and adding up their areas*])
    - One can use the theoretical relationship between the epidemic parameters and AUDPC
- The relationship between *L* and AUDPC (or other weighted integral) can be a straight line or curved (possibly nonlinear)

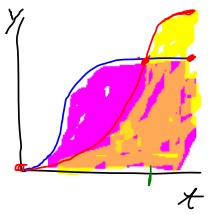


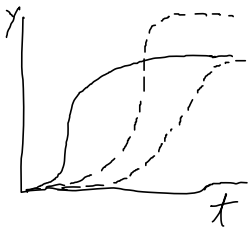
Interpret parameters in *about* the same way as with single-point models ($L = \beta_0 + \beta_1 y$): β_1 shows the *tolerance* or *sensitivity* to the disease



Integral models

- A single predictor variable, but the variable represents an **integration (or summation)** of the *entire* epidemic
- Based on the same premise as the multiple-predictor models: **crop loss is a function of the entire epidemic**
- Usual variable: **$AUDPC = \int y \cdot dt$**
- Model: **$L = \beta_0 + \beta_1 AUDPC$**
- You should realize by now, that AUDPC is really a function of y_0, y_{final}, t_0, t_{final}, and r (for a given disease progress model)
  - Thus, the above model is really a succinct way of expressing the dependence of *L* on the entire epidemic (synthesis [weighting] is done outside the loss model)
  - Model can be modified to give more weight to some times than others

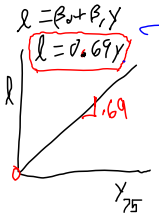




In AUDPC models, the weighted disease average is done before the crop loss model. With multiple point models, the weights are determined within the crop loss model.

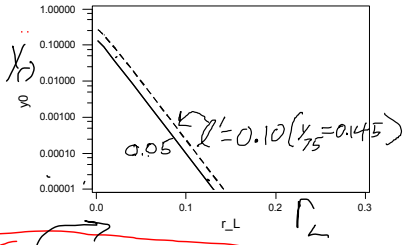
An application

- Previously, we looked at epidemics in terms of initial disease intensity and rate parameter
  - We could evaluate the parameters in terms of resulting epidemics
- Now, we can extend to crop loss
  - Example: what combinations of y_0 and r (for a given time duration) will keep ℓ below some specified value (e.g., $\ell' \leq 0.10$)?



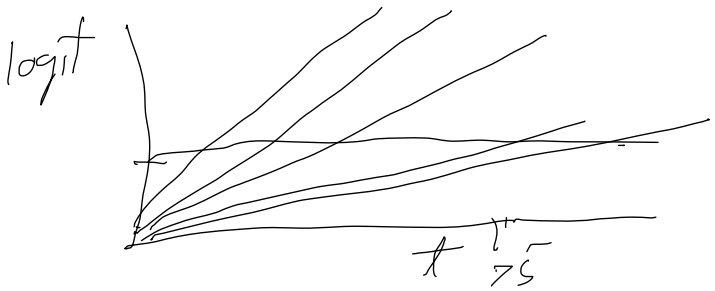
To keep $\ell \leq 0.10$ (10%), y must be $\leq 0.10/0.69 (= 0.145)$ at $t = 75$

If logistic ($K = 1$), there are many combinations of y_0 and r_L that gives $y = 0.145$ at $t = 75$ days.



$$\ln\left(\frac{0.145}{1-0.145}\right) = \ln\left(\frac{y_0}{1-y_0}\right) + r_L \cdot 75$$

$$\ln\left(\frac{y_0}{1-y_0}\right) = -\ln\left(\frac{.145}{1-.145}\right) + r_L 75$$



Crop losses

- The relationship between yield of a crop and plant disease is complicated, and it is unreasonable to expect simple linear models to be satisfactory, or consistent
  - **Crop physiology** as a discipline deals generally (and more thoroughly) with crop growth and development in relation to various environmental and biological factors, including stress (e.g., diseases).
  - Prediction of yield, and hence loss, may require very complicated **mechanistic models** of crop physiology
  - Linking models for crop physiology to population-dynamics models of disease is not trivial, and has seldom been done
- Nevertheless, empirical evidence is strong that **a fair degree of understanding can be obtained by the use of fairly simple models for *L:y*, at least as summaries of more complicated processes**
  - The simple models permit more direct linkage of population dynamics of disease and resulting mean yield of a population

Crop losses-- In a highly influential paper, Boote et al. (1983) considered that plant pathogens (and pests in general) affect crop physiology in seven ways

Not covered in lecture.

1. **Tissue consumers** (e.g., necrotic pathogens)
2. **Leaf senescence accelerators** (e.g., Cercospora on peanut--leaf abscission)
3. **Stand reducers** (e.g., damping off pathogens, seedling diseases)
4. **Light stealers** (e.g., pathogens with lesions)
5. **Photosynthetic rate reducers** (e.g., viruses, biotrophic pathogens)
6. **Assimilate sappers** (e.g., nematodes, root diseases, biotrophic pathogens, systemic pathogens)
7. **Turgor reducers** (e.g., root infecting pathogens, vascular tissue pathogens)

1-4: involve Radiation Interception (RI);

5-7: involve Radiation Use Efficiency (RUE).

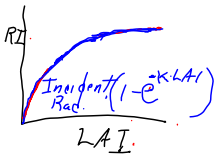
See article by Ken Johnson (1987); several articles in last two decades (see Chapter 12, for more information)

- Each of these processes can be directly studied and quantified (using **physiological models**)
- Pathogens may affect one or several of these
- **All plant growth (and yield production) can be considered a product of RI and RUE**

RI/RUE: impact on crop yield and loss (continued)

- Pathogens (and pests) affect **RI** and/or **RUE**
  - Think of plants as 'factories', with RI being the fuel running the 'machines'; the machines operate at an efficiency equal to RUE
    - **Pathogens affect the 'fuel' supply, or the efficiency of the 'machines', or both**
- RI is a (nonlinear) function of **LAI** of a crop
  - The relation between incident radiation and LAI is described by **Beer's law**
  - Because of shading of lower leaves, many leaves do not intercept much radiation
  - **Reducing the total LAI, or the healthy (disease-free) LAI, may not have much affect on RI, unless LAI is low**
- RUE can be directly and linearly affected by disease intensity (for some diseases)

- **A more useful way of characterizing crop loss may be to quantify, directly or indirectly, RI and RUE**



LAI: Leaf area index [area of host (generally leaf) tissue per unit area]

Not covered in lecture.

RI/RUE: impact on crop yield and loss (continued)

- An innovative approach, first advocated by Waggoner and Berger, is to determine **LAI** (see Chapter 2) and disease severity (***y***), and combine these into an ***integral-type model***

- Since yield (e.g., kilograms/hectare) of a crop is a function of LAI (through the RI), and not a percentage or proportion of LAI, one must estimate (somehow) disease-free LAI ('*healthy*' LAI: **HLAI**)
  - **HLAI = (1-*y*)·LAI** <--One approach (note: LAI = *M*, total plant "size")
  - That is, multiply proportion disease-free by LAI
    - **This is just *H = M-Y*, using earlier notation (for disease severity)**

- One summary of the epidemic is the *integration* of HLAI over time, known as **healthy area duration** (**HAD**) or **green leaf area duration (GLAD)**:

  HAD = ∫HLAI·*dt* <--*Think of the opposite of AUDPC, absolute scale*

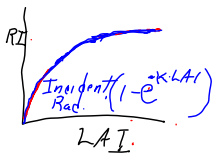
- If LAI was fixed (non-growing host), then there would be an analytical solution to HAD (e.g., if logistic), because HLAI is just ***H=M-Y*** (disease-free intensity, in *absolute* units)-- big AUDPC means small HAD
  - With changing LAI (independent of disease), generally HAD is estimated by obtaining HLAI at several times, and calculating area with mid-point rule (as with AUDPC)

Not covered in lecture.

- Model: ***Yield* = β_0 + β_1HAD**
- This is a very good approach, but could be considered an intermediate step in quantification, because all disease-free leaf area is considered the same (here)
  - Yield goes up with HAD; so, *L* goes *down* with *increasing* HAD
  - However, it is known that all leaves do not intercept the same amount of incident electromagnetic radiation (Beer's law)
    - Often, **Yield:HAD** data do not follow a straight line, and are inconsistent

Not covered in lecture.

- **Direct use of RI:**
  - If one knows LAI, one can calculate RI using Beer's Law
  - If one knows HLAI, one can calculate HRI (**Healthy Radiation Interception**) based on Beer's Law
    - **Incident radiation**, although variable, is generally well known for a latitude/longitude and time of year.
    - **HRI = RI·(1-*y*)**
    -
      - Compare with **HLAI = LAI(1-*y*)**
      - (HRI involves a *nonlinear* transformation of LAI)



- Healthy area *absorption* (HAA): **HAA = ∫HRI·*dt* = ∫RI·(1-*y*)·*dt***
  - *Absorption* being equated with interception (*not quite the same*)
  - Compare with: **HAD = ∫HLAI·*dt* = ∫LAI·(1-*y*)·*dt***
  - Compare with : **AUDPC = ∫*y*·*dt***
- Reminder:
  - AUDPC was an integration of *y* over time
  - HAD was an integration of **HLAI** over time
    (opposite of AUDPC, but on absolute scale)
  - Now, HAA is an integration of HRI over time, with

Not covered in lecture.

- **Yield of a crop** (the *fundamental* concept)**:**

  If RUE is constant over time, then yield is:

  Yield= (RUE)·(HAA)
  - a linear model with slope RUE (β_1), 0 intercept, and HAA as predictor variable
- Often, there is a linear relation between yield (or loss) and HAA

Yield = (RUE)·(HAA)

Not covered in lecture.

- A very useful approach to quantifying crop loss
  - Expandable to multiple diseases simultaneously
  - Based *explicitly* on **RUE** and **RI (or HRI)**
    (through HAA = $\int$HRI·*dt*)
- **Disease affects yield by lowering radiation interception (though reduction in healthy LAI [HLAI]) and/or efficiency in utilizing the radiation**
  - Visualize location of the curve or the slope
- Disadvantages:
  - Need to estimate/measure LAI (difficult)
  - Need LAI and *y* at several times during the epidemic
  - Must either measure intercepted radiation (difficult) or assume published values for incident radiation and use theoretical calculations based on Beer's Law
- Used for some well-studied systems

Crop losses: Summary

Not covered in lecture.

- **Crop loss assessment** is usually considered as part of plant disease epidemiology
  - Some researchers study only crop loss (and not the rest of epidemiology)
- There are various uses for crop loss information
- Traditional methods focused on ***L*** (or **ℓ**) as a function of disease severity as a proportion (***y***) - *still useful*
  - Some newer approaches deal more often with **LAI, HLAI=LAI(1-*y*) = *M*-*Y*, RI, HRI=RI(1-*y*)**
  -
- Use of integral-type models is valuable for understanding crop losses in relation to plant disease
  - The traditional approach (***L*** as a function of **AUDPC**), is still popular (*and useful*)
  - Newer approaches deal with *L* as a function of **HAD** or, even better (if data available), **HAA**
- Other integral approaches are useful for discrete variables (diseased plants)

Reading assignment:

Sections 12.1 to 12.3 (pages 353-360) of textbook (no math in these sections)

Background reading (not required):

Chapter 12 in textbook

References (for background):

Gaunt, R. (1995). Annu. Rev. Phytopathol. 33: 119-144.

Bergamin Filho, A., et al. (1997). Phytopathology 87: 506-515.

Savary et al. (2006). Annu. Rev. Phytopathol. 44: 89-112.

Course conclusions:

- **Plant disease epidemiology continues to be a major field of study around the world**
  - Until durable controls are developed and utilized that totally prevent infection, epidemics will occur, and there will be a need to understand disease dynamics in time and space
  - This understanding can lead to better long-term **strategies** (reduce y_0 or r_* [or Υ]???) as well as short-term **tactics** (prediction of *y* under specified [e.g., control] conditions) for disease management
- **Many other topics can be covered in plant disease epidemiology, some of which are presented in the textbook**
  - Examples: *Sampling* (for estimation or decision making), disease *prediction* algorithms (i.e., disease forecasting), detailed crop loss modeling and analysis, more detailed mechanistic modeling of disease in time and space, calculation of R_0 under complex scenarios, …
- **Topics not in the textbook, but of high importance also:**
  - Examples: Populations of multiple pathogen taxa and/or multiple host taxa; evolution in terms population dynamics and controls; interrelationships between environment and plant disease; stochastic approaches for disease development; etc...