

## Defoliation, Disease, and Growth

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To determine crop loss, plant pathologists usually examine the relation between crop yield and the severity of disease. The relations are often disappointing, however, because the logic of the relation is uncertain, because the effect of a severity is different early and late in the growing season, or because defoliation is not included in severity assessments.

Yield or the dry matter of a plant is largely the integral of photosynthesis in leaves. Although respiration, a few grams of nutrients from the soil, and photosynthesis in organs other than leaves contribute to the eventual yield of dry matter, it is not surprising that Watson (20) found that yield was related to leaf area index,  $L$  (dimensionless square meter of leaf per square meter of land). Yield was, however, correlated with the integral over days of leaf area index or leaf area duration (LAD) (days) rather than with  $L$  itself. In a colloquial analogy, the character of the species and of the environment determine the size of the factory, the leaf area index; then the accumulated product or yield is determined by the size of the factory integrated during a season, the LAD.

During the past decade, the realization that photosynthesis is determined by the absorption of insolation by leaves rather than by their area has permitted a refinement of the relation between leaf area and yield. Generally, Beer's law expresses the transmission of insolation,  $I$  ( $\text{MJ m}^{-2}$ ), through foliage; i.e., the fraction,  $f$ , absorbed is  $[1 - \exp(-K L)]$  where  $K$  varies from about 1 in canopies of horizontal leaves to 0.3 in those of erect leaves. For a range of crops, the production of dry matter,  $W$  ( $\text{g m}^{-2}$ ), is related to leaf area and insolation by

$$W = \int e f I dt + W_o$$

$$= \int e [1 - \exp(-K L)] I dt + W_o \quad (1a)$$

If the conversion efficiency,  $e$  ( $\text{g MJ}^{-1}$ ), is constant during the period of integration and  $W_o$  is negligible,

$$W = e \int I f dt \quad (1b)$$

Insolation is of the order  $20 \text{ MJ m}^{-2} \text{ day}^{-1}$ , and  $e$  is  $1-3 \text{ g MJ}^{-1}$  (12). Thus, a more accurate analogy is: Yield is determined by the energy taken in during the season from the amount falling on a circumscribed catchment. The return from enlarging the factory decreases because enlargement can increase the fraction of energy taken in but not the amount of energy falling on the catchment.

Because plant pathologists are naturally most interested in pathogens and disease, they have concentrated on the increase in the lesions of a disease on leaves or progress of disease severity with passing time. The progress curves of the increasing fraction  $x$  (dimensionless) of disease incidence or severity have been summarized as their relative rate,  $r$ , ( $\text{day}^{-1}$ ) of change (19) or as the area under the disease progress curve (AUDPC, days) (18,19). Whereas LAD is the integral of leaf area index during time, neither the rate of change of  $x$  nor the integral of  $x$  tell the size of the foliar factory, how long it operates or how much insolation it absorbs.

If pathologists want to relate their disease progress curves to growth, it would be logical to subtract the area of diseased leaves from the LAD by integrating the size  $[(1-x)L]$  of the healthy and operating factory during the season. This calculation from the progress of  $x$  and  $L$  gives what we call healthy leaf area duration or HAD (days). Or by a logical adaptation of eq. 1b, the healthy area absorption, HAA ( $\text{MJ m}^{-2}$ ), can be calculated by integrating the fraction  $[1 - \exp(-K L)]$  of insolation absorbed by both healthy and diseased foliage multiplied by  $I$  and by the healthy portion  $(1-x)$ .

## MULTIPLE-POINT MODELS AND HAD

James (7) reviewed the attempts to relate yield to disease and categorized them as critical point, multiple point, and AUDPC models. When the percentage of loss of potato tubers at harvest was compared with the late blight caused by *Phytophthora infestans* at a critical point in time or to the AUDPC, the relation was destroyed by the failure of the point or AUDPC to distinguish early and destructive from late and relatively harmless epidemics. That is, dissimilar epidemics could have similar critical points or AUDPCs. The weighting of disease at multiple points, alternatively, was more closely related to loss.

The multiple-point model is

$$\text{loss} = b_1 x_1 + b_2 x_2 + \dots + b_n x_n \quad (2)$$

where the  $b_i$  are regression coefficients estimated from past epidemics and yields. The  $x_i$  are disease severities at  $t_i$ , and loss is the decrease in yield relative to a healthy crop. Instead of defining loss in terms of disease proportions at various points in time, one could define the loss in terms of the LAD of a healthy standard and the HAD of the diseased crop. If  $L_h$  is the leaf area index of the healthy crop, and if  $L_d$  is the total and  $(1-x)L_d$  the healthy leaf area index of the diseased crop,

$$\text{loss} = 1 - \text{HAD}/\text{LAD}$$

$$= 1 - [\int (1-x) L_d dt] / [\int L_h dt]$$

$$= \{ \int [L_h - L_d + x L_d] dt \} / [\int L_h dt] \quad (3a)$$

If  $L_h = L_d = L$ ,

$$\text{loss} = [\int x L dt] / [\int L dt] \quad (3b)$$

Then if the leaf areas are constant,

$$\text{loss} = [L \int x dt] / [L \int dt] = \text{AUDPC}/t \quad (3c)$$

Alternatively if  $L_h = L_d$  and  $x$  is constant,

$$\text{loss} = [x \int L dt] / [\int L dt] = x \quad (3d)$$

Comparison of eq. 3b and c illuminates the difference between AUDPC and HAD: In a growing crop, an  $x$  early and late in the season add equally to AUDPC, whereas an early  $x$  when leaf area is small subtracts less from HAD than the same  $x$  later when leaf area is large. Eq. 3d shows, of course, that a critical point model will

succeed if both  $x$  is constant and the leaf area of healthy and diseased are equal.

Eq. 3b for equal  $L$  in healthy and diseased crops can be written as an approximation that resembles the multiple point model, eq. 2:

$$\text{loss} = \left\{ \sum [L_{hi} - L_{di} + x_i L_{di}] [t_i - t_{i-1}] \right\} / \text{LAD} \quad (4)$$

where  $L_i$  and  $x_i$  represent approximately the leaf area and portion diseased during the period  $[t_i - t_{i-1}]$  from the  $(i - 1)^{\text{th}}$  to the  $i^{\text{th}}$  critical point. When all  $L_{hi} = L_{di}$ , eq. 2 and 4 specify the same losses if all  $b_i$  equal all  $L_i [t_i - t_{i-1}] / \text{LAD}$ . Alternatively, if  $[L_{hi} - L_{di}] / x_i$  is a constant ratio because the difference between leaf areas grows as  $x$  increases, eq. 2 and 4 specify the same losses if all  $b_i$  equal all  $[ \text{constant ratio} + L_{di} ] [t_i - t_{i-1}] / \text{LAD}$ . Thus, if yield is proportional to HAD, the  $b_i$  may be estimates of past  $L_i (t_i - t_{i-1}) / \text{LAD}$ . Because eq. 4 shows the meaning of  $b_i$ , it provides a means of logically adjusting  $b_i$  for future courses of leaf area index with new cultivars and seasons.

### DEFOLIATION AND HAD

The decrease of  $L_d$  by defoliation makes  $x$  difficult to interpret. If  $x$  is the current, visible portion of the standing crop diseased, one may conclude that disease is less because the  $x$  is less in an epidemic that defoliates all infected leaves than in an epidemic that merely spots and does not defoliate the crop. On the other hand, if  $x$  is the diseased portion of both present and fallen foliage, one is faced with the difficult estimation of the quantity and condition of foliage lost because of disease and senescence; that is, lesions and tissue not present must be estimated and added to the visible ones (15).

If knowing the impact of disease on yield is the goal and HAD is the parameter to be calculated from the observations, the uncertainty and difficulty of estimating total  $x$  are avoided: The  $x$  and leaf areas of eq. 3a are simply the current, visible states of the standing crop. The disease and its impact are logically assessed from host properties that are present and visible, i.e., the amount and duration of the healthy leaf area.

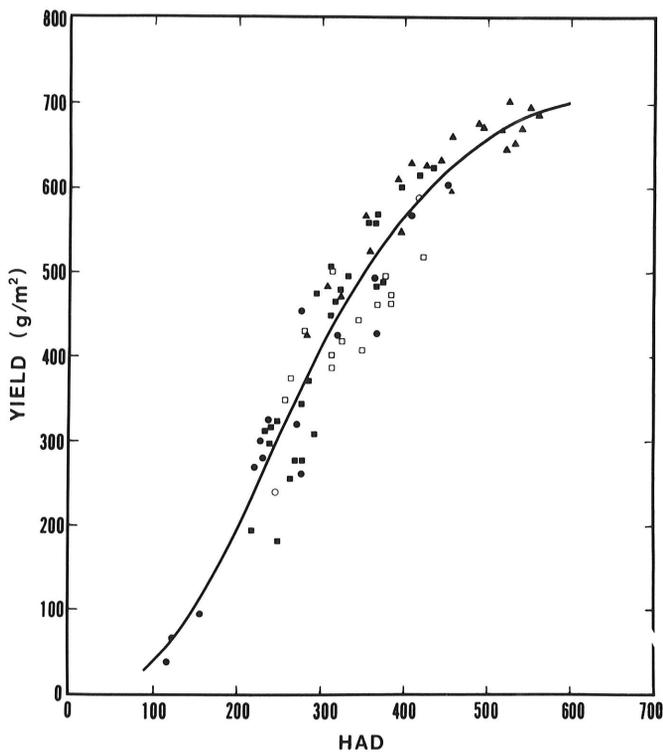


Fig. 1. The healthy leaf area duration HAD and pod yield of 78 crops of peanuts cultivar Florunner grown over a period of 14 yr by five growers. The curve represents a Gompertz equation,  $\text{yield} = \exp\{-3.15 \exp[-.00821 [\text{HAD}-93.71]]\}$ . Data from: (■) Greene and Gorbet (3), (▲) Nickle (13), (●) Wilkerson et al (22), (□) Mangold (10), and (○) Pixley (14).

### THE EXAMPLE OF PEANUTS

Several studies have been made of the leaf area and yield of peanuts that have been defoliated manually and by disease, and they can be used to test the hypothesis that HAD predicts loss from defoliation and disease. We begin with four examples of manual defoliation of one commercial peanut, cultivar Florunner. Wilkerson and co-workers (22) removed 50% of the leaflets from two canopy configurations at several crop stages before two harvesttimes. The leaf area index and light interception for the 15 treatments were measured to determine the effect of time of leaf removal on various plant characteristics, including yield. The coefficient  $K$  for absorption of insolation was 0.412. In a similar investigation, Mangold (10) removed 25, 50, or 75% of the leaflets on several dates. The yield for the 14 treatments was determined at a common harvest date. Nickle (13) removed 25, 50, 75, or 100% of the leaflets uniformly from the canopy on several dates, making 21 treatments. Greene and Gorbet (3) removed 10, 20, 33, or 50% of the tops of peanut canopies by mowing at various times in each of 3 yr and determined yields for 26 treatments.

Although the same cultivar was used in all the experiments, they were conducted by several researchers over a 14-yr period at several locations and under drought as well as normal rainfall. Thus the 76 comparisons provide a severe test of the utility of HAD over a range of environments, leaf area durations, and yields. For the nondefoliated plants of Greene and Gorbet (3) and of Nickle (13), the  $L$  for each week was interpolated between their observations by the model of Wilkerson et al (22). The  $L$  for the defoliated treatments were estimated by subtracting the amount of manual defoliation from the  $L$  of the nondefoliated plants. For each of the 76 treatments, the HAD was calculated by weekly increments:

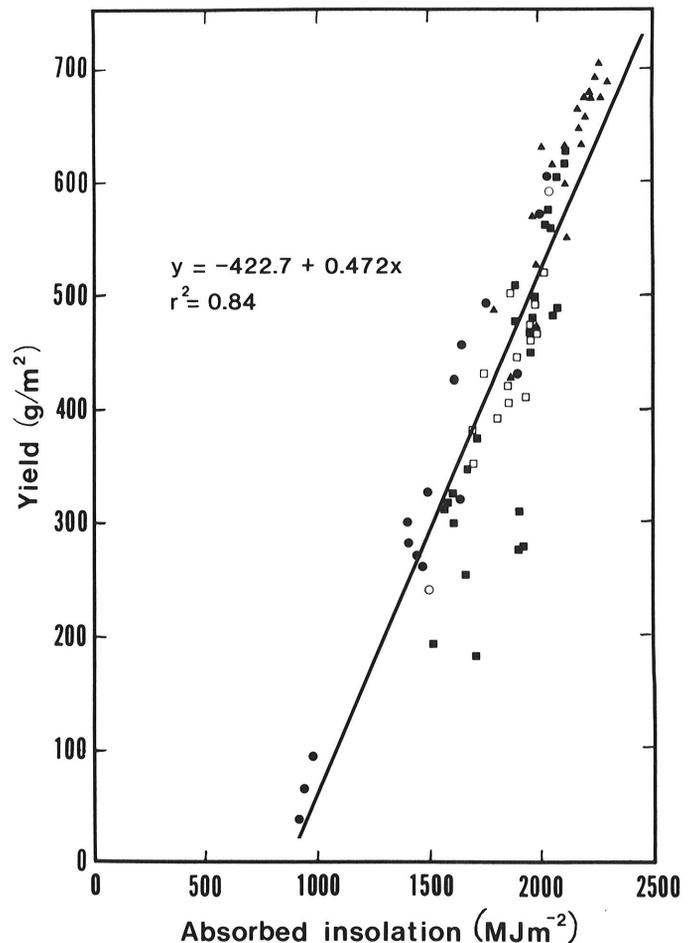


Fig. 2. The healthy leaf area absorption HAA and pod yield of 78 crops of peanuts cultivar Florunner grown over a period of 14 yr by five growers. Data from: (■) Greene and Gorbet (3), (▲) Nickle (13), (●) Wilkerson et al (22), (□) Mangold (10), and (○) Pixley (14).

$$\text{HAD} = \sum \{ [1 - x_i] L_i [t_{i+1} - t_{i-1}] / 2 \} \quad (5)$$

For the 76 cases of manual defoliation,  $[t_{i+1} - t_{i-1}] / 2 = 7$  and  $x = 0$ .

The pod yields for the 76 treatments are related to HAD in Figure 1. Clearly, yield increases with increases in HAD. The decreasing return from very great HAD suggests that yield is more closely related to absorption of insolation than to leaf area itself, and hence HAA was calculated.

$$\text{HAA} = \sum \{ I [1 - x_i] [1 - \exp(-K L_i)] [t_{i+1} - t_{i-1}] / 2 \} \quad (6)$$

Because insolation was not observed, we estimated it as 80% of the insolation at the top of the atmosphere at 30° latitude (9); this  $I$  varies from about 23 MJ m<sup>-2</sup> day<sup>-1</sup> in late March to 30 in late June. Because HAA is an estimate of the integral of eq. 1b, the ratio of yield to HAA should be a conversion efficiency of 1–3 g MJ<sup>-1</sup>.

The linearity of the relation between yield and HAA in Figure 2 and the square of the correlation coefficient, i.e., coefficient of determination, of 0.91 indicate the suitability of HAA as a predictor of the effect of defoliation. The negative intercept of the relation indicates no peanuts are set for very small HAA. The slope of the relation is  $e$  of 0.47 g MJ<sup>-1</sup>. To compare this  $e$  of 0.47 to the  $e$  of other crops requires adjustment for the 6% water in the pods, the dry matter of stem and foliage that equal that in the pods, and the caloric content of peanuts, which is 70% more than, say, wheat (21). After this adjustment, the  $e$  for the peanuts is 1.5, which is comparable to the 1–3 of other crops and provides further evidence of the suitability of HAA as a predictor of yield.

The tests of HAD and HAA as predictors of yield and the specific relations of Figures 1 and 2 were solely from manually defoliated peanuts. Can the concepts of HAD and HAA predict yield from a crop with leaves spotted and defoliated by a pathogen?

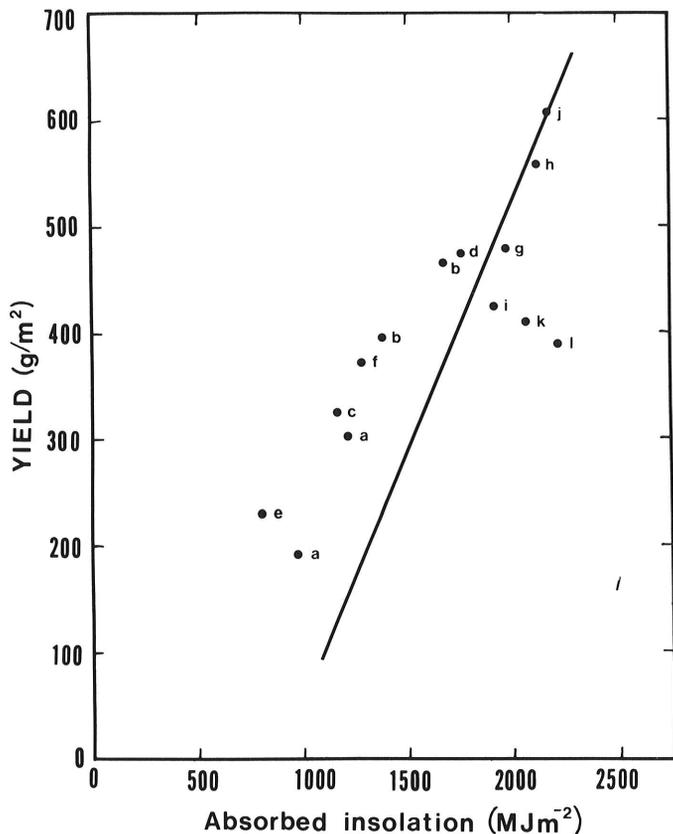


Fig. 3. The healthy leaf area absorption HAA and pod yield of peanut cultivars compared with the relation for Florunner represented by the equation,  $\text{yield} = -422.7 + 0.472 \text{ HAA}$ . Lines grown by Elston et al (2): (a) S38, (b) S38 sprayed with fungicide, (c) TBU, (d) TBU sprayed, (e) F439, (f) F439 sprayed. Lines grown by Pixley (14): (g) F81206, (h) F81206 sprayed, (i) F80202, (j) F80202 sprayed, (k) MA72 × 94-12, (l) MA72 × 94-12 sprayed.

To answer, we again employed eq. 5 and 6, entering an  $x$  for the spotting and remembering that  $L$  is the standing crop of foliage remaining despite defoliation.

Here, we are again fortunate that Pixley (14) protected some Florunner peanuts with a fungicide while allowing others to be naturally infected by *Cercosporidium personatum* and *Cercospora arachidicola*. We calculated HAD days for the sprayed and nonsprayed plants from the  $L$  that he measured at regular intervals. The sprayed plants had  $x$  less than 0.005, HAD of 412 days, and a yield of 590 g m<sup>-2</sup>. The nonsprayed plants had a maximum  $x$  of 0.2, HAD decreased to 237 days by defoliation and disease, and a yield of 240 g m<sup>-2</sup>. Figure 1 shows that these HADs and yields for crops affected by disease fit well within the variation of the manually defoliated crops used to derive the curve drawn on the figure. Similarly, the HAA of 2,053 and yield of 590 of the healthy and the HAA of 1,504 and yield of 240 of the diseased crop fit well within the variation of the manually defoliated crops used to derive the curve of Figure 2.

The five experiments with peanut in Florida provide evidence that yield is simply determined by the duration of healthy leaf area HAD and is linearly related to the healthy leaf area absorption whether the crop is grown by one person or another in one year or another and whether it is defoliated by man or disease. Several qualifications, of course, immediately come to mind, and some can be examined.

Will variety, for example, affect the relation between HAA and yield? In Figure 3, the HAA and yield of three other varieties observed by Pixley (14) are shown by the letters g to l, and Florunner is shown by the curve of Figure 2. The sprayed and nonsprayed varieties indicated by g to j behave as Florunner. Differences in HAA caused by fungicide caused the expected changes in yield.

The peculiar line MA76 × 94-12 indicated by k and l yielded less than Florunner per insolation absorbed, and it actually yielded less when sprayed than nonsprayed. Its behavior suggests that this line partitions relatively more photosynthate to leaves rather than to pods, causing a low harvest index and causing healthy plants with more leaves to yield fewer pods. The curve of eq. 3 can be

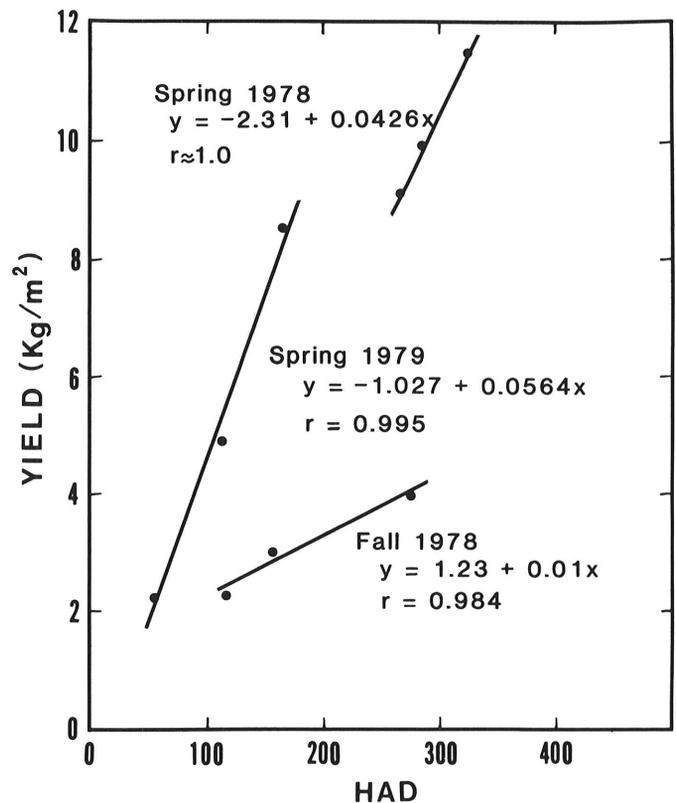


Fig. 4. The healthy leaf area duration HAD and kg m<sup>-2</sup> tuber yield of potatoes grown by Rotem et al (16,17) in three seasons.

considered a measure of the photosynthetic partitioning as well as conversion efficiency of the cultivar Florunner. Breeders have had difficulty obtaining peanut lines with leaf spot resistance that also yield well; i.e., all lines fall below the response of pod yield of Florunner to HAA. Commonly, breeders select lines that have more foliage than susceptible lines at the end of the season, and thus they select lines with high leaf rather than high pod production. Seemingly, lines that are resistant and high yielding might be obtained faster by first selecting ones that partition photosynthate efficiently into pods as shown by a yield/HAA response above Florunner and then incorporating components for greater leaf area and resistance.

The HAA and yield of still three other varieties grown by Elston and co-workers (2) in Africa are also shown on Figure 3. Insolation was estimated for 10° latitude and 80% transmission by the atmosphere, and HAA was calculated from  $L$  without knowing  $x$ . Differences in HAA caused by fungicide caused the expected changes in HAA and yield. Whereas Florunner increased its yield with  $e$  of about 0.5, the African varieties increased theirs with an  $e$  of 0.3; nevertheless, their yields were greater per HAA than Florunner because of a higher intercept of their relation of yield with HAA. The difference in  $e$  between Florunner and Pixley's varieties, on the one hand, and the African crops, on the other, would be less if  $x$  were considered for the African entries. Given the nature of our estimate of  $I$  and other uncertainties, we should not press the comparison too far. Instead we conclude that the conception that yield is related to HAA permits a systematic examination of the peculiarities of varieties.

Although leaves lower in the canopy receive less light, have slower photosynthesis, and produce less yield per area, Wilkerson et al (22) did not find that the defoliation of certain positions caused a markedly different yield than uniform defoliation from all positions. That is, the total HAD or HAA rather than the position of the leaves was evidently the primary determinant of yield, a helpful simplification.

Time of defoliation could be thought to be critical, and the four manual defoliations we examined included defoliations at different times to test that question. The yields of all were, however, fit by the same relation with HAD or HAA, providing no evidence that an HAD or HAA produced by defoliation at one time had a

different effect than at another, which is further helpful simplification.

The important outcome for plant pathology, however, is the similarity of the outcome of reduction of HAD and HAA by manual defoliation to a reduction in HAD or HAA by the severity  $x$  and defoliation of disease. One could easily conjecture that leaves spotted by lesions would have a different respiration, photosynthesis, and contribution to yield than expressed in the simple fraction  $x$ . The evidence of Figures 1 and 2, nevertheless, is that these factors do not greatly affect the relation between HAD or HAA and yield, at least in several peanut cultivars attacked by *Cercosporidium* and *Cercospora* in several years on two continents.

### THE EXAMPLE OF POTATOES

Beginning with the statement, "Studies of crop losses provide another example in which measurement of healthy foliage area makes for a better understanding of the phenomena involved," Rotem et al (16,17) reported the average healthy haulm area during the growth of potatoes attacked by *Phytophthora infestans* and the subsequent yield of tubers. The severity of blight at the end of the seasons varied from about 10% in a crop sprayed with fungicide to 100% in a nonsprayed one. Although they found differences in yield within a season were related to average healthy haulm area, the relations were different for crops grown in spring and fall.

They published enough data about the spring crops of 1978 and 1979 and the fall crop of 1978 to permit our calculating HAD from healthy haulm area. We calculated HAD as the sum of healthy haulm area multiplied by the interval of time and related it to yield in Figure 4. Because growth extended over a longer period in the spring of 1978 than in 1979, Figure 4 shows a difference between the relation of yield to HAD for the two springs as well as between spring and fall. Whereas Rotem et al concluded that yield was related to average healthy haulm area by a single relation, a different relation is required for each year as well as season if HAD is the independent variable.

Eq. 1 and 6 remind us, however, that insolation as well as leaf area duration determine yield. If 80% of the insolation at the top of the atmosphere at 30° latitude reaches the ground,  $I$  changes from 22.6 MJ m<sup>-2</sup> day<sup>-1</sup> on 21 March to 29.5 on 22 June and from 22.3 on 25 September to 11.6 on 22 December (9) and is far from constant for spring and fall crops.

HAA was calculated for a  $K$  of 0.4, which fits transmission of insolation in canopies of potato foliage (1). The  $L$  in eq. 6 was set equal to the healthy haulm plus the lesion area, and the fraction  $(1 - x)$  of healthy area was set equal to (healthy haulm area)/(healthy haulm plus lesion area). When yield is related to HAA in Figure 5, a single regression with slope 8.1 g MJ<sup>-1</sup> fits the data for all seasons with a coefficient of determination of 0.94. Because about 80% of the dry matter of a potato plant is in the tubers (1) and the tubers are about 20% water (21), the slope of 8.1 corresponds to a conversion efficiency  $e$  of 2, which is in the usual range of 1–3 g MJ<sup>-1</sup>. In Figure 4 the HAD of the fall crop failed to produce a proportionate yield because insolation was low, and the very high HAD of the spring crop of 1978 failed to produce a proportionate yield because of the decreasing return in absorption from high leaf areas. A single relation of yield to the absorption of insolation by healthy foliage with an  $e$  similar to that observed by others (1) explains differences among seasons and epidemics.

In another investigation of potatoes Johnson et al (8) reported that the integral of [(1-Defoliation fraction)(1-Blighted × Hopperburned fraction of foliage)] was correlated ( $r=0.8$ ) with the yield of potatoes. Although their integral lacks the  $L$  and  $I$  of the calculation of HAD and HAA, it certainly confirms the importance of defoliation as well as integration over time.

Again, one could easily worry that leaves spotted by lesions would have a different respiration, photosynthesis, and contribution to yield than expressed in the simple fraction  $x$ . The evidence of Figures 4 and 5, however, is that these factors do not greatly affect the relation between HAD or absorbed radiation and yield, at least in potatoes attacked by *Phytophthora*.

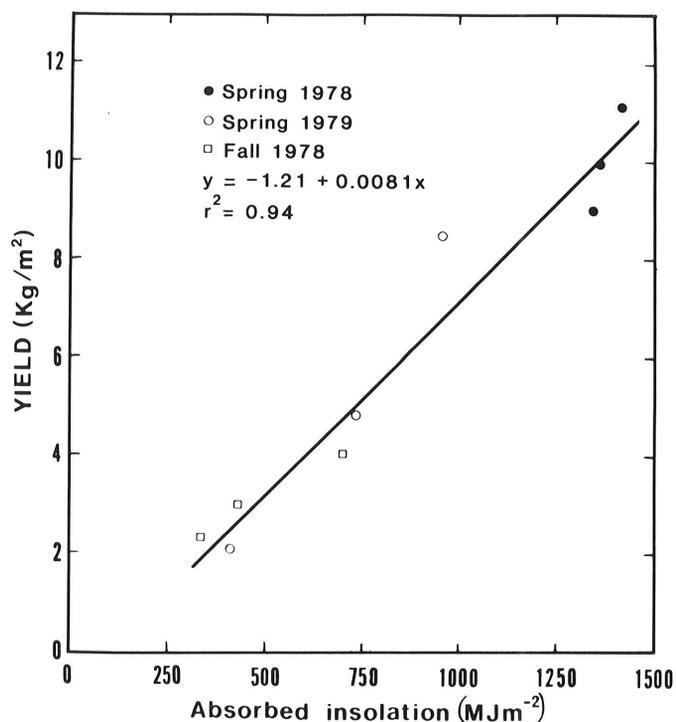


Fig. 5. The healthy leaf area absorption HAA and tuber yield of potatoes grown by Rotem et al (16,17) in three seasons. The curve represents the equation, yield = -1.21 + 0.0081 HAA where the yield is kg m<sup>-2</sup> of tubers.

## THE EXAMPLE OF MAIZE

Beginning with the observation, "Equal increments of differences in disease scores often do not represent equal increments of differences in plant injury as reflected in grain yields," Hooker (6) reported the integral of an index of photosynthesis and growth stage of maize during the filling of grain to predict the decrease in yield caused by manual defoliation or *Helminthosporium turcicum*. The index of photosynthesis incorporated the relative contributions of 10:5:1 from top, middle, and bottom thirds of the canopy of foliage during the filling of grain, and the integral PGI of the product of the two indices increased rapidly at first as defoliation or disease increased and then more and more slowly as the defoliation or disease grew greater. The yield of defoliated as a percentage of the nondefoliated was related to PGI by the regression:  $15.2 + 0.83 \text{ PGI}$  or 0.83% yield per percent of PGI in Hooker's Table 3; this relation with a coefficient of determination of 0.91 is closer than the relation of relative yield to percentage of foliage green, which has a coefficient of determination of 0.79 in Hooker's Table 3. Hooker found that the index of growth stage had little effect.

To test HAA as an alternative predictor of loss, the following were assumed: During the filling of grain, the product of  $K$  and  $L$  was constant at 2, as with  $K = 0.4$  and  $L = 5$ ;  $I$  was steady and its sum during the period was  $1,200 \text{ MJ m}^{-2}$ ; and all dry matter went to the grain. The yield of defoliated and nondefoliated in  $\text{g m}^{-2}$  is related to HAA in  $\text{MJ m}^{-2}$  with a coefficient of determination of 0.81 by the regression:  $124 + 1 \text{ HAA}$ ; i.e., the maize yielded  $1 \text{ g MJ}^{-1}$ , which is in the usual range of  $1-3 \text{ g MJ}^{-1}$  for  $e$ .

The loss caused by *Helminthosporium* was measured in three hybrids. The yield of infected as a percentage of the uninfected plants of the same hybrid can be calculated by the regression equation above, relating yield of defoliated as a percentage of nondefoliated plants to the index PGI. Alternatively, the percentage yield of the infected can be predicted from HAA. Eq. 6 shows HAA is proportional to  $(1 - x)$  when  $K$ ,  $L$ , time, and insolation are constant; that is, under these conditions HAA and yield are proportional to the area above the disease progress curve and loss is proportional to AUDPC. The root mean square difference between the three pairs of actual and predicted yields as percentages of the yields of healthy plants is 16% for prediction from PGI and 8% for the prediction from  $(1 - x)$  justified by eq. 6.

Again, one could easily worry about the location of infected leaves in the canopy and differences in their respiration, photosynthesis, and contribution to yield not expressed in the simple fraction  $x$ . The comparison of the simplicity of eq. 6 with Hooker's careful and detailed analysis that is designed to take these factors into account, however, indicates that they do not greatly affect the relation between HAA and yield, at least in maize attacked by *Helminthosporium*.

## THE EXAMPLE OF WHEAT

Hendrix et al concluded that "The relatively minor role played by cereal foliage in contributing to over-all kernel filling" (5) explained why the manual defoliation decreased wheat yield less than stripe rust. Although they did not report the amount of disease, leaf area, nor defoliation in their bulletin, they clearly stated that the number of plants and the defoliation patterns were precisely the same as those of the inoculated series.

Near Nottingham, England, Green and Ivins (4) observed the effect on wheat of late infestations of take-all (*Gaeumannomyces graminis* var. *tritici*). They measured the total and green area of winter wheat per land area as senescence and take-all reduced them from five in early June to about three total and zero green 2 mo later. They calculated the fraction of photosynthetically active radiation absorbed during the 2 mo by summing  $[1 - \exp(-0.9K L)] / 1.1$  with  $K = 0.44$  and  $L$  the green area index. They wrote that the 26% reduction in radiation interception caused by take-all would account for a similar loss in final dry weight, and in fact, the final reduction in dry weight was 27%, although grain was reduced 55%.

Logically and numerically HAD, and even HAA, differ somewhat from the fraction calculated by Green and Ivins. HAA includes  $I$ , which is about  $15 \text{ MJ m}^{-2} \text{ day}^{-1}$  in Nottingham during the period (11). Also, it assumes senescent as well as green foliage absorbs radiation, and the dry matter is increased by the fraction  $(1 - x)$  of the absorbed radiation. To test HAD and HAA as predictors of loss in wheat, the increments of dry matter in  $\text{g m}^{-2}$  during five periods within the 2 mo were related to HAD in days and HAA in  $\text{MJ m}^{-2}$ . HAA was calculated for  $I = 15$  and  $K = 0.44$ . The regression of yield on HAD had a coefficient of determination of 0.91. The regression:  $-25 + 1.98 \text{ HAA}$ , or  $e$  of  $1.98 \text{ g MJ}^{-1}$ , fit the observations with a coefficient of determination of 0.86. The conversion efficiency,  $e$ , did not differ significantly between healthy and diseased and is in the usual range of  $1-3 \text{ g MJ}^{-1}$ . Although the regression of dry matter on HAA does not fit these data better than a regression on HAD or Green and Ivins' fraction, the logic of all foliage absorbing radiation is compelling, and the opportunity to compare conversion efficiencies adds evidence for the argument.

Again, the ability of the simple concept of HAD and HAA to explain yield of healthy and diseased plants indicates that many complications, such as location of infected leaves in the canopy and differences in metabolism not expressed in  $x$ , do not greatly affect the relation between absorbed radiation and yield, at least in wheat affected by take-all.

## CONCLUSION

The character of the species and the environment determine the size of the factory, the healthy leaf area index; then the accumulated product or yield is determined by the size of the healthy factory integrated during a season, the HAD. Or, more precisely, yield is determined by the energy HAA taken in during the season by the healthy and operating factory from the amount falling on its circumscribed catchment. These conceptions are frameworks for our observations of disease, leaf area, and yield that we can test. If these simple conceptions, like the simple conception that disease increases logarithmically, prove generally true, we shall have standards that will highlight exceptions and differentiate the important from the unimportant processes relating yield to disease.

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