PHYTOPATHOLOGICAL NOTES

Field and Growth Chamber Approach to Epidemiology of Pseudoperonospora cubensis on Cucumbers

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ABSTRACT

Field experiments showed that the outbreak of epidemics caused by *Pseudopercnospora cubensis* on cucumbers depends more on the proximity of a given field to a source of inoculum than on the prevailing temperature. To verify other, inconclusive results reached in field work, mini-epidemics were produced in growth chambers. These epidemics confirmed that the comparative damage depends more on the rate of foliage development of the host than on the weather conditions. Phytopathology 61:736-737.

Additional key words: inoculum, techniques.

The conventional method for studying patterns of disease development is that of field experiments. However, the complexity of field conditions makes it difficult to distinguish between the effects exerted by various factors. Moreover, even when a certain conclusion is reached, it often remains dubious unless verified during many additional years.

Facing this situation in the case of *Pseudoperonosporo cubensis* (Berk. & Curt.) Rost. on cucumbers (*Cucumis sativus* L. 'Bet-Alpha'), we produced epidemics in growth chambers, where the problems in question were studied.

The preceding field work was done on two winter plots (70 m² each) cultivated under plastic tunnels, and three summer-autumn plots (400-600 m²) grown in the open. In each plot, temperature was recorded by a thermograph placed at the plant level. The development of plants was regarded as poor, intermediate, or rich when 33, 66, or 100% of the soil was covered by the foliage, respectively. Disease incidence was assessed by estimating the area of lesions as per cent of the entire foliage area. Analysis of results was done in relation to (i) the effect of proximity of the experimental plot to the source of inoculum; (ii) the effect of seasonal temperatures; and (iii) the effect of plant development.

The effect of inoculum availability on the onset and on the further development of the epidemic was studied by comparing two plots (May-June, Sep.-Oct.) sown next to diseased fields and one plot (June-Sep.) sown in an area apparently free of an inoculum source. It was found that plots cultivated in an infested area became infected soon after sowing, while the comparatively isolated plot remained disease-free for 35 days (Fig. 1).

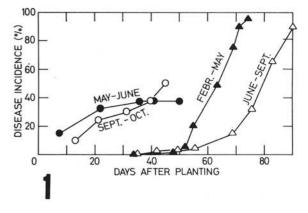
Of the winter plots grown under plastic tunnels, the

Dec.-Feb. plot did not contract the disease. The Feb.sown plot remained disease-free until mid-March, when infected plants were introduced into seven out of ten plastic tunnels. Within these seven tunnels, the disease spread rapidly, but the three noninoculated rows became infected only after the plastic covers were removed from all rows in mid-April.

These results led to the conclusion that the onset of an epidemic is related to the availability of inoculum rather than to seasonal differences. No additional experiments were found necessary to verify this phenomenon.

The seasonal temperatures, whose effect was then analyzed, were expressed as the mean min:max for each experimental period. As seen in Fig. 1, there was no indication that the development of the disease was affected by differences during the season. Since this finding contrasted with reports describing cucumber downy mildew as a hot-climate disease (4, 5), the factor was then studied under controlled conditions.

In order to relate disease development to the degree of foliage development, plots exposed to a similar im-



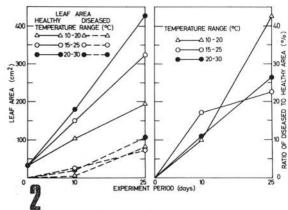


Fig. 1-2. 1) Disease incidence of cucumbers infected with *Pseudoperonospora cubensis* in various seasons, as plotted against plant age with indication of the development of plants. The numbers in parentheses are the mean min:max temperatures in C. 2) The effect of temperature and time on epidemics of *P. cubensis* on cucumbers in growth chambers. (Left) Dimensions of the total and diseased leaf area; (Right) ratio of diseased to healthy area.

pact of inoculum were compared. Of two pairs of such plots (see Fig. 1), plants in the May-June and Sep.-Oct. plots reached a similar grade of plant development. In the second pair, a higher degree of disease was noted on the poorly developed foliage of the Feb.-May plants than on the rich foliage developed in the June-Sep. plot. Since such a conclusion was reached in only two out of five fields, it remained unconvincing; the factor was then studied with epidemics created in growth chambers.

In producing these epidemics, we considered the fact that in the field all events leading to the development of disease occur with a minimum of interference by the researcher. Minimization of such interference in epidemics created in growth chambers was thus an essential condition in the epidemiological studies.

Our epidemics were produced in three growth chambers kept at cycling temperatures with daily min:max of 10:20, 15:25, and 20:30 C, respectively. All chambers worked on similar humidity regimes with a min of 55% relative humidity (RH) during the hottest day-hr, and 12 hr of "dew", produced by humidifiers, during the night period. Wind velocity was kept to a minimum during the night, while an additional blower was working during the day. Light intensity at the 12-hr photoperiod was 1,560 ft-c.

Thirty-five healthy, 1-week-old cucumber plants in 0.5-kg pots were placed in each chamber. As a source of inoculum, one plant with two 4-cm-diam lesions was placed in the center of each chamber for a period of 16 hr; viz, for the first dew period which facilitated sporulation and for the following hours needed for dispersal of the produced sporangia. The set-up was then left without interference. Disease incidence and foliage development were evaluated by tracing the outlines of leaves and lesions on celluloid sheets, and weighing the marked areas. The results were analyzed with the aid of an IBM 360/50 computer, using an analysis of variance for one-way design (3).

Figure 2, left, shows that of the three sets of temperature, the one with a daily min:max of 20:30 C was associated with the greatest increase in total area of leaves (significant at 95% between all treatments) and lesions (significant after incubation of 25 days between 20:30 C and the other two treatments). The data on lesion development indicated that the disease is facilitated by a rather hot climate. However, an opposite indication was found when not the total infected area, but the ratio of infected to healthy area, was calculated. Figure 2, right, shows that this ratio was highest in the 10:20-C treatment; viz, the one under

which the least leaf and a little less disease development occurred. The results of the 10:20-C treatment differed significantly (99%) from the other two treatments after incubation of 25 days. No significant differences were found between the 15:25- and the 20:30-C treatments. In this experiment, the growth chamber results confirmed the field indication that plants with poorer foliage suffer from a comparatively greater damage.

The epidemic patterns of P. cubensis in the field can be explained as follows. The opt temperature for sporulation and infection are in the 15- to 20-C range, but most rapid necrotization occurs at 25 C (1, 2, 6). Since the degree of necrotization is the parameter used in practice for evaluating disease incidence, a rather hot climate is expected to enhance the damage. However, high temperatures also increase foliage development which, according to our results, should decrease the percentage of blighted tissue. The fact that the disease develops best in a hot climate derives from the dependence of epidemic development on the availability of inoculum. Thus, in cool seasons small areas of cucumbers are cultivated and the chance of a large inoculum impact is slight. The damaging development of P. cubensis in summer is therefore related to large and often adjacent areas of cucumbers cultivated in this season, whereby the amount and the transfer of inoculum is increased.

These conclusions, reached by field and growth chamber techniques, stress the importance of the latter in clarification of epidemiological problems.

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