

Effect of Cultivar, Inoculum Dose, and Strain of *Clavibacter michiganense* subsp. *sepedonicum* on Symptom Development in Potatoes

A. L. Bishop and S. A. Slack

Former research assistant and professor of plant pathology, University of Wisconsin-Madison, Madison 53706. Current address of first author: Department of Plant Pathology, Cornell University, New York State Agricultural Experiment Station, Geneva 14456.

Research supported by the College of Agriculture and Life Sciences, University of Wisconsin-Madison, and by Project NC135.

Accepted for publication 27 January 1987 (submitted for electronic processing).

ABSTRACT

Bishop, A. L., and Slack, S. A. 1987. Effect of cultivar, inoculum dose, and strain of *Clavibacter michiganense* subsp. *sepedonicum* on symptom development in potatoes. *Phytopathology* 77:1085-1089.

Inoculation of potato (*Solanum tuberosum*) seed pieces with *Clavibacter michiganense* subsp. *sepedonicum* (= *Corynebacterium sepedonicum*) through wounds beneath sprouts invariably resulted in infection in cultivars Green Mountain, Norland, and Superior at inoculum doses from 10^2 to 10^6 cfu/seed piece, but incidence of infection varied from 70 to 100% in cultivars Katahdin, Ontario, and Russet Burbank. Infection did not affect field stand. Latent period (time from inoculation and planting to wilting of foliage) was significantly shortened by increasing inoculum dose in 1984 but not in 1981. Time of cultivar maturity correlated positively with

length of latent period. The difference between time of symptom onset in infected plants and time of senescence in controls was significantly reduced by increasing inoculum dose. Stunting was a distinct symptom of bacterial ring rot in some cultivars. Strain SS43 of *C. m.* subsp. *sepedonicum* had a significantly shorter latent period than strain SS13 and caused more stunting than strain SS13 in Russet Burbank but not in Katahdin or Norland. Variation in latent period and severity of stunting may affect disease detection in the context of seed potato certification.

Foliar symptoms of bacterial ring rot of potato caused by *Clavibacter michiganense* subsp. *sepedonicum* (5) (= *Corynebacterium sepedonicum* (Spieck. & Kotth.) Skapt. & Burkh.) are known to appear late in the growing season (3,6,10,15). Because detection of diseased plants during field inspections of seed potatoes is one of the most important steps in control of bacterial ring rot, variability of symptom development has a direct impact on disease control. Previous investigators have considered cultivar, temperature, inoculum concentration, interference by other diseases, and inoculum source as factors affecting symptom development (4,11,15-17,20-22). These reports, however, did not provide quantitative data concerning disease development over time, and several lacked appropriate controls. Except for the most recent studies (15,16), inoculum was prepared directly from decayed tuber tissue, such that microorganisms other than *C. m.* subsp. *sepedonicum* may have been included for which there were no appropriate controls. Studies comparing inocula derived from different diseased cultivars were not controlled with regard to inoculum concentration (4,22). Inoculation techniques such as rubbing the cut surface of a diseased tuber on the cut surface of the tuber to be inoculated or dipping cut tubers in a suspension of decayed tissue were used. Both of these methods are fairly efficient, but neither delivers controlled quantities of inoculum.

Parameters in previous field studies included single assessments of incidence of infection and incidence of symptom expression but no quantitative measures of symptom development over time. The effect of inoculum dose on incidence of symptoms was evaluated on two dates in one case, but symptomless infections were not determined (21). Inoculum dose affected incidence of symptomless infection, but disease was assessed only once near the end of the season (15).

Stunting, in addition to wilting, chlorosis, and necrosis, has been reported as a symptom of bacterial ring rot but is infrequently described (1,9,16). The significance of this symptom in disease detection has not been addressed.

Our objective was to determine the effect of pathogen dose and strain and cultivar on latent period (time from inoculation to foliar symptom development) of infections by *C. m.* subsp. *sepedonicum* in field-grown potatoes. The period between symptom expression in inoculated plants and senescence of uninoculated plants (the

time when disease should be most readily detected by visual inspection) was estimated. We have also evaluated the effect of cultivar and pathogen dose and strain on stunting as a symptom of bacterial ring rot.

MATERIALS AND METHODS

Bacterial strains and preparation of inoculum. Strains SS13 (D. Gross) and SS43 (F. Manzer) of *C. m.* subsp. *sepedonicum* were isolated from infected potato (*Solanum tuberosum* L.) plants or tubers. These strains were typical of *C. m.* subsp. *sepedonicum* in culture, Gram stains, serological tests, and eggplant pathogenicity tests. Cultures were maintained by periodic transfer on yeast glucose carbonate slants (YGC) (1% yeast extract, 2% glucose, 2% CaCO₃, 2% agar), and stored for up to 6 mo at 5 C on YGC slants sealed with Parafilm or lyophilized as a suspension in 7% peptone and 7% sucrose for longer periods. Inoculum was cultured at room temperature (about 22 C) on NBY (nutrient broth yeast extract) agar (23). Four-day-old NBY agar cultures were harvested by washing plates with sterile 0.05 M potassium phosphate buffer, pH 7.2. Suspensions were adjusted to $A_{600nm} = 0.1$ with buffer (about 10^8 cfu/ml, estimated by dilution plating). Tenfold serial dilutions of these standardized suspensions served as inoculum.

Inoculation and cultivation of potatoes. Certified seed potato tubers were washed in tap water and sprouted at room temperature for about 2 wk. Two days before planting, tubers were cut into seed pieces (30-70 g) with one eye and were placed in loosely closed plastic bags overnight to heal. Seed pieces were inoculated the next day, 1 day before planting. Potato seed pieces were inoculated by placing 10 μ l of inoculum adjacent to the sprout and inserting a dissecting needle through the inoculum into the tuber tissue to a depth of 0.5-1.0 cm immediately beneath the sprout. The needle was inserted twice more at right angles to the first wound, such that the second and third wounds contacted the first beneath the sprout and withdrawal of the needle drew the inoculum into the seed piece.

Field plots were established at the University of Wisconsin Arlington Experimental Farm in silt loam soil. Fertilizer (6-24-24, 1, 100 kg/ha) and aldicarb (Temik 15G, 3.4 kg a.i./ha) were banded in the furrow at planting in 1981. Fertilizer treatment was the same in 1984, but aldicarb (2.2 kg a.i./ha) was applied after emergence, before second hilling. Seed pieces were hand-planted with the eye uppermost. Postemergence hilling was performed manually.

Metrazin (Sencor, 0.56 kg a.i./ha) was applied before emergence in 1984 to control weeds. All other weed control was performed manually. Rows were 91 cm apart, and plants were on 61-cm centers.

Experimental design. Factorial experiments of cultivar \times inoculum dose and cultivar \times strain were planted in randomized complete blocks. Five cultivars (Green Mountain, Katahdin, Norland, Ontario, and Superior) were compared at four inoculum levels (10^6 , 10^4 , and 10^2 cfu/seed piece and buffer control) of *C. m.* subsp. *sepedonicum* strain SS43 in 1981. Cultivar Russet Burbank was substituted for Green Mountain in 1984. Strains SS43 and SS13 were compared at the highest inoculum level in cultivars Norland and Katahdin in 1981 and in Norland, Katahdin, and Russet Burbank in 1984. Control treatments were inoculated with sterile buffer. Experimental units were groups of 25 plants replicated in three blocks. Plants were scored weekly for percentage of foliage affected by wilting or necrosis. Coding of treatments in 1984 ensured unbiased comparison of controls and inoculated plants.

Stems were examined for the presence of *C. m.* subsp. *sepedonicum* at harvest by cutting at soil level, squeezing them with pliers near the cut end, touching a microscope slide against the sap expressed from the cut surface, and examining Gram stains of the resulting smears with a microscope (17). Smears were considered positive if several 1,250 \times microscope fields with >50 gram-positive coryneform bacteria could be found.

RESULTS

Stand counts. Infection did not affect stand counts in either year. Average stands (all treatments) were 97.7 and 99.1% of planting in

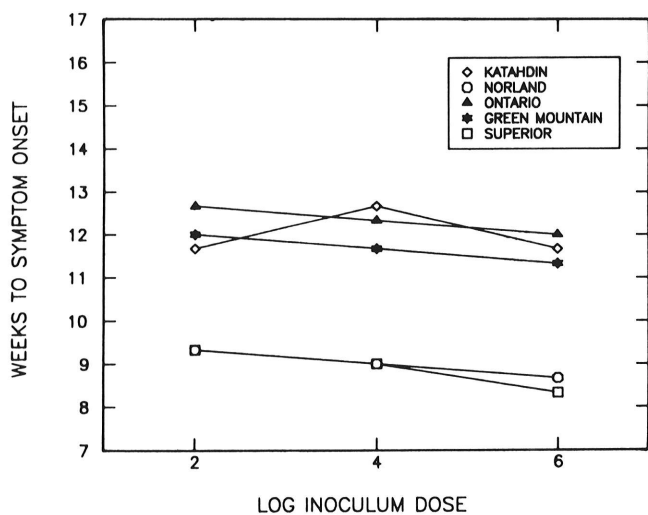


Fig. 1. Effect of inoculum dose of *Clavibacter michiganense* subsp. *sepedonicum* and potato cultivar on latent period, 1981. Significant differences between cultivars were identified as differences in intercepts ($P < 0.01$, $R^2 = 0.920$). Slopes are not significant ($0.10 < P < 0.25$). Points are means of three blocks of 25 plants each.

TABLE 1. Incidence of infection of six potato cultivars inoculated with three doses of *Clavibacter michiganense* subsp. *sepedonicum* strain SS43 in 2 yr as determined by Gram stains of stem smears

Cultivar	Incidence of infection ^a					
	10^6 cfu ^b		10^4 cfu		10^2 cfu	
	1981	1984	1981	1984	1981	1984
Green Mountain	100	nd	100	nd	100	nd
Katahdin	100	100	100	100	96	99
Norland	100	100	100	100	100	100
Ontario	100	97	97	93	75	70
Russet Burbank	nd	88	nd	97	nd	81
Superior	100	100	100	100	100	100

^a Percent infection in three replicates of 25 plants; nd = no data.

^b Inoculum dose in colony-forming units (cfu) per seed piece.

1981 and 1984, respectively.

Effect of inoculum dose on incidence of infection. Stem smears from all plants were Gram-stained at the end of the 1981 season, when uninoculated controls of early cultivars were in an advanced state of senescence; uninoculated controls and plants that did not show wilting symptoms typical of bacterial ring rot, or did so after controls had begun to senesce, were assayed in 1984. All Green Mountain, Norland, and Superior plants were infected at all inoculum levels, whereas some Katahdin, Ontario, and Russet Burbank plants escaped infection (Table 1). Results reported are corrected to 100% incidence of infection.

Effect of inoculum dose on latent period. No significant effect of inoculum dose was found by linear regression analysis of latent period (weeks after planting to symptom onset, where symptom onset = two or more plants with $\geq 5\%$ wilted or necrotic foliage) on log inoculum dose in 1981 ($P > 0.10$). Significant differences among cultivars were identified, however, as differences in intercepts ($P < 0.01$, $R^2 = 0.920$). Norland and Superior had the shortest latent period, followed by Green Mountain, Katahdin, and then Ontario (Fig. 1).

Inoculum dose and cultivar were significant factors affecting latent period in 1984 ($P < 0.001$, $R^2 = 0.904$) (Fig. 2). Linear regression identified differences among cultivars as differences in

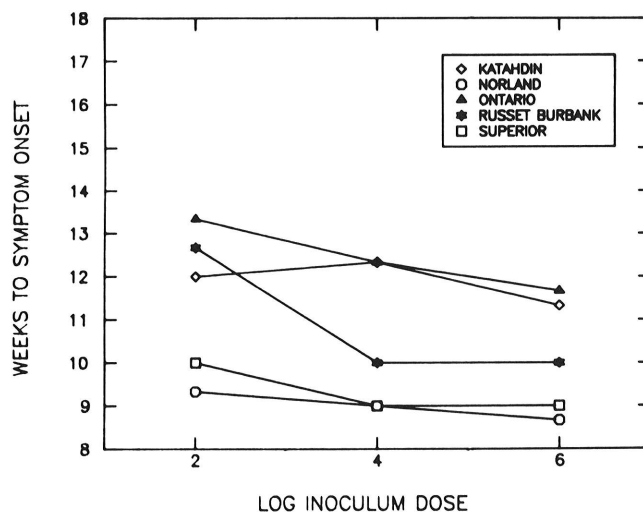


Fig. 2. Effect of inoculum dose of *Clavibacter michiganense* subsp. *sepedonicum* and potato cultivar on latent period, 1984. Significant differences between cultivars were identified as differences in intercepts and slopes ($P < 0.001$, $R^2 = 0.904$). Points are means of three blocks of 25 plants each.

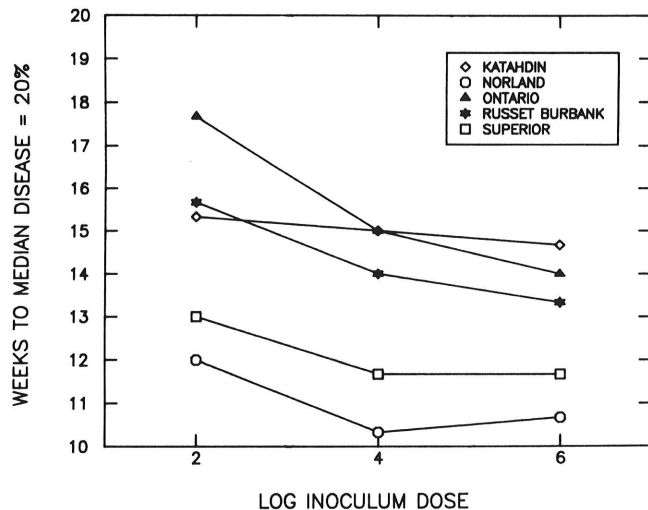


Fig. 3. Effect of inoculum dose of *Clavibacter michiganense* subsp. *sepedonicum* and potato cultivar on advanced latent period, 1984. Significant differences between cultivars were identified as differences in intercepts and slopes ($P < 0.001$, $R^2 = 0.943$). Points are means of three blocks of 25 plants each.

slopes and intercepts in four significantly different lines ($P < 0.05$). Latent period was shorter at high than at low inoculum dose. Slope of the response was greatest in Russet Burbank, followed by Ontario, and then Katahdin, Superior, and Norland. Intercepts for Superior and Norland could not be distinguished from one another but were significantly different from those of the other cultivars.

Assessment of latent period of infection based on a more advanced level of disease (advanced latent period = time from inoculation and planting until 50% of plants have $\geq 20\%$ wilted or necrotic foliage) amplified differences based on cultivar and inoculum dose variation. Advanced latent period was affected significantly by inoculum dose and cultivar in 1984 ($P < 0.001$, $R^2 = 0.943$) (Fig. 3). Linear regression defined a unique relationship of advanced latent period to log inoculum dose for each cultivar. Advanced latent period was shorter at high than at low inoculum dose. Slope was greatest in Ontario, followed by Russet Burbank and then Norland, Superior, and Katahdin. Intercepts correlated roughly with time of maturity of the different cultivars, with early-maturing cultivars (Norland and Superior) showing symptoms earlier than late-maturing cultivars (Katahdin, Russet Burbank, and Ontario). Analysis based on this level of disease was not possible in 1981 because of extensive defoliation of many plants in the 13th week after planting caused by late blight (*Phytophthora infestans* de Bary), before most treatments had reached a median of 20% disease.

Correlation of senescence and latent period. Latent period and advanced latent period in infected plants were highly correlated with the corresponding measures of senescence in the uninoculated controls in 1984 ($R = 0.773$ and 0.893 , respectively). Premature defoliation by late blight prevented similar analysis of data from 1981.

Linear regression of the difference in weeks between time of onset of senescence in controls (two or more plants with $\geq 5\%$ foliage affected) and time of onset of symptoms in infected plants on log inoculum dose indicated a significant effect of dose and cultivar ($P < 0.005$, $R^2 = 0.685$). Differences ranged from 0.7 to 2.7 wk in Superior, Norland, Katahdin, and Ontario. The difference reached 4–5 wk in Russet Burbank at inoculum levels of 10^4 and 10^6 cfu/seed piece (Fig. 4). Analysis of the time separating symptom onset in infected plants and senescence in controls based on more advanced stages of senescence (50% of plants with $\geq 20\%$ foliage affected) and disease (median = 20% disease) was similar to the analysis above, though the effect of inoculum dose was more pronounced. Dose and cultivar were significant factors ($P < 0.005$, $R^2 = 0.656$) (Fig. 5). The response was greatest in Ontario.

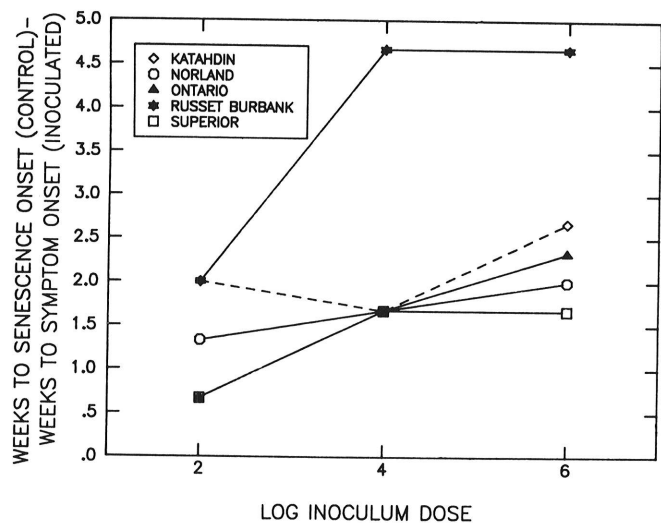


Fig. 4. Effect of inoculum dose of *Clavibacter michiganense* subsp. *sepedonicum* and potato cultivar on difference between time of onset of senescence in controls and time of onset of symptoms in infected treatments, 1984. Dose and cultivar had a significant effect on time to symptom onset ($P < 0.005$, $R^2 = 0.685$). Points are means of three blocks of 25 plants each.

Effect of strain variation on symptom development. Latent period was significantly shorter in plants infected with strain SS43 than in those infected with SS13 in 1981 ($P < 0.025$). Latent period in Norland was shorter than that in Katahdin, regardless of strain. Norland, Katahdin, and Russet Burbank were compared on the same basis in 1984, but a strong interaction term in the analysis of variance ($P < 0.05$) obscured differences between strains. The relationship between SS43 and SS13 was retained despite the interaction; SS43 had a consistently shorter latent period than SS13. Comparison of advanced latent periods distinguished significant differences between strains SS43 and SS13 as well as among cultivars Norland, Katahdin, and Russet Burbank ($P < 0.005$) (Fig. 6). Symptoms developed earlier in plants inoculated with SS43 than in those of the same cultivar inoculated with SS13. Time to median = 20% disease in treatments infected with SS13 was indistinguishable from time to median = 20% senescence in controls.

Effect of inoculum dose and cultivar on plant height. Measurement of plant height (from soil level to canopy top of

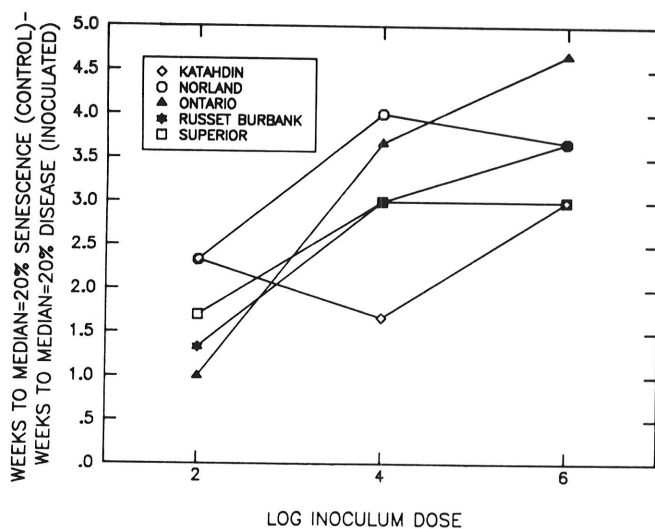


Fig. 5. Difference in weeks between time when controls reached median = 20% senescence (50% of plants with $\geq 20\%$ foliage senescent) and time when plants infected with *Clavibacter michiganense* subsp. *sepedonicum* reached median = 20% disease (50% of plants with $\geq 20\%$ foliage wilted or necrotic) vs. log inoculum dose. Dose and cultivar have a significant effect ($P < 0.005$, $R^2 = 0.656$). Points are means of three blocks of 25 plants each.

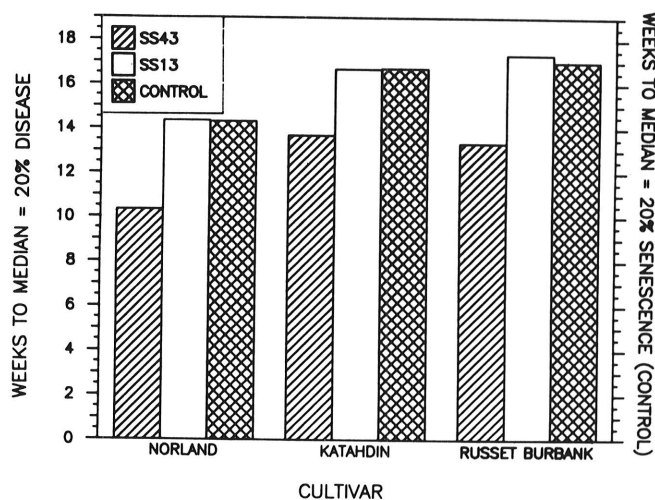


Fig. 6. Effect of strain of *Clavibacter michiganense* subsp. *sepedonicum* and potato cultivar on advanced latent period (infected) and median senescence = 20% (controls), 1984. Symptoms developed more rapidly in plants inoculated with SS43 than in those inoculated with SS13, and Norland displayed symptoms earlier than Katahdin or Russet Burbank ($P < 0.001$); Katahdin and Russet Burbank could not be distinguished on this basis.

individual plants) in cultivar Superior indicated that this cultivar was stunted by infection with *C. m.* subsp. *sepedonicum* in 1981. Treatments were compared by linear regression of percent reduction in height from uninoculated control plants in the corresponding blocks. Inoculum dose had a significant effect on percent reduction in plant height 7 wk after planting ($0.05 < P < 0.10$, $R^2 = 0.927$).

Plant heights of all treatment combinations of cultivar \times dose were measured 7 wk after planting in 1984 (Fig. 7). Effects of log dose and cultivar on reduction in height were significant ($P < 0.001$, $R^2 = 0.843$). Regression analysis indicated separate slopes for Superior and Russet Burbank and a common slope for Norland and Katahdin. Reduction in plant height attributable to infection was not detectable in Ontario.

Effect of strain variation on stunting. Analysis of height reduction in the cultivar \times strain experiment was restricted by the small number of degrees of freedom and the large difference in response between Russet Burbank, Norland, and Katahdin. Analysis of variance indicated a significant interaction between strain and cultivar: Russet Burbank had a greater response to strain difference than Katahdin or Norland; SS43 caused more stunting than did SS13 regardless of cultivar.

DISCUSSION

Previous investigators have treated disease development in bacterial ring rot as a quantal response, scoring plants as either wilted or symptomless (4,15-18,21,22). Data regarding the growth of *C. m.* subsp. *sepedonicum* in planta indicate that the distinction between symptomless and symptomatic infections may be simply a matter of timing, because infections are active throughout the growing season but symptoms do not appear for several months (2). This observation suggested a quantitative parameter, latent period of infection (13,14), which allowed us to discriminate between a variety of cases that could not be distinguished if plants were rated for disease development at only a single time. Latent infection is not an absolute state in this context but is conditioned on time of observation. We interpret instances of symptomless infections as special cases where the latent period is so long that it extends into the period when uninfected potato plants become senescent, are killed by frost, or would be killed with herbicides as a part of the normal production scheme.

Our inability to detect an effect of dose on latent period in 1981, and the small dose effect observed in 1984, may result from the special nature of the potato-*C. m.* subsp. *sepedonicum* interaction. In other systems where the relationship of bacterial dose to latent

period has been described, the host is relatively static developmentally during the latent period (8,14). In potatoes infected with *C. m.* subsp. *sepedonicum*, the latent period encompasses almost the entire period of host growth and development, from vegetative propagule to mature plant. That the small dose response observed in one season should be indistinct in another is not surprising given the variability of the field environment.

The definition of latent period used in 1981 (time until two or more plants had 5% symptoms out of a group of 25) was selected because it was the most advanced level of disease present in some treatments when late blight defoliated much of the plot. It relies on extreme cases to characterize a larger group and is thus subject to greater variability than a more central parameter. The more central parameter, advanced latent period (time until 50% of plants in a treatment showed 20% symptoms), allowed discrimination of dose-response relationships for each of the five cultivars tested in 1984. A disadvantage of advanced latent period is that this stage may not be reached in every season; e.g., frost damaged much of the foliage in the field plot the week after the last treatments reached this level in 1984.

The correlation between latent period and time to senescence in uninfected controls indicated that symptom development might be triggered by physiological changes in the maturing host. The effect of inoculum dose on the interval between symptom onset in infected plants and senescence in uninfected controls indicates that pathogen growth kinetics specific to cultivars may play a role in determining this interval. There are two practical implications of this observation: 1) the senescence of healthy foliage can be expected to interfere with disease detection, particularly at low inoculum doses and in certain cultivars where the interval between symptom onset in infected plants and senescence in uninfected controls is short, and 2) it may be possible to predict the onset of symptoms by using predictive models for host growth and development, given appropriate weather and cultivar data, and a model capable of predicting growth stages of the potato (19).

Comparison of latent periods of infection for strains SS13 and SS43 indicated that, even at high doses, there is potential for the latent period to extend into the onset of senescence, rendering disease undetectable in field inspections. An important limitation of this experiment is our lack of knowledge regarding variation among strains of *C. m.* subsp. *sepedonicum* in the potato crop at large. If more strains not aggressive enough to cause symptoms sufficient for detection are to be found, extensive field surveys will be required, including sampling of fields where ring rot is not detected by visual inspection. Many seed potato programs now limit the number of generations over which a seed stock may be propagated and require that the initial propagation material be tested directly for *C. m.* subsp. *sepedonicum*, minimizing undetectable infections (6,12).

The reduction in incidence of infection observed at low inoculum doses of *C. m.* subsp. *sepedonicum* in cultivars Katahdin, Ontario, and Russet Burbank indicates that there was some resistance to infection in these cultivars. This effect, minimal even at very low inoculum doses, is probably of little consequence in a field setting. Resistance to this type of inoculation does not indicate that similar resistance to natural infections would be found, because our inoculation technique, designed to deliver uniform quantities of inoculum to the sprout, does not mimic the natural mode of infection. In the field, inoculum may reside in the vascular system of seed tubers or be introduced to the cut faces of seed pieces during cutting and handling.

Stunting of infected Superior and Russet Burbank was obvious by casual observation, especially at higher inoculum doses. Stunting in Norland and Katahdin, while detectable by direct measurement, was not obvious in inspections of the plot. Documentation of a significant relationship between inoculum dose of *C. m.* subsp. *sepedonicum* and stunting in this study indicates that stunting may be useful in disease detection in some cultivars.

The most important conclusion to be drawn from these studies is that, in certain predictable cultivar \times dose or cultivar \times strain

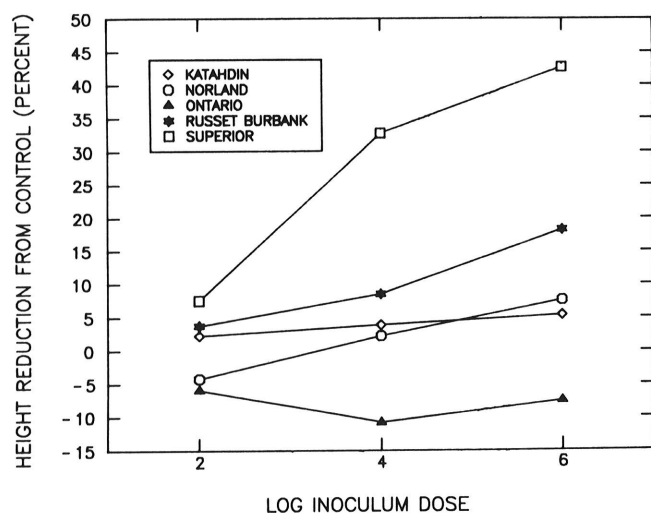


Fig. 7. Effect of inoculum dose of *Clavibacter michiganense* subsp. *sepedonicum* and potato cultivar on reduction in plant height 7 wk after planting, 1984. Effects are significant ($P < 0.001$, $R^2 = 0.843$). Regression analysis indicates separate slopes for Superior and Russet Burbank and a single slope for Norland and Katahdin. Reduction in plant height was undetectable in Ontario.

combinations, bacterial ring rot may be undetectable, or detectable only for a short time, by visual inspection in the field. Detection efficiency may be improved by inspection schedules adjusted to the time of senescence of the cultivar inspected. This would be facilitated by the development of models capable of predicting the time of senescence of potatoes and development of symptoms of bacterial ring rot.

LITERATURE CITED

1. Baribeau, B. 1948. Bacterial ring rot of potatoes. *Am. Potato J.* 25:71-83.
2. Bishop, A., and Slack, S. A. 1981. Population levels of *Corynebacterium sepedonicum* and symptom development of ring rot in potato plants. (Abstr.) *Phytopathology* 71:861.
3. Bonde, R. 1939. Bacterial wilt and soft rot of the potato in Maine. *Maine Agric. Exp. Stn. Bull.* 396. 16 pp.
4. Bonde, R., and Covell, M. 1950. Effect of host variety and other factors on pathogenicity of potato ring rot bacteria. *Phytopathology* 40:161-172.
5. Davis, M. J., Gillespie, A. G., Jr., Vidaver, A. K., and Harris, R. W. 1984. *Clavibacter*: A new genus containing some phytopathogenic coryneform bacteria, including *Clavibacter xyli* subsp. *xyli* sp. nov., subsp. nov. and *Clavibacter xyli* subsp. *cyonodontis* subsp. nov., pathogens that cause ratoon stunting disease of sugarcane and Bermudagrass stunting disease. *Int. J. Syst. Bacteriol.* 34:107-117.
6. De Boer, S. H., and Slack, S. A. 1984. Current status and prospects for detecting and controlling bacterial ring rot of potatoes in North America. *Plant Dis.* 68:841-844.
7. Dykstra, T. P. 1942. Compilation of results in control of potato ring rot in 1941. *Am. Potato J.* 19:175-196.
8. Ercolani, G. L., and Crosse, J. E. 1980. The growth of *Pseudomonas phaseolicola* and related plant pathogens *in vivo*. *J. Gen. Microbiol.* 45:429-439.
9. Guthrie, J. W. 1959. The early dwarf symptom of bacterial ring rot of potato in Idaho. *Phytopathology* 49:453.
10. Kreuzer, W. A., Glick, D. P., and McClean, J. G. 1941. Bacterial ring rot of potato. *Col. Agric. Exp. Stn. Press Bull.* 94. 12 pp.
11. Logsdon, C. E. 1967. Effect of soil temperature on potato ring rot. *Am. Potato J.* 44:281-286.
12. Manzer, F., and Slack, S. A. 1979. Report of the pathology section committee on bacterial ring rot diagnosis. *Am. Potato J.* 56:551-555.
13. Meynell, G. G. 1957. Inherently low precision of infectivity titrations using quantal response. *Biometrics* 13:149-163.
14. Meynell, G. G., and Meynell, E. W. 1958. The growth of microorganisms *in vivo* with particular reference to the relation between dose and latent period. *J. Hyg.* 56:323-346.
15. Nelson, G. A. 1982. *Corynebacterium sepedonicum* in potato: Effect of inoculum concentration on ring rot symptoms and latent infections. *Can. J. Plant Pathol.* 4:129-133.
16. Nelson, G. A., and Howard, R. J. 1982. Effect of the ring rot pathogen and latent potato viruses on ring rot symptoms and yield of potatoes. *Am. Potato J.* 59:213-219.
17. Paquin, R., and G n reux, H. 1976. Effet du climat sur la fl trissure bact rienne de la pomme de terre en relation avec le contenu en sucres des tiges. *Can. J. Plant Sci.* 56:549-554.
18. Racicot, H. N., Saville, D. B. O., and Conners, I. L. 1938. Bacterial wilt and rot of potatoes—some suggestions for its detection, verification and control. *Am. Potato J.* 15:312-318.
19. Sands, P. J., Hackett, C., and Nix, H. A. 1979. A model of the development and bulking of potatoes (*Solanum tuberosum* L.) I. Derivation from well managed field crops. *Field Crops Res.* 2:309-331.
20. Sherf, A. F. 1944. Infection experiments with potato ring rot and the effect of temperature on the disease. *Am. Potato J.* 21:27-29.
21. Starr, G. H. 1947. The effect of different concentrations of bacterial suspensions upon subsequent ring rot symptoms in potato plants. *Am. Potato J.* 24:151-156.
22. Starr, G. H., and Riedl, W. A. 1948. A comparison of *Corynebacterium sepedonicum* inocula from resistant and susceptible varieties. *Am. Potato J.* 25:432-437.
23. Vidaver, A. K. 1967. Synthetic and complex media for the rapid detection of fluorescence of phytopathogenic pseudomonads: Effect of the carbon source. *Appl. Microbiol.* 15:1523-1524.