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Biology of infection of common bacterial blight (Xanthomonas phaseoli (E.F. Sm.) Dows.) on white bean (Phaseolus vulgaris L.).

Lucia Pei Chu
University of Windsor

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BIOLOGY OF INFECTION OF
COMMON BACTERIAL BLIGHT (*XANTHOMONAS PHASEOLI* (E.F.Smn.) DOWS.)
ON WHITE BEAN (*PHASEOLUS VULGARIS* L.)

by

LUCIA PEI CHU

B.S.,1965, M.S.,1967, National Taiwan University

A Thesis

Submitted to the Faculty of Graduate Studies through the
Department of Biology in Partial Fulfilment
of the Requirement for the Degree
of Master of Science at the University
of Windsor

Windsor,Ontario,Canada
1969
DEDICATION

To my parents, Mr. and Mrs. Y. K. Chu, who taught me to be independent, and to learn through personal experiences.
ABSTRACT

The biology of infection of common bacterial blight (Xanthomonas phaseoli (E.F. Sm.) Dows.) on white bean (Phaseolus vulgaris L.) was studied with regard to changes in symptomology, anatomy and leaf pigments during early stages of the post-inoculation period. The susceptibility of 10 varieties of beans to one isolate of common blight was tested. A resistant and a susceptible variety were grown under three light intensities and the development of blight in each was compared. For the resistant variety, reduction in lesion size, and increase in lesion number and pigment value, resulted from reduced light intensity. The opposite occurred for the susceptible variety. The most susceptible and resistant varieties tested were Seaway No. 65 and Clipper, respectively. The development of a stem canker, the progressive disorganization of infected leaf mesophyll, and the invasion of vascular elements were photographed from stained sections.
ACKNOWLEDGMENTS

The writer wishes to acknowledge her sincerest thanks to Professor W.G. Benedict for his expert and innumerable suggestions during the course of these studies and in the preparation of the manuscript.

I also wish to gratefully acknowledge the financial assistance provided by the Department of Biology, University of Windsor, and by Grant No. 728 of Dr. Benedict from the National Research Council of Canada.

Special thanks are due to Miss M. Manton and Mr. R. Winmill for their help in the preparation of the final graphs, and Mr. N. K. Yong for assistance with photographs.
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INTRODUCTION

Common bacterial blight of bean (*Phaseolus vulgaris* L.) is caused by *Xanthomonas phaseoli* (E.F. Sm.) Dows., 1939, whose synonymy is *Bacillus phaseoli* E.F. Sm., 1897, *Pseudomonas phaseoli* E.F.Sm., 1901, *Bacterium phaseoli* E.F.Sm., 1905, and *Phytomonas phaseoli* (E.F.Sm.) Bergey et al., 1923. Common blight was first reported in the United States from New York and New Jersey in 1892 but the disease is widespread in the United States and Canada east of the Continent Divide and in Europe and other continents where beans are grown under environmental conditions favourable for the disease (Walker, 1969). Common blight is an important transit disease of fresh wax- and green-podded varieties.

The first symptoms of common blight are small translucent watersoaked spots on leaves. The spots arise around stomata invaded by the bacterium and are about 1 mm in diameter. The leaf tissue between spots may turn yellow and die to form lesions of necrotic tissue of various shapes and sizes. Small spots also appear on bean pods and some seeds are infected from the pod wall. Watersoaked spots or streaks may appear on stems and petioles and especially on the pulvinus. As common blight progresses, the vascular system becomes discoloured and stem cankers may form at the first node which causes a joint rot and the plant breaks over. Systemic infection extends through the funiculus into seeds which become shrivelled and discoloured.

When infected seeds are overwintered and sown, the bacterium develops in the testa of the germinating seed and con-
taminates the surface of the swollen cotyledons, penetrating cracks in the cuticle and progressing intercellularly until the vascular system of the seedling is reached. Systemic infection gives rise to leaf lesions and stem cankers (Zaumeyer, 1932). Primary local infection can take place through stomata by inoculum from infected cotyledons and overwintered diseased vines. Secondary inoculum from primary lesions is spread by wind-borne rain, dust, implements, man and animals.

Common blight is favoured by high air temperatures and may develop rapidly under favourable conditions (Patel & Walker, 1963). Once inside the plant, the bacterium migrates through the intercellular spaces of parenchyma tissue, dissolving the middle lamella slightly in advance. The affected cells collapse and breakdown of tissue gives lysigenous cavities. In the xylem cells the bacterium advances systemically and, after multiplication, may rupture or dissolve the cell walls and reenter the parenchyma tissues in other parts of the plant. The bacterium may then emerge through stomata on the stem and enter other stomata on the same stem (Zaumeyer, 1932).

Isolates of _X. phaseoli_ vary in degree of virulence when collected in nature and maintained in pure culture but no varieties of _P. vulgaris_ are free from symptoms after heavy inoculation with the bacterium (Coryne et al., 1963). The physiology of host-parasite interaction has been demonstrated by comparing the rate of bacterial increase in resistant and susceptible species of Phaseolus (Scharen, 1959). The bacterial count rises rapidly, declines, and increases again upon the appearance of symptoms. The second rise, however, is much
slower in the tolerant and resistant species than in the susceptible varieties of *Phaseolus vulgaris*.

The purpose of the present study was to investigate the biology of infection of *X. phaseoli* in a "resistant" and a "susceptible" variety of *P. vulgaris* and to determine the effect that light intensity has on symptom expression and leaf-pigment value when the plants were grown and infected under controlled environmental conditions that favoured the pathogen.
MATERIALS AND METHODS

White bean (*Phaseolus vulgaris* L.) seed, varieties California Flat White, Gratiot, Michelite No. 62, Saginaw and Sanilac were obtained from Green Giant of Canada Ltd., Tecumseh, Ontario (1966 crop), Seaway, Seaway No. 65 and Clipper from the Soil Substation, CDA Research Branch, Woodslee, Ontario (1967 crop), and Kinghorn Wax and Red Mexican from the Cell Biology Research Institute, CDA Research Branch, Ottawa, Ontario. These varieties were selected for a broad range of susceptibility to common bacterial blight.

Before sowing the seed, it was disinfected for 5 minutes in a solution of 0.1% HgCl₂ followed by 3 rinses in distilled water. The seeds were sown 2.5 cm deep in steamed Brookston Clay loam with small additions of sheep manure, pulverized limestone and peat moss. The seedlings were grown in 12.5-cm clay pots in plant growth chambers (Benedict, 1964) at 24°C, 70-95% relative humidity and 9100 lumens during a 14-hour day. After 5 to 8 days the seedlings were transplanted, one per pot, into 10-cm pots filled with Perlite and watered twice weekly with 50 ml of chelated fertilizer (15-30-15 NPK plus trace elements) prepared by dissolving 7.5 gm/l distilled water.

The pathogen, *Xanthomonas phaseoli* (E.F.Sm.) Dows., obtained from the Cell Biology Research Institute, CDA Research Branch, Ottawa, Ontario, was subcultured on nutrient agar. Inoculum was prepared from 48-hour cultures, with approximately
7.8 \times 10^7 \text{ bacterial cells per ml sterile distilled water.}

Healthy, 7- to 10-day-old seedlings with hypocotyls 3-4 cm and well-developed unifoliate leaves were inoculated in the lighted growth chamber by immersing the upper part of the plant, including the cotyledons, in the bacterial suspension for two hours. Applying the inoculum to the leaf surface by rubbing, spraying or needle punctures consistently gave unsatisfactory infections.

In order to alter light intensity in the growth chambers, a chamber was made into smaller compartments by light-proof partitions and each compartment shaded with varying layers of cheesecloth as required. Light intensities utilized were 1750, 5300 and 9100 lumen/m$^2$.

As symptoms developed on the unifoliate leaves or trifoliate leaflets, the individual translucent watersoaked spots or, later, individual necrotic lesions were counted by transmitted light with a stereoscopic microscope. The spots and lesions on each leaf or leaflet were counted as long as they were discrete, 20 leaves per treatment, and their numbers averaged. The experiments with varying light intensity were repeated three times. The standard deviations of the means were determined for each treatment. Some photographs were taken of infected leaves.

Leaf pigments were extracted from the infected resistant variety Sanilac, susceptible variety Seaway No.65 and from uninoculated leaves and leaflets of these varieties every 4th day following inoculation, a total of 3 extractions. Prior to
extraction, the leaves were weighed as they were excised from the plants in the chamber. The pigments, chlorophyll A and B, and carotenoids were determined spectrophotometrically at 452, 644 and 663 μm, respectively, using a Bausch & Lomb Spectronic 20 and the equations of Röbbelin (Wolf, 1963).

\[
\begin{align*}
\text{Chlorophyll A} &= 10.3 \left( \log \frac{I_{0}}{I} \right)_{663} - 0.918 \left( \log \frac{I_{0}}{I} \right)_{644} \\
\text{Chlorophyll B} &= 19.7 \left( \log \frac{I_{0}}{I} \right)_{644} - 3.87 \left( \log \frac{I_{0}}{I} \right)_{663} \\
\text{Carotenoids} &= 4.75 \left( \log \frac{I_{0}}{I} \right)_{452} - 0.226 (\text{ChA} + \text{ChB})
\end{align*}
\]

During each 29-day experiment, for histological treatments, petiole and leaf pieces were collected on the 3rd, 7th and 14th day after inoculation and fixed in Zenker's fluid (Gatenberg and Beams, 1950). Dehydration utilized tertiary butyl in the alcohol series. Embedding was in tissuemat. The plant material was sectioned both transversely and longitudinally at 10 μm on a Leitz-Wetzler Rotary microtome. Sections were deparaffinized, and stained with Flemming's triple, safranin-fast green, ruthenium red, periodic acid-Schiff's reagent, and toluidine blue O following standard procedures (Johansen, 1940; Pearse, 1960). The stained plant parts were examined microscopically and appropriate photographs made.
RESULTS

I. Symptomology

A. The diseased plant

When 2-week-old bean plants were infected, the first symptoms to appear after 2 or 3 days were angular, water-soaked spots on the lower surfaces of the unifoliate leaves. Some veinlet-clearing also occurred at this time (Plate I). About 1 week later, the water-soaked spots became necrotic lesions 122 mm in size surrounded by a chlorotic zone 2-3 mm in width in susceptible varieties (Plates II and III, Figs. 4 and 6). At this time reddish-brown lesions on the petiole and pulvinus caused the leaf to wilt (Plate II, Fig. 4). Also, elongate lesions on the epicotyl caused stem cankers with bacterial exudate. Similar lesions occurred at the cotyledonary node which caused the plant to break over later.

B2. Effect of light on symptom expression in a resistant and a susceptible variety

Typical symptoms for the resistant variety Sanilac and the susceptible variety Seaway No. 65 are shown in Plate III, Figs. 5 and 6, respectively. The small water-soaked spots in Sanilac gave enlarged necrotic lesions whereas in Seaway No. 65 the small spots became necroses surrounded with conspicuous chlorotic areas. Shading with 6 and 12 layers of cheesecloth, Plate IV and V, respectively, reduced the size of the necrotic lesions on the resistant
variety particularly with 6 layers of cloth shade.

II. Histology

A. Stem-canker formation

The formation of a stem canker is shown in Plates VI-VIII. The epidermal cells in the vicinity of the infection at first hypertrophied and then collapsed as cell division in the underlying cortex tended to wall off the lesion.

B. Stomatal penetration

In Plate IX, Fig. 16 the bacterium invaded the sub-stomatal cavity on the lower leaf surface and multiplied intercellularly in the mesophyll. Later the bacterium was found throughout the mesophyll. Some disorganization of the tissue occurred (Plate IX, Fig. 17). A petiole is shown in Plate XI, Fig. 20 infected by means of stomatal penetration and intercellular migration of the bacterium.

C. Cellular dissociation

The dissolution of pectic substances in the walls between adjacent mesophyll cells was seen in advance of bacterial migration in the tissue by staining sections of the tissue especially with ruthenium red (Plate X, Plate XI, Fig. 21 and Plate XII).

D. Vascular invasion

Invasion of the vascular system by the bacterium was confirmed by finding bacterial cells in the xylem elements (Plate XIII and XIV).

III. Varietal susceptibility

Results of tests of the susceptibility of ten bean
varieties to common blight showed that most varieties probably fall into one of two groups based upon the number of leaf lesions (Table 1) which appear during the first 10 days after infection. The more susceptible varieties tested produced 100-200 lesions per leaf 4 days after inoculation. The number of lesions increased only slightly during the next 6 days, except in variety California Flat White which showed another increase in lesions 4 days after the initial rise in lesion number. Such susceptible varieties included California Flat White, Gratiot, Michelite No.62, Saginaw, Seaway and Seaway No.65. The more resistant varieties tested, namely, Clipper, Kinghorn Wax, Red Mexican and Sanilac, produced 0-50 lesions per leaf 4 days after inoculation with an increase during the next 4 days and a further increase thereafter, except in variety Clipper which consistently developed only a few lesions per leaf (Fig. 27).

IV. Leaf-pigment analyses

The effects of varied light intensity on the leaf pigments in an infected resistant and an infected susceptible variety during the first 12 days following inoculation are shown in Table 2 and 3, and Figures 28-30.

Compared with the control, the infected leaves of the resistant variety contained significantly more chlorophyll A, B and carotenoids at each light intensity studied. This occurred at each analysis except for two leaf samples, both of which were grown under 12 layers of cloth shade for 4 days following inoculation, when the chlorophyll A and carotenoid
content was much less in the infected leaves. In contrast, the infected leaves of the susceptible variety contained significantly less chlorophyll A, B and carotenoids at each light intensity studied, with the exception of all analyses for those leaves which were grown under 12 layers of cloth shade.
DISCUSSION

In 1962 Klement and Lovrekovich in their studies on host-parasite relations of bean pods infected with bacterial pathogens concluded that no bacteriostatic reactions are induced in the sensitive host plant, but that the multiplication of phytopathogenic bacteria non pathogenic to bean were inhibited by a postinfectional defense mechanism and, saprophytic bacteria were inhibited by a factor preinfectionally present in the bean. Later, in 1968, Klement reviewed the fate of phytopathogenic bacteria in compatible and incompatible hosts. Following infection, a primary specific factor was the lack of ability of the pathogen to induce a defense reaction. Then secondary nonspecific factors resulted in the formation of toxins and enzymes that initiated disease development. Conditions for further multiplication of the bacterium were thereby insured. Winstead and Walker (1954) and Husain and Kelman (1958) had shown that pectic and cellulolytic enzymes of Pseudomonas solanacearum (Granville wilt) in pathogenesis involves the breakdown of host tissue. Smith (1958) showed that two other species of bacterium, Xanthomonas phaseoli and X. translucens (black chaff of wheat) produced pectin glycosidase and pectin methyl esterase only in infected plants.

In the present investigation, Xanthomonas phaseoli, the causal agent for common bacterial blight of bean, was assured entry through stomata when the seedlings were inoculated by immersion in a bacterial suspension in the lighted
growth chamber. A differential effect of light intensity on disease severity could be induced by post-inoculation shading of especially the more resistant varieties of bean. The development of stem canker, an important aspect of the disease, can be shown by serially sectioning a portion of the infected stem and staining the sections with various dyes. The dissolution of pectic materials in advance of cellular dissociation in the mesophyll could be shown by staining infected tissues with ruthenium red. Systemic movement of the bacterium was evident because bacterial cells could be found in xylem elements of all parts of the infected plants. The degree of susceptibility of a variety could be found by inoculating seedlings and counting the number of lesions produced per leaf within 10 days of inoculation. By extraction and analysis of the chlorophyll A and B, and carotenoids the effect of the bacterium on leaf pigments during early stages in the development of the disease could be studied.

The results show that bean variety Clipper is virtually resistant to the strain of *X. phaseoli* used in these experiments. The results correspond to those of Gallegly and Walker (1949) and Buddenhagen and Kelman (1964), who showed that low light intensity following inoculation with bacterial wilt of tomato caused an increase in disease development by *Pseudomonas solanacearum* under similar conditions.
SUMMARY

1. Typical symptoms and signs of common blight were obtained successfully when white bean seedlings were immersed in a suspension of the bacterium *Xanthomonas phaseoli* and grown in a suitable controlled environment for disease development.

2. A differential host-parasite relationship in a resistant and a susceptible variety was induced by varying the light intensity during early stages of the post-inoculation period. Bean varieties more tolerant to common blight showed a variation in size and number of lesions, and in pigment values, when grown in increasing shade than less tolerant varieties.

3. Standard histochemical techniques were utilized to study stem-canker formation, stomatal penetration, cellular dissociation and vascular invasion by the bacterium causing common blight of bean.
LITERATURE CITED


PLATES I-XIV

Figures 1-26, inclusive

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PLATE I

Comparison of a healthy versus a diseased unifoliate bean leaf with first symptoms 3 days after inoculation.

Fig. 1. Healthy leaf. X 7

Fig. 2. Diseased leaf showing veinlet-clearing near the midrib. X 7
PLATE II

Comparison of healthy versus diseased bean leaves and stem 7 days after inoculation.

Fig. 3. Healthy plant. X 1

Fig. 4. Inoculated unifoliate leaves with necrotic and chlorotic lesions. Petiole and pulvinus lesioned. Epicotyl with stem canker. Infected cotyledon and hypocotyl lesions. X 1

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PLATE III

Comparison of leaf symptoms on a resistant and a susceptible bean variety lighted with 9100 lumens/m² 14 hours per day.

Fig. 5. Resistant var. Sanilac. X 7

Fig. 6. Susceptible var. Seaway No. 65. X 7

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PLATE IV

Comparison of leaf symptoms on a resistant and a susceptible bean variety lighted with 5300 lumens/m² 14 hours per day.

Fig. 7. Resistant var. Sanilac. X 7

Fig. 8. Susceptible var. Seaway No. 65. X 7.
PLATE V

Comparison of leaf symptoms on a resistant and a susceptible bean variety lighted with 1750 lumens/m² 14 hours per day.

Fig. 9. Resistant var. Sanilac. X 7

Fig. 10. Susceptible var. Seaway No. 65. X 7
PLATE VI

Stem-canker formation

Fig. 11. Early stage showing hypertrophy of epidermal cells and initial division of cortical cells to wall off the lesion. X 3,000. Safranin-Fast Green.

Fig. 12. A few hypertrophied epidermal cells have collapsed, cortical cells have undergone division to wall off the lesion. X 3,000. Safranin-Fast Green.
PLATE VII

Stem-canker formation (continued)

Fig. 13. Some hypertrophied epidermal cells have collapsed and further cell division has occurred in the cortex. X 3,000. Safranin-Fast Green.

Fig. 14. The canker is evidenced by the collapse and discoloration of infected epidermal cells. Further cell division in the cortex has occurred. X 3,000. Safranin-Fast Green.
PLATE VIII

Stem-canker formation (concluded)

Fig. 15. Sunken lesion caused by collapsed and discoloured infected epidermal cells walled off by cell division in the underlying cortex. X 3,000. Safranin–Fast Green.
Bacterial invasion of the stomata and intercellular spaces of bean leaf

Fig. 16. Stomatal invasion in the lower epidermis and multiplication in intercellular spaces of the leaf mesophyll. X 1250. Toluidine Blue O.

Fig. 17. Invasion of intercellular spaces with some disorganization of the leaf mesophyll. X 625. Safranin-Fast Green.
PLATE X

Comparison of healthy and infected bean-leaf mesophyll tissue.

Fig. 18. Healthy mesophyll. Paradermal section. X 1250. Periodic acid-Schiff's reagent.

Fig. 19. Infected mesophyll showing disorganization of tissue and cell contents. Paradermal section. X 1250. Periodic acid-Schiff's reagent.
PLATE XI

Bacterial infection of bean petiole and the resulting disorganization of tissue.

Fig. 20. Petiole infected by stomatal penetration. X 1250. Ruthenium Red.

Fig. 21. Disorganization of chlorenchyma in petiole. X 1250. Safranin-Fast Green.
PLATE XII

Comparison of healthy and infected bean petioles.

Fig. 22. Healthy. X 1250. Ruthenium Red.

Fig. 23. Infected tissue and the open stoma through which the bacterium invaded the substomatal and intercellular spaces. X 1250. Ruthenium Red.
PLATE XIII

Vascular tissue invaded by the bacterium.

Fig. 24. Xylem elements in a vascular bundle of the stem. X 1250. Flemming's triple stain.

Fig. 25. Xylem elements in a leaf petiole. X 1250. Flemming's triple stain.
PLATE XIV

Vascular tissue invaded by the bacterium.

Fig. 26. Xylem elements in a young leaf petiole. X 1250, Toluidine Blue O.
TABLES 1-3

Figures 27-30, inclusive
<table>
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<th>Variety</th>
<th>No. of Local Infections per Leaf</th>
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<tr>
<td>Mexican Wax</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>Citrus mexican</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Red Sanjiao</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Kianghion</td>
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<td></td>
</tr>
<tr>
<td>Melonette</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pint White</td>
<td>65</td>
<td></td>
</tr>
<tr>
<td>Seaweed California</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seaweed Sanjiao</td>
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Each number is the mean of twenty leaves ± standard deviation.

Table 1. Compartive Susceptibility of ten bean varieties to common blight.
Table 2. Effect of three light intensities upon the development of leaf pigments in bean, variety Sanilac, resistant to common blight

<table>
<thead>
<tr>
<th>Pigment</th>
<th>Days After Inoculation</th>
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<tr>
<td>No Shading 9100 lumens /m²</td>
<td>Chlorophyll A</td>
<td>C 4.69 ± 1.14 *</td>
<td>5.80 ± 1.05</td>
<td>6.23 ± 0.91</td>
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<td></td>
<td></td>
<td>I 5.25 ± 1.04</td>
<td>5.90 ± 0.97</td>
<td>6.82 ± 1.08</td>
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<tr>
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<td>Chlorophyll B</td>
<td>C 2.83 ± 1.03</td>
<td>4.13 ± 0.96</td>
<td>4.84 ± 1.01</td>
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<td>I 4.11 ± 0.98</td>
<td>4.78 ± 1.05</td>
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<td>Carotenoids</td>
<td>C 3.34 ± 1.38</td>
<td>3.62 ± 1.30</td>
<td>3.91 ± 1.29</td>
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<td></td>
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<td>I 3.54 ± 1.34</td>
<td>4.18 ± 1.24</td>
<td>5.91 ± 1.34</td>
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<td>Chlorophyll A</td>
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<td>I 4.28 ± 1.20</td>
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<td></td>
<td>I 2.55 ± 1.10</td>
<td>5.80 ± 0.84</td>
<td>5.95 ± 1.09</td>
</tr>
<tr>
<td>Chlorophyll B</td>
<td>C 3.77 ± 1.04</td>
<td>3.92 ± 0.84</td>
<td>4.54 ± 0.97</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>I 3.99 ± 0.99</td>
<td>4.53 ± 0.89</td>
<td>4.72 ± 1.01</td>
</tr>
<tr>
<td>Carotenoids</td>
<td>C 3.41 ± 1.37</td>
<td>3.82 ± 1.31</td>
<td>4.24 ± 1.41</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>I 2.09 ± 1.22</td>
<td>4.15 ± 1.28</td>
<td>4.95 ± 1.32</td>
</tr>
</tbody>
</table>

* Each number is the mean of three experiments ± standard deviation

C : Control

I : Inoculated

** single thickness of cheesecloth

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Table 3. Effect of three light intensities upon the development of leaf pigments in bean, variety Seaway No. 65, susceptible to common blight

<table>
<thead>
<tr>
<th>Pigment</th>
<th>Days After Inoculation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>4 Days</td>
</tr>
<tr>
<td>Chlorophyll A</td>
<td>C 5.48</td>
</tr>
<tr>
<td></td>
<td>I 4.10</td>
</tr>
<tr>
<td>Chlorophyll B</td>
<td>C 4.33</td>
</tr>
<tr>
<td></td>
<td>I 3.09</td>
</tr>
<tr>
<td>Carotenoids</td>
<td>C 3.58</td>
</tr>
<tr>
<td></td>
<td>I 2.87</td>
</tr>
<tr>
<td>Chlorophyll A</td>
<td>C 4.48</td>
</tr>
<tr>
<td></td>
<td>I 4.01</td>
</tr>
<tr>
<td>Chlorophyll B</td>
<td>C 3.73</td>
</tr>
<tr>
<td></td>
<td>I 3.15</td>
</tr>
<tr>
<td>Carotenoids</td>
<td>C 3.00</td>
</tr>
<tr>
<td></td>
<td>I 2.69</td>
</tr>
<tr>
<td>Chlorophyll A</td>
<td>C 4.26</td>
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<tr>
<td></td>
<td>I 4.29</td>
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<tr>
<td>Chlorophyll B</td>
<td>C 3.14</td>
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<tr>
<td></td>
<td>I 3.41</td>
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<tr>
<td>Carotenoids</td>
<td>C 2.68</td>
</tr>
<tr>
<td></td>
<td>I 2.97</td>
</tr>
</tbody>
</table>

* Each number is the mean of three experiments ± standard deviation

C : Control
I : Inoculated

** single thickness of cheesecloth

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Fig. 27. A comparison in 10 bean varieties of the number of local lesions per leaf which developed during the first 10 days after inoculation with the bacterium causing common blight. Standard deviation from the means are shown by vertical lines in the graph. (See also Table 1).
Fig. 28. A comparison of the chlorophyll A in controls and infected resistant and susceptible bean varieties grown under increasing shade for 12 days after inoculation with the bacterium causing common blight. (See also Tables 2 and 3). (layers=single thickness of cheesecloth)
Fig. 29. A comparison of the chlorophyll B in controls and infected resistant and susceptible bean varieties grown under increasing shade for 12 days after inoculation with the bacterium causing common blight. (See Tables 2 and 3). (layers=single thickness of cheesecloth)
Fig. 30. A comparison of the carotenoids in controls and infected resistant and susceptible bean varieties grown under increasing shade for 12 days after inoculation with the bacterium causing common blight. (See Tables 2 and 3). (layers=single thickness of cheesecloth)
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