II. REVIEW OF LITERATURE
A comprehensive and critical review on bacterial blight (translated into English by Hiroshi Fujii) which covered the publications on this disease up to 1962 has been published by Tagami and Mizukami (1962). This comprehensive review has been freely drawn up in the present review. Additional information has been added on the basis of published literature since 1962 till March, 1977.

A. MORPHOLOGICAL FEATURES AND PHYLLOPLANE MICROFLORA OF RICE CULTIVARS IN RELATION TO DISEASE DEVELOPMENT

1. Morphological features of the host in relation to disease development

Kiryu and Mizuta (1955a; b) analysed morphological features of resistant, intermediate and susceptible cultivars and found that cultivars with few, short, narrow leaves with less horizontal spread of the leaves were more resistant than those with luxuriant growth or spreading leaves which tended to increase humidity amongst plants while wounds and frequent contact of leaves favoured disease dissemination and development. Broad leaf types were associated with more disease (Sulaiman and Ahamed, 1965).

According to Singh and Rao (1971), height, leaf angle and indica and japonica type of grain did not hold any consistent relationship with resistance or susceptibility in pin-prick inoculations. Rolled and narrow leaves conferred resistant reaction whereas flat and broad leaf characters showed resistance very rarely. Mutant with dark green,
glabrous and slow senescent leaves showed high frequency of resistant plants than light green, pubescent and rapidly senescent leaves. According to them, the pubescent leaves provided sticking surface to the pathogen and slow, senescent leaves might have had a carbon/nitrogen ratio which did not favour the growth of the pathogen. Fang et al. (1963) also found that late maturing and glabrous cultivars were more resistant.

But, amongst the different morphological features of rice cultivars studied by Premalatha Dath (1974) under pin-prick and spray inoculation methods, no correlation was obtained between disease development and narrow or broad, thick or thin, and pale or dark green leaves. However, a strong correlation was observed between hairy and glabrous leaf texture and disease development in spray inoculation. Hairy cultivars suffered significantly more disease than glabrous cultivars under natural field conditions, though both were equally susceptible when pin-prick inoculated with the pathogen. Large number of different types of hairs were found arising over the veins and also in the interveinal zones on the surface of the lamina of hairy cultivars and they were lacking on the surface of glabrous ones. Though both the cultivars had marginal hairs, they were more in number, bigger in size and closely placed in hairy leaves whereas in glabrous leaves, the marginal hairs were few in number, small in size and widely placed. However, she failed to observe any
clear-cut differences in the internal anatomical features between the hairy and glabrous cultivars. Therefore, she presumed that the hairs helped in the successful entry of the pathogen either by providing more entry points to the pathogen when the hairs broke due to mechanical injury, or by providing more congenial microclimate over the leaf surface.

2. Phyllopance microflora in relation to disease development

Pandey (1970) observed that saprophytic bacteria largely belonged to Erwinia and Pseudomonas were not antagonistic to X. oryzae in vitro. Moreover, the culture filtrate of the Pseudomonas sp. supported the growth of the pathogen in a medium free from organic nitrogen.

However, some of the epiphytic bacterial (International Rice Research Institute, 1970; Isaka, 1973; Nwigwe, 1973; Hsieh and Buddenhagen, 1974; Uma Gupta, 1975) and fungal flora (Uma Gupta, 1975) were found to reduce the disease development when mixed with the inoculum and then inoculated on susceptible rice plant by employing various inoculation methods. The proportion of epiphytic bacterium with X. oryzae in the inoculum seemed to influence the disease development. The higher the proportion of the epiphyte in the mixture the greater was the reduction in infection (International Rice Research Institute, 1970; Isaka, 1973; Nwigwe, 1973; Hsieh and Buddenhagen, 1974).
The inhibition of the pathogen in vitro was either due to lowering of the pH to a level uncongenial for the multiplication of the pathogen (Hsieh and Buddenhagen, 1974; Uma Gupta, 1975) or due to the production of some antibiotic principle (Uma Gupta, 1975).

Spraying the epiphytic antagonists before inoculating the test plants with the pathogen was more effective than spraying the antagonists after inoculation in reducing the disease intensity. When the pathogen was inoculated simultaneously with the antagonists, the infection was either totally inhibited or reduced to some extent depending upon the antagonists involved (Isaka, 1973; Uma Gupta, 1975).

Isaka (1973) reported that the phylloplane bacteria increased especially in August. The number of bacteria on phylloplane showed a increasing tendency more rapidly by heavy fertilization than by the normal fertilization and the number of phylloplane bacteria were more on diseased leaves than on healthy leaves. The suppressing effect of the phylloplane bacteria was independent of the virulence of X. oryzae isolate used for inoculation.

It was surprisingly difficult to recover X. oryzae by agar techniques from leaf surfaces showing bacterial ooze (Buddenhagen, 1969), or from leaves showing too advanced stages of infection (Rao and Srivastava, 1971). Presence of high phage population was throught to be the reason for the failure in isolating X. oryzae by these authors.
Induction of resistance was studied by Watanabe et al. (1976) in rice plant - *X. oryzae* system by primarily inoculating with incompatible strains or non-pathogenic bacteria and then challenging with a compatible strain. Lesion enlargement was markedly inhibited by the preliminary inoculation with incompatible strains of *X. oryzae* but not with compatible strains. Inhibition was higher in the region located closer to the site of primary inoculation and was significant even in the tissue located 5 cm apart from the site of primary inoculation. Inhibition was highest when challenge inoculation was made 3 days after inoculation and remained effective until 9 days after pre-inoculation. The non-pathogenic bacteria, however, did not induce detectable resistance in the region located more than 3 cm from the site of inducing interaction when challenge inoculation was made 6 or more days after the primary inoculation. According to these authors, neither nutritional competition nor production of antibacterial substance by these bacteria seemed responsible for this type of induced resistance, at least not with *X. citri* and *P. tabaci*.

B. HOST NUTRITION IN RELATION TO DISEASE DEVELOPMENT

1. Nitrogen

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Kuwazuka, 1933; 1938; Japanese food crop disease and control, 1947; Hashioka, 1951; Yamanaka et al., 1952; Kondo and Komura, 1953; Yasu and Iida, 1956; All prefectures in Hokuriku, 1959; Inoue, 1960; Takano et al., 1960; Inoue and Tsuda, 1961; Kulkarni, 1962; Indian Council of Agricultural Research, 1964; Sulaiman and Ahamed, 1965; Sulaiman et al., 1965; Mizukami, 1966; Mukherjee et al., 1966; Park, 1965; 1966; Ray et al., 1967; All-India Coordinated Rice Improvement Project, 1970; Central Rice Research Institute, 1970; Devadath and Padmanabhan, 1970; Mahmood and Singh, 1970; Pandey, 1970; Padhi and Chakrabarti, 1971; Ten Have and Kauffman, 1972; Chattopadhyay and Mukherjee, 1973; Premalatha Dath, 1974; Padhiit and Mishra, 1975; Uma Gupta, 1975). However, according to Srivastava (1972), heavy fertilization was only complementary to epidemics and was not the primary cause.

As far as the individual lesions were concerned, higher nitrogen in the soil or culture solution did not seem to affect the size of the lesions (International Rice Research Institute, 1966; 1974; Ou et al., 1971). In contrast, Devadath and Padmanabhan (1970), Matsuaki et al. (1972) and Uma Gupta (1975) observed an increase in the spread of the lesion with an increase in the level of nitrogen. Further, Matsuzaki et al. (1972) reported that the percentage of infected leaves were much less in plants with low nitrogen as compared to plants grown under normal levels of nitrogen.
2. Combination of nitrogen, phosphorus and potassium

Combinations of NPK fertilizers also influenced the disease development. Insufficient phosphate or potash and late or heavy top dressing of these two fertilizers increased the disease (Soga, 1918). Disease incidence was high with heavy application of nitrogen, phosphorus or potash, but the incidence was brought down with the application of the latter two or when the three fertilizers were applied in combination, especially with potash (Matsuda, 1928; Kuwazuka, 1933). Sulaiman et al. (1965) observed that application of nitrogen along with lower doses of potash, tended to increase the disease. But when higher doses of potash were applied, the disease incidence decreased.

At every level of nitrogen, addition of potassium over 60 kg/ha reduced the lesion length. Least lesion length was obtained at N_{180}K_{180} whereas maximum length was recorded at N_{180}K_60 combinations (Devadath and Padmanabhan, 1970). Nitrogen in combination with phosphate increased the disease (Sulaiman et al., 1965). However, application of 40 lb of nitrogen, 20 lb of phosphate and 20 lb of potash per acre reduced the incidence and intensity of the disease (Sulaiman and Ahamed, 1965; Sulaiman et al., 1965). Potassium and phosphorus at twice the normal level tended to stimulate the lesion development (Kim and Cho, 1970).

Disease was severe in poorly drained paddy fields wherein the fertilizers were available to the crop for longer
time (Kojima et al., 1954; Kojima, 1954). In flooded areas, differences produced by fertilizers were not distinct, but when not flooded, the disease was severe in more nitrogen applied fields (Yasu and Iida, 1956).

3. Reaction of rice cultivars at different levels of fertilizers

Nitrogenous fertilizers, especially, inorganic ones increased the disease but the reaction varied with rice cultivars (Kondo and Komura, 1953; Central Rice Research Institute, 1970). The lesion development was not significant between two nitrogen levels but it was significant among the cultivars (Ou et al., 1971). With addition of potassium the lesion length in IR 8 was brought down significantly than in Taichung (Native) 1 (Ranga Reddy and Sridhar, 1975; Uma Gupta, 1975). However, according to Nishihara (1959), the varietal differences for each fertilizer were less distinct than the local environmental differences.

4. Effect of other fertilizer elements

With regard to other essential elements besides nitrogen, phosphorus and potassium, excess or late fertilization of shale, silicate and magnesium tended to increase the disease occurrence (Kojima et al., 1954, 1955; Inoue, 1960; Takano et al., 1960; Inoue and Tsuda, 1961). The severity of the disease was proportional to the amount of shale soil applied. In this case, the base absorption
capacity \( \text{SiO}_2, \text{Mg} \) of rice plants increased and fertilizer became active in the later period (Kojima et al., 1954). Heavy disease occurrence was seen where shale was the mother rock and this was related to a higher clay content, much salt like silicate and magnesium and a high fertilizer holding capacity which might have caused absorption at a late growth stage (Kojima et al., 1954; 1955; 1959).

Excess silicate and magnesium increased the susceptibility, especially late fertilization with potash and silicate (Inoue, 1960; Inoue and Tsuda, 1961). According to Kumamoto and Hayashi (1959), calcium silicate did not influence the disease.

5. Fertilizer deficiency

Disease increased when the fertilizer was exhausted (Mizukami, 1957) and particularly at the active tillering stage (Nasuda and Takeuchi, 1960).

C. VARIATION IN VIRULENCE OF SINGLE COLONY ISOLATES

There was not only a great deal of variation in virulence between the isolates of the causal bacterium, but also wide variations existed among the colonies of the individual isolate. It was found that among single celled colonies obtained from a virulent clone of the bacterium, several non-virulent strains were recovered (Tagami and Mizukami, 1962; International Rice Research Institute, 1967; 1969; Ou, 1972; Una Gupta, 1975).
X. oryzae was liable to dissociate in the medium into wild and mutant colony types. The colonies of the wild type were characterised by opaque, wax yellow or whitish yellow, slimy and larger colonies. In contrast the mutant type colonies were translucent and yellow or deep yellow in appearance and small in size (Goto and Okabe, 1967; Nwigwe, 1973; Premalatha Dath, 1974). The wild type colonies were more virulent than the mutant type (Goto and Okabe, 1967; Goto, 1972; International Rice Research Institute, 1972; Reddy, 1974; Premalatha Dath, 1974; Raoof, 1975).

Goto (1972) obtained colonial mutants of 7 bacterial isolates. The phage susceptible wild colony type (WS) was highly virulent. On the contrary, most clones of phage resistant mutant colony type (TR) were either virulent or showed marked attenuation in virulence. But the virulence of phage resistant wild colony type (WR) was entirely different from that of 'WS' and 'TR' types. Some clones were as virulent as those of most virulent 'WS' clones and others were as low in virulence as the least virulent 'TR' ones. On the contrary, Nwigwe (1973) observed no appreciable differences in the virulence of the colony types.

D. MULTIPLICATION OF THE PATHOGEN IN RICE CULTIVARS

Multiplication of the pathogen after entry was closely related to the resistance or susceptibility of the host. In susceptible cultivars, the pathogen could proceed to
logarithmic period after 24-48 hr of initial lag phase and increased rapidly as much as hundreds of thousands at which the disease was initiated. In resistant cultivars, the pathogen began to increase similarly after initial lag phase, but stationary period soon followed before they multiplied enough for disease initiation, and the disease was retarded (Sekiya and Watanabe, 1957; Watanabe et al., 1957).

In susceptible cultivar which was inoculated by wounding, the bacteria did not multiply but rather decreased at first, and thereafter, they rapidly increased until or even after symptoms appeared. In resistant cultivar, similar tendency was seen at first, but the following increase was not so remarkable. Bacteria ceased to increase after symptoms appeared (Mizukami and Seki, 1956; Sekiya and Watanabe, 1957; Watanabe et al., 1957; Kusaba et al., 1957; Mizukami, 1961; Watanabe and Asaumi, 1975).

Reddy and Kauffman (1973) determined the multiplication and movement of *X. oryzae* periodically in resistant (BJ 1) and susceptible (Taichung (Native) 1) cultivars of rice. In both the cultivars the pathogen multiplied equally well at the site of inoculation. One cm away from the inoculation point, however, a lower rate of multiplication was observed in BJ 1 than in Taichung (Native) 1. Multiplication and movement of the bacterium was faster in Taichung (Native) 1 than in BJ 1 where only restricted
lesions developed. The mechanism of slow multiplication and movement in BJ 1 was, however, not known.

Lesion initiation and expansion were more rapid in the susceptible cultivars JC 70 and IR 8 than in the resistant cultivar Zenith. In advanced lesions, the bacterial population was always highest at the advancing tip than in the older parts, and 3-week-old lesions contained few or no X. oryzae cells, indicating that the 'organism' preferred the living parts of the host tissue to the dead portions (Nwigwe, 1973).

According to Devadath (1969), the multiplication of the bacterium in rice cultivars was closely related to the degree of virulence of the isolate. Virulent isolate multiplied more rapidly in resistant and susceptible cultivars than the less virulent isolate. Further, the multiplication of both the isolates was more in the susceptible cultivar than in the resistant cultivar. The virulent isolate multiplied and reached a population of $10^5$ cells/ml to produce visible symptoms in both the susceptible and resistant cultivars though the multiplication rate was less in resistant cultivar whereas the less virulent isolate reached a population level of $10^5$ cells/ml only in susceptible cultivar to produce symptoms but never reached to that level to produce symptoms in the resistant cultivar.
According to Premalatha Dath (1971), multiplication of the wild colony type (virulent) was many times more than that of the mutant colony type (less virulent) in resistant and susceptible cultivars. Multiplication of both the colony types was less in the resistant cultivar than in the susceptible cultivar. The frequency of attenuation to mutant type from wild type was less in the resistant cultivar than in the susceptible cultivar. However, the back mutation to wild type from mutant type was many times more in the resistant cultivar than the susceptible cultivar.

Mohiuddin and Kauffman (1975) studied the multiplication of X. oryzae isolates in compatible and incompatible host-isolate combinations. It was observed that population trends at the inoculation point were generally similar both in the compatible as well as in the incompatible host-pathogen system. In DJ 1, isolate H66 which was less virulent had a similar multiplication pattern as that of H100 which was moderately virulent on this cultivar. Population trends of both isolates started at about $1 \times 10^5$ bacteria/leaf disc at 30 min and increased to 100 times ($1 \times 10^8$ bacteria/leaf disc) at 6 days after inoculation. Similar trend was noticed with both the isolates on Wase aikoku.

The population trends of the bacterium were markedly different at a distance of 1 cm below the point of inoculation as compared with those at the inoculation point. In the compatible host-isolate system, the bacterial population was
higher than that in the incompatible host-isolate systems when assayed at 6-10 days after inoculation. In Taichung (Native) 1 the population trends were similar for both the isolates as the host-isolate system was compatible with both the isolates.

Each isolate had a remarkably similar population trend at the inoculation point in BJ 1, Wase aikoku and Taichung (Native) 1. Population trends were also similar at 1 and 3 cm below the inoculation point for the compatible system.

A decline in the population of the bacterium was observed in Wase aikoku and Taichung (Native) 1 at 8 and 10 days after inoculation at 1 cm and 12-14 days after inoculation at 3 cm from inoculation point which was probably due to the necrotic lesion advancing into these areas in many of the leaves. On BJ 1, necrosis did not develop so rapidly.

Goto (1972) mixed wild (WS) and mutant (TR) colony types in different proportions and inoculated to rice plants to study the multiplication of these colony types in relation to disease development. Results showed that the disease severity was reduced as the proportion of WS were reduced in the mixed inoculum. Addition of TR to WS did not significantly depress symptom expression. Reduction in disease severity was accompanied by decrease in the total
population which appeared to reflect only the proportion of WS in the inoculum. The proportion of TR in the population varied as TR proportion in the inoculum, but was inversely related to the disease severity and the total population. The disease severity was essentially a reflection of the proportion of WS in the inoculum and the total population in the diseased tissue was dependent on the growth of WS. Interactions occurred in the infected leaf tissues and that the multiplication of TR colony type was restricted by the rapid growth of WS colony type. The less virulent clones back mutated to virulent clones and when this back mutation became dominant in some of the inoculated leaves, the disease extended faster.

The multiplication of virulent and less virulent isolates mixed in different proportions was studied in a resistant cultivar BJ 1 by Reddy and Kauffman (1974). It was found that the multiplication pattern of H_{144} (less virulent) in BJ 1 was similar individually or when mixed at 95:5 and 50:50 ratio of H_{144}:H_{100} isolates (virulent). The population trends of H_{100} was similar in various combinations except at a mixture of 95:5 ratio of H_{100}:H_{144} where the population was reduced 10 fold as compared to other combinations. The total population of both the isolates at this combination, however, was similar to the total population at other combinations and when the isolates were individually inoculated.
Fujii (1976) reported that when virulent strain was mixed with avirulent strain and inoculated to rice leaves, multiplication of virulent strain was inhibited by the addition of avirulent strain. When avirulent strain alone was inoculated, it multiplied during 10-15 days within 2-3 cm distance around the inoculation points though no visible lesion was formed on the leaf. When avirulent and virulent strains were mixed and inoculated, bacterial population of avirulent strain in leaves was much increased, and at the same time avirulent strain was distributed within wider area in leaves.

E. ROLE OF PLANT ROOTS IN THE ACTIVATION OF THE PATHOGEN

Wakimoto (1957) had demonstrated for the first time the swarming action of the pathogen on rice roots by immersing the roots of rice plants in the bacterial suspension. The bacteria swarmed onto the roots leaving the suspension transparent. The swarmed bacterial cells from such roots could be re-suspended when the roots were shaken in clear water. This swarming phenomenon was non-specific and could be observed both on host and non-host plants. It was presumed that such action was due to the bacterial response to sugars, amino acids, or oxygen in water secreted by plant roots (Wakimoto, 1957; Mizukami, 1959; 1961).

According to Mizukami (1961), the phenomenon was closely related to the chemical properties of water, such as
low and high pH, various solutes such as calcium phosphate, calcium hydroxide, calcium nitrate, straw ash and heavy metallic fungicides. The bacterial cells present in water were likely to swarm to the parts of high metabolic activity of an iron enzyme, a kind of a peroxidase (\( Fe^{++} \rightarrow Fe^{+++} \)). It was further seen that *X. oryzae* swarmed to the parts at which the redox potential (\( Eh \)) was very high.

Mizukami (1959; 1961) had reported that the static state of the pathogen buried in soil and bacterial cells that were weakened or lost the reproductive ability in the culture medium by the treatment of heat, dilute bactericides and ageing, recovered and became virulent when the bacterial cells succeeded to contact the roots of rice plant. Thus, the roots of rice plant seemed to play an important role in turning the static bacterial cells to aggressive cells to infect the host plant. Though the pathogen could multiply to some extent on the roots of other plants, the rate was low (Mizukami, 1961).

**F. ROLE OF BACTERIAL EXUDATE, TOXIN AND METABOLITES ETC. IN DISEASE DEVELOPMENT**

*X. oryzae* was known to produce a viscous matrix when grown on agar media. This substance dissolved in distilled water was precipitated by the addition of acetone. This exudate was identified as a neutral heteropolysaccharide by means of the paper chromatographic analysis upon the hydrolysate of exudate. The sugars identified were galactose,
glucose and xylose. In addition, other reducing substances, presumably sugars and a uronic acid were also present in the hydrolysate (Mizukami, 1959).

Devadath and Premalatha Dath (1970) isolated the exudate (polysaccharide) from 2 strains of \textit{X. oryzae} which could induce wilting of the rice cuttings. They concluded that it was a non-specific phenomenon caused by the blockage of the xylem vessels in plants. They also isolated the polysaccharide from the kreseked plants which gave a positive response in wilting tests, thus providing an indirect evidence that the slime material (polysaccharide) was the wilt inducing factor.

A toxic slime substance capable of causing wilting in rice cuttings was also isolated by Kuo \textit{et al.} (1970) from cultures of \textit{X. oryzae}. At the concentration of 10 ppm the purified slime caused the rice cuttings to wilt in one hour. The slime was non-specific, heat resistant and water soluble polysaccharide.

The polysaccharide blocked the acid fuschine transport of rice cuttings, dextran with a molecular weight of 150,000 could also induce similar wilting. Enzyme studies gave no sign of the presence of an active site. No damage of cell permeability by the polysaccharide was found. Since the wilting was produced even by very low concentration (10 ppm) of the polysaccharide it was thought to be caused
by toxigenic action rather than by plugging of xylem vessels (Fan and Kuo, 1972).

Egwa et al. (1968) isolated a metabolite of \textit{X. oryzae} (Phenylacetic acid) which depressed the growth of young roots of rice seedlings.

A toxin (appeared to be phenolic in nature) was isolated from culture filtrates and infected leaves. It was not host specific and was more effective against fungal than bacterial diseases. Spraying of rice plants with 500 ppm of this toxin increased respiration and inhibited shoot and root growth (Purushothaman and Prasad, 1972).

A water soluble inhibitor of bacterial multiplication was produced in rice leaves inoculated with strains of \textit{X. oryzae}. In leaves inoculated with an incompatible strain the inhibitor could be detected within 24 hr after inoculation, increased rapidly reaching the maximum amount by 8th day, then gradually decreased. In leaves inoculated with compatible strain, on the contrary, the inhibitor could not be detected until the 5th day after inoculation, the inhibitor then gradually increased until the last stage of lesion enlargement. The amount of inhibitor produced was much less with compatible strain than with incompatible strain. When the water extract from leaves showing resistant reaction was diluted to \( \frac{1}{4} \) and \( \frac{1}{16} \) concentration with distilled water, inhibiting activity was
decreased correspondingly, and almost disappeared by diluting to 1/8 concentration. The water extract from the healthy leaves also showed a slight inhibitory action, but it disappeared by 1/4 dilution (Watanabe and Nakanishi, 1976).

According to Uehara (1960), a kind of phytoalexin was produced which inhibited the multiplication of the pathogen. The production of phytoalexin was non-specific, and was decreased by treating the plants with ether. At the wounded parts of the leaves, phytoalexin prevented the pathogen from attaching to wounds. The action of phytoalexin was distinct on resistant cultivar than on the susceptible cultivar (Mizukami, 1956).


When *X. oryzae* was inoculated to resistant and susceptible cultivars, the accumulation of P^{32} around the inoculated portion was not remarkable in both the cultivars indicating that the mutual interaction between host and pathogen was not distinct (Kusaba *et al.*, 1957).

Mizukami and Murayama (1960) studied the mechanism of resistance by exposing the plants to ether and immersing in water and inoculating with the pathogen before and after the treatments. There was no difference in lesion size between treated and untreated plants when inoculated after treating the plants. On the contrary, when the plants were treated
after inoculation, the lesion size was increased on treated plants indicating that the resistance of rice plant against bacterial multiplication was reduced and/or the favourable nutrition for bacteria was produced in the plant by this treatment. Leucine, valine, methionine, alanine, glutamic acid, aspartic acid and an undetermined aminoacid "b" were found in both treated and untreated plant sap but the total contents of the aminoacids, especially, leucine and valine were increased in the treated plants than untreated ones. Moreover, undetermined substance "a" which developed a light green coloured spot when sprayed with 0.2 per cent ninhydrin ethanol solution was also found in the treated plants. The juice of the treated plants supported the growth of the pathogen on culture media indicating that necessary nutrients for the multiplication of the pathogen were produced in the treated plants.

Studies carried out at the International Rice Research Institute (1967) showed that resistant cultivars had a low reducing sugars : nitrogen ratio while susceptible ones had a high ratio.

According to Misawa and Miyazaki (1972), in the inoculated leaves, contents of reducing and nonreducing sugars slightly increased at the early stages of infection. Moreover, crude starch content increased while total carbohydrate and hemicellulose carbohydrates decreased after the infection. There was a high accumulation of free ammonia,
low contents of water soluble protein and water insoluble protein nitrogen in the inoculated leaves. As the disease advanced, total nitrogen content decreased gradually. According to the results of molecular weight classification of water soluble protein, the contents of high molecular weight protein, especially "fraction I protein", decreased and low molecular weight protein increased by the infection. At early stages of infection, there was an increase in the acid soluble and inorganic phosphorus contents and there were high contents of total phosphorus and insoluble phosphorus in the inoculated leaves.

The levels of nitrogen in the leaves had little effect on lesion development in IR 8 since plants receiving either high or low nitrogen level exhibited similar lesion development. However, high nitrogen levels showed considerably more susceptibility than did the low nitrogen levels in IR 26. This indicated that high levels of nitrogen applied to the semi-dwarf rice might not directly increase disease by making the tissue more susceptible but indirectly by increasing the number of leaves for the bacterium to infect and by changes in microclimate around the plants which favoured the disease build-up (International Rice Research Institute, 1974).

Watanabe and Asaumi (1975) observed a marked increase in the oxygen uptake after the first appearance of the symptoms, and showed a value 1.7 times high when compared to
healthy tissues. A high correlation between the number of bacteria and the degree of oxygen uptake was noticed in a susceptible cultivar. In the resistant cultivar, a rapid increase in oxygen uptake during the earlier phases of infection was not observed and the increase at the later phases of infection was limited only 1.3 times when compared with that of healthy tissues. On the other hand, in the rice leaves inoculated with the incompatible bacterium X. phaseoli, neither the disease occurrence nor the increase in oxygen uptake was observed. These authors, therefore, presumed that the enhanced uptake of oxygen was essentially a host response to the pathogen rather than a reflection of pathogen respiration.

According to Moses et al. (1975), the susceptible cultivar and the susceptible F₂ segregants showed relatively more total nitrogen, reducing sugars and total sugars while the resistant cultivars and the resistant F₂ segregants showed higher ratios of reducing and total sugars to total nitrogen.

Ranga Reddy and Sridhar (1975) recorded higher concentrations of phenols, reducing and non-reducing sugars and aminoacids in the highly susceptible cultivar Taichung (Native) 1 than the less susceptible IR 8. Inadequate supply of potassium favoured an accumulation of these substances in the leaves of both cultivars than in the leaves of plants generously supplied with potassium. The levels of phenolic
compounds and aminoacids decreased in the diseased leaves of Taichung (Native) 1 while that of IR 8 showed an increase and the increase being more in plants supplied with higher concentrations of potassium. In the infected leaves of these cultivars, both the reducing and non-reducing sugars were generally low.

Miyazaki et al. (1976) studied the cellulose, invertase, xylanase, phosphatase, b-amylase (α-amylase), pectinase (pectin methyl esterase and polygalacturonase), lipase, lecithinase and protease enzymes in healthy and diseased tissues. These enzymes were more in diseased tissues than the healthy tissue. On the contrary polygalacturonase was not activated by the infection. Enzyme activities increased more in the early stage of infection than the late stage (8 days after inoculation) except β-amylase. However, cellulose activity was more in the late stages of infection. Increased activities of the enzymes seemed to be mainly originated from the enzymes produced by the pathogen. The protease activity of pH 3.7 was remarkably increased by the infection and a positive relationship was observed between protease activity of pH 8.7 of seven isolates of the pathogen and their pathogenicity.

But, according to Damodaram Naidu et al. (1977), there was no relation between totals of phenols, reducing and non-reducing sugars, aminonitrogen and nitrogen in the leaf tissue and the development of bacterial blight in resistant or susceptible cultivars.
H. INTERACTION BETWEEN HOST, PATHOGEN AND ENVIRONMENT IN DISEASE DEVELOPMENT

1. Age of the host

In multi-needle or clip inoculation tests, it had been observed that in certain cultivars the resistance increased with an increase in the age of the plant (Muko et al., 1953; Kuhara and Sekiya, 1957; Goto, 1965; International Rice Research Institute, 1968; Devadath, 1969; Devadath and Padmanabhan, 1969; All-India Coordinated Rice Improvement Project, 1970; International Rice Research Institute, 1972; Horino and Ezuka, 1973; Ezuka et al., 1974; Kauffman et al., 1974; Premalatha Dath, 1974; Reddy, 1974; Uma Gupta, 1975). At heading stage, when the resistance increased, the difference between resistant and susceptible cultivars became apparent (Muko et al., 1953; Wakimoto and Yoshii, 1954; Kuhara and Sekiya, 1957). On the contrary, in certain other cultivars the susceptibility increased as the plant age increased (Devadath, 1969; Devadath and Padmanabhan, 1969; Kauffman et al., 1974).

But, according to the work done at the International Rice Research Institute (1965; Ou, 1966; Ou et al., 1971) none of the resistant, moderately resistant and susceptible cultivars showed a striking difference in their reaction between seedling and flag leaf stages.

According to Reddy (1974), bacterial blight reaction changed at different stages of the crop. Seedling reactions
and adult plant reactions were not always correlated. The disease severity was reduced as the age of the plant advanced in certain cultivars and in other cultivars it was differentially affected by plant age. Some of the cultivars which have usually low disease levels in the field at adult plant age had more per cent kresek when infected at the nursery stage. On the other hand, some other cultivars, which were susceptible at adult plant age, had only lesser kresek. But some other cultivars did not show marked difference between both the stages of infection. Therefore, it was concluded that the tissue susceptibility as measured by pin-prick inoculation under greenhouse conditions did not actually represent the natural field reaction at different ages of the crop (All-India Coordinated Rice Improvement Project, 1971). However, Premalatha Dath (1974) and Uma Gupta (1975) observed that with an increase in the age of the plant there was an increase in the disease under natural field conditions.

Even within a given cultivar, the resistance of the plants was known to increase with an increase in the age (Reitsma and Schure, 1960; Sulaiman and Ahamed, 1965; Sulaiman et al., 1965; Devadath, 1969; All-India Coordinated Rice Improvement Project, 1971; Horino and Ezuka, 1973; Miah, 1973; Ezuka et al., 1974; Watanabe, 1975). But reports were not lacking to say that the susceptibility increased as the plant age increased (Mahmood and Singh, 1970; Kuo et al., 1971).
2. Effect of inoculum concentration

Inoculum concentration greatly influenced the incubation period, percentage of plants infected and lesion development. On susceptible cultivars, the disease was initiated even by dilute inoculum (Kuhara, 1956; Mizukami and Seki, 1956; Sekizawa, 1963).

Few bacterial cells when placed on wounds did not incite infection whereas abundant inoculum resulted in high percentage of infection. The minimum concentration to incite the infection was $10^4$ cells/ml (Mizukami, 1961). Premalatha Dath (1974) observed no significant differences in lesion length among different concentrations from 0.05 OD to 2.0 OD. However, according to Tagami et al. (1961; 1964), Yoshimura (1963) and Yoshimura and Tagami (1967), the number of bacterial cells on the seedlings not only influenced the disease intensity but also the time of outbreak of the disease after transplanting.

The inoculum concentration in the soil seemed to influence the appearance of kresek seedlings (Premalatha Dath, 1974) and the minimum concentration required to initiate kresek was $10^2$ cells/ml (Watanabe, 1975).

It was observed that more the concentration of the inoculum and less the age of the plant, more severe were the symptoms noted and the symptom development was also earlier. With less concentration and with an increased age of the
host not only the initiation of the disease was delayed but
the intensity of the disease was also less (Devadath, 1969).
But according to Reddy (1974), the reaction of susceptible
or resistant cultivars was not influenced by the age of the
host or the inoculum concentration. However, the tolerant
cultivars at high inoculum concentrations \(10^9\) cells/ml
reacted as susceptible and at low concentrations \(10^6\) cells/ml
the same cultivars reacted as moderately resistant-
moderately susceptible at the seedling stage. Fluctuations
of reactions were not so wide in this group when inoculum
concentrations of \(10^6\) and \(10^9\) cells/ml were used at the adult
plant age.

On the contrary, Kuo et al. (1971) reported that
when 6 day old seedlings were inoculated, a much higher
concentration was required for successful infection, but the
aged plants (2 month old) became infected even when the
inoculum concentration was as low as \(10^1\) cells/ml.

Kauffman et al. (1973) observed that bacterial
populations of \(10^7\) cells/ml or greater were adequate to
give 100 per cent leaf infection on resistant and susceptible
cultivars. Below this concentration, infection frequently
declined rapidly. The disease scores in both susceptible
and resistant cultivars decreased and the incubation period
was slightly prolonged with lower inoculum concentrations.
An optimum concentration of $10^6$, $10^7$, $10^8$, $10^9$ cells/ml was required to inoculate 14, 37, 48 and 58 days old plants respectively according to Cho (1975).

3. **Effect of climatic factors on disease development**

a) **Rainfall**

The intensity of the disease was also associated with the amount of rain (Kuwazuka, 1933; 1942; Goto et al., 1955; Yasu and Narikawa, 1956; Fujikawa et al., 1957; Yasu et al., 1959; Tagami and Mizukami, 1962; Sulaiman and Ahamed, 1965; Mizukami, 1966; All-India Coordinated Rice Improvement Project, 1969; Buddenhagen, 1969; Kauffman et al., 1971; Ten Have and Kauffman, 1972; Kauffman and Rao, 1972; Isaka, 1973).

The number of rainy days were reported to be more important than the amount of rain for severe infection (Tagami and Mizukami, 1962; All-India Coordinated Rice Improvement Project, 1969; Isaka, 1973; Premalatha Dath, 1974; Uma Gupta, 1975). The time of rain also played an important role in the severity of infection. Rain during June-July (Kuwazuka, 1933; 1942; Fujikawa et al., 1957; Isaka, 1973), heavy rain from the end of May to the beginning of June was associated with high incidence of the disease in nursery stage and again from tillering to flag leaf stage (Goto et al., 1955). On the contrary, cool and rainy days in September inhibited the disease development (Yoshimura, 1959).
Though the disease was of importance only in the rainy season (June-December), moderate to severe infections had also been observed in the winter crop (December-May) in the double cropped areas. Despite sub-normal rainfall, the disease appeared in severe form in most of the plots in north and south India during the monsoon season of 1965 (Srivastava, 1967). Premalatha Dath et al. (1973) reported that rains coming either at the time of inoculation or immediately after did not interfere with the disease development. On the other hand, rains after inoculation were found to be more congenial for the development of infection. 

Premalatha Dath (1974) studied the effect of weather factors on the development of lesion in pin-prick inoculated host plants in 40 consecutive fortnightly transplantings of four rice cultivars. She failed to get any correlation between the number of rainy days and the amount of rainfall with lesion development. But, in a study on the spread of the bacterial blight in rabi and kharif seasons under natural field conditions, she found that weather factors like high rainfall and more number of rainy days favoured more spread of the disease.

b) **Humidity**

High relative humidity was reported to be necessary for the seed transmission (Srivastava and Rao, 1964) and subsequent disease build-up (Kuwazuka, 1933; 1942;
Kiryu, 1954; Kobayashi et al., 1954; Tanaka, 1958; Srivastava and Rao, 1964; Premalatha Dath, 1974). Low humidity at the time of inoculation greatly influenced the disease development (Kuhara, 1956; Aoyagi et al., 1960). Presence of dew drops or cloudy humid weather were also favourable for invasion and infection and this explained why the disease occurred in mountaneous basins in Japan (Tagami, 1968).

Sulaiman and Ahamed (1965) observed that a relative humidity of 93 per cent was congenial while 91 per cent was an adverse factor for the development of the disease. According to Chattopadhyay and Mukherjee (1973), uniform mean relative humidity of 89.6-92.8 per cent encouraged the disease development. Uma Gupta (1975) found that high disease intensity was associated with a mean relative humidity of 79.3-90.1 per cent while 60.3-77.0 per cent mean relative humidity was associated with low disease intensity.

Premalatha Dath (1974) felt that relative humidity played a secondary indirect role in the lesion development in the pin-prick inoculated plants. Results obtained at the International Rice Research Institute (1974) also indicated that clip inoculation was effective even under conditions of low relative humidity. However, according to Premalatha Dath (1974) high humidity was one of the associated climatic factors in the secondary spread of the disease amongst the plant populations under field conditions.
According to Kiryu and others (1954 cited by Tagami and Mizukami, 1962) an average 10 O'Clock temperature of above 25°C in July and a minimum temperature of above 21°C prevailed in endemic areas. When the annual mean temperature was 14°C or when July mean temperature was 24°C the disease occurrence was severe. However, occasionally disease was also seen when the July mean temperature was 22°C (Kuwazuka, 1933; 1942). Successive high 10 O'Clock temperature during the last 10 days in May was associated with incidence of the disease in nursery stage (Goto et al., 1955). The optimum temperature for the disease development in fields was assumed to be anywhere between 22°-27°C (Goto et al., 1955; Fujikawa et al., 1957). High temperature in tillering stage, low temperature in August and early September and warm autumn were suited for blight occurrence (Goto et al., 1955; Yoshimura, 1959).

Tagami (1963) considered that 'fine' weather with high temperature which was seen in 'warm' places in normal years was unfavourable for this disease. The water temperature in nursery, in an early period of paddy field and also the atmospheric temperature in autumn similarly acted sometimes favourably and other times unfavourably for infection and spread of the disease.

During hot and dry season in mid-summer the disease development was usually arrested temporarily and the pathogen
on rice plants also decreased even in the case of early outbreak in warmer and cooler regions of Japan (Goto et al., 1955; Tagami and Mizukami, 1962; Yoshimura and Tagami, 1967). However, after the mid-summer, the weather factors such as cloudy or rainy days and typhoons helped the disease to progress again (Yoshimura and Tagami, 1967). According to Tagami (1968), much rain in summer not only aided the dispersion of the pathogen and secondary infection directly but also caused lowering of temperature as a result of reduced sunshine hours, thus furthering infection in warm places.

High temperature in Japan was rather favourable and prolonged the period of infection and disease development (Tagami and Mizukami, 1962). In cooler regions of Japan, low temperature in September was considered unfavourable (Yoshimura, 1959; Tagami, 1968).

The low disease incidence at flowering in rabi season in India had been attributed to the hot dry weather at the time of maturity of the crop (Srivastava et al., 1966; All-India Coordinated Rice Improvement Project, 1969; Ten Have and Kauffman, 1972).

According to Sulaiman and Ahamed (1965), the optimum temperatures for the occurrence of the disease were 23.4°C and 30.0°C of minimum and maximum respectively but 22.4°C and 34.0°C temperatures of minimum and maximum respectively were one of the adverse factors for the disease development.
Chattopadhyay and Mukherjee (1973) reported that a moderate but uniform temperature range of 30.4°-32.7°C favoured the disease development. The high disease incidence according to Uma Gupta (1975), was associated with a mean maximum temperature range of 29.6°-31.8°C and a mean minimum temperature range of 22.3°-25.4°C, while low disease incidence was associated with a mean maximum temperature range of 31.4°-36.4°C and relatively a very low mean temperature range of 13.4°-20.2°C.

Spread of the disease amongst plant population under field conditions seemed to be favoured by a mean minimum and a mean maximum temperatures of 24.3°C and 34.0°C. But a temperature of 18.2°C and 37.2°C respectively were adverse for the spread (Premalatha Dath, 1974).

Temperature at the time of inoculation seemed to be a critical factor for the lesion development (Kuhara, 1956; Aoyagi et al., 1960). Lesion development was faster at higher temperature range of 25°-28°C than at low temperature range of 17°-21°C (Muko et al., 1957; International Rice Research Institute, 1967; Ou et al., 1971). The symptoms appeared within 4 days at higher temperature (28°±2°C) and within 12 days at low temperature (21°±2°C). The kresek symptoms appeared within 14 days at higher temperature but were not visible even after 40 days at low temperature (International Rice Research Institute, 1967; Ou et al., 1971). According to Premalatha Dath (1974), the most
critical factor in lesion development in pin-prick inoculated plants appeared to be the temperature. A mean temperature range of $21.3^\circ\text{C}$-$32.7^\circ\text{C}$ was favourable for rapid lesion development whereas a mean minimum temperature of $16.7^\circ\text{C}$ and below and a mean maximum temperature of $37.4^\circ\text{C}$ and above adversely affected the lesion development.

Temperature regimes ranging from $25^\circ/20^\circ\text{C}$-$35^\circ/35^\circ\text{C}$ (day/night) promoted lesion development in IR 8. Kresek also developed at these temperatures regimes except at $35^\circ/35^\circ\text{C}$ with a 75/75 per cent relative humidity where kresek did not develop. In IR 26, however, lesion development was noticeably favoured at high temperatures (International Rice Research Institute, 1974).

d) Sunshine

Low sunshine in July and August was associated with severe disease in Japan (Soga, 1918; Fukuoka Agr. Exp. Sta., 1920; Goto et al., 1955; Yasu and Narikawa, 1956; Yasu et al., 1959; Tagami and Mizukami, 1962; Tagami, 1968). Comparatively less sunshine hours (Maruyama, 1908; Chattopadhyay and Mukherjee, 1973; Premalatha Dath, 1974; Uma Gupta, 1975) were found to encourage the disease development. Less sunshine hours over the growth period of the crop not only favoured the multiplication of the pathogen but also weakened the plant (Chattopadhyay and Mukherjee, 1973). On the contrary, Yoshimura (1959) concluded that
severe disease in Hokuriku area during 1958 was due to more sunshine in nursery or early paddy field stage.

According to Premalatha Dath (1974), number of sunshine hours per day did not show any correlation with lesion development in pin-prick inoculated plants though the correlation was obtained in the spread of the disease under natural field conditions.

Mention was made by Sulaiman and Ahamed (1965) that first infection usually appeared on plants under shade of trees.

Preliminary experiments conducted at the International Rice Research Institute (1968) showed that rice plants pre-disposed to total darkness for 2-3 days and then grown in reduced light (partially shaded) developed smaller lesions than those exposed to full light.

Premalatha Dath (1974) studied the effect of exposure of plants to shade and light at different intervals both before and after inoculation on the lesion development. She found that plants grown under continuous shade encouraged more spread of the lesion as compared to those grown under full day light. There was a reduction in lesion length when plants were shifted from shade to light before inoculation than after inoculation at different intervals. But there was a gradual increase in lesion length when the plants grown under continuous light condition were shifted to shade at
different intervals before than after inoculation. Exposure 
of plants from shade to light, in general, increased the 
lesion length as compared to plants shifted to shade from 
light.

Results reported at the International Rice Research 
Institute (1974) revealed that high light intensity (50 klx) 
generally favoured more disease than did low intensity (20 klx) 
though symptoms appeared under both the light intensities.

Tagami (1968) concluded that it was very difficult to 
separate out each environmental factor and attribute it to be 
responsible for disease development under field conditions, 
for instance, much rain not only aided the dispersion of the 
causal bacterium and secondary infection directly, but also 
caused lowering of temperature as a result of reduced sunshine 
hours and increased the humidity thereby favouring infection.

e) Interaction of rice cultivars and meteorological factors 
in relation to disease development

All the four cultivars tested viz., BJ 1, IR 8, 
CR 129-118 and Taichung (Native) 1 responded in the same way 
as similar trends were reflected in lesion lengths, in relation 
to weather factors, irrespective of their differential 
susceptibility in pin-prick inoculated plants (Premalatha Dath, 
1974). On the contrary the work done at the International 
Rice Research Institute (1974) showed that IR 8 and IR 26 
reacted differentially under different environmental conditions.