The little-leaf disease of eggplant could only be transmitted by graft, dodder or by vector leafhoppers as is typical for other mycoplasma like disorders. The dodder-bridge method has successfully been employed for transmission of bayberry yellows (Raychoudhury, 1953), *Chrysanthemum* flower distortion virus (Brierley and Smith, 1957), Aster yellows and peach-X disease (Carling and Milliken, 1977). From the comparison of minimum incubation period in test plants as well as the per cent success, it is found that graft inoculation technique is more successful than the vector and dodder-bridge transmission. The vector leaf hopper requires a minimum acquisition feeding of 4h and a latent period of 15 days to be infective with an infective feeding of 2h or more. Such latent period in the vectors is required for the spread of other mycoplasma diseases. The clover phyllody disease transmitted by *Eusalis plebegus* requires 30 days latent period after an acquisition feed of 2 days before being infective (Cousin *et al.*., 1960). The *Catharanthus* little-leaf MLO requires 18 days latent period in vector leaf hopper after an acquisition feed of 6h or more (Kar and Panda, 1990).
General symptoms of the disease are similar in the naturally infected and artificially inoculated plants. The symptoms always remain true through successive generations of disease, when naturally infected plants are used as the primary source of inoculum. These observations indicate that the little-leaf syndrome in nature is not an outcome of mixed infections but is due to pure mycoplasmal infection. Keeping the shoot in darkness for 4 days prior to inoculation and 2 days after, enhances the disease. The symptoms are always restricted to the apices of the branches on which the grafts are mounted. Other existing branches remain healthy though new branches arising from the base of infected shoots exhibit disease symptoms. This indicates that the disease is systemic and primarily affects the meristematic tissue. Movement of the pathogen within the plant body probably occurs with the flow of photosynthates. The main symptoms of the disease are, initial greening of flowers accompanied with reduction of laminar area and sprouting of dormant buds so that numerous little-leaves are produced on slender branches. The apical dominance ends and the plants become completely vegetative with little-leaves only. Thus the reduction in laminar area prove to be the most useful singular characteristic to judge the severity of the disease. The overall internal morphology of stem, petiole and pedicel in healthy and diseased plants are comparable but for the presence of bluish-violet patches in the phloem of diseased stem sections with lacmoid staining which is peculiar.
Lacmoid reaction being specific for callose (Goove, 1865), indicates its accumulation in the phloem cells of diseased plants. This might be an outcome of localisation of pathogen in the phloem tissue as is typical for MLOs in general (Doi et al., 1967). The greater proliferation of internal phloem in diseased plants might be in response to the damage caused in external phloem due to growth of the pathogen.

Experiments on host range shows that the pathogen infects 15 species of plants distributed in 5 unrelated families. Many yellows type of diseases like tomato bigbud (Hill, 1943), cranberry false-blossom (Kunkel, 1945) and aster yellows (Kunkel, 1937), similarly have wide host range covering plants distributed in widely unrelated families. Removal of sources of infection can be helpful in avoiding disease spread into the new crop (Barnett and Gibson, 1977).

Out of the plants susceptible to eggplant little-leaf, Datura fastuosa, Solanum nigrum, S. xanthocarpum, Catharanthus roseus and Argemone mexicana often suffer in nature from little-leaf syndrome. Cross inoculation test using these naturally infected plants as the source, produce typical little-leaf syndrome in eggplant. This indicates that these weeds are the major alternative hosts for eggplant little-leaf MLO and probably act as the focus for its natural spread. Out of these four Solanum xanthocarpum which is a biennial herb very common in this locality, serves as the host for growth and oviposition of the vectors (Bindra and Sohi, 1969). Therefore, S. xanthocarpum appears to qualify as the most
important natural source of eggplant little-leaf disease found in the district.

A total of 79 species distributed in 5 families have been reported earlier as alternate hosts for brinjal little-leaf disease (Anjaneylu and Ramakrishnan, 1973; Konai, 1984; Varma et al., 1965). In this study 2 new species of alternate hosts, Cyamopsis tetragonoloba and Zinia elegans are tracked. Thus the possibility exists that the MLO responsible for eggplant little-leaf probably has a much wider host range than is normally assumed. One of the species tested Sesamum indicum suffers in nature from a phyllody disease with symptoms alike with eggplant little-leaf. However, artificial inoculation of healthy Sesamum indicum with eggplant little-leaf MLO failed to produce any disease symptom on the test plants. This indicates that Sesamum phyllody and eggplant little-leaf are caused by two different pathogens although the disease symptoms are identical and that, Sesamum is immune to eggplant little-leaf pathogen.

Earlier workers reported a positive correlation between the rise in vector population after rains and the natural spread of eggplant little-leaf disease (Chakrabarty and Choudhury, 1973; Panda and Kar, 1980; Srinivasan and Chelliah, 1977). But experiments on variation in leaf hopper population and disease spread indicates that maximum disease incidence and rapid spread succeeds the phenomenal rise in
the vector population observed during Oct-Nov. (Panda and Kar, 1990). The most rapid spread rather coincides with the secondary rise in field population of vectors during Feb-Mar. Thus it appears that this secondary rise in vector population after winter is actually responsible for rapid natural spread of the disease. Probably the primary rise in the vector population after rains during Oct-Nov, provides the disease focus into the crop fields by transmitting the disease from weed hosts to eggplants planted in that season. Depletion of the weed hosts during winter restricts the secondary wave of vectors to the eggplant fields and facilitates the rapid spread. This view is also supported from the observation that disease spread is very low in test plots without artificial disease focus. (Panda and Kar, 1992)

Restriction of the disease spread from April onwards is probably due to an adverse effect of temperature on the pathogen and the course of disease development. Even mild symptoms reversal during these months are noticed in plants infected earlier. These results are in conformity with the earlier works on the thermotherapy of MLO incited diseases (Anjaneyulu and Ramakrishnan, 1973; Kunkel, 1941; Maramorosch et al., 1970; Nanda et al., 1979). Diseased plants with prolonged infection died due to stem necrosis which occurred independently in the different branches of the same plant. This may have been due to damage caused by infection to the phloem tissues of the stem impairing the process of translocation. This view would agree with the
anatomical observations on diseased stem phloem tissue and the earlier finding that MLO is localised in the phloem tissues (Kar et al., 1982) because of favourable condition of growth in the phloem element in the form of slightly higher osmotic pressure and alkaline pH.(Doi et al., 1967).

Within broadly similar pattern of dry matter accumulation in healthy and little-leaf infected eggplant are many important differences. It is obvious from the results that infection significantly alters the growth and morphogenetic responses of the plant. The RGR and NAR decrease rapidly in healthy plant with time and growth. The LAR and SLA decrease very slightly with significant rise in LAD and BMD. Reduction in LAR with age has been observed as an usual feature in grasses (Higgs and James, 1969), and other plants (Pandey and Sinha, 1977). Thus the observed decrease in LAR might be an ageing stimulus. The NAR is a measure of increase in dry weight of the plant per unit leaf area per week and is thus an index of overall photosynthetic efficiency. The reduction in RGR appears to be an outcome of reduction in NAR rather than LAR. The progressive shading of lower leaves due to increased growth and leafiness of plants may be a factor in the decline in NAR and RGR(Panda and Kar, 1992,b). Fall in RGR with time has been observed in several other plants(Panda and Kar, 1991; Pandey and Sinha, 1977; Thorne, 1960). Thorne, (1960) observed greater
decline in RGR in those species which had shown initially higher values.

The RGR and NAR generally remain low in diseased plants. An increase in the values over control is observed during the 3rd harvest interval. This may have been the outcome of improved photosynthetic efficiency of the lower leaves, as the characteristic progressive shading of lower leaves in healthy plants is absent in diseased plants due to progressive reduction in the size of the new upper leaves. Subsequent rapid decline in both NAR and RGR may be due to the senescence and abscission of fully expanded lower leaves. This view is supported by the observed decline in BMD values, GA concentration and rise in ABA concentration under pathogenesis. Concomitant upon the decrease in RGR and NAR, the LAR and SLA increase significantly. Such rise probably is a compensatory phenomenon since the amount of chlorophyll per unit area of leaf decrease under pathogenesis. This compensatory rise in SLA, LAR and LAD under pathogenesis will be helpful in maintaining the RGR in face of lower amount of chlorophyll per unit area, because in condition unfavourable for photosynthesis, leaves have the first call on its products (Newton, 1963).

The LWR decreases under disease up to 3rd harvest but improves later to the normal level. This decrease may be the outcome of flow of products from leaves to meet the growth demands of the plant in face of reduced NAR. As the NAR
improves so does LWR, indicating greater accumulation of photosynthates in the leaves. This trend of accumulation of photosynthates in the diseased leaves is also maintained at the later symptomatological stages despite the decrease in NAR and corresponding RGR. Since the outward movement of photosynthates from the leaves occurs in the phloem, the MLO growing in and damaging the phloem tissues, would impair the outward translocation of photosynthates from the leaves to other parts of the plant. Had the photosynthates been stored in individual leaves, there would have been no increase in SLA. Hence it is assumed that the photosynthates are stored in the vicinity of leaves in the condensed inflorescences borne in the individual leaf axils. The stored photosynthates probably induce floral phyllody and proliferation of flowers into small little shoots. The process repeated often over a period of time, results in the typical witchesbroom syndrome which is a characteristic feature of the disease. This hypothesis would accommodate the observed rise in SLA and LAR, and decrease in NAR. Diseased plants show lower BMD rates than healthy. The overall reduction in BMD and RGR is comparable. Product of these two parameters give the projected yield (Kevt et al., 1971). It would therefore be expected on theoretical grounds alone that, pathogenesis would reduce the normal yield in terms of weight by about 40% of control. This conjecture agrees with the actual total dry weight data of diseased plants being 35% of control at the last harvest. The USR and URR show similar trend with
the RGR and NAR up to the 3rd harvest interval. The URR remains constant below the healthy level while the USR remains above it. This is consistent with the above view that photosynthates accumulate in the shoots under disease syndrome (Panda and Kar, 1991, b).

The Sw to Rw ratio shows slight increasing trend in the later stages of disease. From this it can be concluded that disease induces greater accumulation of photosynthates in the roots in comparison to the shoots at the early phase. This trend however is reversed in the late stage. Further, a correlation between root and shoot dry weights for healthy and diseased yields a more positive 'r' value for the diseased, indicating that the root system is more dependent on the shoot system (Panda and Kar, 1992, a).

Allometric relationship between root and shoot (Hunt, 1975, 1978), yields completely dissimilar k values in healthy and diseased at all harvests. It decreases in healthy during the 2nd harvest interval, increases during the 3rd and again decreases afterwards. But in diseased, it decreases rapidly in time till the third harvest interval and then increases slightly. From these results it is suggested that the pathogen first spreads in the shoots, and then moves down with the flow of photosynthates into the roots. As a result of the spread and mal-functioning of the roots, the growth in shoot gets further affected with production of
short shoots that accumulate little dry matter, thus yielding a high value of $k$.

The allometric relationship $'a'$ is an useful index of morphogenetic behaviour of plant (Whitehead and Myerescough, 1962). It indicates the increase in dry weight surplus to that required for maintaining the morphogenetic proportions of the plant as an efficient photosynthetic form. The higher value of $'a'$ in the diseased plants at the first harvest interval indicates lesser consumption of assimilates for the growth of leaves, shortly after inoculation. The lower values of $'a'$ after appearance of symptoms indicates a greater consumption of assimilates in the further growth of leaves with little going to other parts of the plant. This implies theoretically a continuation of vegetative phase of growth with no immediate prospect of flowering. Such a situation actually arises in diseased plants as discussed earlier. In healthy plants higher than unity values of $'a'$ beyond the third harvest indicates diversion of higher proportion of assimilate for flowering and fruition (Whitehead and Myerescough, 1962).

The infection brings about profound changes in the physiology and metabolic status of the host. Its laminar area is reduced and leaves become thinner in comparison with the healthy. The dry matter per unit leaf area reduces progressively with time under disease, whereas it increases with time in the healthy. Such reduction in the
dry matter content of the leaf under disease may be associated either with the observed reduction in the number of mesophyll cells (plates 4), increase in the rate of respiration (Fig 5.18), decrease in the rate of net photosynthesis (Fig 5.16), or all of them together. Reduction in the number of mesophyll cells (Esau, 1956), increase in the rate of respiration (Jansen, 1972) and decrease in the rate of net photosynthesis (Hall and Loomis, 1972; Irvine, 1971), have been reported for mosaic symptoms of viral origin. The per cent water content as well as total plant hydration increase in the diseased plant with symptoms advancement. As against this, the LWC decreases although the per cent water content of leaf tissue and laminar hydration remain constant with an increase in the RWC. Probably the low LWC originates in the reduced dry matter of the diseased leaves.

The increase in RWC might be due to (i) distribution of dry weight in the diseased leaf in the soluble and simpler forms, (ii) reduced rate of transpiration due to stomatal mal-function, or some other factor. Stomatal mal-function leading to reduced rate of transpiration has been reported for Catharanthus little-leaf disease (Kar, 1987). Impaired translocation leading to accumulation of photosynthates within and in the vicinity of leaves as discussed earlier might be a contributing factor too. Such accumulation of photosynthates besides serving as a factor for shift in the osmotic balance, will also have a bearing on the
characteristic proliferation of axillary branches due to disease, that would function as sinks for a portion of such photosynthates. This conjecture would agree with the findings on ecology discussed earlier.

The chlorophyll content of leaves decrease under infection whereas the carotenoid content show little change (Panda and Kar, 1988). The rate of degradation of chl a is greater than that of chl b lowering the chl a/chl b ratio in the disease. Such decrease in the content of chlorophyll has been reported for periwinkle leaf infected with MLO associated with aster yellows and peach-x disease (Carling and Milliken, 1977). Further, they also found that the reduction in total chlorophyll was quantitative for both disorders. Thus basing on the response of chlorophyll pigmentation in leaves, it appears that the MLOs have a tendency to disrupt the photosynthetic apparatus like the viruses. In most cases of virus infection there is a marked decrease in the chlorophyll content of leaves (Irvine, 1971; Jansen, et al., 1972; Kabi et al., 1979; Kar et al., 1979; Mandahar and Garg, 1977; Nambiar and Ramakrishnan, 1968; Nanda et al., 1979; Sridhar et al., 1976) with an accompanied breakdown of the chloroplast lamella (Pares, 1972; Pares and Bertus, 1978) and it is suggested that intact chlorophyll molecule must be present for chloroplast lamella (Benson, 1974).
To study the situation more clearly absorbance, fluorescence emission, fluorescence excitation spectra and DCPIP-Hill reaction rates, of isolated chloroplasts from healthy and diseased leaves were analysed. The DCPIP-Hill reaction activity per g fr leaf tissue or per unit chlorophyll, progressively decreased under pathogenesis. Since the DCPIP-Hill reaction activity is a function of the PS II of chloroplast, the reduction of activity must originate either in the failure of PS II complex or impairment in the coupling of photolysis of water with the PS II which would obstruct the normal inflow of electrons into PS II.

The study of absorption properties of chloroplast provides information about the organizational status of the organelle (Raval et al., 1984; Panigrahi and Biswal, 1979). It also indicates the presence of different pigment species on the thylakoid membrane. Recent information from studies of absorption properties has been used to analyse the stacking status of the organelle (Choudhury and Biswal, 1984; Behera and Choudhury, 1986). The absorption spectrum (Fig 5.13) of chloroplast isolated from the healthy leaves shows a red band with a peak absorption value at 676 nm and a blue band with a peak absorption value at 436 nm. The chl b band is prominent around 650 nm and the carotenoid shoulder band is distinct at 475 nm. The half-band width and blue to red ratio are 25 nm and 2.31 respectively.
The absorption spectrum of chloroplast prepared from the diseased leaves show similar blue and red peaks at 436 and 675nm respectively. However, the height of the blue peak has significantly increased. The chl b hump and the carotenoid shoulder band are not so prominent. The absence of a shift in the diseased sample indicates that the process of membrane disorganization is relatively less severe (Mishra et al., 1989; Raval et al., 1984). The half band width of disease is same as the healthy although blue to red ratio has increased from (control) 2.31 to 2.65 (diseased) suggesting no ultrastructural changes of chloroplast (Raval et al., 1984; Panigrahi and Biswal, 1979) in the diseased sample.

Fluorescence analysis of chloroplast provides information about the structural organization in relation to pigment complexes in the thylakoid membrane (Biswal and Biswal, 1988). Information about the coupling of light absorption and photochemical reactions associated with reaction center of different photosystems can also be obtained by monitoring the fluorescence characteristics (Swain et al., 1980).

The chl a fluorescence emission spectra of chloroplast at room temperature (Fig 5.14), isolated from the healthy leaves, shows a distinct peak at 685nm (F685) and a shoulder around 735nm (F735). The measurement of fluorescence
property of isolated chloroplast was done on chl basis, so that meaningful correlation could be made between the fluorescence property and photochemical activities, such as the DCPIP-Hill reaction. Although there is no significant difference in the qualitative nature of the spectra prepared from the chloroplast of diseased leaves from that of healthy leaves, there is significant change in the fluorescence intensity. (Table 5.13).

It is known that F585 originates mainly from PS II (Papageoorgiou, 1975) and F735 from PS I (Van Grondelle, 1985). The higher value of F685 to F735 in the diseased sample indicates that the increase in the ratio is mainly due to loss of fluorescence emission by long wavelength absorbing pigments of PS I (Lebedev et al., 1986).

Study of excitation characteristics has been used to explain the spatial arrangement and coupling of different pigment molecules to the thylakoid membranes (Swain et al., 1990). Peaks of the excitation spectrum at 439, 471, 485 and 676 nm are attributed to chl a, chl b, carotenoids and chl a red band respectively (Panda and Biswal, 1989; Mishra et al., 1989). The level of chl a fluorescence depends on its proximity or coupling with other accessory pigments, mainly chl b and carotenoids in the chloroplasts (Papageorgiou, 1975). These pigments exist as complexes with a definite spatial arrangement on the thylakoid membranes. If the complexes are altered because of change in the
pigment composition of chloroplast the quantum migration from other pigments to chl a is affected, resulting in a change in fluorescence. This is reflected in the peak height in the excitation spectra of accessory pigments (Panda, 1987).

The excitation spectra of chloroplasts isolated from both types of leaves show distinct peaks at 439, 471, 485 and 676nm, besides several shoulder bands compared to the excitation spectra of healthy, there is, in diseased leaves significant change in relative peak heights of spectra in almost all the peaks, with 676nm being the most significant. However, the similarity in the ratio of 471 to 439 and 676 to 439 (Table 5.14) indicates equally strong coupling between chl a and accessory pigments in both healthy and diseased preparations (Panda, 1987).

This implies that the impaired DCPIP-Hill reaction activity probably originates in the disruption of coupling between photolysis of water and PS II. Several models for the three dimensional structure of PSII have been proposed (Murata and Miyayo, 1985; Swain et al, 1990). All the models agree that Mn plays the most important role in transferring the electrons from photolysis of water to the electron hole of PS II (Swain et al., 1990) although the specific site and the protein binding Mn remains obscure. The smallest isolated PS II preparation which retain water photolysis activity contains at least 6
intrinsic polypeptides, none of which can be ruled out as the location of the Mn cluster. (Ghanotakis et al., 1987). But there is a good deal of evidence pertinent to the Mn cluster which more or less agree that there are 4 Mn ions involved (Cheniae and Martin, 1970) in the electron transfer process. Therefore the micronutrient composition of the leaf sample was analysed which shows (Table 5.18) reduction in the Mn level of diseased leaves by about 90%. This observation strongly indicates impairment of photosynthesis through the failure of coupling between the photolysis of water and electron flow into the PS II, although the overall chloroplastic integrity remains undisturbed due to disease.

Deranged carbohydrate metabolism is a major phenomenon of the infective process leading to accumulation of starch in the matured leaves from the 4th week onwards. But the sugar level which increases initially, decreases later to a level below healthy. Concomitant upon the rise in sugar, the tissue respiration increases and is maintained at a higher rate throughout pathogenesis as found for a variety of pathological conditions (Daly, 1976). Accumulation of starch under pathogenesis can occur from disturbed starch hydrolysing enzymes (Balls and Martin, 1938; Kabi et al., 1979; Kar et al., 1979), or from some types of resistance offered by the disease to free translocation of photosynthates (Jansen, 1972; Kar et al., 1983). In order to study these
possibilities, the starch content of matured leaves along with the activities of major starch hydrolysing enzymes - amylases were estimated at the beginning (L-) and end (L+) of active photosynthetic period. It was found that L+ samples contain less whereas L- samples contain more starch than their corresponding healthy samples. The amylase activity pattern closely follows that of starch although the proportion of enzyme activity to that of starch is low in diseased L- samples. This indicates that factors other than amylases are strongly involved in starch accumulation. The lower starch content of L+ diseased samples may be a reflection of reduced photosynthetic efficiency indicated by low DCPIP-Hill reaction activity. But the higher than normal rate of starch in L- samples indicates that photosynthates from other portions of shoot probably migrate at the end of photosynthetic period and are fixed during the night as starch in the matured leaves owing to a failure in the normal translocation process. Destruction of phloem tissue due to MLO growth discussed earlier supports this view. The absence of starch accumulation in the first two weeks following infections may be due to intact phloem and normal translocation, and/or canalisation of a larger proportion of the photosynthates to meet the demand for enhanced respiration. The maintenance of higher respiratory rates in face of low photosynthesis may also account for
the lower level of sugar in the later stages of disease in face of accumulated levels of starch.

Accumulation of soluble and storage carbohydrates (Jansen; 1972, Orlob and Arny, 1961, Singh and Srivastava, 1974), their paucity (Kabi et al., 1979; Kar et al., 1979; Lodh et al. 1971), or unchanged levels (Sridhar et al., 1976; Vidyasekharan and Kandaswamy, 1972) have been reported for various virus and yellows types of diseases. Thus it appears that the changes in the carbohydrate metabolism under virescence syndrome does not follow a definite pattern.

Phenolic compounds have been shown to play key roles in the resistance of plants to disease causing pathogen (Cruickshank, 1963; Tomiyama, 1963; Kuc, 1972). Polyphenols are known to be powerful inhibitors of IAA oxidase (Hare, 1964), and may in turn be able to reverse the enzyme activity and confer resistance via auxin-sparing action on plants that produce them in response to infection (Lee and Tourneau, 1958). The observed pre-symptomatological rise in phenols may have a functional significance in symptoms development whereas the steep post-symptomatological rise may be a resistance stimulus.

In the initial phases of disease development, the NADH2-cytochrome system is enhanced whereas in the later stages, other oxidative enzyme systems are activated (Millerd and Scott, 1962; Goodman et al., 1987). These
include polyphenoloxidase (Barbara and Wood, 1972), ascorbic acid oxidase (Fric and Majernick, 1964), peroxidase (Lobenstein, 1963; Menke and Walker, 1963; Shankarlingam, 1980; Tripathy et al., 1975) and catalase (Peterfi et al., 1971a,b). In the present investigation, catalase activity in matured leaves remains low under infection with a sharp decline at 2 weeks after inoculation. Decrease in catalase activity has also been reported for several mosaic infections (Kar et al., 1985; Seyonara et al., 1972; Tosic, 1971). The peroxidase activity also declines steadily under infection with symptom advancement. The extent of decline in catalase is more severe in the early stages whereas the maximal decline in the peroxidase occur in the late phase. It has been suggested that catalase activity is positively correlated with chlorophyll during senescence stress (Kar and Mishra, 1976; Patra et al., 1978). No such correlation existed between chlorophyll and catalase activity under disease indicating that the change in catalase activity is a response to stress induced by the pathogen, independent of chlorophyll content. Catalase being peroxide-destroying and peroxidase, peroxide-utilising enzymes (Brenan and Frankel, 1977), their low activity level would spare peroxidase and super peroxides. Accumulation of these would form singlet oxygen (Brenan and Frankel, 1977) oxidise sulphhydryl group (Stonier and Yuan, 1973), and induce ethylene production (Brenan and Frankel, 1977), which stimulate senescence or cause high oxygen tension in tissue.
that triggers senescence (Frankel and Garrison, 1976). Such a view agrees with the generalization (Seigel and Porto, 1981) that the transition in plants from juvenility into senescence is accompanied by progressive shift from a reducing state to an oxidative state. Thus the observed low rate of BMD and early senescence of little leaves may be an outcome of disturbed hydroperoxidase level.

The polyphenoloxidase and peroxidase attack similar substrates (Kar and Mishra, 1976; Powell and Hildebrand, 1970). The slight enhancement in the PPO activity during early phases of disease development in face of reduced hydroperoxidase might be a response to meet the need for the recycling of reducing equivalents in the tissues. This view would agree with the observed reduction of polyphenoloxidase in diseased open proliferating flower buds where the hydroperoxidase increases several folds; and the increase in PPO in diseased embryonic tissue which reduce peroxidase unlike diseased open proliferating flower buds. The diseased closed flower buds show enhancement only in catalase but reduction in peroxidase and PPO. The total level of hydroperoxidases in proliferating flower buds and diseased embryonic tissues remains high and comparable. The higher activity level ensures (Mishra and Kar, 1985) the protection of these tissues against accumulation of oxidants and prevents accelerated ageing of the tissue due to disease stress (Kar et al., 1985). The low hydroperoxidase activity associated with early
senescence in diseased closed flower buds supports the above postulate.

Increase (Lodh *et al.*, 1971; Kar *et al.*, 1970; Mang *et al.*, 1974; Jaiswal and Bhatia 1971; Kar *et al.*, 1980), decrease (Panda and Kar, 1991; Somner, 1957), or unchanged (Tosic, 1971) levels of amino acid and a decrease in the level of protein (Kar *et al.*, 1983, Panda and Kar 1991; Lodh *et al.*, 1971) have been reported for various virus and mycoplasma diseases. The protein and amino acid content progressively reduce under pathogenesis. Work on metabolism of growing tissue during induction of growth shows that some endogenous metabolites exist in the cells in relatively inactive pools remote from the main course of metabolism. The pool of total free amino acid in the tissue is an example of one such inactive pool. Plant cells pass more of their carbon than is commonly supposed, through protein prior to its use in respiration. Sugar and simple nitrogenous compounds, or nitrogenous groups seem to move to some localised protein synthesizing center and there, form protein without liberating intermediate amino acids so that they can freely mingle with the stored aminoacid of the cells. The AA formed near the protein synthesising site, donot leave the site before they are bound into the protein. Therefore the metabolically active protein moiety may breakdown and its products, the AA, may be recycled or may be stored as such, or are converted into nitrogen-
rich storage products. By process of deamination and deamidation carbon frameworks of nitrogen compounds are introduced into the respiratory cycle at one point or another (Bidwell *et al.*, 1964; Steward and Bidwell, 1966). When this mobile and storage forms of nitrogen are reused, their nitrogen is donated at respective centre and the necessary additional carbon for protein formation comes more immediately from sugar than from the AA, which are present in bulk, free in cells. This has been shown by experiments in which radioactive sugar and AA were used (Bidwell *et al.*, 1964; Steward *et al.*, 1958). Keeping in view, the above fact, the results obtained in the present investigation are explained as follows. The decrease in the content of AA in the tissue results from the introduction of carbon skeleton of AA into the respiratory cycle to meet the highly enhanced rate of tissue respiration observed under pathogenesis. The growing need for respiratory substrates probably triggers a general proteolysis, depleting the levels of protein which is accentuated by non-availability of sugars to contribute the carbon skeleton for proteins. This view would also accommodate starch accumulation in face of low photosynthetic efficiency where the accumulated starch acts as the sink for most of the photosynthates. The low protein content probably restricts the normal growth of foliar tissue and results in stunting which is a characteristic symptom of the disease.
Harmones are considered to be mediators of physiologic process. They appear to modulate specific environmental clues into biochemic messages. Plant hormones, unlike animal hormones, are more concentration specific than target specific (Amen, 1974). Fluctuations in the concentration of endogenous auxins have been reported for many diseases (Pegg, 1976; Sequeira, 1973; Van Andel and Fuchs, 1972; Veldstra, 1968). These variations however, do not conform to any specific pattern and hence may be specific for a particular host-pathogen combination. In the present investigation, endogenous auxin concentration of eggplants increased under pathogenesis with symptom advancement. Of the two activity peaks obtained, one corresponded with the marker IAA and the other with Indole-3 acetaldehyde (IAAL) separated on solvent system on paper (Schneider et al., 1972). Hence, it is construed that the observed peaks are for IAA and IAAL. The auxins are degraded in hosts due to oxidation by IAA-oxidase (Hare, 1964) and peroxidase (Galston et al., 1968). Polyphenols are known to be powerful inhibitors of IAA oxidase (Hare, 1964). The accumulation of auxins under pathogenesis therefore might originate in the reduced levels of peroxidase in foliar, embryonic and diseased closed flower buds along with the rise in phenolic compounds.

Some of the morphological, physiological and biochemical changes observed in eggplant little-leaf syndrome may have their origin in the higher level of auxin produced under
pathogenesis. It is reported that IAA increase respiration and growth (Rowan et al., 1972) and that the rate of growth and respiration are closely connected (Audus, 1960). Indole-3 acetate regulates the synthesis of peroxidase (Galston, 1967; Glasziou, 1969) and increase in IAA concentration increases the activity of this enzyme to which it owes its own destruction (Glaston and Davis, 1970). Therefore it is suggested that increase in foliar tissue respiration and peroxidase activity of proliferating flower bud under pathogenesis may be the effect of higher level of auxins. However, Sequeira (1973) and Van Andel and Fuchs (1972) after evaluating the role of auxins in many other plant diseases have made the following observations. In general there is little evidence that auxin produced during pathogenesis plays an important role in initiating pathogen or in determining the nature of host pathogen interaction. Instead, changes in the auxin activity during pathogenesis probably results from alterations in the host metabolism induced by the pathogen.

Another objection to the involvement of IAA in symptomatology during disease development is the absence of apical dominance in diseased plants in spite of high auxin concentration. The relative inaction of auxin in apical dominance may be due to drastic reduction in GA levels and an augmentation in the levels of ABA, accompanying the rise in auxin concentration. The GA has been considered the primary determinant in apical
dominance, apical control and geotropic responses in shoots of many plants (Pharis et al., 1972). In many other plants GA promotes the auxin action in apical dominance (Jacobs and Case, 1965; Scott et al., 1967; Shein and Jackson, 1972; Thomaszewski, 1970). Cleland (1968) stressed that GA exerts greatest influence on subapical meristem of a plant where the mitotic activity is largely regulated by its concentration (Sachs et al., 1959) and controls the synthesis and/or release of amylase, catalase and pyrophosphatase. The reduction in the level of catalase and lower proportion of amylase than accumulated starch would dictate, may be an outcome of lower GA concentration seen under infection. The GA also promotes RNA and the total protein content of tissue and brings about a change in the pattern of enzyme development (Mc. Comb et al., 1970). These metabolic changes in response to GA maintain rather than initiate increased growth. Endogenous levels of GA maintain flowering in Citrus (Glodschimdt and Monselise, 1972), and slight quantitative variation may shift the balance between vegetative and reproductive growth. This agrees with the observed shift from reproductive to vegetative phase of growth under pathogenesis. Abscisic acid lowers RNA and protein content (Poulson and Beevers, 1970), and inhibits flowering (Sembdner et al., 1972). The ABA may also inhibit the GA synthesis (Beevers et al., 1970; Smith and Sadri, 1970) and inhibit auxin action (Addicot, 1972; Adanson et al., 1972)
though the opposing influence of auxin, IAA and ABA are biochemically non-competitive.

Many plants exposed to stress condition respond by rapid accumulation of ABA-like substances (Weight and Hiron, 1969; Mizzrahi et al., 1970; Mohanty et al., 1979; Steadman and Sequeira, 1970). The pathogenesis resembles stress phenomenon and the increase in ABA-like substances in diseased plants fits into this framework. It is therefore suggested that changes in the endogenous hormonal balance probably is responsible for the symptomatology through their effect on altered metabolism.

It is known that mycoplasmas depend on sterols for growth (Doi et al., 1968; Maramorosch, 1972) and that mevalonic acid is the precursor for biosynthesis of terpenoids in plants (Barnes et al., 1968), where farnesyl pyrophosphate forms the branch point for the synthesis of sesquiterpenes and steroids (Britton, 1976). The steroids are synthesized via sequalane pathway (Siperstein, 1970). The ABA is thought to be derived from carotenoids in light (Taylor, 1968), and through mevalonic acid pathway in a manner analogous to sequalane pathway in darkness (Millborrow, 1974). Farnesyl pyrophosphate adds one molecule of isopentenyl pyrophosphate to form geranyl geranyl pyrophosphate which is the precursor of GA and carotenoids biosynthesis. The GA is synthesized via kaurene pathway (Barendse, 1975) and carotenoids via phytotene (Britton, 1976). Therefore, it
is probable that mycoplasma infection interferes with the biosynthesis of GA by selective inhibition of pathway, leading to its synthesis or by cannalisation of precursors through sequalane pathway to meet its sterol requirements. The latter proposition, although explains the observed rise in ABA-like substances, fails to accomodate the unchanged levels of carotenoids in diseased plants. Therefore the former proposition, appears more acceptable. Such situation would agree with Pegg's (1976,a,b) view that change in activity of endogenous growth substances under pathogenesis probably results from an alteration in host metabolism.

Several, morphological, physiological and biochemical changes in higher plants are caused by deficiencies or toxicity of micro-nutrients. Mass spectroscopic analysis of foliar micro-nutrient composition shows that diseased leaves have very low concentration of Fe and Mn and very high concentration of Zn, Cu and Mo in comparison with healthy. These changes are interesting in face of observed biochemical anomalies. The chlorophyll content of chloroplast is reduced under iron deficiency (Terry, 1978). The Mn is implicated in in vitro photolysis in PSII (Bishop, 1971). Chloroplast contains 1 atom of Mn/14-600 chlorophyll, but only 1 Mn/50-100 chlorophyll is assumed for the pool associated with PSII and needed for maximum O₂ evolving activity where the O₂ evolving enzyme complex seemingly contains 4 Mn atoms (Cheniae and Martin,
Usually, two groups of iron containing proteins in cells are well defined. They are: (i) Fe-sulphur proteins where Fe is coordinated to inorganic S and/or to thiol group of cysteine, and (ii) hemoproteins containing Fe inserted into porphyrin skeleton. A third group include Fe-super oxide dismutase and many less characterised proteins (Sandman and Bager, 1983). One type of Fe-sulphur protein having 2 tetranuclear Fe-clusters (4Fe-4S) with centre surrounded by 4 cysteine residues is present in thylakoids. These iron-sulphor proteins are denoted as ferredoxins when they act exclusively as electron carriers (Bohme and Boger, 1982). Heme proteins or iron-porphyrins form cytochromes for electron transfer of which cyt b559, cyt.b563 (Boger and Bohme, 1982), cyt C553 and cyt f553 (Bohme et al., 1980) are found in chloroplasts for photosynthetic electron transport. Excess of Cu results in an inhibition of the photosynthetic electron transport by reciprocal formation of cyt C553 (Bohner et al., 1980) and decomposition of chloroplast membranes (Sandman and Boger 1980a). This happens due to competition of redox active Cu with peroxo compounds within chloroplasts. The radicals originating from these metal catalysed Fenton type reaction cause peroxidative degradation of membrane lipids (Sandman and Boger, 1980). Many other heavy metals like Cu, exert inhibition along photosynthetic electron transport chain. Their favoured target is water splitting system. Copper also inhibits electron flow at the reducing
site of PSI presumably by interfering with ferredoxin (Shioi et al., 1978). Thus, it appears that the change in the foliar micro-nutrient status, particularly the severe paucity of Mn and Fe and abundance of Cu, result in the loss of water-splitting ability of PSII and long wavelength absorbing pigment from PSI seen under pathogenesis.

Of the several superoxide dismutase (SOD), the Cu,Zn and Mn containing SODs are found in higher plants (Asada et al., 1977). The Mn-SOD is found in mitochondria (Jackson et al., 1978), and Cu-Zn-Mn SOD in chloroplasts (Bridges and Salin, 1981). All types catalyse superoxide radicals O$_2^-$ to H$_2$O$_2$ and O$_2$. Renger (1977) proposed an interesting line of thought on the possible reaction leading to free O$_2$, mediated by Mn involving a SOD from chloroplast as a part of the O$_2$ evolving system. Even with this possibility the extreme paucity of Mn under disease syndrome would naturally inhibit the system.

Peroxidase and catalase are hemoproteins with protoheme as the prosthetic group (Sandmann and Boger, 1983) and are localised in leaf peroxisomes (Talbert et al., 1983). Therefore, the paucity of Fe may have led to the lower levels of these enzymes under pathogenesis. In fact, their low activity is used to test Fe deficiency in plants (O'Sullivan et al., 1979). The concentration of ABA in plants is known to increase
under stress and nutrient deficiencies (Mizzrahi and Richmond 1972). This increase in ABA level has a feedback effect on mineral ions into the xylem and thence to shoots causing relatively greater changes in the concentration of ions in the shoots of the plants suffering from deficiency (Moorby and Besford, 1983). Thus it appears that the spectacular rise in ABA-like substances during the course of disease development is intimately associated with the observed deficiency of some micro-nutrients. Probably the deficiency arises from reduced rate of absorption and/or inhibition of transport into shoots from roots since the retranslocation rate of Fe, Mn, Zn, Cu from the senescent tissue to growing tissue is very high (Marschner, 1983).