A. INTRODUCTION

The sequence of events leading to establishment of a facultatively parasitic pathogen is essentially different from those in obligate parasites. In an obligate parasite, successful parasitism is dependent on a genetically compatible reaction and is accomplished by the establishment of a stable haustorial relationship by the pathogen on the compatible host. The pathogen can thrive only on the living cells of the host. A compatible host–parasitic reaction is characterized by hypersensitive reaction which results in death of invaded cells as a response to attack by the pathogen, consequently the pathogen cannot establish infection. A substantial amount of work has been done on the biochemistry of infection in case of obligate parasites including viruses. Metabolic changes in the post infection stage in response to attack of such a pathogen have been recorded.

A facultative parasite after penetration into the host plant, normally does not enter into haustorial relationship with the host. It usually kills the host tissue in advance of penetration. The mechanism of killing the host cells may involve enzymes or toxins or both. Facultative parasites are capable of secreting a large array of enzymes which may be constitutive or adaptive. Evidences of toxins have been noticed and their roles in pathogenesis
have been investigated in a number of such pathogenic fungi. Nevertheless the invading pathogen is often limited in its attack by either mechanical barrier of the host plant or chemical barrier which may be produced by the host plant in response to attack of pathogen. In majority of cases the pathogen is arrested after certain advancement. Resistant reaction is determined by the quickness of check of progress of pathogen and the extent of attack. Hence it may be assumed that pathogen which is facultatively parasitic is likely to induce some metabolic changes in the host tissue even though it may be affected either through enzymes or tissues which will kill them. Resistant reaction may be produced in response to attack. But sufficient investigation has not been carried out with facultative parasites in this context. Biochemical or metabolic changes induced as a result of infection have not been fully explored.

_Helminthosporium oryzae_, the incitant of brownspot disease of rice produces discrete brown necrotic lesions which are sharply defined. The pathogen is also host specific. Quantum of infection is determined by the number of lesions as well as their sizes. Investigations by Dasgupta (1968) have shown that two distinct factors are involved one concerning with penetration and another with lesion expansion. Factors concerned with penetration are largely mechanical or anatomical, whereas biochemical or internal
factors determine the course of reactions concerned with lesion expansion. Delimitation of lesion is determined by the defensive mechanism of the host plant, it is proposed to investigate some pertinent biochemical or metabolic changes that might be induced in response to attack of pathogen in the resistant and susceptible varieties. Studies of Akai et al. (1958-66), Asada (1956-59, 1962, 1967), Oku (1958-60, 1962, 1965), Shishiyama et al. (1969) on the biochemistry and pathophysiology of brownspot of rice have indicated that changes may occur in the rice plant due to infection of \textit{N. oryzae}. It is hoped that findings would be helpful in determining the induced changes and reactions that characterize a resistant and a susceptible plant and the mechanism of disease resistance.