INTRODUCTION

Tea [*Camellia sinensis* (L.)O.Kuntze] forms the backbone of the economy of North-East India. Being a perennial the tea plant possibly interacts with, and samples more environmental (both physical and biological) problems than does any other plant. Black rot caused by *Corticium theae* Bernard is one of the foliar diseases of tea (Plate I) which is very common in the plains but rare in the hills. It attacks all teas from their seedling stage upwards. The disease persists in the same areas for years, if not controlled, causing gradual deterioration in the health of the tea and loss in crop. It thrives best and persists for years in the badly ventilated places, often where air movements are prevented by bamboo plantations, under overdense shade or areas surrounded by jungle, especially where the air in rainy season is hot, moist and still. Black rot is more prevalent on tea which has been cut across without any cleaning out and unskiffed and unpruned tea. *C. theae* produces on the leaves large patches covering about half and sometimes the entire leaf area. Colour on the uppersurface of the affected area at the early stage is reddish-brown, similar to sun-scroch damage, later it is a mixture of brown, yellowish-brown and grey; the undersurface is light brown or greyish-white and usually covered with a net work or cream to brown mycelium. Diseased leaves often remain attached, to other leaves and stems, held together by small cushions or films of pinkish-white or cream coloured mycelium. The fungus produces on the stem, thick cords of mycelium, up to about 3 mm across, dark purplish-brown on the older portions of the stem and dull white to light brown on the green portions at the top.The fungus produces minute resting bodies (sclerotia) in the cracks and crevices of the stem towards the end of the rainy season. Fructifications appear, during the rainy season, as white, dusted patches on the undersurface of mature, green leaves. The fungus spread not only by direct contact from bush to bush but also by wind, birds as well as by workers of the tea garden

In nature plants survive in the face of attack by many microbial organisms that threaten their survival and attempt to use them as a food source by employing several layers of defense response. The interaction between plants and their pathogens is complex and may be very specific to a given combination of the plant and the fungus. The defence strategies of plants against their pathogens are manifold and include the use of antifungal chemicals. On the other hand, pathogens have evolved mechanisms to evade these chemicals. Heath (1980) has argued effectively for the differentiation of the responses of plants to pathogens based on host and non-host interactions.

Plate1

In such relationships it has long been recognized that responses are characterized by the early accumulation of phenolic compounds at the infection site and that limited development of the pathogen occurs as a result of rapid (hypersensitive) cell death (Fernandez and Heath,1989). Regardless of the reasons for cell death, it is thought that rapid accumulation of phenols may result in the effective isolation of the pathogen at the original site of ingress. These responses include the formation of lignin, the accumulation of cell-wall appositions such as papillae, and the early accumulation of phenols within host cell walls. Numerous studies suggest that low molecular weight phenols, such as benzoic acids and the phenylpropanoids are formed in the initial response to infection. Most research on resistance mechanisms has shown that the plant uses defenses that are activated after infection to stop pathogen development (Dixon and Harrison,1990). Many biochemical changes occur in plants after infection, and some of these have been associated with the expression of defense they have activity against pathogen *in vitro.* One of the best and longest-studied defense response of plants to infection is the induced accumulation of antimicrobial, low-molecular weight secondary metabolites known as phytoalexins (Hammerschmidt, 1999; Harborne, 1999; Greyer and Kokubun, 2001).

Host and parasite iteraction can also be correlated with lock and key function. Notches on the parasite i.e. 'key' are those factors required to allow colonization of plant tissue and/or to overcome host resistance factors. In order to colonize host tissue and reproduce, a successful parasite must have accumulated the genetic information to eliminate, overcome, avoid or escape all of the host defenses encountered. Although plants do not produce antibodies, as animal do, against invading pathogens, still some kind of immunological response may be operating in plants. The striking similarities of cell surface chracteristics have been critically emphasized. The complexity of the interactions that affect the selection of parasites and allow their establishment and survival among host cells is manifested in the frequency and variability of cell surface antigens. Some intriguing research work suggests that antigenic similarity between host and pathogens may be a prerequsite for compatible reactions or in other words, successful establishment of the pathogens in host depends upon some kind of molecular similarity between two partners (Devay and Adler, 1976). However, only

certain key-common antigens are important in host-parasite compatibility (Chakraborty and Purkayastha, 1983; Alba and Devay,1985; Chakraborty and Saha, 1994; Chakraborty et.al, 1995; 1997; 2002). It has been observed that with increased antigenic disparity, the response of host may prevent further activity of the parasite (Chakraborty,1988; Purkayastha, 1994).

The basic objectives of the present investigation are (a) screening of Tocklai and UPASI tea varieties for resistance to *C.theae;* (b) determination of level of phenolic compounds in the leaves of resistant and susceptible varieties before and after infection with *C.theae*; (c) assay of peroxidase (PO) and phenyl alanine ammonia lyase (PAL) activities in the leaves before and after infection with *C.theae ;* (d) ascertaining the antifungal activity of phenolics associated with differential host response to infection; (e) estimation of host-parasite proteins before and after infection and analysis by SDS-PAGE; (f) extraction of antigen from mycelial and cell wall preparations of *C.theae*; healthy, artificially inoculated and naturally black rot infected tea leaves; (g) raising of polyclonal antibody against antigens of tea leaves and *C.theae*; (h) detection of serological cross reactivity between *C.theae* and tea varieties following immunodiffusion tests and enzyme linked immunosorbent assay (ELISA); (i) detection of *C . theae* in artificially inoculated tea leaves by ELISA; (j) detection of pathogen using dot blot and (k) determination of the cellular location of cross reactive antigens (CRA) in tea leaf tissues, mycelia and sclerotia of *C..theae* using immunofluorescence

Before going into the details of the present work, a brief review in conformity with this study has been presented in the following pages.