STUDIES ON PERMEABILITY CHANGES IN COTTON
(GOSSYPIUM HIRSUTUM) CAUSED BY
VERTICILLIUM ALBO-ATRUM*

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ABSTRACT

The present work demonstrates permeability changes in Verticillium-infected cotton tissues. Permeability was determined by measuring the exosmosis rate of electrolytes. The roots and leaves showed more electrolytic leakage as compared to the stem. This leakage while continues in the leaf tissue, a fall was recorded later for the roots. The stem tissues were affected least. The ash analysis of the diseased leaves showed a marked decrease in the potassium and sodium ions as compared to the healthy tissues. Calcium showed increased accumulation in diseased tissues. There was a decrease in total nitrogen content of diseased leaves while the infected root and stem tissues did not show much difference from the healthy tissues. The total carbohydrate content of the diseased leaves was considerably low but the roots showed an increase over the healthy tissues.

INTRODUCTION

Similarities in physiological and biochemical events in diseases caused by a variety of pathogens indicate a common trigger. Kuc (1966) suggested a mechanism based on enzyme induction and repression mediated through effects on nucleic acids. Altered permeability of the cell membranes is another attractive trigger as it can be induced by a variety of physical, chemical and mechanical stimuli. The first act of the parasite, comments Sempio (1959), is to open the door of the larder. Bollard and Matthews (1966) state that the first effect on host tissue appears to be an increase in permeability which makes nutrients available to the parasite. Alteration in cellular permeability leads to destruction of turgor which inevitably disturbs the water balance. The regulation of the 'milieu interior' is disturbed. An impairment of the membrane would alter compartmentalization of normal

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biochemical functions in host cells. Mixing of enzymes and substrates separately compartmentalized in a healthy cell could initiate the oxidation reactions suggested to be associated with necrosis.

Hutchinson (1913) working with Rangpur tobacco wilt caused by *Pseudomonas solanacearum* first reported altered cellular permeability. Thatcher (1939, 1942, 1943) established the loss of permeability before the appearance of external symptoms in rusts, powdery mildews, soft and dry rots and wilts and postulated that increased permeability of the plasma membrane or host cytoplasm would make available nutrients to parasites resulting in susceptible reactions. Decreased permeability, on the other hand, would restrict the nutrient supply and result in resistance. However, Thatcher's conclusion on decreased permeability favouring resistance is now contested by Wheeler and Hanvey (1968).

Gottlieb (1944) found that fluid in vascular bundles of tomato plants infected by *Fusarium lycopersici* increased the water permeability of the medullary cells of tomato shoots almost three-fold. Gaurman (1958) believed that altered permeability of leaf cells was a primary factor in wilt diseases. The semi-permeability of the plasma membrane in tomato plants is injured by fusaric acid in such a way that metal ions, amino-acids and peptides escape from within the cell into the transpiration stream of the cell-walls; hence the physical precondition for osmotic pressure and, therefore, of turgor are destroyed.

Sadasivan (1955) suggested that toxins/antibiotics interfered with enzyme systems and produced derangement of key metabolites starting a chain of derangements. Linskens (1955) detected leakage of ions like K, Ca, Na and amino-acids from lycomarasmin and fusaric acid afflicted leaves. Gnanam (1956), Sadasivan and Kalyanasundaram (1956), Sadasivan and Saraswathi-Devi (1957) have determined the ionic imbalance in *Fusarium*-infected cotton plants.

In recent years, victorin, a toxin produced by *Helminthosporium victoriae*, has been shown to have a primary effect on cell permeability (Wheeler and Luke, 1963). Increased respiration in victorin-treated tissues has been shown to be caused by the leakage of ions, salts and acids from the tonoplast as a result of toxin action on mitochondria resulting in augmented respiration (Black and Wheeler, 1966). Prior leaching in water of the victorin-treated tissue removes the ions and then no increase in respiration occurs (Amador and Wheeler, 1966). Further victorin added to isolated mitochondria does not affect the release of electrolytes or oxidative phosphorylation. Electron