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PATHOGENESIS OF *RHIZOPUS ORYZAE* WENT AND GEERLINGS IN FRUIT-ROT DISEASE OF BRINJAL (*SOLANUM MELONGENA* L.)

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Rhizopus oryzae Went and Geerlings have been reported to cause rot diseases of apple, banana, mango, potato, tomato[9], garlic[5], and sugarbeet[7] in storage and fields in various parts of India. During a survey of vegetable fields around Gwalior (M.P.) from November to March (1977 and 1978) a soft rot of brinjal fruits was observed. The incidence of disease was recorded on both young and mature fruits during this period. However, the disease incidence was relatively low on younger fruits. Isolation from the diseased lesions showed *Rhizopus oryzae* Went and Geerlings (IMI No. 233341) to be the causal organism of this fruit-rot.

In nature the disease starts as a soft watery rot accompanied by tissue decoloration of the brinjal fruits, which later on develop browning, and the symptoms progress rapidly in the healthy tissues. Later the pathogen covers the fruit surface with effuse growth and the entire fruit rots in about 15 20 days. Pathogenicity experiments, using Granger and Horne's[8] method of inoculation, proved the Koch's postulates. The organism was found to be a wound pathogen as the infections of fruits were mainly caused through the surface wounds (Fig. 1). Since, the rot syndrome

Fig. 1. *R. oryzae* infected fruit.

exhibited much involvement of macerating enzymes in producing the disease, the enzyme complex *in vivo* was also investigated. For these studies, extracts from the diseased tissues were obtained at different periods of incubation for the assays of enzymes of the pectolytic and cellulolytic moieties. The preparation of extracts and assay procedures followed were similar to those reported elsewhere[1,2,4].

The results of these studies (Table I) showed that the pectolytic enzyme moiety, of the pathogen *Rhizopus oryzae*, consisted of polygalacturonase (PG), pectin methylgalacturonase (PMG), and pectinmethylesterase (PME) whereas, cellulase (C_1 and C_x) represented the cellulolytic moiety. The quantitative production of PG and PMG was relatively high at 5 and 10-day stages of the disease than that of PME. Quantities of cellulase were higher at 10 and 15-day stages

Table I

Periodic levels of pectolytic and cellulolytic enzymes produced in vivo by R. oryzae

Incubation period (days)	Relative enzyme activity (REA)			
	PG	PMG	PME	Cellulase
5	67	63	12	36
10	50	59	14	77
15	21	27	10	59
20	20	14	10	27

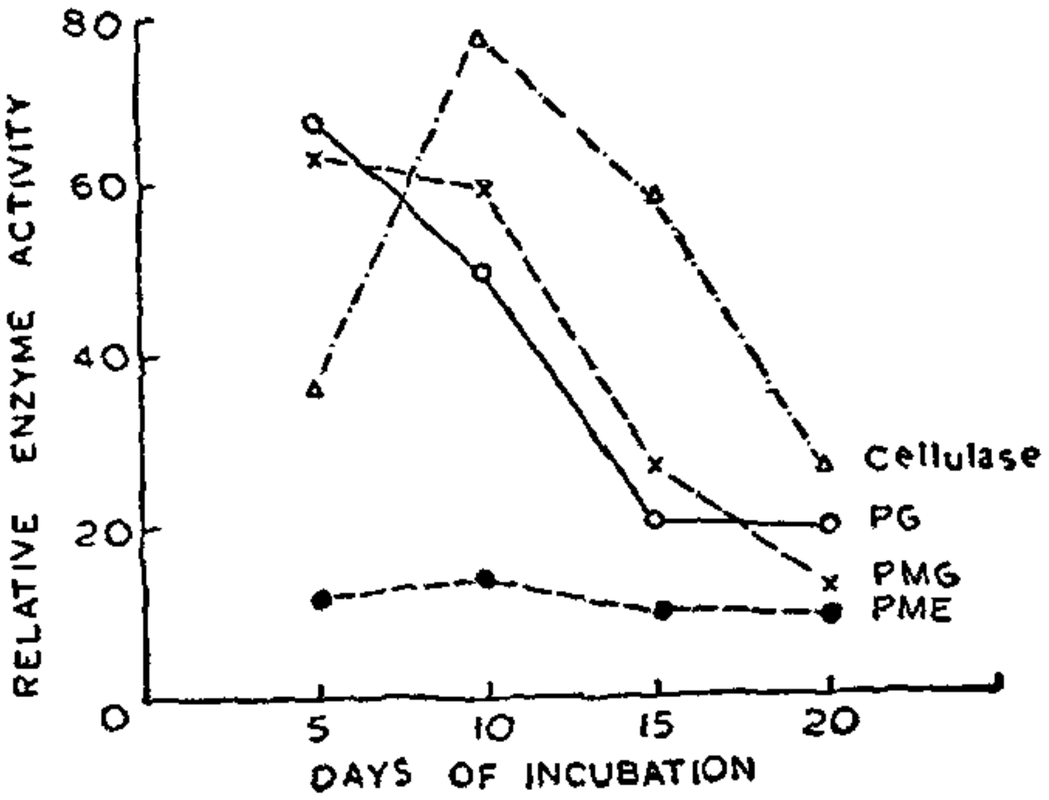

FIG. 2. Enzyme levels at different incubation periods, produced by *R. oryzae, in vivo.*

Production of higher amounts of PG and PMG at the initial stages of pathogenesis indicated their direct involvement in maceration of host tissues and degradation of pectic materials contained. Subsequent production of cellulase further facilitated degradation of the cell walls and helped to create the free nutrient pool, besides forming spaces for the growth of pathogen and invasion of the host tissues. PME was not found to be significantly involved in pathogenesis in this case (Fig. 2). However, it appeared that the quantities of these enzymes decreased to a sufficiently low level with the progress of rot.

Rhizopus oryzae Went and Geerlings, though reported to parasitize several hosts[6–9], is being reported here for the first time as a fruit-rot pathogen of brinjal. During its pathogenesis PG, PMG, and cellulase were mainly responsible in producing the disease syndrome.

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TWO HYPERPARASITES ON *ACROSPORIUM DENDROPHTHOAE*

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RECENTLY Ramachar and Bhagyanarayana[2] reported the conidial stage of a powdery mildew on *Dendrophthoae falcata* (Linn. f.) Etting. as *Acrosporium dendrophthoae*. In their further effort to collect the perfect stage, of the fungus two hyperparasites were observed and the same are reported in this note.

The mycoparasites were isolated in culture by the dilution plate technique and colonies cultured on potato sucrose agar medium. A microscopic examination revealed them to be as *Alternaria alternata* (Fr.) Keissler and *Cladosporium spongiosum* Berk. & Curt. (Figs. 1B and C).

Both *A. alternata* and *C. spongiosum* were found to enter into the ectotrophic mycelium, conidiophores and conidia (Fig. 1A). Due to the infection, plasmolysis took place resulting in the complete collapse and disintegration of the mildew fungus. It was noted that the spores obtained from the infected areas lost their viability and failed to germinate.

A careful survey of the literature[1,3,4,5,6] shows the presence of *Ampelomyces* sp. (= *Cicinnobolus* sp.) as a hyperparasite on the powdery mildews. There is no record of *A. alternata* hyperparasitic on powdery mildews and *C. spongiosum* hyperparasitic on *A. dendrophthoae* and is therefore to be taken as first records.

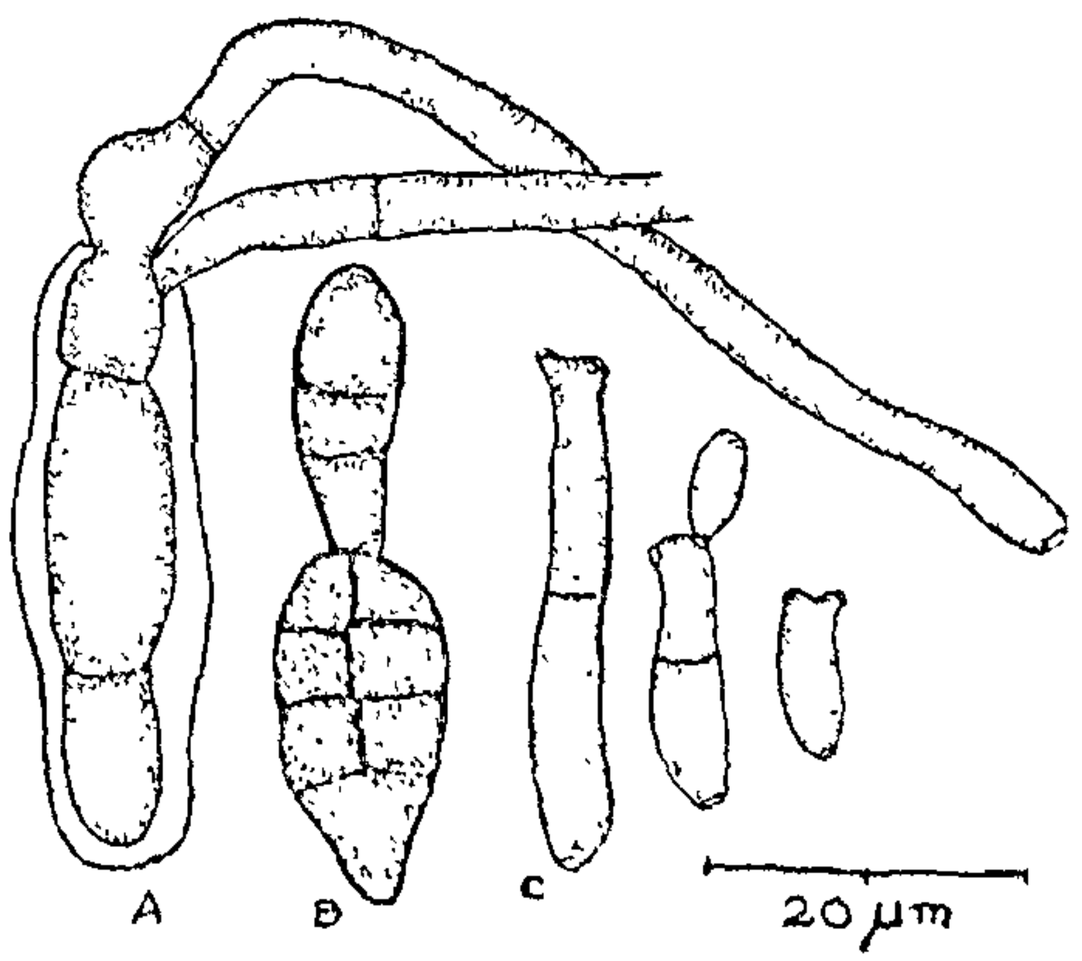

FIG. 1. (A) An infected conidium of *Acrosporium dendrophthoae* showing internal septate mycelium and the conidiophores. (B) Conidia of *Alternaria alternata*. (C) Conidia of *Cladosporium spongiosum*.