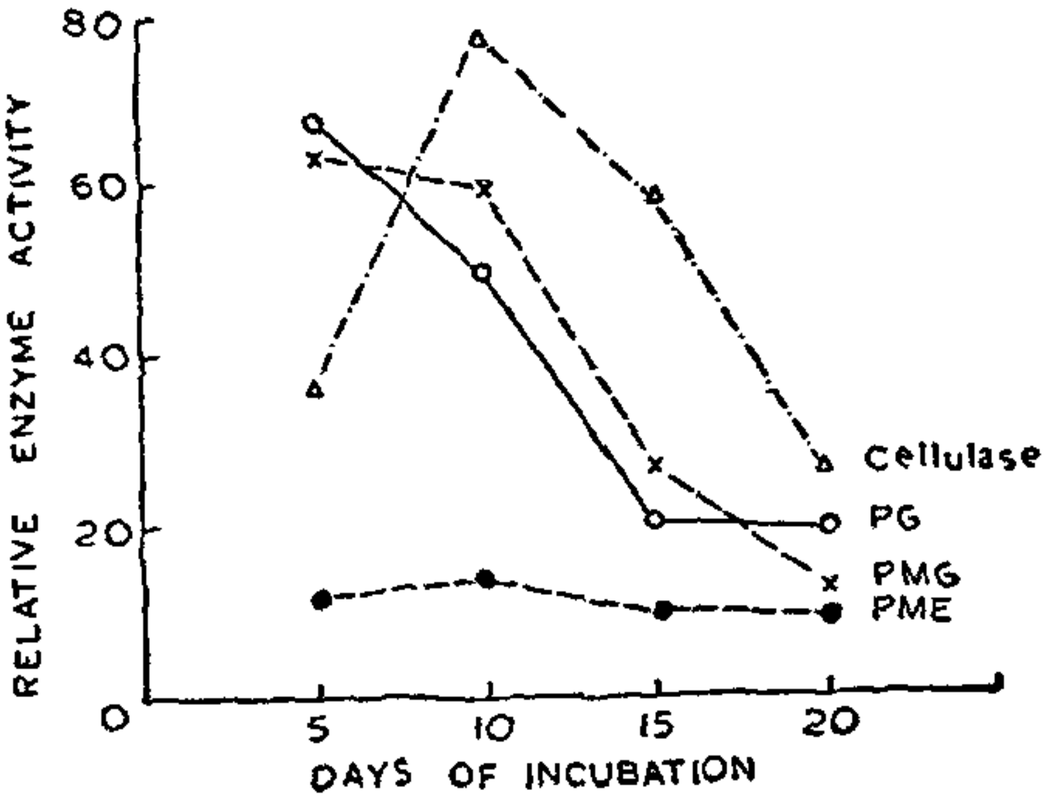

FIG. 2. Enzyme levels at different incubation periods, produced by *R. oryzae, in vivo.*

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.

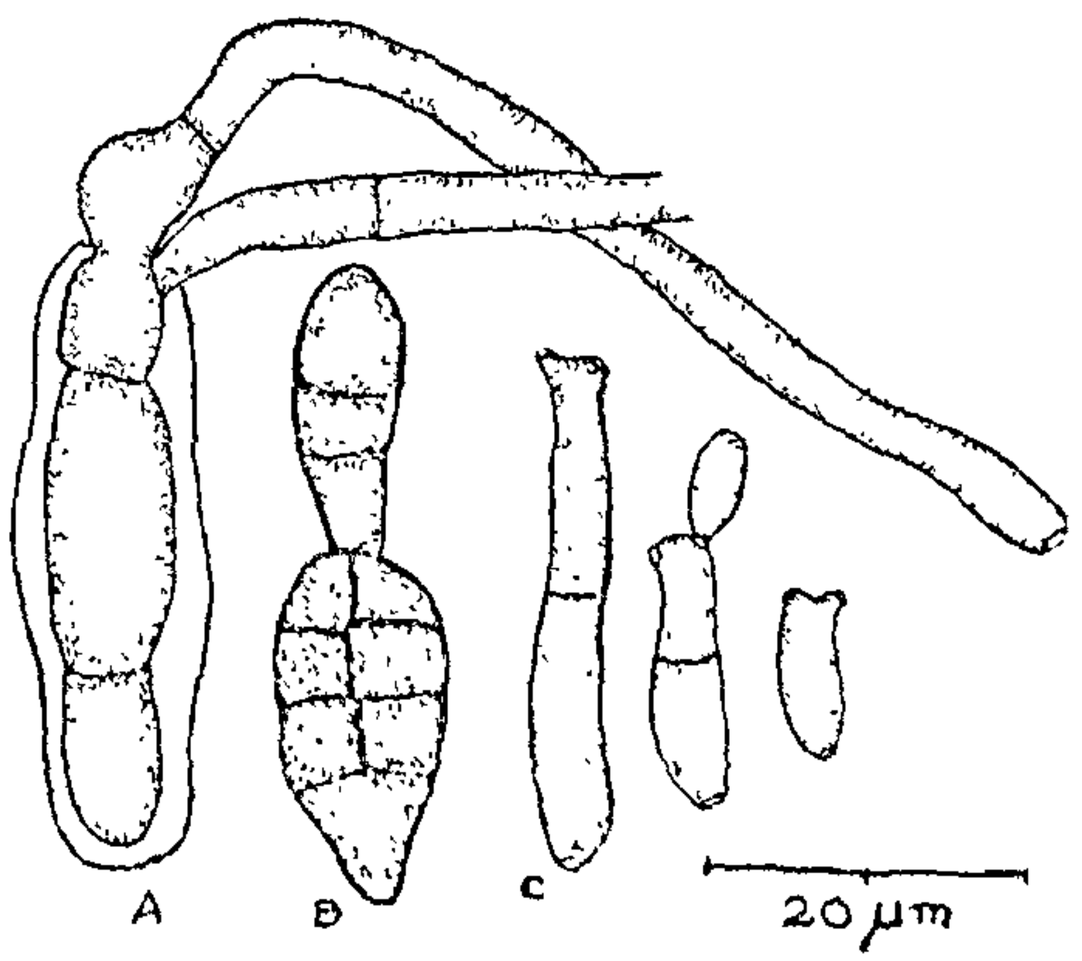

FIG. 1. (A) An infected conidium of *Acrosporium dendrophthoae* showing internal septate mycelium and the conidiophores. (B) Conidia of *Alternaria alternata*. (C) Conidia of *Cladosporium spongiosum*.

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OCCURRENCE OF PEACH YELLOWS IN INDIA

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Yellows disease of peach is one of the oldest diseases known in the history of plant virus diseases. The disease was recorded as early as 1791 in Philadelphia[5] and its first report was published in 1888[6]. The disease is of great economic importance as it appeared in an epidemic form for six times between 1791 to 1888 in various parts of America. In 1974, 3 peach trees in the vicinity of IARI Regional Station, Kalimpong, were observed with symptoms of peach yellows as described by Hartzell[4]. The incidence of the disease varied in different orchards from 5 to 30%. The results of transmission studies are reported in this note.

The scions collected from naturally affected trees were wedge-grafted on 5 seedlings of a local variety on peach var. Holton, in 1975. The first symptoms of the disease appeared in three seedlings and four Holton peach in autumn of 1976 as premature unfolding of the leaf buds on one or two branches. Numerous upright growing shoots bearing small slightly chlorotic leaves developed on infected branches, thus giving a 'Witches broom' appearance (Fig. 1). The small yellowish leaves were dotted with red spots and continue to grow after the fall of normal leaves. Twigs developed from the affected branches have a tendency to grow vertically. The larger leaves of affected plants were mottled. Similar symptoms were also seen on peach seedlings which were graft-inoculated with the diseased material from glasshouse-grown plants and also by the dodder, *Cuscuta reflexa*.

It was observed that the flower and leaf buds in the naturally affected trees as well as glasshouse-inoculated plants develop earlier than in healthy trees. The fruits on the affected branches of outside trees or on infected plants in the glasshouse were larger but of inferior quality with a bitter taste and ripen earlier than those on healthy trees. The affected branches died within 2–3 years following infection.

In 1977–78 some plants of different peach varieties were brought from Simla (H.P.) to study the host range of peach X disease recently reported from this region[1]. It was observed that one plant of each variety J. H. Hale and July Alberta among this lot carried yellows infection suggesting that this disease may be present in Himachal Pradesh also. A disease with similar symptoms was recorded on *Prunus puddum* in Simla at Plant Introduction Station and designated as paja rosettee[3] which appears to be peach yellows. Hence the growers must be cautious while using *P. puddum* as a root stock in Simla Hills.

The symptoms of the disease on peach as described above closely resemble those of peach yellows described by Hartzell and of little peach rejected by Cation[2]. The disease, in question, differs from little peach as fruits on little peach affected trees are smaller and ripen late. Further, the typical foliage and 'Witches broom' symptoms developed on inoculated

Fig. 1