It is common practice to group catalase among the respiratory enzymes, although its actual function is poorly known at present[5]. The activity of catalase generally decreased in the infected host[11]. It seemed that the host cell catalase is enclosed in high molecular protein during the process of sporulation so that the activity of the enzyme can only be exerted under appropriate concentrations after the sporulation of the fungus. The lower content of catalase in the case of virus infection has also been explained on the basis of its multiplication[10].

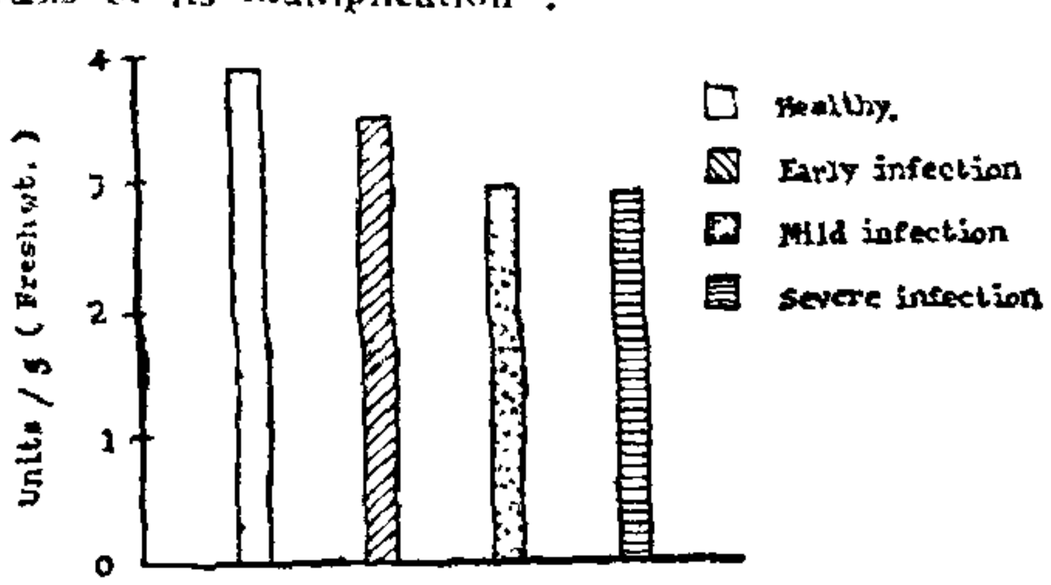

FIG. 1. Catalase activity of healthy and infected diseased samples of leaves.

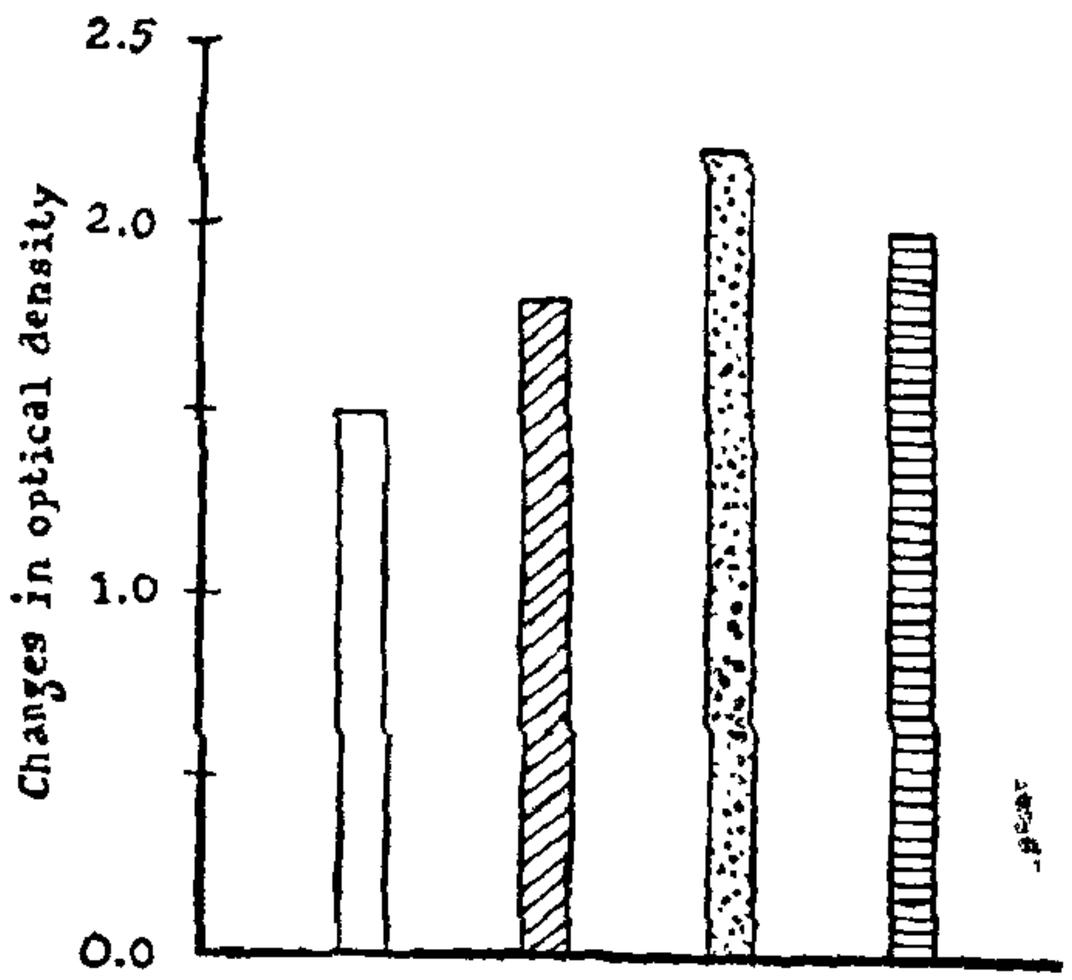

FIG. 2. Peroxidase activity of healthy and infected diseased samples of leaves.

Several workers have demonstrated an increase in peroxidase activity in different virus and bacterial infected plants[4,7,12,13,14,15,16,18]. Loebenstein and Linsey[4] showed a positive relation between peroxidase activity and developing of vein clearing in infected sweet potatoes. As yet, the physiological role of peroxidase, even in normal metabolism, is not clearly understood, although it is known to catalyse the oxidation of phenolic substances and aromatic amines to quinones in the presence of H_2O_2[17]. An increase in content of peroxidase due to infection discussed for viruses and bacteria might also be true for infection caused by *Gloeosporium pestis* Massee.

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BACTERIAL LEAF SPOT AND STEM BLIGHT—A NEW DISEASE OF SAFFLOWER IN INDIA CAUSED BY *PSEUDOMONAS SYRINGAE*

A BACTERIAL disease of Safflower, *Carthamus tinctorius*, L. occurred in severe proportions during October–November, 1977 in Coimbatore immediately after the rains. Plants (3–4 week old) in their rosette stage exhibited severe blight which appeared as dark necrosis in their shoot tips and stem tissues. The interior tissues of the stem were rotten (Fig. A), which

often extended below the soil line into the roots. The leaves also showed necrotic spots and streaks (Fig. B); the tissue in the centre of the leaf spot became translucent and appeared encircled with a dark brown or black margin. The affected plants usually collapsed due to progressive rotting and in most cases only the black stem was left in the field.

FIG. A

This report summarizes studies on the symptoms, pathogenicity and biochemical characters of the causal organism. These studies indicate that this disease is the same as the disease of safflower reported by Erwin *et al.*,[2] in 1964 from California. No other bacterial disease is known to occur on safflower and this is the first time this disease has been found in India.

Isolations from macerated infected marginal tissues of leaf spots or stem lesions consistently yielded rough white bacterial colonies on nutrient agar. The colonies turned cream coloured to yellowish as they matured and were readily pathogenic to safflower. For inoculation, the bacterium was multiplied in nutrient broth for 48 hours on a rotary shaker; the cell suspension was adjusted to an O.D. of 0·05 at 420 nm (approximately 10^6 cells/ml.) and used as inoculum. The different methods of inoculation suggested by Erwin *et al.*,[2] were tried using safflower seedlings of 21 days. The plants were inoculated either by infiltrating the lamina with bacterial suspension or by injecting the bacteria into the petioles with a hypodermic needle or by rubbing the carborundum dusted leaves with the cell suspension. The inoculated plants were incubated in a moist chamber at 25–27° C. The inoculated plants showed the appearance of leaf spots in 2 days and were killed in 5–7 days due to rotting of the leaf and stem tissues.

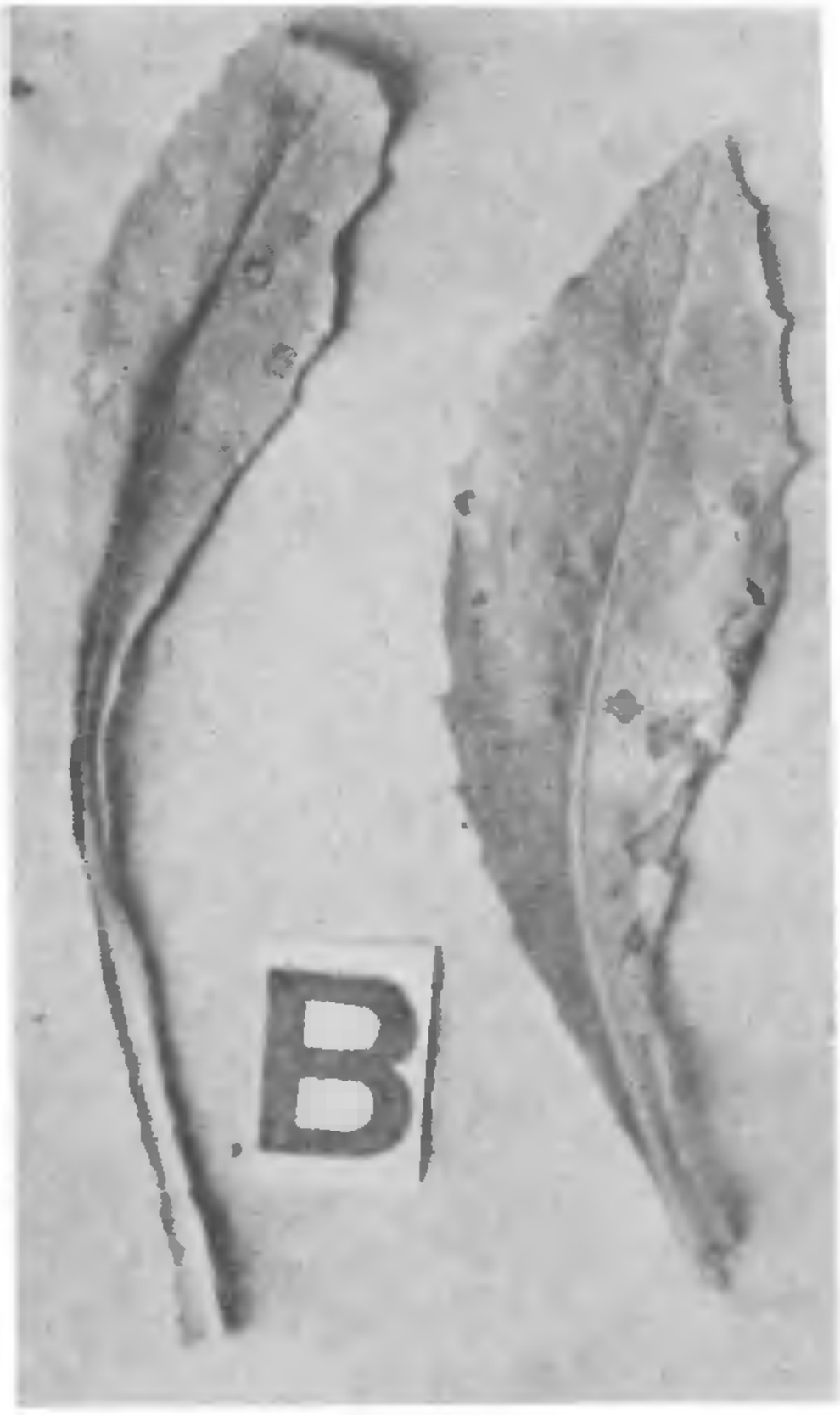

FIG. B

The causal organism is a rod-shaped bacterium; cells varied between 0·6 × 1·5-2·0μ in size. It is a Gram negative organism and is motile with several polar flagella. On medium King B[3] the isolate produces a green fluorescent pigment. It produces acid, but not gas, from glucose, sucrose, mannose, galactose, mannitol and glycerol and does not produce acid from maltose or lactose. Nitrites are not formed from nitrates. Neither indole nor H_2S is produced. Gelatin is readily liquefied. The organism was also screened using the LOPAT determinative tests developed by Lelliot, Billing and Hayward[4]. It was tested for its pathogenicity on the following other hosts: *Lycopersicon esculentum*, *Dolichos lab-lab*, *Vigna sinensis*, *Zea mays*, *Sorghum vulgare*, *Pennisetum*

typhodes and the tests were positive for pathogenicity on every host. The results from host range[1], and LOPAT determinative tests agree with that reported for *Pseudomonas syringae*. Therefore, the causal organism of the safflower stem blight in India is identified as *Pseudomonas syringae* van Hall.

The authentic cultures of this isolate have been deposited at the Indian Type Culture Collection at Indian Agricultural Research Institute, New Delhi and at the Type Culture Pool at the Tamilnadu Agricultural University (item 58).

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DIRECT STYLE AND STIGMA NEOFORMATION FROM STIGMA EXPLANTS OF *NICOTIANA TABACUM* L.

STIGMA along with a small portion of the style from flower buds of approximately stage 2 was inoculated on Nitsch medium[2]. The cultures were grown in Erlenmeyer flasks at 23° ± 2° C with continuous illumination of 3,000 lux.

Slight enlargement in size of excised stigma was noticed six days after inoculation and minute outgrowths arose from all over its surface after 10 days. No development was observed on the styler part during this period. The outgrowths from the stigma developed into thinner elongations with swollen tips at the free end, each, though smaller in size resembling a true style and stigma (Fig. 1). A number of these miniature styles with stigmata developed after 3 weeks, covering the entire surface of the stigma explant. Some of these stigmatoid structures repeated the same type of development (Fig. 2).

Some callus developed from the cut end of the style during the 4th week. But this neither developed further nor differentiated in the original culture or on subculturing on Nitsch medium. The tissue along with newly formed styles and stigmata gradually turned brown showing senescence.

Formation of complete flowers directly from the explants of the epidermis without any callus formation was recorded[3]. Development of embryoids from pollen without callus formation was also observed[2]. In the process of differentiation and development of organs, function of certain genes could be specific both in terms of and the time or stage of development of the organ[1]. The style and stigma develop to perform a specific function of receiving male gametes and conducting the male gametophytes to the ovules. During their growth, presumably, only part of the genome may be functioning. In the present case of the stigma explant this part of the genome seems to continue to function *in vitro* (Nitsch medium) as it promotes growth of structures which simulate the true style and stigma. Under the culture conditions mentioned above, the differentiated state of stigmatic tissue appears to contain information for development of both stylar and stigmatic structures and to be stable for at least two generations of those structures, though on a miniature scale.

FIG. 1. Stigmatic explant with neostyles and stigmata.

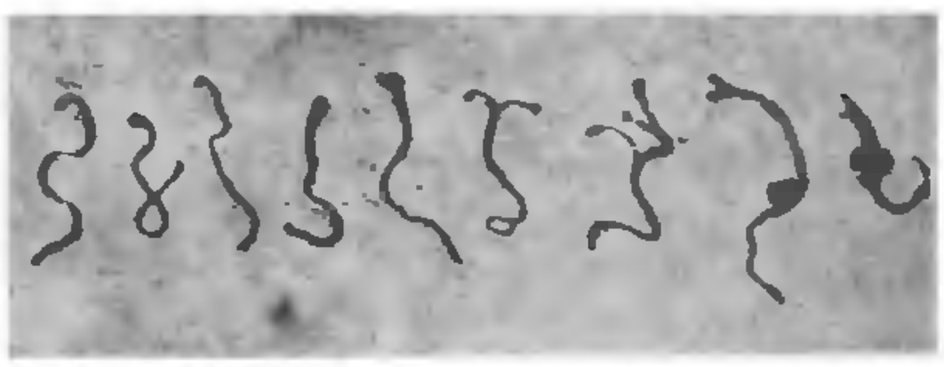

FIG. 2. Outgrowths taken out from stigma explant, bulbous, flattened, clavate, bifid and trifid miniature stigmata.