A NEW FUNGAL DISORDER OF AUSTRALIAN ACACIA

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The authors observed a severe blight disease on phyllodes of Australian acacia (*Acacia melanoxylon* R. Br.) growing in an artificially developed forest in the campus of Jiwaji University, Gwalior. The disease generally started from the tip but rarely from any other part of the phyllodes and covered more than half the surface in advanced stages (Fig. 1 *a* and *b*). Maximum incidence of the disease was observed in the rainy seasons of 1979 and 1980 when the disease was recorded on 15–20% plants. On a severely affected plant 5–10% phyllodes were found to be diseased.

The pathogen produced pink colonies on Czapek–Dox medium. Hyphae 2·5–3·0 μm broad; conidiophores simple; microconidia hyaline, ovoid; macroconidia sickle-shaped, 1–3 septate, size: 1 septate—14–24 × 2·0–3·2 μm, 2 septate—19·2–22·5 × 2·5–3·5 μm, 3 septate—24·2–26·0 × 3·0–3·8 μm; Chlamydospores terminal or intercalary, generally solitary but occasionally in pairs, spherical, rough walled, 8–14 × 8·10 μm.

On the basis of above characters the fungus was identified as *Fusarium oxysporum* Schlecht. The culture is deposited in C.M.I. herbarium (I.M.I. No. 247185).

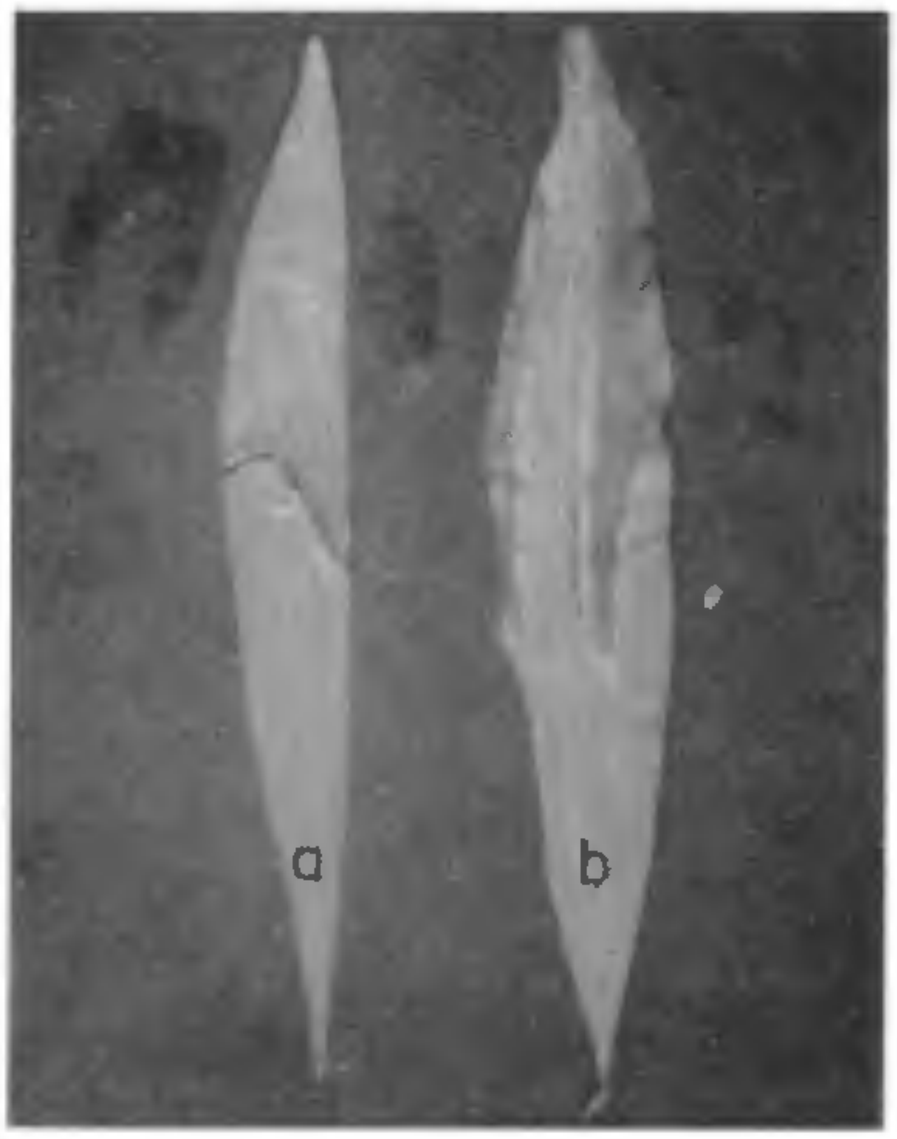

Fig. 1 (*a* and *b*). Symptoms of the disease, *a*—naturally infected and *b*—on artificially infected lodes of Australian acacia.

Pathogenicity of the fungus was tested on thoroughly washed intact phyllodes. Typical symptoms of the disease started to develop after 8 days of inoculation, but no such symptoms appeared in the level experiments conducted concurrently. The same pathogen was re-isolated from artificially inoculated phyllodes.

Several varieties of *F. oxysporum* have been recorded on different plants in India[1], but association of the fungus with Australian acacia has not been reported so far.

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AN INSTANCE OF RARE SEED SETTING *CURCUMA LONGA* L.

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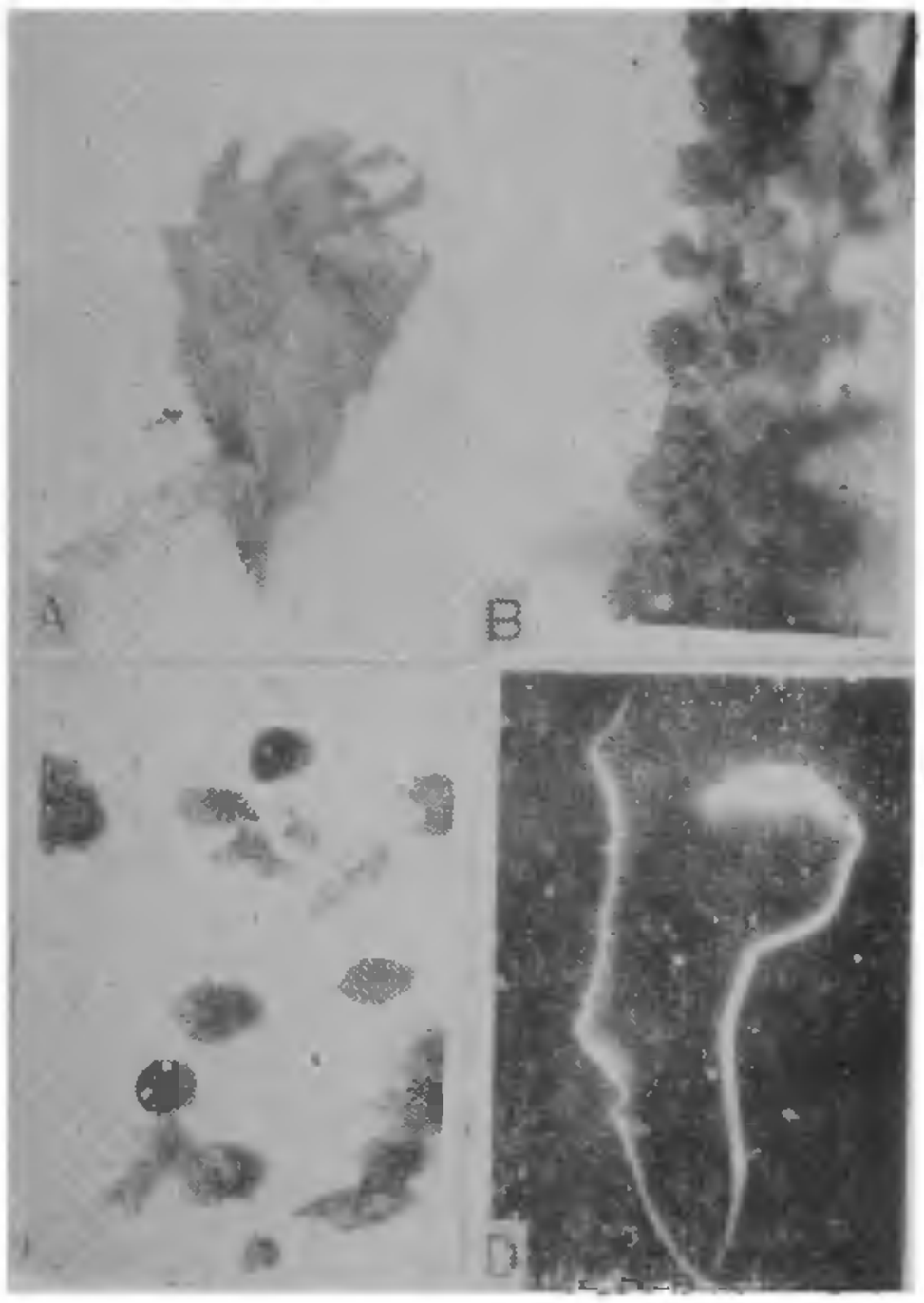
Curcuma longa L. (Turmeric), a vegetatively propagated spice crop is reported to be a non-seed setting species[1]. It is also reported that flowers of this species are adapted for insect pollination[2].

Preliminary studies showed that over 50% of the pollens were viable (iodine test) and showed *in vitro* germination. Ovules showed swelling on hand pollinations. Hence large-scale observations were carried out to detect the possible natural seed set. Screening of over 1,000 inflorescences of *C. longa* (var. Rajapuri) showed one inflorescence bearing seeds. It was not possible to trace the mother rhizome of the seed producing inflorescence. The details of rare seed set are reported below.

The inflorescence had a seed-bearing elongated axis in one of its bracts (Fig. A). The papery capsule cover was lost during handling. Fig. B shows close-up photograph of the seed-bearing axis. Seeds were arilate brownish black, with a smooth polished testa and globose in shape (Fig. C) out of six seeds only one germinated (Fig. D). However the seedling produced



FIGS. A–D. Fig. A. Part of the inflorescence bearing arnormal seed set. Fig. B. Close up of the axis bearing seeds. Fig. C. Seeds of *C. longa*. Fig. D. Germinating seed.

was albino and whithered off in few days. The albino condition may be the result of the obliteration of sexual reproduction.

The stray case of seed set is significant in turmeric improvement. Large-scale screening and seed collection seems to be feasible. If such seeds give normal healthy plants, it will help considerably in improvement work since the seed progenies will provide genotypic variability lacking in vegetatively propagated plants.

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April 7, 1981.

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REPLICATION OF INFLUENZA VIRUS IN THE PRESENCE OF VITAMIN C

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THE effect of vitamin C on the replicating ability of animal viruses has been controversial. There are reports demonstrating positive inhibitory effect on the multiplication of viruses[1–4] whereas others contradict this finding[5–6]. Ascorbic acid is widely used as prophylactic and therapeutic agent against the common cold caused by viruses as in the case of influenza virus. However, there is no evidence to show that vitamin C inhibits influenza virus *in vivo*. Therefore, the effect of this vitamin on the infectivity and replication of influenza virus in mice and chicken embryonated eggs was investigated.

Materials and methods

Influenza A_2 Hong Kong/68 (H_3N_2) obtained from Pasteur Institute was propagated in allantoic cavity of embryonated white leghorn chicken eggs[7] and quantitated by (HA) technique[8].

Swiss Albino male mice aged 5–6 weeks and weighing 25–30 g each was obtained from Biological Evans Limited, Hyderabad.

The groups of mice (six in each group) of which one was fed on normal diet lacking vitamin C and another vitamin C containing diet (10 mg/day/animal) one week before inoculation of the virus (pretreatment) and continued till the termination of the experiment. One more group of six mice served as virus free control.

The above mice were infected with 2 HA units of influenza A_2 Horg Kong virus intraperitoneally (i.p.) and the control group of mice received virus free allantoic fluid[9].

The mice were sacrificed at 48 hr p.i., the testes and lungs were removed aseptically and the HA titre of the extracts of these organs was determined[9]. It was earlier demonstrated that influenza virus replicated in lungs and testes of infected mice[9].

Results and Discussion

The effect of vitamin C on the hemagglutination activity of influenza A_2 virus was determined by treating 4 HA units of virus/ml with 500 mg of vitamin C (final concentration) for 90 minutes at 37° C. The virus showed no reduction in number of HA units,