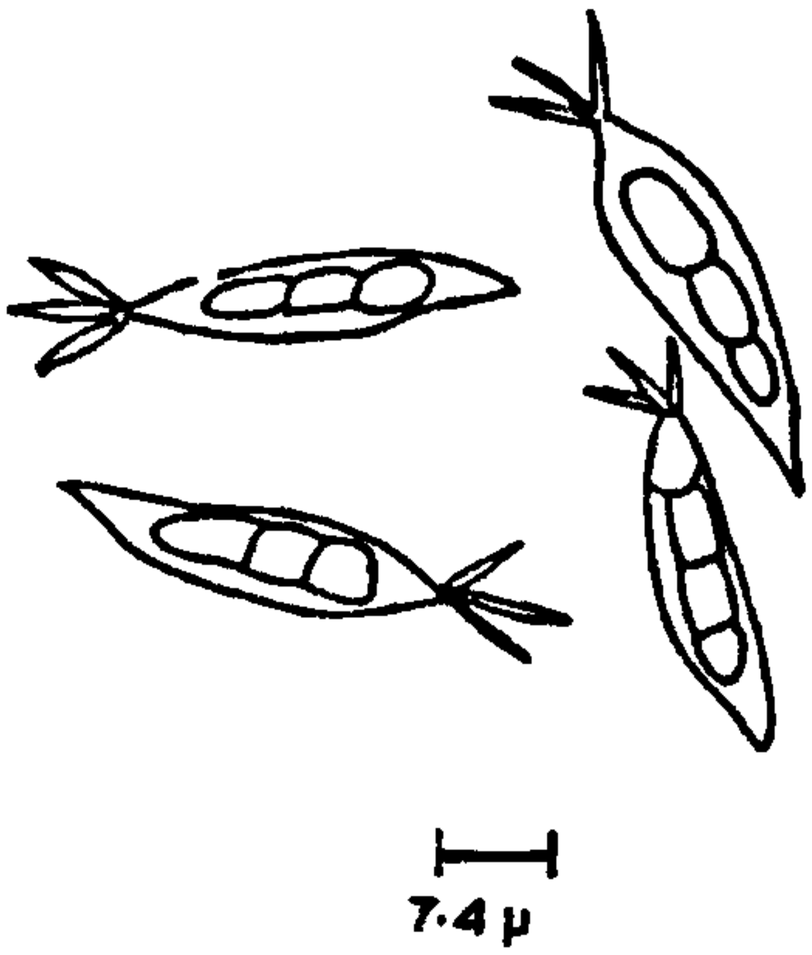

Figure 1. Camera-lucida diagram of conidia of *P. versicolor* (Speg.) Steyaert.

The authors thank the Director, CMI, Kew, England, for confirming the identification of the fungus.

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TRANSMISSION OF *RHIZOCTONIA SOLANI* (KUHN) IN SEEDS OF BEAN (*PHASEOLUS VULGARIS* L.)

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RHIZOCTONIA SOLANI (Kuhn) is pathogenic to bean, *Phaseolus vulgaris* L., causing web blight[1]. Hedgecock[2] observed mycelium and sclerotia of *R. solani* in the seed coat of bean. *R. solani* has so far not been reported to be associated with bean seed in Egypt. Hence its occurrence, location in different seed parts, and transmission through seed and soil were studied.

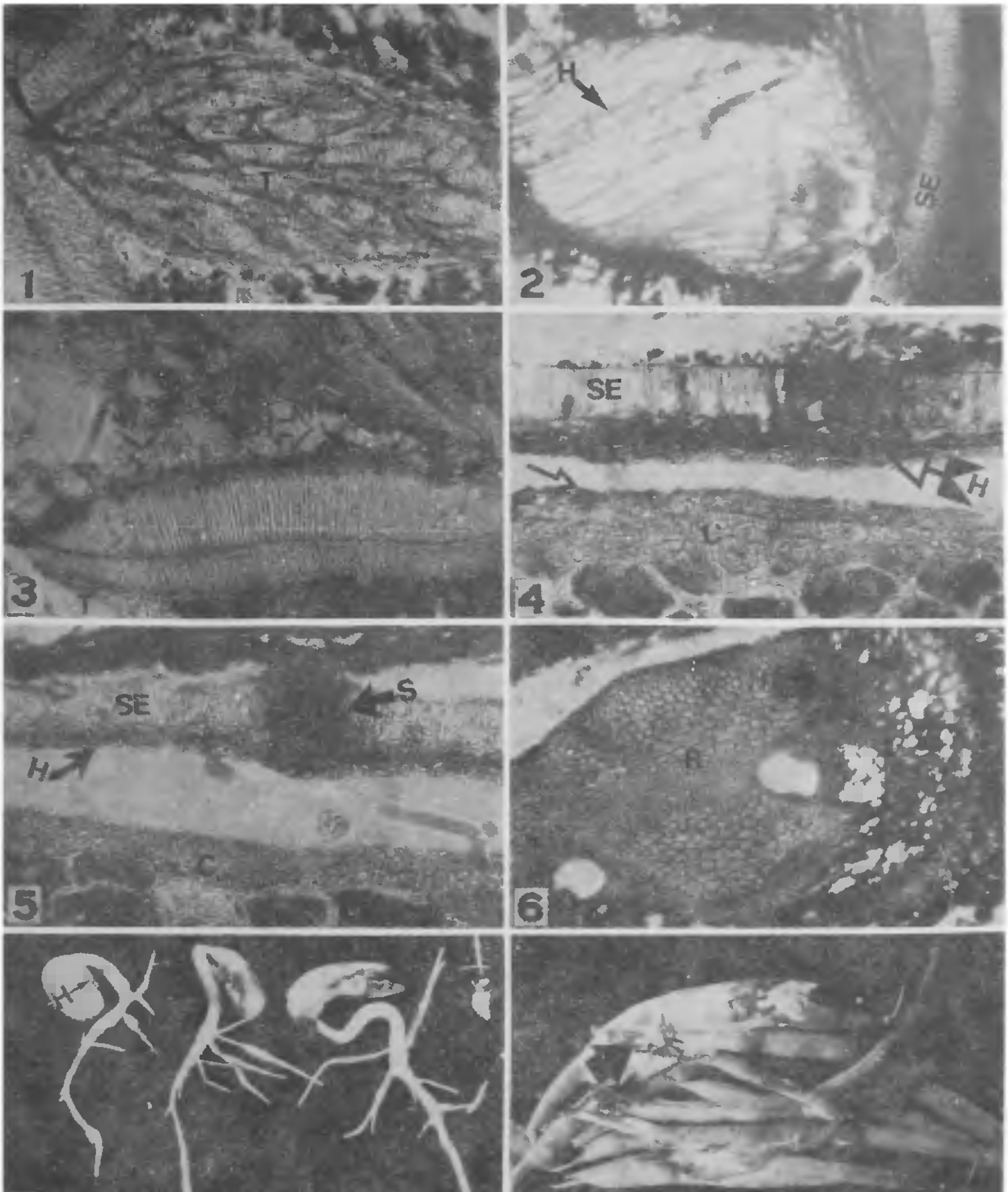
Seeds were collected from Alexandria, Beheira and Giza in Egypt. Incidence of seed infection was determined using the standard blotter paper method[3]. For location of the pathogen in different seed parts, seeds soaked in water were dehulled; seed coat, cotyledon and embryo axis were separated. The separated components were placed on water agar in petri plates and incubated according to ISTA[3] procedures. Highly infected seeds and seedlings were selected and histologically processed following the methods described earlier[4,5]. To study transmission of the pathogen through seeds, a pot culture test[6] was conducted using sterilized soil.

The pathogen was recovered in high percentage from seed coat and cotyledons, and in lower percentage from embryo axis. Histological studies indicated that seed penetration occurred mainly through the hilum tracheids (figure 1). Direct penetration through the intact seed coat was also observed in many seeds (figure 4).

The fungus colonizes the bean seed in all parts; hilum (figures 2 and 3) and cotyledon (figure 4) were the main sites of infection. Many reports indicate that the cotyledons are the main target of infection of many fungi that infect the embryo of legumes. Sclerotia were observed in the seed coat in some seeds (figure 5). In infected cotyledons, parenchyma cells of the outer part of the cotyledons were shrunken and devoid of cell contents (figures 4 and 5).

In 4-day-old seedlings from infected seeds fungal hyphae were observed on the seed coat, below the palisade layer, in the cotyledonary tissue, and in most tissues of the radicle (figure 6). Sclerotia were not observed deep in the tissues.

Transmission studies showed that infected seeds produced diseased seedlings, and 27% of the seedlings died within 4 days from germination.



Figures 1–8. *Rhizoctonia solani* in seed of *Phaseolus vulgaris*. **1,** Fungal hyphae penetrating the seed through the hilum tracheids. **2,** Hilum tissues colonized by fungal mycelium. **3,** Extensive growth of mycelium in the hilum area. **4,** Cross-section of seed showing direct hyphal penetration through the seed coat. Hyphae can be seen located between the seed coat and the cotyledon and have invaded the outer parts of the cotyledon (arrow). **5,** Formation of sclerotia in the seed coat, with fungal mycelium between the seed coat and the cotyledon. **6,** Cross-section of infected seedling showing mycelium colonizing radicle tissues. **7,** Symptoms of infection in young seedlings. Note stunted growth of the seedling on the left due to the infection. **8,** Green bean pods, 24 h after storage, showing heavy fungal growth and pod rot. (C, Cotyledon; H, hyphae; R, radicle; S, sclerotia; SE, seed coat; T, tracheids.)

Symptoms on seedlings are sore shine, root rot, stem rot, necrotic leaves, dark pathces on cotyledons, and leaf spots (figure 7). Green pods had infection in many parts, and the hyphae colonizing most of the pods surfaced after 24 h of incubation in storage conditions (figure 8), causing pod rot.

Results also indicated that the pathogen was transferred from infected seeds to the soil. This means that infected seeds can cause spread of the pathogen in soil, where they are not known to occur. The danger of transmitting the pathogen through seeds has been reported earlier[12, 13].

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SELECTION FOR RED ROT RESISTANCE IN SUGARCANE

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RED rot is the most destructive disease of sugarcane in India. Many important varieties have gone out of cultivation because of this disease. Control methods are ineffective. Sugarcane being a highly heterozygous and polyploid species, one needs large progenies/seedlings for a search for disease-resistant varieties or clones. Degree of parental resistance had no effect on frequency of resistance in seedlings[1,2], but results contrary to this have also been reported[3,4]. The present study was therefore undertaken to study the role of parental resistance in the progenies of 14 intervarietal crosses.

Seven parental varieties, viz. the moderately resistant CP 44-101, Co 7637 and Co 1148; the moderately susceptible Co 7620 and Co 617; the susceptible Co 62174; and the highly susceptible Co 7717 were studied[5]. The fluff (true seed) of 14 crosses (table 1) was sown in a glasshouse in February 1982. The seedlings were transplanted in the field on 25th May 1982. Plant-to-plant and row-to-row distances were kept 75 and 90 cm respectively. Seedlings were harvested in February 1983 and ratoon crop was maintained. After six months standing canes were inoculated by the standard plug method[6]. The inoculated canes were split open longitudinally after 100 days and were graded into 5 grades (1, highly susceptible, to 5, resistant) on the basis of linear spread, condition of the top leaves, width of the lesion, and presence or absence of white spots, as suggested by Srinivasan and Bhat[7].

Table 1 shows the percentages of seedlings with the different grades of red rot resistance. Higher percentage ($\geq 20\%$) of resistant/moderately resistant seedlings was recorded in five crosses, all of which involve CP 44-101 as one of the parents.

Correlation between mid-parental reaction and percentage of resistant and moderately resistant clones among the progenies was positive and highly significant ($r = 0.55^*$ and 0.86^{**}), whereas correlation with percentage of susceptible and highly susceptible clones was negative and highly significant

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