genous cells *C. geophilum* can be separated from these two fungi. The swollen conidiogenous cells of this species may be regarded as rudimentary ampulliform swellings as in *Chrysosporium pannicola* and *Myceliophthora fergusii*. The present fungus is classified in *Chrysosporium* because of its conidia with broad bases. This fungus grew well on moistened human hair and Sabouraud's dextrose agar at 35°C. The keratinophilic nature of this fungus was further determined by the method of Agrawal and Kushwaha[9].

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LARGE CARDAMOM—A NEW HOST FOR *PESTALOTIOPSIS VERSICOLOR* (SPEG.) STEYAERT

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LARGE cardamom (*Amomum subulatum* Roxb.) is an important cash crop of Sikkim occupying the largest area and highest production in India. During a survey of large cardamom diseases several foliar diseases were noticed. Recently symptoms of leaf blight, which could severely damage the foliage and the crop, were also noticed in plantations of large cardamom around Gangtok.

Minute grey spots with chlorotic haloes developed on the leaves mostly from the tip or margin. The spots were amphigenous and irregular in shape, with a prominent reddish-brown margin surrounded by chlorotic haloes. The spots were 130–200 mm × 26–45 mm in size, but under favourable conditions the entire leaf blade was blighted very fast. After some time, the central necrotic portion turned straw-coloured and appeared studded with numerous black dots on both the surfaces. Several such spots often coalesced to give a blighted appearance to the foliage. In the rainy season the necrotic tissues of the infected leaf withered away.

Repeated isolations from the infected leaves yielded a fungal pathogen, which was identified as *Pestalotiopsis versicolor* (Speg.) Steyaert. Colonies on potato dextrose agar are white, with aerial mycelium diffuse towards irregular advancing edge but denser towards the older part of the colony. Acervuli develop from yellowish clumps of hyphae and produce greenish-black spore masses. Diurnal zonations of mycelial growth and acervulus formation are apparent. No pigmentation or discoloration of the mycelium was visible on the reverse side. Conidia (21.5–27.5 × 4.35–8.75 μm) 4-septate, with 3 middle cells dark or brown and end cells hyaline. Apical cell bears 2 or 3 appendages while basal cell ends in a simple pedicel or a single basal appendage (figure 1).

Pathogenicity of the fungus was established by spraying spore and mycelial suspensions on healthy leaves. Humidity above 85% RH and temperature between 23 and 28 °C favoured development of the disease. Typical disease symptoms appeared on both younger and older leaves 6–10 days after inoculation, but infection on younger leaves appeared early. The fungus has not been reported earlier on *Amomum subulatum*[1,2]. Therefore this report constitutes a new host record for *P. versicolor* and also a new disease record for large cardamom. Earlier the fungus has been recorded on many hosts, including *Mangifera indica*, *Eugenia jambolana*, *Terminalia tomentosa*, *Typha angustata*, *Musa paradisica* var. *robusta*, *Jatropha curcas* and *Aloe vera*. The diseased specimen and culture have been deposited at the herbarium of CMI, Kew, England, under reference No. IMI 276700.

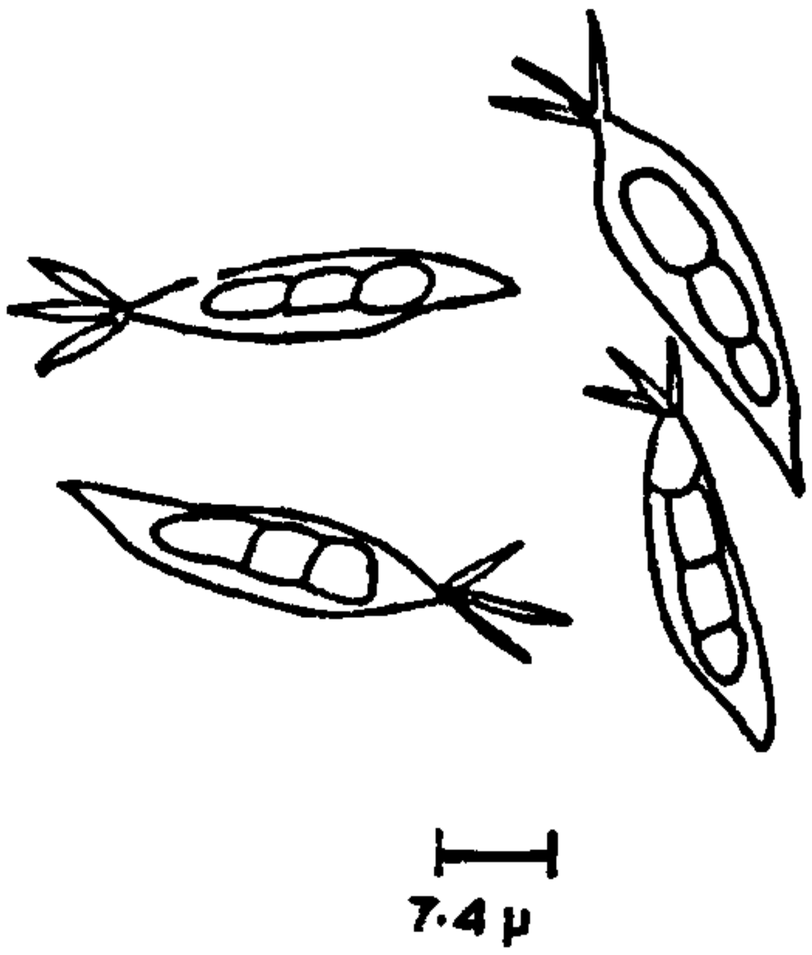

Figure 1. Camera-lucida diagram of conidia of *P. versicolor* (Speg.) Steyaert.

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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.