from buffaloes. The identification was further confirmed as the faeces of the young calves became positive for the eggs of *S. papillosus*. The finding of *S. papillosus* larvae in the milk of buffaloes is, obviously, a first record.

Neoascaris vitulorum (Figs. 4 and 5)

In all, 3 larvae were recovered from the milk samples of 2 buffaloes on 8 and 24-day post-parturition. Larvae, with a bluntly rounded anterior and gradually tapering posterior end, measured 425–510 in length and 14–20 in maximum width. The 3 lipped condition was apparent at the anterior end. Nerve ring and excretory pore were situated at 78–81 and 81–90 distance respectively from the anterior end. The oesophagus measured 136–146 in length and 10–12 in maximum width. The tail measured 38–42 in length. The faeces of these suckling calves later became positive for the eggs of *N. vitulorum*.

Tongson[6] encountered two types of *N. vitulorum* larvae (third stage) in the milk of buffaloes in Philippines : (1) Smaller sized found in majority, measured 442–628 μ and (ii) A few larger sized measured 1150–1580 μ in length. Our material resembled the smaller sized *N. vitulorum* larvae described by Tongson[6]. Present report, on the occurrence of *N. vitulorum* larvae in the milk of buffaloes, is evidently a first record from India.

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DRY ROT OF GINGER CAUSED BY *MACROPHOMINA PHASEOLINA* (TASSI) GOID.

THE cultivation of ginger (*Zingiber officinale* Roxb.) is seriously handicapped by the incidence of soft rot or rhizome rot disease caused by *Pythium* sp. in several tracts of Kerala. The fungus causes pre-emergence and post-emergence rotting of rhizomes during the South-West monsoon from June to September. Under Kasaragod conditions the total rainfall received during June to September 1972 was 2521·1 mm with a mean temperature range of 23·0–30·8° C and a mean relative humidity range of 90·4–95·0%. During the course of investigations on soft rot disease, incidence of another type of rot in mature rhizomes of ginger was noticed from late October onwards till the time of harvest, *i.e.*, February. This rot was found to persist even during storage. During this period (October 1972–February 1973) the total rainfall received was 236·1 mm with a mean temperature range of 20·0–32·7° C and a mean relative humidity range of 87–93%. A perusal of the literature shows that no similar disease has been recorded in ginger and hence reported here.

Affected plant under field conditions shows slight yellowing of leaves in the initial stages, and in advanced stages the whole plant presents a blighted appearance. Unlike in soft rot disease, the base of the stem does not decay and as such the plant does not snap away at the collar region on a slight pull. During advanced stages of the disease, the rhizomes appear shrunken and the inner tissues show discolouration and start disintegrating. Later the inner core of the rhizome shows dark sclerotia of a fungus adhering loosely to the fibrous tissues. The affected tissues do not exhibit any wet rot. Such rhizomes shrink and dry up. Hence this disease is termed as dry rot (Fig. 1).

FIG. 1. Longitudinal section of dry rot-affected ginger rhizome.

Two fungi, *viz.*, *Fusarium* sp. and another sclerotial form were isolated consistently from affected rhizomes. On potato sucrose agar medium *Fusarium* sp. sporulated abundantly while the sclerotial form produced large number of sclerotia. Both in culture and infected tissues the sclerotia appeared as black, minute, anastomosed dark hyphae, the interior of which is light brown to dark in colour with thick walled cells. Sclerotia were either

globose, oval, oblong, or irregular in shape and 20–200 $\mu \times 28$–172 μ in size.

For pathogenicity tests fresh rhizomes of ginger variety 'Maran' were sown in beakers of 500 ml capacity containing garden soil mixed with oat meal inoculum at 3:1 ratio. Both the isolates were tested individually and in combination. The moisture content was maintained at 50% water holding capacity of the soil. The inoculated beakers were incubated at room temperature (28–30° C). In eight days rhizomes inoculated with the fungus producing sclerotia showed darkening and shrinking to a depth of 5–10 mm. Similar result was obtained when both the isolates were inoculated in combination; but no synergistic action was noted. Pathogenicity tests with *Fusarium* sp. alone gave negative results. Thin hand sections of the infected rhizomes showed both inter and intracellular hyphae. Reisolations from the deeper portion of the affected tissue yielded sclerotial form only.

Under field conditions the rhizomes, which are injured either mechanically or by pests like rhizome weevil grubs, showed extensive colonisation by the sclerotial fungus. Therefore it appears that in majority of cases initial injury of rhizomes predisposes them to the fungal invasion, and subsequent colonisation.

The organism has been identified as *Macrophomina phaseolina* (Tassi) Goid., by Dr. Mordue of C.M.I., London (IMI. 172541). Only the sclerotial form of the fungus was produced in culture maintained in this laboratory. *M. phaseolina* has not so far been recorded as a causative organism of dry rot of ginger. The only dry rot that has been reported from ginger is the one caused by *Diplodia natalensis* (Wilson and Balagopal, 1971). Thus this appears to be a new record on ginger. Detailed studies on the fungus and its control measures are under way.

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FIRST RECORD OF SPIDER MITE *TETRANYCHUS LUDENI* ZACHER TRANSMITTING DOLICHOS ENATION MOSAIC VIRUS

DOLICHOS enation mosaic virus (DEMV), first reported on *Dolichos lablab* L. in 1948[1], is a virulent leguminous strain of tobacco mosaic virus with a wide host range. It is easily transmitted only by sap. So far it is not known to be carried either through seed or by any vector[1,4]. During studies on DEMV infection in some legumes such as *D. lablab*, *Phaseolus mungo* L., *P. aureus* Roxb., and *Glycine max* (L.), Merr., at the S.V. Agricultural College, Tirupati, in 1972–73, it was observed that even the healthy controls exhibited foliar symptoms of DEMV. Further tests on the local lesion host cluster bean (*Cyamopsis psoraloides* DC)[3] confirmed the presence of the virus also in the healthy series. A vector was, therefore, suspected and it was observed that only webbing red spider mites were seen both in the healthy and infected plants of these crops. Critical mite transmission studies as suggested by Slykhuis[7] were, therefore, undertaken with DEMV on field bean (*D. lablab*) var. Local Red in insect proof nylon cages in the glasshouse with appropriate controls.

In the first experiment, the nymphs/adults of these mites actively feeding on DEMV-infected *D. lablab* leaves (Fig. 3) were carefully collected and transferred to 8-day old field bean plants at 30 mites per leaf on both the primary leaves. After allowing them to feed for 4 days, they were killed by spraying miticide. Mite damage was evident as white specks on the leaves. These plants exhibited conspicuous stunting (average height reduction of 38·2%) as compared to the control (Fig. 1) 30 days after transferring mites. Eleven out of 13 plants developed typical foliar abnormalities and symptoms (Fig. 2 *a* to *e*). These symptoms were identical with those observed on *D. lablab* by sap inoculation with DEMV[4]. The saps extracted individually from all the 13 plants, in turn, produced systemic infection on field bean and local lesions on cluster bean (1·1 to 140·3 lesions per leaf) thereby proving that this mite acts as a vector for the transmission of DEMV.

In the next experiment, the nymphs/adults of the mite on DEMV-infected *D. lablab* plants collected carefully were macerated in glass mortar with pestle at 500 mites per ml of distilled water. This mite extract when rubbed on young field bean and cluster bean plants with 'celite' as abrasive[4] produced characteristic DEMV symptoms while controls receiving extracts of similar mites collected from healthy plants did not reveal any symptoms. This time, the average number of lesions per leaf worked out to 4·6 indicating pro-