from the tribal areas of Medak district, Andhra Pradesh was conducted. All the samples collected were categorised into insect-damaged and non-insect damaged. The associated fungi with Kesari grains were isolated, identified and the relative abundance of fungi was determined by counting percentage of the total number of colonies of all fungi. The moisture content of these grains was determined by hot air oven method. The samples were observed under long wave (360 nm) UV light for bright greenish-yellow fluorescence[1]. The BGY positive samples were extracted for aflatoxins by the technique employed by Pons *et al*[2]. The confirmation of aflatoxins was carried out accurately by comparing the R_f values of sample extracts with those of standards on the same silica gel plates in different solvent systems. Chemical confirmation was also done with trifluoroacetic acid[3].

Aspergillus flavus group was dominant followed by *Pencillium* and *Fusarium* among the fungi associated with the grains. Moisture content of the samples is in the range of 9–21%. Out of 66 samples collected 36 were insect-damaged and 30 non-insect damaged. Among all the samples screened only 26 gave BGY fluorescence of which 12 are of insect and 7 non-insect (normal) damaged samples were contaminated with one or more aflatoxins. The aflatoxins in these samples ranging in concentration from 12 to 115 ppb. Among all the aflatoxins detected aflatoxin B_1 was recorded the highest (115 ppb) and the lowest aflatoxin G_1 (12 ppb) was in insect and normally damaged samples respectively. Aflatoxin G_1 was present only in one normally damaged sample. All the samples containing toxin were BGY positive[4]. In 75% of insect damaged and 57% of normally damage samples the aflatoxin was above the tolerance level of 20 ppb. Even though the Kesari grains are very hard, the damage caused by insects was very high. Aflatoxin incidence was significantly higher in insect-damage samples, probably due to the invasion of toxigenic fungi into the damaged seeds[5].

Kesari itself nas a volatile toxic alkaloid which causes paralysis. But when it is contaminated with aflatoxins, the resultant synergetic toxic effect is more hazardous to human health. Scientists and educationists should warn the tribals, of the possible adulteration of Kesari dal with 'tur', or 'chana' due to its close resemblance and also mixing with powdered chana by greedy traders.

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1. Fennell, D. I., Bothast, R. J., Lillehoj, E. B. and Peterson, R. E., *Cereal Chem.*, 1973, **50**, 404.
2. Pon's W. A., Cucullu, A. F., Lee, L. S., Robertson, J. A., Franz, A. O. and Goldblatt., L. A., *J. Assoc. Official Anal. Chem.*, 1966, **49**, 554.
3. Stack, M. E. and Pohland, A. E., *J. Assoc. Official Anal. Chem.*, 1975, **58**, 114.
4. Lillehoj, E. B., Kwolek, W. F., Fennell, D. I. and Milburn, M. S., *Cereal Chem.*, 1975, **52**, 403.
5. Aucamp, J. L., *J. Stored Prod. Res.*, 1969, **5**, 245.

POLYGALACTURONASE PRODUCTION BY *RHIZOCTONIA SOLANI* KÜHN—THE CAUSAL ORGANISM OF SHEATH BLIGHT DISEASE OF RICE.

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SHEATH blight disease caused by *Rhizoctonia solani* Kühn is one of the major diseases of rice in tropical Asia. The pathogen is a soil-borne facultative saprophyte and survives as sclerotia or mycelia with a wide spectrum of host range. The various isolates of *R. solani* are known for their production of the enzyme polygalacturonase[1]. This enzyme plays an important role in pathogenesis. However, its production by the rice isolates and its relationship with the virulence have not been examined. In this paper, a report on the production of polygalacturonase by two differentially virulent isolates of *R. solani* obtained from rice is presented.

The least virulent (R_1) and virulent (R_5) isolates *R. solani* collected from the University Botany Laboratory Culture collection, University of Madras, Madras and University of Agriculture Sciences, Bangalore, were used. The virulence of the pathogens was assessed on the basis of disease severity index[2]. Culture filtrates for enzyme assay were obtained periodically from culture grown on liquid media at 32° C. The enzyme polygalacturonase activity was determined by the viscosimetric method and the reaction components were temperature-equilibrated and mixed in the following proportions: 5 ml of 1.6% sodium polypectate, 1 ml of 0.5 M acetate buffer (pH 4.5) and the enzyme solution with distilled water were mixed to a final volume of 8 ml. Five ml of this reaction mixture were added to a viscosimeter at 0 time. The activity was converted to viscosimetric units by calculating the reciprocal of the time required for 1

ml of the enzyme solution to reduce the viscosity[3] of the substrate by 50%. Enzymatic activity was also determined colorimetrically by estimating the reducing sugars[4]. The reaction mixture was the same as that used for the viscometric method. The developed colour was read at 720 nm. The standard curves of glucose were prepared to estimate the enzyme activity.

In the present study, the optimum production of enzyme was standardized with 2 ml of substrate concentration in the presence of 1 ml of enzyme concentration at pH4.5. Moreover, a close correlation (table 1) was obtained among the differentially virulent isolates of *R. solani* with the production of the enzyme polygalacturonase. The virulent isolate recorded a miximum production of this enzyme even during the earlier period of incubation, when compared to that of the least virulent isolate R_1. Similar observation has been made earlier by Geypens[5], with various isolates of *R. solani* other than rice. The work of Weinhold and Motta[6] suggests the possible damage of cell wall before penetration of the pathogen by the cell wall degrading enzymes. The earlier detection of polygalacturonase from the virulent isolate of *R. solani* further reiterates the involvement of this enzyme in the primary armoury of the pathogen during pathogenesis. More detailed investigation of isozymes of polygalacturonase present both in culture filtrates and in host tissue may be helpful in determining the virulence of the isolates.

TABLE 1

Polygalacturonase production by the virulent (R_5) and least virulent (R_1) isolates of R. solani

Days of incubation	Least virulent (R_1)	Virulent (R_5)
Enzyme activity (Units)		
3	83	335
5	225	300
7	200	285
10	300	95
Growth (Mg dry wt)		
3	5	8
5	20	21
7	36	45
10	30	41

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1. Bateman, D. F. and Millar, R. L. *Annu. Rev. Phytopathol*, 1966, **4**, 119.
2. Ramalingam, P., Ph.D. Thesis. The University of Madras, Madras, India. 1981.
3. Bateman, D. F. *Phytopathology*, 1963, **53**, 1178.
4. Nelson, N., *J. Biol. Chem.*, 1944, **153**, 375.
5. Geypens, M., *Ann. Phytopathol.*, 1978, **10**, 355.
6. Weinhold, A. R. and Motta, J., *Phytopathology*, 1978, **65**, 157.

ENTEROCYSTIS BENGALENSIS N.SP. (APICOMPLEXA:ENTEROCYSTIDAE) FROM *PSOCATROPOS* SP. (PSOCOPTERA) OF WEST BENGAL, INDIA.

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WHILE studying the parasitic protozoa of arthropods of West Bengal, India, I encountered several species of gregarines and microsporidians. This paper describes the only aseptate gregarine that I found from the midgut of the *Psocatropos* sp. (Psocoptera).

The hosts were collected from the animal room, dissected in 0.5% NaCl solution and examined under a microscope. Smears of the infected midgut contents were made on slides and fixed in Schaudinn's or Bouin's fixatives and subsequently stained with Heidenhain's haematoxylin. The gametocysts, isolated from the hindgut of the host, were kept in the moist chamber for further development. The liberated oocysts were examined with Lugol's iodine under the oil immersion lens. All measurements (given in μm) are the mean with the range within parenthesis (total number of individuals measure = n).

The earliest form was an aseptate, small, spherical or ovoid body with a round nucleus; it was attached to the midgut epithelium by a depression (sucker?) on its surface (figure 1). These forms were later released into the midgut lumen and transformed into elongate gamonts. Mature gamonts were aseptate and elongated with a round anterior end tapering gradually towards the posterior end (figure 2); they were 90 (49–140) × 24.6 (14–42) (n=25). The nucleus was spherical with an ovoidal nucleolus, situated near the centre or anterior region and rarely in the posterior part of the gamonts; it was 8.4 (4.7–16.3) (n=25) in diameter. The cytoplasm of live specimens appeared deep brown and contained large spherules; it was covered by a clear pellicle. Locomotion was by streaming movement of the protoplasm followed by