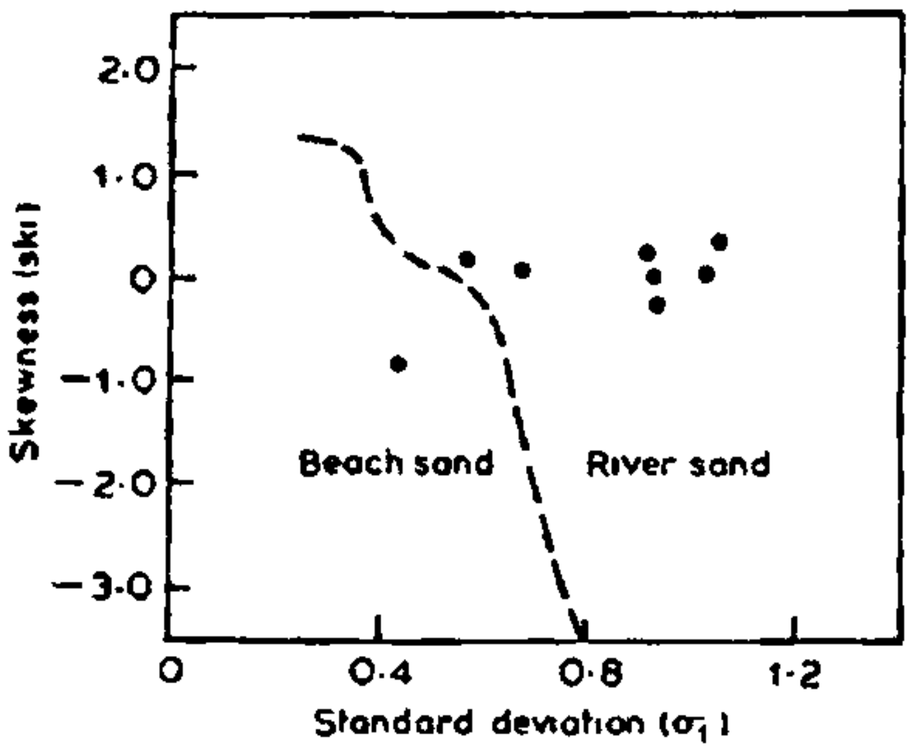

Figure 3. Plot of skewness and standard deviation, after Friedman, 1961, for the Warkalli sediments.

Varkala indicates that these were mostly deposited by streams (figure 3).

On the basis of the occurrence of drift assemblage of silicified fossil tests in the Warkalli sediments (type area) it is surmised that during a regressive phase of the sea, possibly of the Late Miocene, the sediments including lignitic pieces and fossils were eroded and redeposited in the littoral area. This further suggests that the overlap sequence at Varkala type area should necessarily be younger (Late Miocene-Pliocene) than the sediments occurring around Kundara, Padappakara, Paravur and Edavai.

Thanks are due to the State Ground Water Department for the bore-hole samples and to the Regional Research Laboratory, (CSIR) Trivandrum for extending SEM facilities.

24 November 1986; Revised 2 February 1987

1. King, W., *Rec. Geol. Surv. India*, 1882, **15**, 93.
2. Paulose, K. V. and Narayanaswami, S., *Geol. Soc. India Mem.*, 1968, **2**, 300.
3. Ramanujam, C. G. K. and Rao, K. P., *The Geophytology*, 1977, **2**, 160.
4. Rajendran, C. P., Raha, P. K. and Kar, R. K., *J. Geol. Min. Met. Soc. India*, 1986 (in press).
5. Rasheed, D. A. and Ramachandran, K. K., *Proc. 7th Colloq. Micropal. Stratigraphy*, 1978, p. 299.
6. Ghosh, S. K. and Raha, P. K., *J. Geol. Soc. India*, 1983, **24**, 655.
7. Menon, K. K. *Proc. Indian Acad. Sci.*, 1967, **65**, 20.

EFFECT OF PENICILLIC ACID ON INTESTINAL BRUSH BORDER OF RABBITS

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PENICILLIC acid, a secondary metabolite was first isolated from the culture filtrate of *Penicillium puberulum*[1]. Scott[2] reported the production of penicillic acid by several food-borne fungi. Penicillic acid has been considered as a potential environmental health hazard when it was isolated from agricultural products[3–5]. Cytotoxic[6], hepatotoxic[7] and carcinogenic[8] effects of penicillic acid have been reported earlier.

Investigations particularly on long-term feeding experiments using other mycotoxins indicated that mycotoxins affect small intestinal cell wall[9,10], which is a primary site exposed to potential toxicants. Since the cytotoxic effect of penicillic acid had already been proved, it was felt that ingestion of *Penicillium cyclopium* and the toxin penicillic acid will initially affect the cell wall of the small intestine. Hence, in the present investigation an attempt has been made to find out the changes of intestinal brush border glycoproteins and lipid parameters which will give an insight into the mode of action of penicillic acid.

Penicillium cyclopium (the strain was isolated in the laboratory from a fungal contaminated feed and confirmed by IARI, New Delhi. This strain found to produce pencillic acid as a major secondary metabolite) was grown on 1 litre of Raulin-Thom medium for 14 days and penicillic acid was isolated from the culture filtrate using the method of Bentley and keil[11]. The purity of penicillic acid was tested by NMR, IR and UV spectral analyses along with an authentic sample (a gift sample from Dr E.B. Lillehoj, Agricultural Research Southern Region, Lousiana). Contaminated diet for our experiments was prepared by growing *P. cyclopium* in sterilized bread at 20 to 22°C for 14 days. The fungus was then inactivated by the addition of chloroform and later removed completely by drying. The bread was then powderd and mixed with normal rabbit diet in the ratio of 1:2 (w/w) (contaminated diet containing 1 mg penicillic acid per 10 g of bread).

Table 1 *Glycoprotein and lipid components of intestinal brush border of normal and experimental rabbits. Sialic acid is expressed as nmol/mg membrane protein, other values expressed as µg/mg membrane protein. Values are the average of six individual experiments ± S.D*

Components studied	Control	Contaminated diet fed	Toxin treated
Hexoses	53.91 ± 4.16	42.42 ± 5.96[b]	39.87 ± 3.42[a]
Hexosamines	30.48 ± 3.35	20.78 ± 3.02[b]	18.54 ± 2.66[b]
Fucose	15.30 ± 1.21	10.86 ± 1.83[b]	9.46 ± 1.46[a]
Sialic acid	10.28 ± 1.84	7.66 ± 1.21[d]	7.36 ± 0.98[b]
Cholesterol	69.25 ± 5.67	58.33 ± 3.07[c]	55.58 ± 4.62[b]
Phospholipids	149.12 ± 10.32	130.79 ± 8.17[b]	125.07 ± 8.89[b]

Statistically significant variations as compared with controls are indicated by [a]$P < 0.001$; [b]$P < 0.01$; [c]$P < 0.02$; [d]$P < 0.05$.

Albino female rabbits (weighing 650 – 850g) were randomly divided into three groups of six animals each. One group served as control which was fed normal laboratory diet. The second group was fed with normal diet mixed with *P. cyclopium* infected bread in the ratio of 1:2 (w/w). The third group was given intraperitoneally with pure toxin dissolved in saline at a dosage of 2.0 mg/kg body weight every alternate day. After the experimental period of six months the animals were starved for 24 hr but allowed free access to drinking water and killed by decapitation. Immediately after killing, the abdominal and thoracic cavities were quickly opened and the small intestine was removed. The intestinal brush border of the rabbits was prepared for analysis by the method of Cooper and Kent[12]. Glycoproteins of the brush border were precipitated by the method of Glossmann and Neville[13]. The pellets were dried overnight at room temperature in a desiccator. For delipidation of the pellets, the method of Folch *et al*[14] was adopted. The lipid extract was used for the estimation of cholesterol[15] and phospholipids[16]. The delipidated membrane pellets were dissolved in 0.1 N sodium hydroxide and aliquots were taken to assay protein[17], hexoses[18], hexosamines[19], sialic acid[20], and fucose[21].

Table 1 shows intestinal brush border glycoprotein and lipid components of both control and experimental rabbits. Total hexoses, hexosamines, fucose, sialic acid, cholesterol and phospholipids were found to be decreased significantly both in contaminated diet fed and toxin-injected animals when compared to control animals.

In all life processes, transport of nutrients across the membrane plays a significant role. Membrane-bound enzymes are essential for the transport of nutrients and maintain the intracellular concentration of electrolytes. Mycotoxins like patulin[9], yellow toxin produced from *P. cyclopium*[10] and citrinin[22] have been reported to affect intestinal membrane and lower the activity of membrane-bound enzymes. Inhibition of ATPase activity during pencillic acid administration has also been reported earlier[23]. Investigations were carried out to elucidate the intestinal membrane alterations during toxicoses. Patulin[9] and sterigmatocystin[24] were found to reduce the intestinal amino acid absorption. The above findings were explained on the basis of membrane damage produced by the mycotoxins. The reduced levels of glycoproteins observed in the present investigation may be due to the interaction of penicillic acid with the membrane and its components leading to membrane damage resulting in the release of membrane components into the surrounding fluids. The reported cytotoxic[6], hepatotoxic[7] and reduction in the membrane bound ATPase activity[23] during penicillic acid treatment lend support to the above findings. The decreased cholesterol and phospholipid levels are an additional evidence for membrane damage during penicillic acid toxicosis.

Grateful thanks are due to CSIR, New Delhi for financial support.

25 August 1986; Revised 16 January 1987

1. Alsberg, C.L. and Black, O.F., *U.S. Dept. Agr. Bur. Plant. Ind. Bull.*, 1913, 270, 5.
2. Scott, P.M., In: *An Encyclopedia Handbook*, (eds) D.Wyllie and L.G. Morehouse, Marcel Dekker, New York and Basel, 1978, Vol.1, 311.
3. Kurtzman, C.P. and Ciegler, A., *Appl. Microbiol.*, 1970, 20, 204.

4. Pero, R.W., Harven, D., Ownes, R.G. and Snow, J.P., *J. Chromatogr.*, 1973, **65**, 501.
5. Bacon, C.W., Sweeney, J.G., Robbins, J.D. and Burdick, C., *Appl. Microbiol.*, 1973, **26**, 155.
6. Umeda, M., *Jpn J. Exp. Med.*, 1971, **41**, 195.
7. Chan, P.K., Hayes, A.W., Meydrech, E.F. and Ciegler, A., *Toxic. Appl. Pharmacol.*, 1980, **55**, 291.
8. Dickens, F. and Jones, H.E.H., *Br. J. Cancer*, 1965, **19**, 392.
9. Devaraj, H., Radha Shanmugasundaram, K. and Shanmugasundaram, E.R.B., *Curr. Sci.*, 1982, **51**, 602.
10. Ramani, N. and Shanmgasundaram, E.R.B., *Curr. Sci.*, 1981, **50**, 946.
11. Bentley, R. and Keil., J.G., *J. Biol. Chem.*, 1962, **237**, 867.
12. Cooper, J. R. and Kent, D. W., *Biochim. Biophys. Acta*, 1978, **513**, 364.
13. Glossmann, H. and Neville, D.M., Jr., *J. Biol. Chem.*, 1971, **246**, 6339.
14. Folch, J.,Lees, H. and Slones, G.H., *J. Biol. Chem.*, 1957, **226**, 497.
15. Parekh, A.C. and Jung., D.H., *Anal. Chem.* 1970, **42**, 1423.
16. Fiske, C.H. and Subbarow, Y., *J. Biol. Chem.*, 1925, **66**, 375.
17. Lowry, O.H., Rosebrough, N.J., Farr, A.L. and Randall, R.J., *J. Biol. Chem.*, 1951, **193**, 265.
18. Niebes, P., *Clin. Chim Acta*, 1972, **42**, 399.
19. Elson, L.A. , and Morgan, W.T.J., *Biochem.J.*, 1933, **27**, 1824.
20. Aminoff, D., *Biochem. J.*, 1961, **81**, 384.
21. Dische, Z. and Shettles, L.D., *J. Biol. Chem.*, 1948, **175**, 595.
22. Ramadoss, C.S. and Shanmugasundaram, E.R.B., *Indian. J. Biochem. Biophys.*, 1973, **10**, 290.
23. Chan, P.K., Timothy, P.D. and Hayes, A.W., *Toxicol. Appl. Pharmacol.*, 1979, **49**, 365.
24. Meerarani, S. and Shanmugasundaram, E.R.B., *Curr. Sci.*, 1984, **53**, 92.

A NEW LEAF SPOT OF *COMMIPHORA WIGHTII* A MEDICINAL PLANT, CAUSED BY *PHOMA* SP.

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COMMIPHORA WIGHTII (Arnott) Bhand is an important medicinal plant and is popularly known as guggal which is the oleogum resin exudate of this plant. It is widely distributed in Karnataka, Maharashtra, Gujarat and Rajasthan states of India. It is reputed for its numerous medicinal properties[1–4]. We have extensively survyed the guggal-growing areas of Rajasthan and encountered a severe leaf spot disease occurring in serious proportions. The leaf spot covering two-third portion of leaves, makes the leaves dry, and which in later stages wither away. This might be affecting overall growth of the plant. This is the first report on leaf spot disease of *C. wightii* caused by a new species of *Phoma*.

The diseased leaf showed symptoms of black concentric ring spots, which in initial stages of infection shows brown discoloration of tissues, but later takes the shape of black concentric rings. Isolations were made from diseased tissues of leaves in petri plates. The small bits of infected tissues along with adjoining healthy areas were dipped in mercuric chloride (0.1%) for surface sterilization for 1–2 min, washed thrice in sterile water and plated on PDA medium poured in petri dishes and incubated at 20–25°C for 7 days. Pure cultures of the fungus were transferred on PDA slants. In pathogenicity tests, fungus produced the symptoms after seven days of inoculations. Morphological characteristics of fungus were studied and it was identified as *Phoma glomerata*. The identification was confirmed by CMI, Kew, England (Herbarium IMI No. 2999459). The fungus is a species of *Phoma* close to *P. glomerata*. Studies on morphological and taxonomic details of the fungus are in progress.

We thank the authorities of Sukhadia University and Dr C. E. Punithalingam, Principal Taxonomist, CMI, U. K. Financial assistance from ICAR, New Delhi in the form of a project is gratefully acknowledged.

24 October 1986; Revised 23 January 1987

1. Anon. *Wealth of India* (*Raw materials*) New Delhi, 1950, Vol. II, p. 313.
2. Kirtikar, K. R. and Basu, B. D., *Indian medicin-*