1. Malik, N. N. and Chughtai, M. I. D., *Pak. J. Sci.*, 1979, **3,** 127.
2. Vora, K. A., Pradhan, R., Amin, A. R. and Modi, V. V., *Curr. Sci.*, 1986, **55,** 622.
3. Abdulla, M. H. and Omer, F., *Mycopathologia*, 1981, **73,** 9.
4. Amin, A. R., Vyas, P. and Modi, V. V., *Indian J. Exp. Biol.*, 1984, **22,** 220.
5. Sahasrabudhe, S. R., Amin, A. R. and Modi, V. V., *Appl. Microbiol. Biotechnol.*, 1985, **21,** 365.
6. Amin, A. R. and Modi, V. V., *Folia Microbiol.*, 1987, **32,** 24.
7. Amin, A. R., Modi, V. V., Udupa, S. R. and Chadha, M. S., *Proc. DAE Symp. on newer approaches to biological applications*, M. S. University of Baroda, India, 1984.
8. Modi, V. V. and Amin, A. R., In: *International biosystems*, (ed.) D. L. Wise, CRC Press, Florida, 1987.
9. Amin, A. R., Shah, T., Modi, V. V., Udupa, S. R. and Chadha, M. S., Paper presented at the VII Int. Symp. Biotechnology, New Delhi, India, 1984.
10. Milstein, O., Vered, Y., Shragina, L., Gressel, J., Flowers, H. and Huttermann, A., *Arch. Microbiol.*, 1983, **135,** 147.
11. Milstein, O., Vered, Y., Sharma, A., Gressel, J. and Flowers, H., *Appl. Environ. Microbiol.*, 1983, **46,** 55.
12. Sturani, E., Costantini, M. G., Zippel, R. and Alberghina, F. A. M., *Exp. Cell. Res.*, 1976, **99,** 245.
13. Sturani, E., Magnani, F. and Alberghina, F. A. M., *Biochim. Biophys. Acta*, 1973, **319,** 153.
14. Christoffersen, R. E. and Laties, G. G., *Proc. Nat. Acad. Sci. USA*, 1982, **78,** 4060.
15. Amin, A. R., Ph.D. thesis, M. S. University of Baroda, January, 1987.
16. Wolf, D. H., *Adv. Microbial. Physiol.*, 1980, **21,** 267.
17. Mortegani, E. and Alberghina, L., In: *The microbial cell cycle*, (eds) P. Nurse, and L. Streibova, CRC Press, Florida, 1984, p. 163.
18. Baird, W. M., Sedwich, J. A. and Boutwell, R. K., *Cancer Res.*, 1971, **31,** 1434.
19. Moffat, N. K. and Jensen, H. M., *Exp. Cell. Res.*, 1968, **52,** 216.
20. Evans, F. J. and Soper, C. J., *Lyodia*, 1978, **41,** 193.
21. Hennings, H. and Boutwell, R. K., *Cancer Res.*, 1970, **30,** 1203.
22. Christman, M. F., Morgan, R. W., Jocobson, F. S. and Ames, B. N., *Cell*, 1985, **41,** 753.
23. Tanguary, R. M., Comato, R., Lettre, F. and Vincent, M., *Can. J. Biochem., Cell Biol.*, 1983, **61,** 414.
24. Baszezynski, C. L., Walsen, D. B. and Atkinson, B. G., *Can. J. Biochem., Cell Biol.*, 1983, **61,** 395.
25. Silver, J. C., Andrew, D. R. and Pekkala, D., *Can. J. Biochem., Cell Biol.*, 1983, **61,** 447.

EFFECT OF CHEMICALS ON THE MORPHOLOGY OF *SCLEROTIUM ROLFSII* SACC CAUSING FOOT-ROT DISEASE OF BARLEY

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MORPHOLOGICAL changes in fungi occur generally during unfavourable conditions. Fungi change their original structure to resist the action of chemicals and very little work has been done on this aspect[1–3]. In the present communication, two phenolic compounds (picric acid and 2, 4–dinitrophenol), one vitamin (riboflavin) and two amino acids (tryptophan and proline) causing considerable morphological changes in *Sclerotium rolfsii* Sacc., a foot-rot pathogen of barley at higher concentrations, are reported.

Required quantities of chemicals were mixed separately in Erlenmeyer flasks (250 ml), containing 100 ml autoclaved molten potato dextrose agar (PDA) to obtain the desired concentrations (100, 500, 1000, 5000, 10,000 ppm). Twenty ml of such amended medium was poured on each sterilized plate (90 mm diam) and allowed to solidify. Fresh mycelial discs (3 mm diam) were cut from actively growing margin of the fungus and transferred singly at the centre of the plates. The control sets did not receive any such chemical. Plates were incubated at 25 ± 1°C for 2 weeks. Changes in hyphal morphology, growth pattern and sclerotial morphology of the pathogen were observed.

Application of riboflavin, tryptophan and proline at 100 ppm and picric acid and 2, 4–dinitrophenol at 1000 ppm resulted in spare growth of the fungal colonies with stranded radiating hyphae appearing from the central inoculum. The number, shape and the size of sclerotia were also slightly reduced.

With 500 ppm riboflavin, tryptophan and proline and 5000 ppm picric acid and 2, 4–dinitrophenol, zonation appeared and the growth of the colonies were markedly arrested with irregular margin.

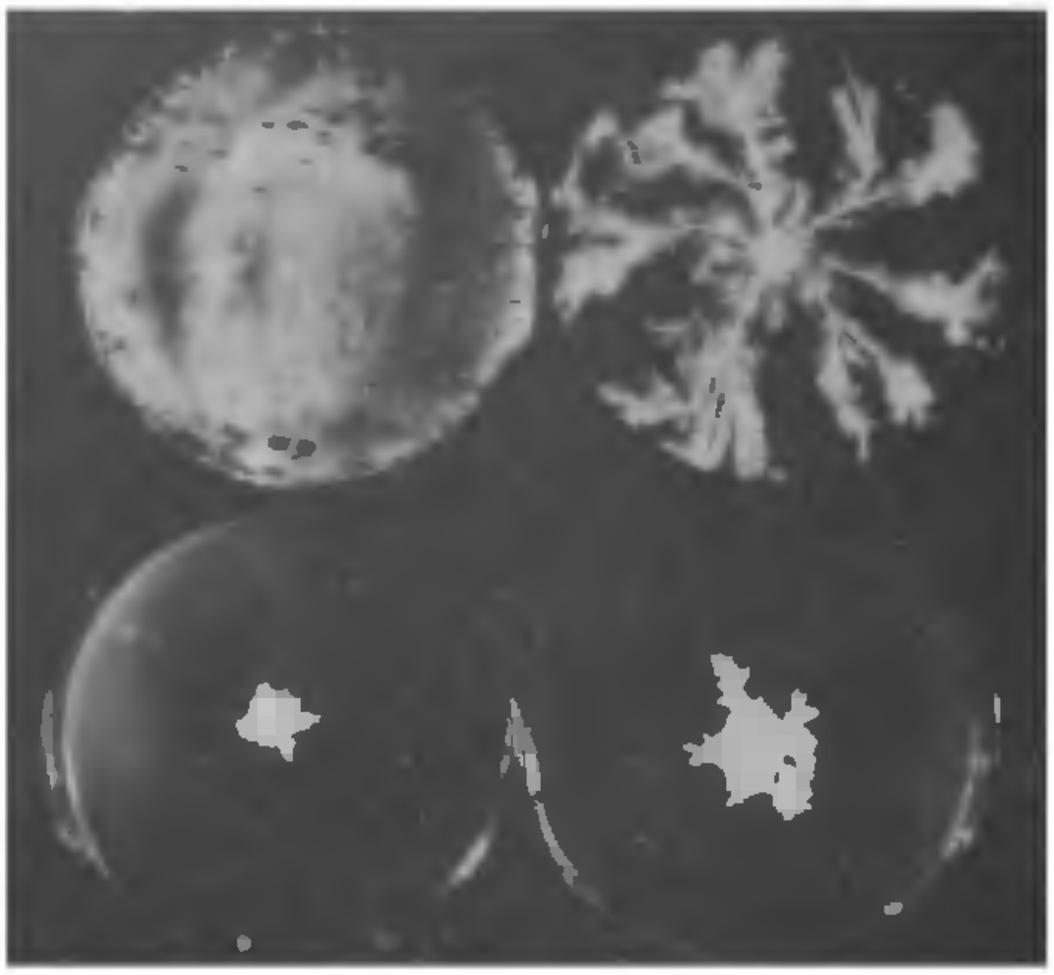

Figure 1. Effect of different concentrations (0, 1000, 5000 and 10,000 ppm) of 2, 4-dinitrophenol on *Sclerotium rolfsii* Sacc.

Colony surface became glossy and the hyphae aggregated into thin-stranded structures (figure 1). A few sclerotia of very irregular shape and size was also seen.

With 1000 ppm riboflavin, tryptophan and proline and 10,000 ppm picric acid and 2, 4–dinitrophenol, hyphal growth was almost arrested and the fungus failed to develop any sclerotia.

Picric acid and 2, 4–dinitrophenol required more than five times the concentration of amino acids and vitamins for the same morphogenetic effect. Other amino acids and vitamins did not change the morphology of the pathogen and the growth was almost similar to the control in these treatments.

The hyphae which were narrow, thin-walled and large-celled in the untreated control became short-celled and thick-walled at all the concentrations tried.

The hyphal and sclerotial characters change when a fungus is subjected to poison stress[1]. One of the remarkable features of such changes is the reduction/elimination of the ability to produce sclerotia[3].

The present results show that the test fungus can tolerate higher concentrations of 2, 4-dinitrophenol, picric acid and other chemicals. Though they are effective against a large number of microorganisms, they are effective only at very high concentration in the case of *S. rolfsii* Sacc (figure 1). Development of tolerance to a chemical by the pathogen may be due to the decrease in permeability of the fungal cell to the chemical and the conversion of the chemical into an inactive form by the pathogen[4]. It may also be due to the selective effect of the chemical which is more active only with selective fungi[1].

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1. Dwivedi, R. S. and Pathak, S. P., *Indian Phytopathol*, 1981, **34**, 238.
2. Grover, R. K. and Moore, J. D., *Phytopathology*, 1961, **51**, 399.
3. Shatla, M. N. and Sinclair, J. B., *Phytopathology*, 1963, **53**, 1407.
4. Dekkar, J., *Annu. Rev. Phytopathol.*, 1976, **14**, 405.

IN VITRO SYNTHESIS OF ECTENDOMYCORRHIZAE OF *PINUS PATULA* WITH *AMANITA MUSCARIA*

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Mycorrhizae or fungus-root associations are the norm for most vascular plants[1]. Many plants depend on their mycorrhizal structures for adequate uptake of nutrients and survival in natural ecosystems[2–4]. The forest trees like pines possess ectomycorrhizal roots and the fungi producing ectomycorrhizae are primarily Agaricales and Gasteromycetes[5]. *Amanita muscaria*, a member of Agaricales is reported to form ectomycorrhizal association with many species in *Pinus*[6].

It is believed[7,8] that hyphal connections between the sporophore and the host plant could not be taken as a proof for mycorrhizal association and only synthesis experiments under controlled conditions can furnish conclusive proof for the mycorrhiza-forming ability of a given fungus, *in vitro* synthesis of mycorrhiza of *Pinus patula* with *Amanita muscaria* was attempted.

The fruitbodies of *A. muscaria* (L. ex Fr.) Pers. ex Hooker found in association with the roots of *P. patula* Schlecht & Cham present in the New Pine plantations of Kodaikanal, Tamil Nadu were collected and used for inoculation studies. Pure mycelial cultures were isolated from the surface-sterilized stipe tissue grown on 2% Hagem's nutrient agar as modified by Modess[8] and subsequently subcultured