Table 1 *Reaction of mixed races of Xanthomonas campestris pv. malvacearum*

Treatment	Reaction on differentials							
	I	II	III	IV	V	VI	VII	VIII
A. Monoinoculation								
1. *Xcm*-V$^-$ (avirulent)	HR	HR	HR	HR	HR	HR	HR	HR
2. *Xcm*R-8	SR	SR	HR	SR	HR	HR	HR	SR
3. *Xcm*R-32	SR	SR	SR	SR	SR	SR	HR	SR
B. Coinoculation								
4. *Xcm*-V$^-$ + *Xcm*R-32 (1:1)	SR	SR	SR	SR	SR	SR	HR	SR
5. *Xcm*-V$^-$ + *Xcm*R-32 (2:1)	HR	HR	HR	HR	HR	HR	HR	HR
6. *Xcm*-V$^-$ + *Xcm*R-8 (1:1)	SR	SR	HR	SR	HR	HR	HR	SR
7. *Xcm*-V$^-$ + *Xcm*R-8 (2:1)	HR	HR	HR	HR	HR	HR	HR	HR
8. *XcmR*-8 + *Xcm*R-32 (1:1)	SR	SR	SR	SR	SR	SR	HR	SR
9. *XcmR*-8 + *Xcm*R-32 (2:1)	SR	SR	HR	SR	HR	HR	HR	SR

SR, susceptible reaction; HR, hypersensitive reaction; I, Acala-44 (no genes for bacterial blight resistance); II, Stoneville 2B-S9 (polygenes); III, Stoneville-20 (B_7 + polygenes); IV, Mebane B-1 (B_2 + polygenes); V, 1–10.B (B_{In} + polygenes); VI, 20–3 (B_N + polygenes); VII, 101–102. B (B_2B_3 + unknown); Gregg (unknown).

changed in a mixture of races at 1:1 ratio *i.e.* *Xcm*R-32:*Xcm*R-8 behaved as *Xcm*R-32 and a mixture of *Xcm*-V$^-$:*Xcm*R-8/*Xcm*R-32 behaved as *Xcm*R-8/*Xcm*R-32 respectively. However, at 2:1 ratio of the less virulent/avirulent: virulent genotype mixture the HR on a cv dominated, started earlier and inhibited the SR (table 1) *i.e.* these mixtures behaved as avirulent in the presence of *Xcm*-V$^-$ and as race-8 in the presence of *Xcm*R-8, and not as *Xcm*R-32. The results also emphasise that the incompatible reaction may be used by the host to eliminate or curtail the development of certain races specially in mixed infections.

It may be mentioned that the reaction of the mixed races was synergistic, at least, on cv I (assessed in terms of lesion size), which was susceptible to both the races of a mixture. The increase in lesion area ranged from 48–73% 14 days after inoculation. It was concluded that the disease reaction of mixed races of *Xcm* was synergistic or mixed on cvs susceptible to both the races; but the reaction was hypersensitive/antagonistic if one of the races of the mixture was incompatible to the cv under test. The results also point out the dangers of the use of mixed races for screening of segregating populations for resistance breeding programmes. For bacterial blight resistance breeding programmes it is, therefore, suggested to use, at least, an established virulent mixture of races or preferably pure cultures of *Xcm*R-32, which are capable of attacking at least five bacterial blight resistance genes namely[3] B_7, B_4, B_2, B_{In} and B_N.

The authors are grateful to Dr M. M. Payak, Head, Division of Mycology and Plant Pathology for his interest and to Dr R. P. Singh for helpful discussions.

26 March 1984

1. Brinkerhoff, L. A., *Annu. Rev. Phytopathol.*, 1970, **8,** 85.
2. Innes, N. L., *Biol. Rev.*, 1983, **58,** 157.
3. Verma, J. P., Singh, R. P., Borkar, S. G., Sinha, P. P. and Prasad, R., *Ann. Agric. Res.*, 1980, **1,** 98.
4. Verma, J. P., Chowdhury, H. D. and Singh, R. P., *Z. PflKrankh, PflSchutz*, 1979, **86,** 460.
5. Verma, J. P. and Singh, R. P., *Indian Phytopathol.*, 1975, **28,** 459.
6. Verma, J. P. and Singh, R. P., *Z. PflKrankh. PflSchutz*, 1976, **83,** 748.

A NOTE ON THE FRUIT BODY PRODUCTION OF *TRICHOLOMA GIGANTEUM* MASSEE

V. S. DADWAL and JAMALUDDIN
Regional Forest Research Centre, P.O. Bilhari, Jabalpur 482 020, India.

LIKE *Agaricus* and *Volvariella*, the species of *Tricholoma* are also edible[1]. Some species like *T. mangolicum* and *T. matsutakes* are collected and used in enormous quantities in Japan[2].

Only eight species of *Tricholoma* namely *T. cremoriceps* Berk, *T. giganteum* Massee, *T. nelaleucum*

(Pers.) Fr., *T. subpulverulentium* (Pers) Fr., *T. nudum* (Bull.) Fr., *T. leucocephalum* (Fr. Sensu) Lange, *T. georgii* (Clus) Fr. and *T. lobayense* Heim have been recorded from India[2-5]. During the survey of agaric flora of local forest, *Tricholoma giganteum* was collected from Jabalpur. The morphological characters of *T. gigantcum* Massee were compared with *T. lobayense* Heim. The salient features of morphological similarity of both the species agree in habitat, length of stipe, size of basidia and basidiospores. Pegler[6] also indicated the similarity of *T. giganteum* with *T. lobayense*. Uptil now no attempt has been made to domesticate the *Tricholoma* spp. in India. The present studies aim to find out suitable substrate for the cultivation of *T. giganteum*.

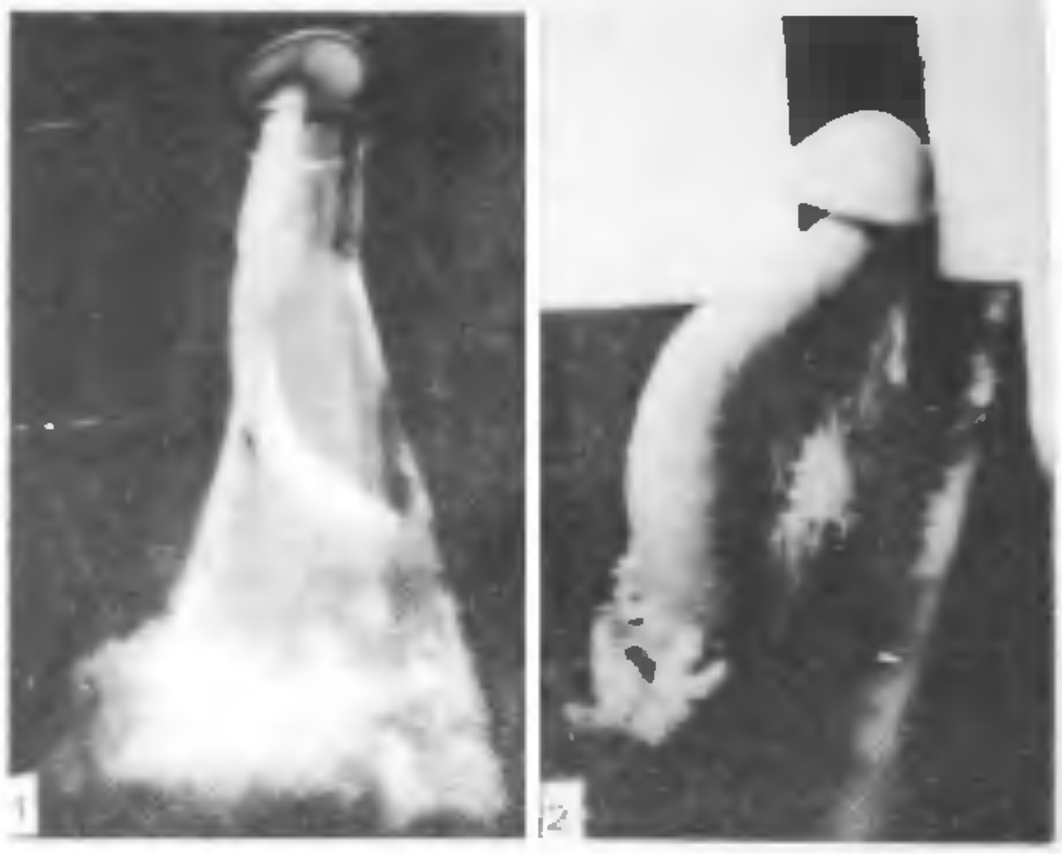
Preparation of spawn

The spawn of *T. giganteum* was prepared on wheat grain as described by Munjal[7].

Production of fruit bodies on different substrates

The preliminary experiment was carried out in 500 ml Erlenmeyer flasks[8]. The suitability of different substrates like barley meal, maize meal, rice husk, sorghum meal, kodo meal, and paddy straw was tested for the sporophore production of *T. giganteum*. The compost (soil + sand, 2 : 1) was supplemented with the above mentioned plant products (5% of total compost). Thus each flask contained 200 g compost, 10 g plant products and 70 ml distilled water. The experiment was laid in triplicate. The flasks were sterilized at 15 lb pressure for 30 min. on two consecutive days and inoculated with 20 wheat grains of one-month old spawn. The flasks were incubated in BOD incubator at 30° ± 2° and 80% humidity for two weeks for spawn running, which was completed in two weeks; then one inch layer of steam sterilized casing soil (farm yard manure + sand, 4 : 1) was added in each flask. The fruit bodies developed after 10–15 days of casing. A number of fruit bodies emerged from the casing soil but only one or two developed to size (figure 1). The fruit bodies were collected in 20–30 days of casing. The yield of sporophores in different substrates is presented in table 1.

The results of table 1 indicate that barley meal and maize meal are the best sources; the fruit bodies could not be developed in paddy straw. The quantity of sporophores was less in rice husk, sorghum meal and kodo meal as compared to *i.e.* barley meal and maize meal.



Figures 1, 2. **1.** Fruit body production of *T. giganteum* in flask containing 5% barley meal with husk. **2.** Fruit body production of *T. giganteum* in trays containing synthetic compost.

Table 1 *Effect of various substrates on the sporophore production of T. giganteum.*

Substrates	Yield/600 g of substrate (Fresh wt. of fruit bodies in gram)
Barley meal	34.5
Maize meal	23.5
Rice husk	18.6
Sorghum meal	21.0
Kodo meal	5.5
Paddy straw	Nil
Control	Nil

In another experiment, cultivation of *T. giganteum* was tried on synthetic compost similar to that used for the cultivation of *Agaricus bisporus*[9]. The experiment was carried out in wooden trays (18″ × 10″ × 6″). One-month old spawn (approximately half bottle) was mixed thoroughly in one tray. The compost was pressed uniformly and covered with moist newspaper sheet. The trays were kept at room temperature (28–30°) during August–September with 80% humidity. The spawn running was completed within 15 days. The casing was done with steam-sterilized casing soil (farm yard manure + sand 4 : 1) and sprayed with uniform amount of water till the small button of sporophores appeared. The mushroom started developing after ten days of casing (figure 2). The results of the experiment in trays were quite encouraging as 15 to 19 fruit bodies developed in each tray which proved that *T. giganteum*

can be cultivated on the synthetic compost like *Agaricus bisporus*.

The fresh sporophores were fed to white albino rat in the laboratory for one week. No abnormality could be detected in them.

The authors are thankful to Director, Royal Botanical Garden, Kew, England for his help in identification of the organism.

13 December 1983; Revised 23 May 1984

1. Philips, R., *Mushroom and other fungi of Great Britain and Europe*, Tappan Printing Company, Hong Kong, 1981.
2. Sinha, M. P. and Padhi, B., The genus *Tricholoma* in Orissa, *Indian Mushroom Science* I. First National Symposium on Survey and cultivation of edible mushroom in India, 1978, p. 457.
3. Sathe, A. V. and Rahalkar, S. R., *Bio Vigyanam. J. Life Sci.*, 1975, **1**, 75.
4. Bilgrami, K. S., Jamaluddin and Rizwi, M. A., *Fungi of India*, 1979, 1981. Part I and II Today and Tomorrows Printers and Publishers, New Delhi.
5. Chakarvarty, D. K. and Sarkar, B. B., *Curr. Sci.*, 1982, **51**, 531.
6. Pegler, D. N., 1982, Personal Communication, Royal Botanical Garden, Kew, England.
7. Munjal, R. L., *Indian J. Mushroom.*, 1973, **1**, 1.
8. Purkayastha, R. P. and Chandra, A. *The mushroom J.*, 1976, **40**, 112.
9. Munjal, R. L. and Seth, P. K., *Bulletin of the Indian Mushroom Grower's Association, 1977.* Mushroom cultivation of India. 1–30.

SIGNIFICANCE OF LIPID PEROXIDATION IN LIVER INJURY AFTER HEAVY METAL POISONING IN RATS

S. V. S. RANA and AJAY KUMAR
School of Environmental Contamination and Toxicology, Department of Zoology, D.A.V. College, Muzaffarnagar 251 001, India.

FORMATION of lipo-peroxides at selective subcellular sites due to an alteration in the antioxidant activity of the cell has been considered to play a significant role in a number of pathological processes that involve the liver. The product thus formed is malonaldehyde or malonaldehyde like substance[1]. Malonaldehyde occurring in living tissues or in biological specimens undergoes metabolic transformation and can be determined by thiobarbituric acid. Many heavy metal salts severely effect the liver as they are metabolised by the hepatic microsomal system. Excessive intake of iron salts is known to stimulate lipid peroxidation[2], whereas zinc inhibits this process[3]. Recently a few observations have been made on malonaldehyde in brain and other tissues[4–6], however, the effects of other and environmentally important metals like mercury, lead, cadmium, molybdenum, copper, chromium and manganese on lipid peroxidation in liver have not been studied. The role of these metal ions on aggravation of liver lipids was reported[7]. Since, peroxidative damage to membranes also encourage triglyceride accumulation[8], role of lipo-peroxides was determined in the liver of rats (*Rattus rattus* albino) fed on few heavy metals *viz* mercury, lead, cadmium, molybdenum, copper, chromium, manganese and zinc.

Ninty laboratory bred male albino rats (*Rattus rattus* albino), 90 days old, weighing 100 ± 10 g were randomly allocated into 9 groups, each of 10 rats. Each rat was housed separately, fed on standard laboratory diet (Hindustan Lever Ltd., Bombay), tap water *ad libitum* and maintained under standard laboratory conditions. Rats of groups A, B, C, D, E, F, G and H received a sublethal dose of 0.005, 0.005, 0.5, 1.0, 0.1, 0.05, 0.25 and 5.0 g/kg body weight of Hg, Pb, Cd, Mo, Cu, Cr, Mn and Zn respectively daily by gavage, for a period of 30 days in addition to laboratory diet whereas the animals of control group I received the laboratory diet alone and tap water *ad libitum*. The dose levels were applied after making primary toxicological tests[9] like oral LD_{50}.

On expiry of treatments, all the rats were starved for 24 hr and then killed by decapitation. Pieces of liver from each rat were quickly excised and immediately frozen at -4°C. 10% (w/v) homogenates of the liver were prepared in 0.9% sodium chloride solution. Temperature near 0°C was maintained throughout the period of homogenisation. The homogenates were centrifuged for 120 min at $1500 \times g$ and the clear supernatant fluids were processed for the estimation of malonaldehyde[10]. The student *t* test[11] was applied to calculate the statistical significance between control and experimental values.

Present results indicate that lead, cadmium and zinc failed to promote the formation of lipid peroxides in the liver of rats whereas mercury, molybdenum, copper, chromium and manganese affected membrane fluidity and induced formation of aldehydes (table 1).

Free metal ions of mercury, lead, cadmium etc