Table 2 *Comparison of mean number of pistillodes/capsule and seed set in control thrum and pistillode mutant of T. subulata.*

	Number of capsules observed	Number of capsules with pistillodes	Number of pistillodes/ capsule	Number of seeds		Aborted seeds	
				without pistillode capsules	with pistillody capsules	without pistillody capsules	with pistillody capsules
Control (Thrum)	200	—	—	30 (20–35)	—	2 (1–2)	—
Mutant	182	76	3 (1–8)	20 (15–24)	13 (9–18)	5 (2–7)	8 (5–11)

following irradiation and is shown to be due to single recessive gene[10-12]. Manga[12] noted some of the mutants that had the stamens modified to 'carpel' and in others the mutant had multiple carpels with occasional formation of seeds. Kihara[13] obtained pistillody in hybrid plants of *Aegilops caudata* × *Triticum aestivum* and found the original carpel to be functional. In the mutants presently studied fertility was greatly reduced by the induction of pistillody.

It is not clear why thrum plants alone are susceptible to pistillody. Whether this is due to the heterozygous nature of thrum (Ss) or due to teratogenic effect of the mutagen is not known at present.

7 April 1984

1. Rama Swamy, N. and Bahadur, B., *Incomp. Newslett.* (Netherlands), 1981, **13**, 20.
2. Rama Swamy, N. and Bahadur, B., *Proc. 3rd Indian Paly. Conf.*, HAU (Hissar), Allied Publishers, 1982 (in press).
3. Bahadur, B. and Rama Swamy, N., *J. Palynol.*, 1982, **18**, 127.
4. Rama Swamy, N. and Bahadur, B., *J. Biol. Res.*, 1983a, **3**, 57.
5. Rama Swamy, N. and Bahadur, B., *XV Int. Congr. Genet.*, 1983b, **I**, 261.
6. Rama Swamy, N. and Bahadur, B., *Incomp. Newslett.*, 1984a, **16**, (in press).
7. Rama Swamy, N., Bahadur, B. and Arthi, C., *Incomp. Newslett.*, (USA), 1983a, **15**, 7.
8. Rama Swamy, N. Bahadur, B. and Arthi, C., *Incomp. Newslett.*, (USA), 1983b, **15**, 8.
9. Rama Swamy, N. and Bahadur, B., *Indian J. Bot.*, 1984b, **7**, (in press).
10. Kamara, O. P. and Nilan, R. N., *J. Hered.*, 1959, **50**, 159.
11. Bhatia, C. R. and Swaminathan, M. S., *Genetica*, 1963, **34**, 58.
12. Manga, V., *Proc. Indian Acad. Sci.*, 1977, **B86**, 93.
13. Kihara, H., *Cytologia*, 1951, **16**, 177.

REACTION OF MIXED RACES OF *XANTHOMONAS CAMPESTRIS* PV. *MALVACEARUM* (E. F. SMITH) DYE

J. P. VERMA and S. G. BORKAR
Division of Mycology and Plant Pathology, Indian Agricultural Research Institute, New Delhi 110012, India.

BACTERIAL blight of cotton is induced by *Xanthomonas campestris* pv. *malvacearum* (*Xcm*). Mixtures of races (genotypes) of *Xcm* are generally used for screening segregating breeding materials[1-3]. However, a mixture of races may give a synergistic, mixed or antagonistic reaction[4]. The present report demonstrates the effect of different populations of the constituent races of *Xcm* on the reaction of mixed races on different cotton cvs with different bacterial blight resistant genes.

The methods used have been described earlier[4-6]. The isolates used were *Xcm*R-32 (race-32), *Xcm*R-8 (race-8) and *Xcm*-V$^-$ (avirulent race-32; virulence lost by repeated transfers on artificial media in about 7 years[6]). The aqueous suspension of *Xcm* isolates was adjusted to 0.1 or 0.2 $E_{620\ nm}$ and then mixed accordingly. The results (table 1) showed that the concentration of the constituent *Xcm* cells played an important role in the reaction of the mixed races. Normally *Xcm*-V$^-$ gave a resistant hypersensitive reaction (HR, a rapid necrosis within 24 hr followed by tissue collapse) on all the cvs; *Xcm*R-32 gave HR on cv VII, while *Xcm*R-8 gave HR on CVS III, V, VI and VII and susceptible reaction (SR) on the remaining cvs (table 1). The reaction of the virulent genotype was not

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. melaleucum*