

Table 2 Combined effect of rifampicin and septran against *M. ulcerans* infection in mouse I.V. model

Treatment	Dose mg/kg	No. of mice	Survivors on day 30 of challenge	Percentage survivors on day 30 of challenge	Mean survival time + standard error
Untreated control		20	0	0	7.0 ± 0.6
Septran	30	12	0	0	12.0 ± 0.35
Rifampicin	2.5	11	0	0	8.3 ± 0.8
Rifampicin + Septran	2.5 + 30	14	0	0	10.0 ± 1.0
Rifampicin	5.0	12	0	0	16.2 ± 1.6
Rifampicin + Septran	5.0 + 30	11	11	100	82.9 ± 7.1
Rifampicin	10	11	10	91	85.0 ± 9.3
Rifampicin + Septran	10 + 30	12	12	100	113.2 ± 9.5
Rifampicin	15	10	10	100	87.5 ± 5.5
Rifampicin + Septran	15 + 30	15	15	100	144.0 ± 5.8

week were given upto a total of ten. The mean survival time of each group was calculated and other parameters of study were followed.

In the rat foot pad model the natural decrease in inflammatory response percentage of untreated control group was only 18 upto 14th week of infection. The value of the corresponding period with rifampicin alone was 62 and that of septran alone was 40, 56 and 56 in increasing doses. The corresponding values with combination was much higher and reached up to 81 when 20 mg of septran and 3 mg of rifampicin were used. This clearly indicated that there was additive action of the drugs when used in combination.

In the second experiment with mice, the results presented in table 2 clearly show that the combination therapy, even when used the other way round (*i.e.* septran dose was kept constant and rifampicin dose was varied) and through a different route, has definite effect in arresting the pathological action of the pathogen and the MST of the infected animals was considerably increased upto 144 days when 15 mg of rifampicin and 30 mg of septran were used. Corresponding values of untreated control were only 7 days. The pathological score of the visceral organ also indicated the effectiveness of the treatment since the lesions were comparatively far less than in the controls. This also indicated the effectiveness of the drug combination in mice.

This combination of drug therapy with rifampicin and septran succeeded with many other drug combinations and may help in the treatment of this infection, along with excisional surgery.

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1. MacCallum, P., Tolhurst, J. C., Buckle, G. and Sissons, H. A., *J. Path. Bact.*, 1948, 60, 93.
2. Feldman, R. A., *Int. J. Dermat.*, 1974, 13, 353.
3. Wolinsky, E., *Am. Rev. Resp. Dis.*, 1979, 119, 107.

A BACTERIAL LEAF SPOT OF *BAEL* (*AEGLE MARMELLOS* CORREA) IN RAJASTHAN AND A REVIVED NAME OF THE BACTERIUM

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BROWN spots were found on leaves of *Bael* trees in villages Baparawal and Barapal, 26 and 29 km respectively from Udaipur city during the month of August and September 1980-82. A bacterium was isolated from the infected leaves and this was purified by dilution plate method.

In nature brown spots were formed on leaves, twigs and fruits and under humid conditions yellow bacterial ooze was seen. The spots often coalesce and shot holes were observed in the infected leaves. By artificial inoculation, using carborandum abrasion technique, symptoms could be reproduced.

The bacterium is rod-shaped, gram negative, motile with single polar flagellum, capsulated, asparagine not utilised as sole source of carbon and nitrogen, pro-

duces H_2S , NH_3 , catalase positive, hydrolyses starch and liquefies gelatin. Out of 34 host plants, belonging to 13 families, the bacterium infected and produced symptoms on the principal host *Bael* and on sour lime (*Citrus aurantifolia* Swingle) spots were produced but there was no canker formation.

The bacterium is identified as *Xanthomonas campestris* (Pammel) Dowson but pathovar is yet unnamed. The bacterium resembles *X. bilvae* Patel *et al*¹, a name which is no longer accepted under the provisions of the International Code of Nomenclature as most of the species of *Xanthomonas* were reduced to pathovars of *X. campestris* by Dye *et al*², and due to lack of a type culture, *X. campestris* pv. *bilvae* was not accepted in to ISPP, list. The present isolate (IMIB 8600) resembles the organism of Patel *et al*¹ and a revived name *X. campestris* pathovar *bilvae* (*nom rev*) is proposed as per Rule 28A of the International Code of Nomenclature of Bacteria 1976³.

Thanks are due to Dr J. F. Bradbury, CMI, UK for his help in identifying the bacterium and valuable suggestions and for depositing this culture (IMI B8600) at the National Collection of Plant Pathogenic Bacteria, Harpenden, UK³. Thanks are due to Dr R. M. Singh, Dean for his encouragement.

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1. Patel, M. K., Allayyanavaramath, S. N. and Kulkarni, Y. S., *Curr. Sci.*, 1953, 22, 216.
2. Dye, D. W., Bradbury, J. F., Goto, M., Hayward, A. C., Lelliott, R. A. and Schroth, M. N., *Rev. Plant Pathol.*, 1980, 54, 153.
3. Bradbury, J. F. (Personal communication).

IN VITRO HYBRIDIZATION IN AN INCOMPATIBLE CROSS—*BRASSICA JUNCEA* × *BRASSICA HIRTA*

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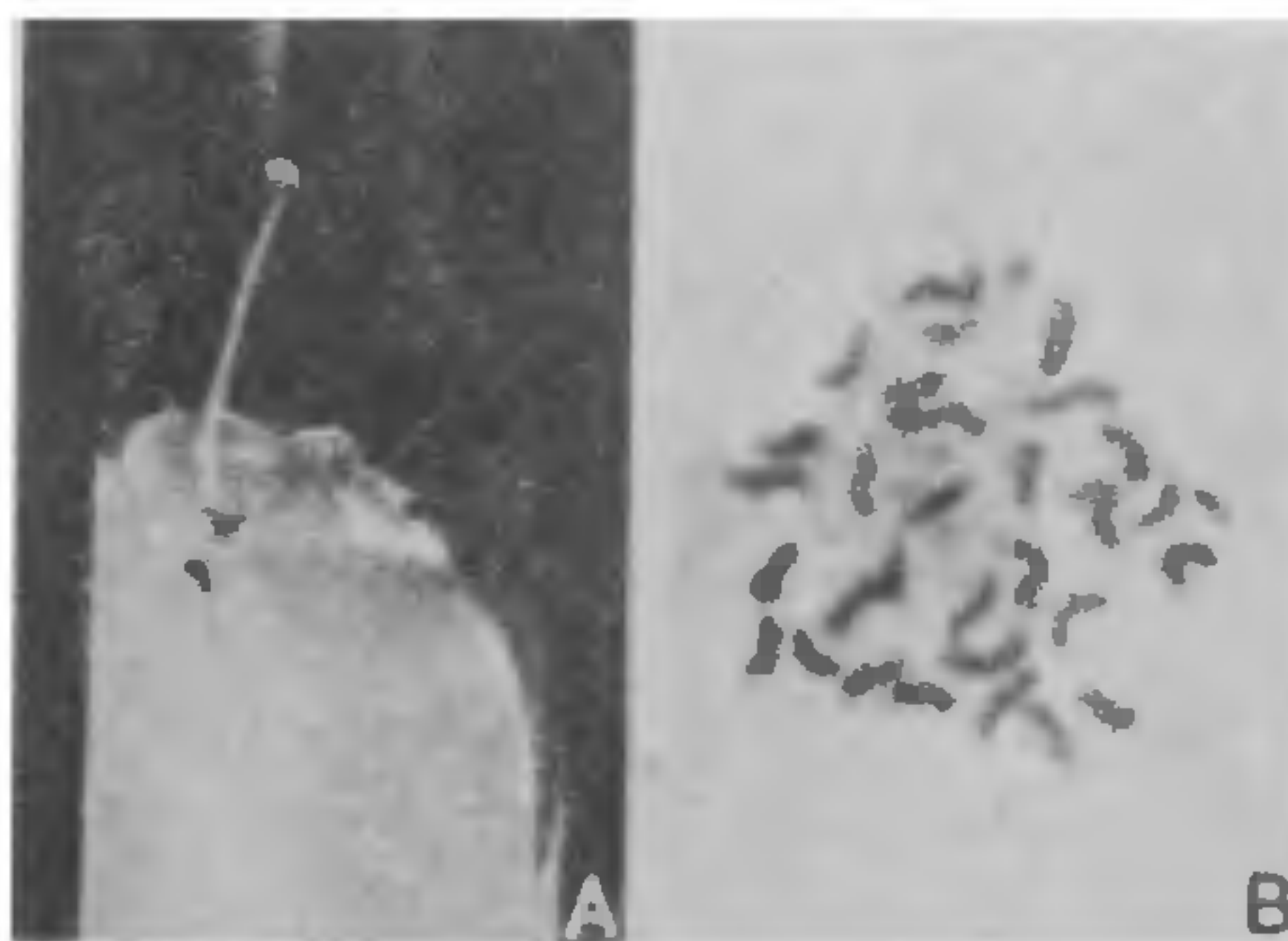
BRASSICA as an oilseed crop occupies second position in India, and is only next to groundnut. The commercially grown species (*B. juncea*, *B. napus*, *B. campestris*) are mostly susceptible to leaf blight (*Alternaria brassicae*). White mustard (*Brassica hirta* or *Sinapis*

alba) though resistant to blight, is incompatible and so far attempts to cross *B. juncea* ($2n = 36$) × *B. hirta* ($2n = 24$) using conventional breeding methods have not been successful¹. However in the present study, by resorting to the culture of young ovules, hybrid plantlets ($2n = 30$) have been obtained *in vitro* and the technique described.

The flower buds of *B. juncea* and *B. hirta* were emasculated two days before anthesis, and were cross pollinated two days after emasculation. Immature ovules [10–15 days after pollination (DAP)] were aseptically excised and cultured on Murashige and Skoog's medium (MS)² supplemented with indole acetic acid (IAA 2 mg/l) + kinetin (kin 0.5 mg/l) + casein hydrolysate (CH 500 mg/l). All the manipulations were conducted under sterile conditions in a laminar flow chamber (Klenzaid, Bombay), and the

Table 1 *In vitro* growth response of parental as well as hybrid ovules (15 DAP) of *Brassica* cultured on MS + IAA (2 mg/l) + kin (0.5 mg/l) + CH (500 mg/l)

Ovules (Parentage)	No. of ovules cultured	No. of ovules forming plants	Percentage of plantlet formation
<i>Brassica juncea</i>	60	36	60.00
<i>Brassica hirta</i>	45	20	44.44
<i>B. juncea</i> × <i>B. hirta</i>	260	6	2.31
<i>B. hirta</i> × <i>B. juncea</i>	210	4	1.90



Figures A and B. *In vitro* cultures of the hybrid ovules (15 DAP) of a cross *Brassica juncea* × *B. hirta*. A. Plantlet from a hybrid ovule 25 days after culture on MS + IAA (2 mg/l) + kin (0.5 mg/l) + CH (500 mg/l). B. Root tip squash of a hybrid showing an intermediate chromosome number ($2n = 30$).