

infection in the seed bed. The black rot does not cause any disagreeable odour. Satyavir *et al.*<sup>7</sup> also reported a bacterial rot of raya (*B. juncea*) which in the initial stage causes dark coloured streaks at the stem base. These streaks gradually girdle the stem, its tissues become soft and there is collapse of the plant. Leaves are also infected, the lower leaves being attacked first. A yellow fluid oozes out from the diseased tissues. Isolations yielded a yellow pigmented bacterium, which was identified as a species of *Xanthomonas*. Healthy plants become infected after 12-15 days following inoculation. No particulars about the inoculation method, characteristic properties of the bacterium and its host range have been furnished.

However, considering the pattern of flagellation, staining reactions and physiological and biochemical characteristics mentioned above as well as the rapidity with which it causes foul smelling rots on *Brassica* and other host species tested, the bacterium is identified as a species of *Erwinia* belonging to carotovora group<sup>1,2</sup> inciting stalk rot of *Brassica*. Perusal of literature shows that this disease has not been reported so far from anywhere.

We are grateful to Dr. V. V. Chenulu, Professor and Head, Division of Mycology and Plant Pathology, I.A.R.I., New Delhi, for providing necessary facilities and encouragements. Sincere thanks are also due to Mr. K. S. Jhala, Joint Director of Agriculture, Jodhpur (Rajasthan), for help in surveying the *Brassica* fields.

Division of Mycology and  
Plant Pathology, I.A.R.I.,  
New Delhi 110 012,  
January 24, 1980.

T. P. BHOWMIK.  
B. M. TRIVEDI.

1. Buchanan, R. E. and Gibbons, N. E., *Bergey's Manual of Determinative Bacteriology*, 8th ed., Williams and Wilkins Co., Baltimore, 1974.
2. Dowson, W. D., *Plant Diseases Due to Bacteria*, Cambridge University Press, 1957.
3. Dye, D. W., *New Zealand J. Sci.*, 1962, 5, 393.
4. Hugh, R. and Leifson, E., *J. Bact.*, 1953, 66, 24.
5. King, E. D., Ward, M. K. and Raney, D. E., *J. Lab. Clin. Med.*, 1954, 44, 301.
6. Patel, M. K., Abhyankar, S. G. and Kulkarni, Y. S. K., *Indian Phytopath.*, 1949, 2, 58.
7. Satyavir, Kaushik, C. D. and Chand, J. N., *PANS*, 1973, 19, 46.

### EVIDENCE OF COMPLEMENT FIXING ANTIBODIES AGAINST EQUINE RHINOPNEUMONITIS VIRUS IN ARMY HORSES AND MULES

SEROLOGICAL surveys against Equine Herpes Virus 1 (EHV1) also known as Equine Rhinopneumonitis Virus (ERV)<sup>1</sup> have been conducted in several countries<sup>2-7</sup>. In India the incidence of ERV infection was first reported at Saharanpur in aborted foetuses, on the basis of histological lesions<sup>8</sup>. Subsequently Kentucky D-strain of ERV was isolated at Hissar, in suckling hamsters<sup>9</sup>. Abortions in some mares have been reported recently from Hissar and a few other places in the country<sup>10</sup>. However, systematic survey has not been undertaken to assess the extent of its endemicity in India. The present report is a preliminary study on the detection of complement fixing (CF) antibodies to ERV among army horses and mules.

Four hundred and thirtyeight serum samples of 333 horses and 105 mules, received in this laboratory for screening against glanders, were simultaneously processed for the detection of CF antibodies to ERV. The sera were inactivated at 56°C for 30 minutes in a water-bath. Hamster adapted lyophilized antigen (EHV1 Kentucky D Strain), positive and negative sera against ERV were obtained from Dr. T. Shimizu (National Institute of Animal Health, Japan). The antigen was reconstituted with distilled water, mixed with an equal volume of 1.8% sodium chloride solution and then centrifuged at 10,000 r.p.m. for 20 minutes. The supernatant was titrated for determining CF units of the antigen. Hyper-immune serum against ERV and antishoop erythrocyte haemolysin were raised in the laboratory in rabbits. Pooled serum complement from ten guinea pigs was titrated before each run of the test proper. Two units each of complement (100% haemolytic unit) and antigen were employed in the test. Complement fixation test (CFT) was carried out in microtitre plates using Veronal buffer as diluent<sup>11,12</sup>. The highest dilution of the serum showing +++ (60 to 80% CF reaction) indicated evidence for the presence of CF antibodies.

Table I indicates that 47 (10.73%) of 438 serum samples were positive for CF antibodies. Out of 333 horse sera, 44 (13.21%), and out of 105 mule sera, only 3 (2.86%) were found to show the presence of CF antibodies against ERV. CF titres obtained were 1:16 in 2 (0.60%), 1:8 in 12 (3.60%) and 1:4 in 30 (9.01%) of positive horse serum samples. Mule sera demonstrated high anticomplementary activity, with CF reaction in an insignificant number of samples.

It has been elucidated that a 50% CF reaction indicates a significant CF titre<sup>3</sup>. In our studies, however,

TABLE I

Distribution of Complement Fixing Antibodies to Equine Rhinopneumonitis Virus in army horses and mules

Class of animal	No. of sera tested	C.F. Titre			Total No. of positive sera	Total No. of negative sera
		1/4	1/8	1/16		
Horses	333	30 (9.01)	12 (3.60)	2 (0.60)	44 (13.21)	289 (86.79)
Mules	105	1 (0.95)	2 (1.90)		3 (2.86)	102 (97.14)
Total	438	31 (7.08)	14 (3.20)	2 (0.46)	47 (10.73)	391 (89.27)

Percentages shown in parentheses.

the CF reaction showing a minimal non-haemolysis of 60 to 80% sheep erythrocytes by the visual observation, was considered to reveal the presence of CF antibodies, which agrees with the findings of Petzoldt<sup>11</sup>. Shimizu *et al.*<sup>3</sup> reported 1:4 and above as significant titre on the basis of 50% CF reaction in their study on serological survey of horse population in Japan. We used in our study ERV antigen (EHVI, Kentucky D Strain), received from Dr. T. Shimizu and therefore a CF titre of 1:4 and above was considered as an indication for the presence of CF antibodies. Besides, it has also been reported, that the titre for EHVI (ERV) may be affected on account of antigenic cross relationship with other herpes viruses, viz., EHV2 and EHV3<sup>6</sup>.

The authors are grateful to Dr. P. K. Ramachandran, Director, for the interest and encouragement, in the work, to Major RD Verma of Central Mil. Vet. Lab., Meerut, for the supply of serum samples and to Dr. T. Shimizu of National Institute of Animal Health, Japan, for sending us CF antigen, positive and negative sera of the virus.

Department of Microbiology,  
Defence Research and  
Development Establishment,  
Gwalior 474 002,  
February 25, 1980.

A. M. JANA.  
G. PANDYA.  
K. M. RAO.

1. Doll, E. R., Bryans, J. T., Mc Collum, W. H. and Crowe, M. E. W., *Cornell Vet.*, 1957, 47, 3.
2. Bagust, T. J., *Vet. Bull.*, 1971, 41, 79.
3. Shimizu, T., Ishizaki, R. and Matumoto, M., *Jap. J. Exp. Med.*, 1963, 33, 133.
4. De Boer, G. F., *Arch. ges. Virusforsch.*, 1966, 19, 23.

5. Duxbury, A. E. and Oxeer, D. T., *Aust. Vet. J.*, 1968, 44, 58.
6. Bagust, T. J., *Ibid.*, 1972, 48, 47.
7. Von Benten, C. and Petzoldt, K., *Berl. Munch Tierarztl. Wschr.*, 1977, 90, 176.
8. Sharma, G. L., Lall, J. M. and Bhalla, N. P., *Indian J. Vet. Sci.*, 1965, 35, 18.
9. Garg, D. N., Manchanda, V. P., Chauhan, H. V. S., Chandiramani, N. K. and Singh, I. P., *Indian J. Anim. Sci.*, 1977, 47, 371.
10. Manchanda, V. P. and Garg, D. N., *J. Remount. Vet. Corps.*, 1975, 14, 185.
11. Petzoldt, K., *Dt. Tierarztl. Wschr.*, 1967, 74, 252.
12. Bagust, T. J. and Pascoe, R. R., *Arch. ges. Virusforsch.*, 1972, 36, 240.

#### INDUCED MUTATIONS IN RELATION TO HETEROCHROMATIN IN *DROSOPHILA MELANOGASTER*

THE X-chromosome of *Drosophila melanogaster* carries a large block of heterochromatin in the proximal part which extends at least up to the region designated as section 20 in the polytene maps<sup>1</sup>. This region of the chromosome is more prone to radiation induced breaks and lethal mutations<sup>1</sup>. The visible mutation frequencies are also higher for the genes located near this heterochromatic region<sup>2-4</sup>. In addition to this major heterochromatic block, the X-chromosome is having intercalary heterochromatic regions<sup>5</sup>. The present investigation was aimed at analysing the role of heterochromatin in the induction of sex-linked recessive visible mutations by 4 different mutagens in *D. melanogaster*.

The mutagens used included gamma rays and three alkylating agents, viz., N-methyl-N'-nitro-N