E). Intersexual flowers and flowers with reduced number of stamens bore pollen which were normal in shape and size. After the 4th week of treatment, plants started bearing normal male flowers. However, plants sprayed with 960 ppm of Ethrel took 6 weeks to revert to the production of normal flowers.

The data presented here demonstrate that the flower sex-expression of male plants of *Cannabis sativa* can be altered by treatment with Ethrel. It has been suggested that Ethrel decomposes in the plant tissues to release ethylene[2,3]. It is recently claimed that some effects of auxin are exerted through an ethylene evolution mechanism[4]. It is likely that Ethrel (a source of ethylene) exerts its effect on sex-expression by manipulating the endogenous level of auxin[5].

*Zusammenfassung.* Ethrel induziert auf männlichen Hanfpflanzen (*Cannabis sativa* L.) weibliche Blüten, welche nach der Bestäubung Früchte ausbilden.

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[2] A. R. COOKE and D. I. RANDALL, Nature, Lond. *218*, 979 (1968).
[3] L. J. EDGERTON and G. D. BLANPIED, Nature, Lond. *219*, 1064 (1968).
[4] S.P. BURG and E.A. BURG, Proc. U.S. natn. Acad. Sci. *55*, 262 (1966).
[5] Acknowledgments: We are grateful to Prof. B. M. JOHRI for interest and facilities and to Amchem Products Inc., Ambler (Pa., USA), for a gift sample of Ethrel.

## Catalase and Peroxidase Activity in Sugarcane Infected with Sugarcane Mosaic Virus

Virus infection usually brings about drastic changes in the physiological processes of the host cell. Among the physiological effects of virus infection on host plants, changes have been observed mostly in over-all respiratory activity[1]. Changes in the activity of oxidative enzymes have also been studied both in fungus diseased[2] and virus infected plants[3,4]. Very little work, however, has been done with mosaic infected sugarcane plants. YAMAFUGI et al.[5] studied the respiration and catalase activity in mosaic diseased sugarcane plants. In the present investigation, the effect of virus infection on catalase and peroxidase activity has been studied in 6 different varieties of sugarcane.

Leaves from healthy and mosaic infected plants of 6 sugarcane varieties, viz., B.O.11, B.O.32, B.O.47, Co. 527, Co. 1347 and Co. S. 416, were taken separately and analyzed for catalase and peroxidase activities. 4 samples were taken in each trial. Catalase activity was measured by the method of DEKOCK et al.[6], with slight modifications. For enzyme preparation, 1 g of leaf tissue was crushed in a chilled mortar with 10 ml of phosphate buffer (pH 6.8) and a pinch of acid-washed sand. This homogenate was made up to 25 ml volume with glass-distilled water. A series of flasks containing 5 ml of 1.5% sodium borate and 1.5 ml of phosphate buffer (pH 6.8) was prepared. At zero time, 1 ml of homogenate was pipetted into each flask. The reaction was stopped in successive flasks after 1, 2, 3 and 4 min by rapidly adding 10 ml of $N$ $H_2SO_4$. The remaining perborate was then titrated with 0.05 $N$ $KMnO_4$ to the first pink colour which lasted for 30 sec.

Peroxidase activity was measured by the method given by PERUR[7]. Enzyme preparation was made by taking 1 g of sugarcane leaf and grinding it in a chilled mortar with 5 ml of glass-distilled water. The homogenate was made up to 50 ml and, after mixing well, it was filtered through cheese-cloth. This filtrate was the enzyme preparation which was used for the measurement of activity. In a test-tube, 10 ml of acetate buffer (pH 4.5) was taken. To it 1 ml of enzyme preparation and 0.5 ml of 1% pyrogallol were added. The contents were thoroughly mixed. At zero time, 0.5 ml of 0.05 $N$ hydrogen peroxide was added and change in optical density was measured at 430 nm, using filter No. 43 in AIMIL Biochem. Absorptiometer (manufactured on Hilger pattern) at the end of 10 min. Data obtained are presented in Figures 1 and 2. The results showed that catalase activity was slightly weaker, whereas the peroxidase remained slightly higher in diseased samples of all the 6 varieties.

It is customary to class catalase among the respiratory enzymes, although its real function is poorly known as yet[2]. The activity of catalase generally decreases in virus infected plants[4,8]. YAMAFUGI et al.[5], in one of their experiments, found that catalase activity was weaker in the mosaic infected sugarcane. According to them, the

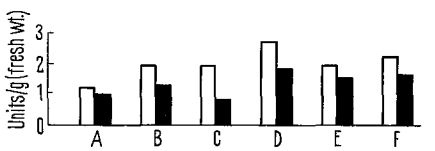


Fig. 1. Catalase activity of healthy (□) and mosaic diseased (■) samples.

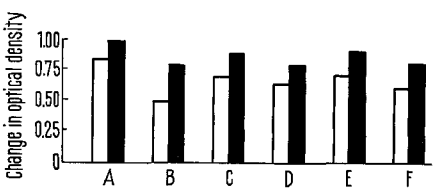


Fig. 2. Peroxidase activity of healthy and mosaic diseased samples. (A) B.O. 11; (B) B.O. 32; (C) B.O. 47; (D) Co. 527; (E) Co. 1347; (F) Co. S. 416. Dark-coloured rectangles show diseased samples and empty rectangles show healthy samples.

[1] G. LOEBENSTEIN and N. LINSEY, Phytopathology *51*, 533 (1961).
[2] Z. KIRALY and G. L. FARKAS, Phytopath. Z. *34*, 341 (1959).
[3] G. DOBY, Z. Pflkrankh. Pflpath. Pflschutz *22*, 401 (1912).
[4] R. M. VAGER, Zh. Obsch. Biol. *16*, 298 (1955).
[5] K. YAMAFUGI, K. So and K. NAGANO, Biochem. Z. *315*, 405 (1943).
[6] P. C. DE KOCK, K. COMMISIONG, V. C. FARMER and R. H. E. INKSON, Plant Physiol. *35*, 599 (1960).
[7] N. G. PERUR, Current Sci. *31*, 17 (1962).
[8] N. GIMESI and B. POZSAR, Annls biol. Univ. Hung. *2*, 90 (1954).

host cell catalase is enclosed in the high-molecular virus protein during the process of virus multiplication, so that the activity of the enzyme can only be exerted under appropriate conditions after the splitting-up of the virus.

As early as 1900, WOODS[9] commented on disturbances of the oxidase and peroxidase systems in tobacco plants infected with mosaic. He showed that this enzyme was more active in light coloured areas of mosaic tobacco leaves. A little later CHAPMAN[10] observed increased activity in both tomato and tobacco plants. BUNZELL[11], in the same year, reported increased peroxidase activity in curly-top virus infected sugarbeet. In recent years also, several workers have demonstrated an increase in peroxidase activity in different virus infected plants[1,4,12,13]. LOEBENSTEIN and LINSEY[1] showed a positive relation between peroxidase activity and development of vein clearing in infected sweet potatoes. As yet, the physiological role of peroxidase, even in normal metabolism, is not clearly understood, although it is known to catalyse the oxidation of phenolic substances and aromatic amines to quinones in the presence of hydrogen peroxide[14,15].

*Zusammenfassung.* Vom Zuckerrohr-Mosaik-Virus befallene Zuckerrohrblätter weisen eine höhere Peroxidase- und eine niedrigere Kataleseaktivität auf gegenüber den Enzymaktivitäten gesunder Blätter.

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[9] A. F. WOODS, Science *11*, 17 (1900).
[10] G. H. CHAPMAN, Mass. Agric. expl. Station (25th Annal Report) *31*, 41 (1913).
[11] H. H. BUNZELL, U.S. Dept. agric. Bur. Pl. Ind., Bull. *277*, 1 (1913).
[12] G. B. ORLOB and D. C. ARNY, Phytopathology *51*, 768 (1961).
[13] S. R. CHANT, Experientia *23*, 676 (1967).
[14] J. BONNER, *Plant Biochemistry* (Academic Press, New York 1950).
[15] This research has been financed in part by a grant made by United States Department of Agriculture under No. P.L. 480, to whom grateful thanks are due.

## DISPUTANDUM

### A Possible Role of Guanine-Deaminase Inhibitor

Xanthinuria is a rare disorder characterized by the excretion of very large amounts of xanthine in urine and a tendency to form xanthine stones. DENT and PHILPOT[1] described the only well-documented case of xanthinuria. Since then approximately 30 additional cases have been reported[2].

In mammalian systems xanthine has 3 known precursors, hypoxanthine, guanine and guanosine. Relatively little xanthine originates from xanthosine, since it is a very poor substrate for nucleoside phosphorylase. The major source of xanthine are hypoxanthine, which is oxidized by xanthine oxidase to yield xanthine, and guanine, which is deaminated by guanine-deaminase to yield xanthine. Xanthine thus formed is further oxidized by xanthine oxidase to give uric acid. Any change in the metabolic condition of either or both of these enzymes, namely guanine-deaminase and xanthine oxidase, will eventually result in changes in the levels of xanthine in the tissue. In this report, the author suggests a possible explanation for the increase of xanthine levels in the tissue causing its excretion in increased amounts with the urine resulting in xanthinuria.

In earlier studies, KUMAR et al.[3,4] found the presence of a naturally occurring inhibitor of guanine-deaminase and xanthine oxidase in various rat tissues. In these studies it was also reported that the inhibitor was of protein nature associated with the nuclear and the heavy mitochondrial particles. If it was assumed that the levels of this inhibitor in the tissues were somehow changed to the extent that it no longer had any effect on the activities of these enzymes, the enzymes will favour the forward reactions – the deamination of guanine and oxidation of hypoxanthine to give xanthine. The xanthine thus produced will be further oxidized by xanthine oxidase into uric acid. It is likely that the rate of formation of xanthine under such circumstances will be faster than the rate of oxidation of xanthine into uric acid, thereby resulting in increased quantities of xanthine. The excess xanthine will in turn be excreted unmetabolized along with the urine, causing xanthinuria. Although no definite clinical tests have been conducted to establish this as the cause of xanthinuria, some of the studies done on the biopsy samples obtained from the liver of a man having initial stages of xanthinuria and from normal human liver, have shown decreased levels of the inhibitor in the particulate fractions obtained from the liver of the man with xanthinuria as compared to normal human liver, suggesting a possible role of the inhibitor in maintaining a metabolic balance in the purine catabolism.

Further studies in this direction are in progress, the results of which will be published elsewhere.

*Zusammenfassung.* Diskussion über die Frage der Ätiologie der Xanthinurie.

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[1] C. E. DENT and G. R. PHILPOT, Lancet *1*, 182 (1954).
[2] J. B. WYNGAARDEN, in *The Metabolic Basis of Inherited Disease* (Eds. J. B. STANBURY, J. B. WYNGAARDEN and D. S. FREDRICKSON; McGraw Hill, New York 1960), p. 761.
[3] S. KUMAR, K. K. TEWARI and P. S. KRISHNAN, Biochem. J. *95*, 797 (1965).
[4] S. KUMAR, V. JOSAN, K. C. S. SANGER, K. K. TEWARI and P. S. KRISHNAN, Biochem. J. *102*, 691 (1967).
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