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.

K. S. BHARGAVA, R. D. JOSHI and G. P. SRIVASTAVA

Department of Botany, University of Gorakhpur, Gorakhpur (India), 15 October 1969.

[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.

S. KUMAR[5]

New York State Research Institute for Neurochemistry and Drug Addiction, Wards Island, New York (N.Y. 10035, USA), 28 August 1969.

[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).
[5] Present address: Department of Pediatrics, Methodist Hospital of Brooklyn 506 Sixth Street, Brooklyn (N.Y. 11 215. USA).