samples, the cause of such variability being largely unknown so far (Fig. 1).

Stabilization of enzymes could be expected to be brought about by using (*a*) extraneous proteins or polyamides, (*b*) reducing agents, (*c*) chelating compounds and related substances, and (*d*) competitive inhibitors.

While in our case, albumin, polylysine, a di-alkyl analogue of 6.6 nylon (group *a*); glutathione, cysteine, ascorbic acid (group *b*); ethylene diamine tetraacetic acid (versene), 8-hydroxyquinoline sulphonic acid, triethylene tetramine, glyceric acid, glycerine (group *c*) afforded scarcely any significant protection at 37° C. for ten days in *M* phosphate at *p*H 6 (enzyme concentration about 0·5 per cent), sodium salicylate prevented completely any loss of oxidase and dehydrogenase activities[1] at 0–5° C. and almost completely at 37° C. (Fig. 1). Salicylic acid has been reported to act as an inhibitor, though not a very powerful one[3], and our own experiments confirmed that this inhibition was competitive in nature, with $K_i = 1{\cdot}35 \times 10^{-4}\,M$ (spectrophotometric assay at *p*H 8·2, taking $K_s = 5{\cdot}3 \times 10^{-6}\,M$[4]). It is also noteworthy that Keeser[5] reported on 'activation' by sodium salicylate of xanthine oxidase in guinea pig liver after incubation for 2 hr. at 37°; in the light of our findings, this observation may be due to stabilization rather than to activation. Of substances related to salicylic acid (all acids in form of sodium salts), salicylamide, sulphosalicylic acid, salicylaldoxime and gentisic acid showed only slight to moderate effects; but *p*-aminosalicylic acid, acetyl salicylic acid (aspirin) and benzoic acid came near or close to salicylate itself, which remained the best. Our findings may be compared with those of Veitch and McComb[6], who have very recently found a stabilizing effect with *m*-toluic acid on D-amino-acid oxidase.

Salicylate could be removed from the enzyme by dialysis, even after prolonged association. This was proved by the fact that the ratio of ultra-violet absorption at 280 mμ to that at 295 mμ (at which wave-length the salicylate ion has an absorption maximum) returned to the value found with untreated samples, and that $^{14}CO_2H$-labelled salicylic acid was removed almost quantitatively (to about 0·25 mole per mole of total xanthine oxidase) by three consecutive dialysis procedures.

Two points remain to be mentioned: one concerns the low activity established with our xanthine oxidase preparations[1] when using cytochrome-*c* as electron acceptor, which did not increase on adding salicylate, but was inhibited by high concentrations of the reagent; in contrast, 8-hydroxyquinoline augmented this activity slightly, confirming the original observation by Doisy *et al.*[7], who suggested interaction between the iron of the enzyme and the chelating agent. The second point refers to the moderate stabilizing effects of uric acid[8] and 2 : 6-diamino-8-phenyl-purine, the latter a powerful new inhibitor of xanthine oxidase.

Details of these investigations, which include the use of salicylate during the purification process[9], and some evidence for the possible mechanism of loss of activity and stabilization by salicylate will be published elsewhere.

Our thanks are due to Dr. V. C. E. Burnop for the synthesis of labelled salicylic acid, Messrs. D. A. Gilbert and P. G. Avis for the preparation of xanthine oxidase samples, the Research Department of British Nylon Spinners for the nylon analogue, Mr. G. M. Timmis for the purine derivative, and Mrs. K. Tussler for technical assistance.

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Presence of Deoxyribonuclease in Silkworm Polyhedral Virus

Working on the supposition[1,2] that polyhedral virus would be derived from the deoxyribonuclease-controlling gene, I have now been able to demonstrate the presence of a fairly large amount of deoxyribonuclease in polyhedral crystals.

Larvæ of a pure line, *P*44, were injected with a viral solution and polyhedra isolated in the usual way from surviving diseased individuals. Isolation was, however, carried out under cooling conditions and the last sediment washed with 2 *M* sodium chloride, in order to remove remaining cell debris. 5 mgm. of purified polyhedral virus was dissolved in 0·6 ml. of 0·025 *M* sodium carbonate and centrifuged, and the supernatant was carefully neutralized with hydrochloric acid to *p*H 7·5. The enzymic activity of the polyhedral solution was determined viscosimetrically, using spleen deoxyribonucleic acid as substrate. The velocity constant, *k*, of decomposition expressed as a monomolecular reaction at 35° C. was 0·00105. In the determination of deoxyribonuclease, 0·005 *M* magnesium sulphate was added. The polyhedra showed a very feeble nuclease action in the absence of magnesium ions, and it is assumed that they contain almost no activators. Another solution of the same polyhedral virus preparation was then mixed with 0·02 mgm. of crystalline, pancreatic trypsin and kept at 5° C. for 80 min. The *k*-value was raised to 0·00146 by this treatment. In supplementary tests, I observed, however, that the deoxyribonuclease is somewhat inactivated by trypsin at higher temperature. It seems that the nuclease exists at least partly in a special combination with other proteins in the polyhedron. Deoxyribonuclease was also demonstrated in the polyhedra prepared from strain *P*21.

In one control experiment, deoxyribonuclease of digestive organ-free tissues of host larvæ was estimated in a similar manner. It was found that the nuclease activity of a tissue preparation corresponds to only 5 per cent of that of crystalline polyhedral virus.

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