versible changes in the enzymes. In neither case would the added sulfhydryl compounds have any reactivating effect.

References

1. LAZAROW, A. *Proc. Soc. Exptl. Biol. Med.*, **61**, 441 (1946).
2. *Ibid.*, **66**, 4 (1947).
3. LUKENS, F. D. W. *Physiol. Revs.*, **28**, 304 (1948).
4. PIETRO, A. *Rev. soc. argentina biol.*, **21**, 259 (1945).
5. LAZAROW, A., PATTERSON, J. W., and LEVY, S. *Science*, **108**, 308 (1948).

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Enzymatic Hydrolysis of Dextran

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The preparation of blood volume extenders from dextran currently is based on partial acid hydrolysis of the bacterial polysaccharide, with subsequent solvent fractionation of the hydrolysate to yield products with average molecular weights approximating those of plasma albumins (*1, 2*). Ingelman (*3*) and Nordström and Hultin (*4*) have reported the presence of enzymes capable of hydrolyzing dextran in filtrates from cultures of *Cellvibrio fulva, Penicillium lilacinum, P. funiculosum,* and *Verticillium coccorum.* The main effect of these enzymes on the polysaccharide is the production of reducing sugars, with the result that they have been of no practical value in the preparation of partially degraded dextrans.

For the past year we have been inoculating dextran-salts solutions with various soil samples, and also allowing such solutions to be exposed to airborne contaminants, in an attempt to isolate organisms capable of attacking dextran. Among a number of such isolates we obtained an *Aspergillus* sp. which has proved useful for the purpose of degrading various dextrans. Filtrates from cultures of the mold in media containing dextran are highly active in splitting the polysaccharide. Only a small amount of reducing sugar is produced during the time required for the filtrates to degrade the polysaccharide into fragments with an average molecular weight in the region of 75,000. By analogy with the α-amylases (*5*), the enzyme formed by this mold probably should be termed an endo-dextranase, since it appears to have a preference for splitting glucosidic linkages remote from end groups. We have found the enzyme to be active against dextrans obtained from 4 different strains of *Leuconostoc.* Active concentrates of the enzyme have been prepared by fractional precipitation of mold filtrates with ammonium sulfate.

A particular advantage of this method of hydrolysis is that the enzyme solution can be allowed to act directly on fermented culture media containing dextran. This eliminates the preliminary precipitation of the polysaccharide with alcohol, as called for in the scheme employing acid hydrolysis (*1*). Since the enzymatic hydrolysis proceeds at room temperature, it is easy to obtain partially hydrolyzed dextrans free from objectionable color.

A typical experiment was carried out as follows: Eight liters of a 20% sucrose medium was fermented with *Leuconostoc mesenteroides* 683, yielding a solution containing dextran in a concentration of 6.1%. Eighty ml of the mold filtrate was added, and enzymatic action allowed to proceed for 40 min, during which time the viscosity of the solution decreased rapidly. The action of the enzyme was stopped by adding alkali to pH 9. The solution was then fractionated with alcohol in the usual manner (*1*), yielding 400 g of a white product with an average molecular weight of 71,000, as determined by viscometry (*6*).

References

1. GRONWALL, A., and INGELMAN, B. U. S. Pat. 2,437,518 (Mar. 9, 1948); *Nord. Med.*, **21**, 247 (1944).
2. *Editorial. Chem. Eng. News*, **29**, 650 (1951).
3. INGELMAN, B. *Acta Chem. Scand.*, **2**, 803 (1948).
4. NORDSTRÖM, L., and HULTIN, E. *Svensk Kem. Tid.*, **60**, 283 (1948).
5. MYRBACK, K., and THORSELL, W. *Ibid.*, **54**, 50 (1942).
6. INGELMAN, B., and HOLLING, M. S. *Arkiv Kemi*, **1**, 61 (1949).

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Blocking of Action of Acetylcholine by Barbiturates

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The barbiturates have proved to be an important group of depressants of the central nervous system since their introduction for clinical trial in 1912 on the basis of the studies of Loewe (*1*), Juliusburger (*2*), and Impens (*3*). In anesthetic doses all the barbiturates inhibit convulsions such as occur in strychnine poisoning, tetanus, and epilepsy. Notwithstanding their continuous use in chemotherapy since their introduction in 1912, evidence of their mode of action has been scanty. Thus Schütz (*4–6*) has shown that there is a progressive decline of cholinesterase activity of human serum and in guinea pig serum, muscle, and certain nervous tissues, during prolonged treatment with a barbiturate. Heinbecker and Bartley (*7*) have shown in their studies with peripheral nerves that phenobarbital increases threshold and at the same time prolongs recovery time of neurones after impulse propagation. Eccles (*8*) showed that phenobarbital blocked two-neurone transmission in the spinal cord primarily by increasing the extent of local depolarization required for initiation of a propagated impulse by motoneurone soma. The author's interest in the

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