of tobacco-mosaic virus by 10M-acetic acid but not by mineral acids (Fraenkel-Conrat, 1957); the acetic acid decreases the surface tension value to the region of over 30 dyn/cm (*International Critical Tables*, 1928). However, even smaller surface-tension value changes can have significant effects on the equilibrium. Thus a decrease of 2.5 dyn/cm in the value of 47 dyn/cm in blood serum results in a greater than 20% decrease in the equilibrium-constant value; such changes can be induced in human sera by freezing and thawing and by increased lipoprotein concentrations (Lewin, 1972). It is relevant that 6M-urea solution reduces the value of the surface tension from approx. 72 dyn/cm to 63 dyn/cm at 25°C while guanidinium chloride solutions reduce it even more (Lewin, 1972). Hence the contribution of high urea and of high guanidinium chloride concentrations to a lowering of interface tension, and therefore to de-adherence of hydrophobic groups, is sufficiently significant to be taken into account when considering helix $\rightleftharpoons$ coil transformations.

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Inhibition of δ -Aminolaevulate Dehydratase by Pyridoxal Derivatives and by Cyanide

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δ -Aminolaevulate dehydratase (EC 4.2.1.24) was purified from extracts of *Rhodospseudomonas spheroides* grown photosynthetically, and assayed as usual by incubating it with substrate (δ -aminolaevulate) in tris buffer, pH 8.5, and determining the product (porphobilinogen) with the Ehrlich reagent (Nandi, Baker-Cohen & Shemin, 1968; S. van Heyningen & D. Shemin, unpublished work). When 1 mM-pyridoxal phosphate was added to the incubation medium the enzyme was inhibited by 30%. At 0.05 mM-pyridoxal phosphate inhibition was almost negligible. Surprisingly the kinetics of inhibition were found to be almost perfectly non-competitive, the K_m for δ -aminolaevulate being

0.8 mM both in the presence and in the absence of inhibitor. The inhibition was not increased by prolonged incubation and activity was fully recovered when pyridoxal phosphate was removed by dialysis.

Nandi & Shemin (1968) showed inhibition of the enzyme by several keto acid analogues of the substrate (e.g. laevulate), but in these cases the inhibition was competitive and presumably due to the binding of the inhibitor at a lysine residue, which they suggested was at the active centre. The effect of pyridoxal phosphate seems to be a specific one of the pyridoxal molecule and not of a carbonyl, since pyridoxal and pyridoxamine phosphate both inhibited to about the same extent as pyridoxal phosphate; but pyridine, benzaldehyde and pyridine-4-carboxaldehyde did not inhibit significantly at 1 mM concentration.

Shemin (1970) has suggested that pyridoxal phosphate, which is a cofactor of δ -aminolaevulate synthetase, which precedes δ -aminolaevulate dehydratase in the pathway of porphyrin biosynthesis, may be involved in a concerted reaction *in vivo* between the two enzymes. Our current work does show some evidence for interaction between pyridoxal phosphate and δ -aminolaevulate dehydratase.

We have also found inhibition of the enzyme by 1 mM-sodium cyanide, and this inhibition shows competitive kinetics. Such competitive inhibition by cyanide has been said to be evidence for a Schiff-base mechanism of the type suggested for this enzyme by Nandi & Shemin (1968); but we consider that it is equally well explained by direct Schiff-base formation between cyanide and δ -aminolaevulate, thereby preventing reaction between the δ -aminolaevulate and the enzyme.

We are grateful to Mr S. L. Guber for his help and to the National Institutes of Health (Grants AM13091 and 09105), American Cancer Society (Grant P.547) and the National Science Foundation (Grant GB7892).

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The Primary Structure of Yeast Aldolase

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Fructose 1,6-diphosphate aldolases fall into two distinct classes that possess different molecular and catalytic properties (Rutter, 1964). Class 1 aldolases occur in animals and higher plants and are tetrameric molecules with molecular weights of about 160 000; they are not inhibited by chelating