The work was supported by a grant from Roche Products Ltd.

Bernheim, F. (1941). *J. Pharmacol.* **71**, 344.

Fuller, A. T. (1937). *Lancet*, i, 194.

Golberg, L. (1967). *J. Soc. cosmet. Chem.* **18**, 421.

Trefouel, J., Trefouel, Mme. J., Nitti, F. & Bovet, D. (1935). *C.R. Soc. biol., Paris*, **120**, 756.

The Enzymic Deacetylation of *p*-Acetamidobenzoic Acid

By M. R. FRANKLIN, J. W. BRIDGES* and R. T. WILLIAMS. (*Department of Biochemistry, St Mary's Hospital Medical School, London W.2*)

Earlier work (Bridges & Williams, 1963) suggested that the enzymic deacetylation of *N*⁴-acetyl-sulphanilamide varied with tissue and species. The deacetylation of *p*-acetamidobenzoic acid (AABA) *in vitro* shows similar variations since it has been found in homogenates of rat, guinea-pig and mouse kidney and guinea-pig and mouse liver, but only slight activity was found in rat and rabbit liver and rabbit kidney. The activity when found was similar in both sexes and occurred in the cell-sap fraction (105 000g supernatant).

The AABA deacetylase activity in the rat was found only in the kidney. The activity was considerably increased (150%) by Mn²⁺, but not by other metal ions (Mg²⁺, Ca²⁺, Fe²⁺, Na⁺, K⁺) or a wide range of common cofactors. The effect of Mn²⁺ on the mouse and guinea-pig kidney enzyme was less marked (50 and 30% respectively). The optimum Mn²⁺ concentration was 7.5 mM for the rat kidney enzyme with an optimum pH range of 7.5–8.0. This enzyme preparation did not deacetylate *N*⁴-acetylsulphanilamide, but acetanilide was deacetylated to some extent, although the main acetanilide-deacetylating activity was associated with the microsomes.

The rat kidney enzyme showed an Mn²⁺-dependent and an Mn²⁺-independent activity. Storage of the enzyme preparation (10000g supernatant) at 4° for 4–5 days (alone or in the presence of nicotinamide, glutathione or added Mn²⁺) destroyed the Mn²⁺-dependent activity, but the Mn²⁺-independent activity remained constant. *p*-Chloromercuribenzoate also destroyed the Mn²⁺-dependent activity, but did not affect the activity of the preparation that had been stored at 4° for 5 days. CoA, thioglycollate, Hg²⁺, Cu²⁺ and Zn²⁺ inhibited both the Mn²⁺-dependent and Mn²⁺-independent activity.

These and other observations suggest that the rat kidney AABA deacetylase may possess either

* Present address: Department of Biochemistry, University of Surrey, London S.W.11.

two active sites, one requiring Mn²⁺ for its activity, or a Mn²⁺-binding site that causes allosteric activation of the active site. The Mn²⁺ binding or activated site is much more susceptible to inactivation than the original active site.

This work was supported by a grant from the Ministry of Defence.

Bridges, J. W. & Williams, R. T. (1963). *Biochem. J.* **87**, 19P.

Observations on the Metabolism of Allyl Compounds in the Rat

By J. J. CLAPP, C. M. KAYE and L. YOUNG. (*Department of Biochemistry, St. Thomas's Hospital Medical School, London S.E.1*)

1-Menaphthylmercapturic acid has been isolated from the urine of rats after the injection of various 1-menaphthyl compounds including 1-menaphthyl acetate and 1-menaphthyl benzoate (Hyde & Young, 1968), and these appear to be the first examples of mercapturic acid formation arising from the administration of carboxylic acid esters to animals. It is generally accepted that biosynthesis of mercapturic acids takes place by the mechanism proposed by Barnes, James & Wood (1959) and Bray, Franklin & James (1959a,b), whereby the administered compound or an active derivative reacts with glutathione to give an *S*-substituted glutathione, which is subsequently metabolized to the mercapturic acid. As it is known that the esters of allylic alcohols are able to transfer an allylic group to a strongly nucleophilic anion such as a thiol (Albert, 1965) it became of interest to determine whether an ester such as allyl acetate gives rise to a mercapturic acid *in vivo*.

The urine of rats that had been dosed with allyl acetate by subcutaneous injection was shown by paper chromatography and thin-layer chromatography to contain a hydroxypropylmercapturic acid. This compound was identified as 3-hydroxypropylmercapturic acid, i.e. *N*-acetyl-*S*-(3-hydroxypropyl)-L-cysteine, by the gas-liquid-chromatographic examination of its methyl ester by the procedure of James, Waring & White (1966). There was no evidence for the presence of allylmercapturic acid, i.e. *N*-acetyl-*S*-allyl-L-cysteine, in the urine of the dosed animals. Similar results were obtained after the administration of allyl alcohol to rats.

Some allyl compounds were found to give rise to allylmercapturic acid in the organism, however, for this compound was isolated from the urine of rats that had been injected subcutaneously with allyl chloride, and by chromatographic examination was