trifluoromethyl group. In the rat monohydroxylation of the compound appears to occur in the 5-, 6- and 7-positions, and conjugates of these materials are found in the urine. Similar hydroxy metabolites were found in maize growing in soil treated with the labelled compound, both free and in conjugation. In 9-week-old maize 25% of the ^{14}C was present as trifluoroacetic acid; this was confirmed by co-chromatography and isotope dilution as the benzylthiouronium salt. Since the proportion of 6-hydroxy- to the 5- and 7-hydroxy derivatives increased with time it is possible that it is these latter compounds that give rise to trifluoroacetic acid, although no intermediates were detected.

Mechanism of Inhibition of Glycollate Oxidase by Aldehyde Bisulphite Addition Compounds, and by Related Structures

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The mechanism of inhibition of pea glycollate oxidase by aldehyde bisulphite addition compounds has been investigated by using the oxygen electrode. We agree with Zelitch (1957) that inhibition by $\text{HO}\cdot\text{CH}_2\cdot\text{SO}_3\text{Na}$ is due to simple competition with glycollate, but with higher members of the series we find that inhibition is due to the formation of glyoxylate bisulphite, $\text{NaO}_2\text{C}\cdot\text{CH}(\text{OH})\cdot\text{SO}_3\text{Na}$, by reaction of enzymically produced glyoxylate with the inhibitor. Our evidence for this is as follows.

(a) Nine aldehyde bisulphite compounds of widely varying structure, and including glyoxylate bisulphite, all caused 50% inhibition of pea glycollate oxidase within the range $0.8\text{--}2.0\mu\text{M}$. Compounds derived from dialdehydes were significantly more inhibitory than those derived from monoaldehydes.

(b) Spectrophotometric evidence with the addition compound of pyridine-2-aldehyde, or that of $\text{Ph}\cdot\text{C}:\text{C}\cdot\text{CHO}$, showed that the spectrum changed in minutes to that of the free aldehyde when glyoxylate was added. This also occurred when glycollate oxidase generated the glyoxylate from glycollate.

(c) The kinetic behaviour of all inhibitors, except the formaldehyde compound, was not straightforward, and resembled that of glyoxylate bisulphite. Inhibition decreased with time, probably owing to the progressive increase in the concentration of glyoxylate, which may interfere with the binding of glyoxylate bisulphite to the enzyme.

An investigation of 37 compounds structurally related to molecules known to bind to glycollate oxidase showed that some aminomethanesulphonates are also powerful inhibitors of glycollate oxidase. Although we have not investigated this inhibitory

mechanism in detail we suspect that the aminomethanesulphonates are unstable and are converted into a simpler compound, possibly the formaldehyde bisulphite addition compound: (a) With compounds of the type $\text{R}\cdot\text{NH}\cdot\text{CH}_2\cdot\text{SO}_3\text{Na}$, the best inhibitors are those in which $\text{R} = \text{CH}_3$, $\text{CH}_3\cdot\text{CH}_2$, or $\text{Ph}\cdot\text{CH}_2$ (50% inhibition of glycollate oxidase at about $3\mu\text{M}$), whereas compounds in which R is Ph or $p\text{-ClC}_6\text{H}_4$ had to be present at $>333\mu\text{M}$ to cause 50% inhibition. Therefore the most inhibitory compounds are those substituted with electron-donating groups, which will make the nitrogen atom liable to protonation, with subsequent hydrolysis to a simpler compound. (b) The hydrolysis rate of $\text{CH}_3\cdot\text{CH}_2\cdot\text{NH}\cdot\text{CH}_2\cdot\text{SO}_3\text{Na}$ was tested conductometrically at pH 7 and room temperature and the half-life was found to be a few minutes.

Zelitch, I. (1957). *J. biol. Chem.* **224**, 251.

The Effects of 1,1-Di-(*p*-chlorophenyl)-2,2,2-trichloroethane (DDT) and 1,1-Di-(*p*-chlorophenyl)-2,2-dichloroethylene (DDE) on Hepatic Microsomal Oxidase in the Rat and Japanese Quail

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The metabolic conversion of DDT into DDE and DDD [1,1-di-(*p*-chlorophenyl)-2,2-dichloroethane] has been demonstrated in a number of vertebrates including rats (Peterson & Robison, 1964) and pigeons (Bailey, Bunyan, Rennison & Taylor, 1969a). DDT and DDE give rise to similar increases in both liver weight and microsomal drug-metabolizing enzyme activities in rats when administered at 100mg/kg for 3 days (Hart & Fouts, 1965). DDE was both more toxic than DDT and led to more rapid and larger mean liver-weight increases when fed to pigeons at 1000 p.p.m. (Bailey, Bunyan, Rennison & Taylor, 1969b). The latter suggested that increases in avian hepatic microsomal enzymes after DDT administration (Gillette, Chan & Terriere, 1966) could also be primarily due to metabolically produced DDE. A similar pattern may also have been overlooked in rats owing to the high dose rates often employed to produce microsomal enzyme induction.

Here we report the measurement of liver and heart residue concentration (Taylor, Rea & Kirby, 1964) together with hepatic microsomal protein content (Lowry, Rosebrough, Farr & Randall, 1951), cytochrome *P*-450 concentration (Omura & Sato, 1964) and aniline oxidase activity (Kato & Gillette, 1965) after feeding with diets containing various

* Deceased 27 February 1970.