THE METABOLISM OF GLYCINE IN EXPERIMENTAL PORPHYRIA*†

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Ockner and Schmid[1] have recently reported a syndrome of porphyria in rats, which can be induced by feeding a diet containing 0·2% hexachlorobenzene. This syndrome has a strong biochemical resemblance to the syndrome of acute porphyria in man, and provides a convenient experimental preparation for the study of disordered porphyrin synthesis.

We have induced porphyria in this way in adult male white rats and studied the metabolism of C^{14}-labelled glycine by liver homogenates incubated *in vitro*. The liver was incubated in a standard Warburg apparatus in the presence of either C-1 or C-2 labelled glycine, and the respiratory CO_2 trapped on filter paper which had been spotted with saturated KOH. The filter paper with the absorbed radioactivity was then counted in a liquid scintillation counter. In this way the rate of conversion of each of the 2 carbon atoms of glycine to CO_2 could be compared in normal and porphyric rats.

The liver homogenates from the hexachlorobenzene-fed rats showed the following changes when compared with the normal animals (the results given are the means for 7 experiments):

1. A depression in Q_{O_2} (normal 9·57, porphyric 6·74).
2. A slower rate of conversion of glycine-1-carbon to CO_2 (normal 22·73 μA/G. dry weight of liver per 2 hours, porphyric 10·79 μA/G. dry weight of liver per 2 hours).
3. A slower rate of conversion of glycine-2-carbon to CO_2 (normal 6·63μA/G. dry weight of liver per 2 hours, porphyric 1·91 μA/G. dry weight of liver per 2 hours).

The depression of the rate of conversion of the second carbon atom to CO_2 was much greater than that of the first carbon atom, with the result that the ratio $\frac{\text{C-1}\rightarrow CO_2}{\text{C-2}\rightarrow CO_2}$ was increased in the porphyric rats (normal 3·76, porphyric 5·74).

This suggests that in hexachlorobenzene-induced porphyria there is a specific defect in the oxidation of the α-carbon atom of glycine to CO_2. It is known that the α-carbon atom of glycine can form 'active C_1 fragments', and subsequent studies with C^{14}-labelled formate have indicated that this oxidative defect might extend to all 'C_1 fragments'.

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REFERENCE

1. Ockner, R. R. and Schmid, R. (1961): Nature (Lond.), **189**, 499.