liver cholesterol is the result of poor absorption, of mobilization of the sterol to meet some demand imposed by the illness, or of a combination of causes. Gillum in this laboratory has shown[4] that it takes a healthy rat 6 to 9 weeks to clear out its cholesterol ester stores when changed from a cholesterol-rich to a cholesterol-poor diet. This is certainly a very much slower process than that which takes place in the sick animal. Further study of cholesterol-fed animals with infections of known type and virulence seems indicated.

Summary. Rats have been fed diets containing 1% cholesterol from weaning throughout their life span. Their growth, health and time of survival have not differed significantly from those of control animals on the same basic diet without the cholesterol.

12031

Oxidation of Some Substituted Alcohols by Rat Liver.

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The alcohol oxidase of liver can oxidize a number of lower primary alcohols.[1] The effect of substitutions in the alcohol molecule has not been studied. The following is a report on the oxidizability by rat liver suspensions of a number of such substituted alcohols.

Experimental. The liver preparation was made by chopping with scissors, grinding with sand and M/20 phosphate buffer pH 7.8, and squeezing through muslin. The alcohols were obtained from the Eastman Kodak Co. They included ethanolamine, diethanolamine, β-β'-dihydroxyethyl ether, β-β'-dihydroxyethylsulfide, ethylene bromohydrin, tribromoethanol (avertin), glycerol, ethylene chlorohydrin, ethylene glycol, and β-hydroxypropionitrile. The last 3 were the only ones oxidized by the liver preparation. Figure 1 shows the oxidation of various concentrations of these 3 compounds compared with the oxididation of ethyl alcohol. The Schiff test

[4] H. L. Gillum, unpublished data.

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[1] Lutwak-Mann, C., *Biochem. J.*, 1938, **32**, 1364.

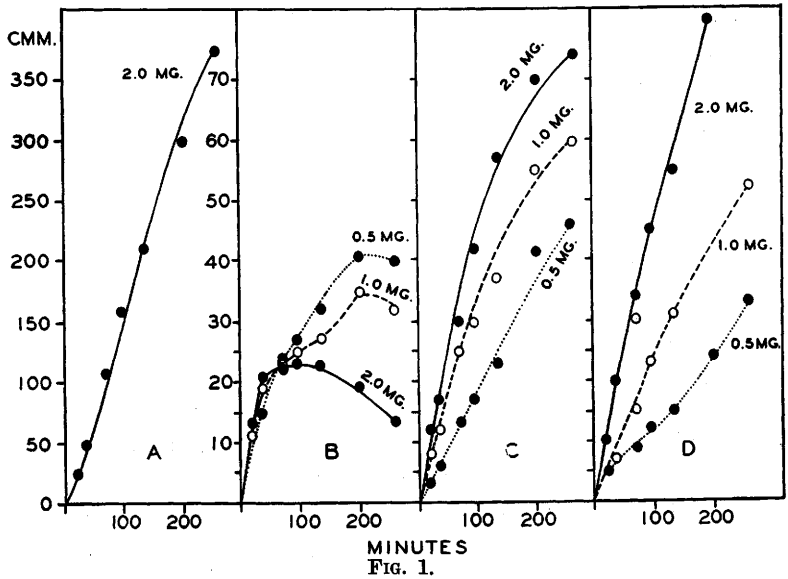

FIG. 1.

The oxidation of various alcohols by rat liver suspension, pH 7.8, 37°. The oxygen uptake of liver alone has been subtracted from the uptake of liver and alcohol. A—ethyl alcohol; B—ethylene chlorohydrin; C-β—hydroxypropionitrile; D—ethylene glycol.

showed that aldehydes were present in concentrations greater than in the control when these compounds were oxidized. The aldehyde formed from ethylene chlorohydrin is evidently toxic because it inhibits the control oxygen uptake of the liver. Tissues lacking the alcohol oxidase, *i. e.*, brain and kidney, fail to oxidize the substituted compounds. None of the compounds inhibit the oxidation of ethyl alcohol. 0.005 M KCN inhibits the oxidations. The pH optimum for the oxidation of the substituted compounds is the same as that for ethyl alcohol.

Summary. Of a series of substituted ethanols and ethanol derivatives, only ethylene chlorohydrin, ethylene glycol, and β-hydroxypropionitrile are oxidized by liver preparations containing an active alcohol oxidase.