strated that a significant amount of fat absorption had occurred. Since it was found in this laboratory that finely emulsified oil particles of approximately 0.5 μ in diameter can be directly absorbed from the rat intestine(1), it seems apparent that the rats did not make effective use of these exogenous emulsifiers to disperse the ingested oil in their digestive fluids more efficiently than they can do without these emulsifiers, nor did the rats make effective use of these exogenous emulsifiers for oil emulsification when their own biliary and pancreatic juices were excluded from the digestive mixtures in their intestines.

The necessity of choline for the turnover of plasma phospholipids has been adequately demonstrated(12-14) and it has also been reported that choline may represent a limiting factor for the formation of phospholipids in the intestine during the absorption of fat from the intestine(15). However, in the work reported in this paper, fat absorption from the choline deficient rat was normal. Furthermore, an adequate choline supplement did not aid the absorption of fat from the ligated intestine in the normal rat from which most of the digestive secretions which might contain choline were excluded. This suggests that under the conditions used here choline had little function in the absorption of fat from the intestinal lumen and that its participation in the metabolism of ingested fat begins after the absorption of fat from the intestine.

Summary. The absorption of orally fed corn oil in approximately physiological amounts by the rat was unaffected by the presence of amounts of purified soybean phosphatide or Tween 80, greatly in excess over that needed to make excellent emulsions by mechanical means. The absorption of approximately physiological amounts of orally fed corn oil by the severely choline deficient rat was similar to that found in the normal rat. The absorption of corn oil after instillation in the rat intestine ligated below the pancreas and at the ileocecal junction was unaffected by the presence of large amounts of purified soybean phosphatide, Tween 80, or choline chloride.

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Lack of Direct Biological Conversion of Glycine to Ethanolamine. (18305)

DAVID M. GREENBERG AND SARAH C. HARRIS

From the Division of Biochemistry, University of California School of Medicine, Berkeley.

Stetten(1) obtained N^{15}-labeled ethanolamine upon feeding the corresponding isotopic glycine and suggested that it was formed by the direct reduction of glycine. This hypothesis has found its way into many textbooks and reviews(2). Subsequently Stetten(3) obtained labeled ethanolamine from N^{15}-labeled serine and proposed instead that serine is decarboxylated to yield ethanolamine. The formation of ethanolamine from serine-β-C^{14} has been demonstrated by Levine and Tarver (4) and also by the authors (unpublished experiments). Since glycine can be readily transformed to serine, it appears quite plausible, as has been proposed by Levine and Tarver(4), that ethanolamine is formed from glycine via serine and not directly. However,

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2. Jukes, T. H., *In Ann. Rev. Biochem.*, 1947, v16, 193.

3. Stetten, D., Jr., *J. Biol. Chem.*, 1942, v144, 501.

4. Levine, M., and Tarver, H., *J. Biol. Chem.*, 1950, v184, 427.

the reverse is also possible and the isotopic experiments that have been reported do not distinguish between the two possible reaction pathways.

The experiment described below, with C^{14}-carboxyl-labeled glycine was performed to test this point. The results indicate that glycine is not directly reduced to ethanolamine and is so transformed only through serine as an intermediate.

Experimental. Three rats were injected intraperitoneally with 500,000 counts per min. (0.52 mg, 2.8 μc) each of C^{14}-carboxyl-labeled glycine. The animals were killed 4 hours after administration, the livers removed and pooled, and the phospholipids extracted, and then hydrolyzed by refluxing at the boiling point with 3.6% hydrochloric acid for 12 hours. The mixture was chilled, the fatty acids filtered off, and the filtrate concentrated *in vacuo* with repeated addition of water to remove the hydrochloric acid. The residue in about 75 ml of water was treated with neutral aqueous lead acetate, and then freed of excess lead with hydrogen sulfide. The filtrate was evaporated to dryness and extracted with small volumes of ethanol. The alcoholic solution was filtered, evaporated to dryness, and taken up in 1 ml of 1 M hydrochloric acid. The solution was mixed with an excess of calcium oxide and ethanolamine isolated by extraction with ether according to the method of Thierfelder and Schulze* (5). It was counted as the oxalate(6). None of the samples gave counts that were significantly above background.

To the phospholipid extract of the liver of another rat, to which the same dose of carboxyl-labeled glycine had been administered, carrier ethanolamine was added and the base was separated from serine by passage through a column of permutit(7) after acid hydrolysis of the phospholipids. The eluted ethanolamine was treated with periodate and the formaldehyde formed collected as the dimedon derivative. This too contained no activity.

Summary. Ethanolamine isolated following administration of C^{14}-carboxyl-labeled glycine contained no radioactivity. This indicates that glycine is not directly reduced to ethanolamine in the body and that this occurs through the intermediate formation of serine.

* Choline also was isolated and counted as the chloroplatinate. It contained no radioactivity.

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A Small Hemagglutinating Component in Preparations of Newcastle Disease Virus.* (18306)

ALLAN GRANOFF, OSCAR C. LIU, AND WERNER HENLE.

From the Children's Hospital of Philadelphia and The Division of Virology, Department of Public Health and Preventive Medicine, School of Medicine, University of Pennsylvania.

The size of the virus of Newcastle disease of chicken (NDV) has been estimated to be between 80-120 mμ by filtration through gradocol membranes(1). This general order of size has been confirmed by electron microscopy(2,3), and ultracentrifugation(2,3). The sedimentation studies with virus obtained from infected allantoic fluids indicated that the hemagglutinating activity of NDV is

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