groups of mice that cod liver oil and oleic acid play rôles similar to glucose.

In the oral administration of radio-phosphorus, in therapeutic attempts, it would seem desirable to administer the P^{32} on an empty stomach and with a 10% solution of glucose in order to obtain greater absorption and retention. The simultaneous administration of iron salts should be avoided.

The greater retention of P^{32} in the young animals can be explained partially on the basis of a more rapid growth, particularly of the osseous system.

Summary. Glucose increased and iron salts decreased the absorption of radioactive phosphorus from the gastro-intestinal tract.

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Choline, Creatine and the "Labile Methyl" Supply.

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Hemorrhagic degeneration, characterized by renal hemorrhage and fatty livers, occurs in young rats on diets low in choline and in methylated compounds possessing choline-like activity.[1] Supplements of choline,[1] methionine[1] or betaine[2] readily prevent this dietary deficiency. The effect of creatine in decreasing the occurrence of renal lesions is shown in Table I. The liver fat is not significantly affected unless both creatine and choline are fed. Prevention of the fatty liver requires more choline than is necessary for the prevention of renal hemorrhage.[1] The experiments in Table I demonstrate that supplements of creatine decrease the severity of but do not prevent hemorrhagic degeneration. This effect differs from that of methionine and betaine in that only partial protection occurs and the degree of protection is not increased by larger supplements of creatine.

The following explanation, which deals with the transfer of essential methyl groups and not with the synthesis of the rest of a

1 Griffith, W. H., and Wade, N. J., *J. Biol. Chem.*, 1939, **131**, 567; 1940, **132**, 627.

2 Griffith, W. H., and Mulford, D. J., unpublished experiments reported to the Division of Biological Chemistry of the American Chemical Society in Detroit, Sept. 9 to 13, 1940.

TABLE I.
Effect of Creatine on Severity of Hemorrhagic Degeneration in Young Male Rats, 38 to 42 g in Weight and 21 to 26 Days of Age, During an 8-day Experimental Period.

	Supplement mg per g of food	No. of rats	Final body wt, g	Kidney wt, % of body wt	Liver fat, % of liver	Renal lesions %
Basal AC 50*		88	50	1.94	19.9	95
+ Creatine	2	42	58	1.43	20.6	74
+ "	5	58	57	1.42	17.5	67
+ "	10	21	61	1.41	18.0	67
+ Choline Chloride	0.4	56	61	1.05	12.5	4
+ Creatine and Choline	5 0.4	21	61	1.00	7.7	0

*Basal diet consisted of casein—18, lard—20, sucrose—48.9, salt mixture—4, calcium carbonate—1, yeast—6, fortified fish liver oil (Natola)—0.1, and agar—2. Average food intake—4.5 to 5.5 g per day.

methylated molecule, is suggested for these results. An adequate supply of compounds containing labile methyl groups is indispensable in the diet of young rats. The occurrence of hemorrhagic degeneration is evidence of a deficiency of choline *and of the labile methyl supply.* Supplements of choline, methionine and betaine contribute to this reserve. On the other hand, the methyl group of creatine is not available for the synthesis of choline. However, dietary creatine does spare the labile methyl supply and makes available for choline formation those methyl groups which otherwise would be used in the synthesis of required creatine.

Choline may or may not be directly involved in all of the various manifestations of hemorrhagic degeneration. It may be required as such for instance in the formation of choline phospholipids or of acetyl choline and it may function indirectly by increasing the labile methyl supply which is required for methylations in metabolism.[3-6] Creatine, unlike choline, apparently does not supplement the stores of available methyl in the body.

[3] du Vigneaud, V., Chandler, J. B., Moyer, A. W., and Keppel, D. M., *J. Biol. Chem.*, 1939, **131**, 57.

[4] du Vigneaud, V., Chandler, J. B., Cohen, M., and Brown, G. B., *J. Biol. Chem.*, 1940, **134**, 787.

[5] Chandler, J. P., and du Vigneaud, V., *J. Biol. Chem.*, 1940, **135**, 223.

[6] Lewis, H. B., Schultz, J., and Gortner, R. A., Jr., *J. Pharm. and Exp. Ther.*, 1940, **68**, 292.