alcohols block conduction before depolarizing, is referable to the abnormally high activity coefficients of methanol and ethanol in non-aqueous solvents, and hence to the high aqueous mole fractions necessary for narcosis. While the alcohols depolarize at aqueous activities of about 0.15-0.30, other organic substances, of a relatively non-polar nature, depolarize only at activities of about 0.8. Such substances show a surface tension depression similar to that of the alcohols at these elevated thermodynamic activities. The distinction is made between narcosis, which requires a given mole fraction of a substance in the non-aqueous phase of the fiber, and depolarization which requires a given depression of the interfacial tension.

1. Laget, P., Posternak, J. M., and Mangold, R., *Méchanisme de la Narcose, CNRS*, Paris, 1951.

2. Bishop, G. H., *J. Cell. Comp. Physiol.*, 1932, v1, 177.

3. Brink, F., *Nerve Impulse*, Macy Foundation, New York, 1951.

4. Lorente de Nó, R., *Studies from the Rockefeller Institute for Medical Research* 131, 1947.

5. Mellan, I., *Industrial Solvents*, Reinhold, New York, 1950.

6. Fenn, W. O., *J. Cell. Comp. Physiol.*, 1935, v6, 469.

7. Ferguson, J., *Proc. Roy. Soc.*, 1939, vB127, 387.

8. ———, *Méchanisme de la Narcose, CNRS*, Paris, 1951.

9. Butler, J. A. V., and Harrower, P., *Trans. Faraday Soc.*, 1937, v33, 171.

10. Butler, J. A. V., *ibid.*, 1937, v33, 229.

11. Smyth, C. P., and Engel, E. W., *J. Am. Chem. Soc.*, 1929, v51, 2646.

12. Hutchinson, E., *J. Coll. Sci.*, 1948, v3, 219.

13. Höber, R., *Compt. Rend. Lab. Carlsberg*, 1937, v22, 205.

14. Wilbrandt, W., *J. Gen. Physiol.*, 1937, v20, 519.

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Influence of Protein Intake on Magnesium Requirement during Protein Synthesis.* (20813)

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The deleterious effect of magnesium deficiency on protein synthesis was reported by Menaker and Kleiner(1). At a 9% level of protein intake (150 to 160 mg N daily), their rats gained about 40% more in 10 days on a complete ration than on a magnesium-deficient one (30.5 vs 21 g) with virtually isocaloric intake. They concluded that magnesium deficiency is approximately as deleterious to protein synthesis as is potassium deficiency, since Cannon *et al.*(2) had found that rats, on complete rations of 216 mg N daily, gain about 35% more in 10 days (about 46 g vs 34 g) than on an essentially isocaloric potassium-deficient ration. Frost and Sandy (3) recently reported that on 120 mg N daily, rats gain virtually as much on a Mg-deficient as on a complete ration the first few weeks, but in 11 weeks rats on a complete ration gain 25 to 30% more weight (about 90 g vs 70 g) than those on a Mg-deficient one. At this low level of N-intake, their rats on a complete ration gain less in 10 days than Menaker's and Kleiner's rats on a Mg-deficient ration of 150-160 mg N daily. An analysis of these data led the author to test whether the Mg requirement is influenced by the level of protein intake.

Method. 24 female Wistar albino rats, depleted 20-25% below initial 250 g (approx.), were divided into 4 groups. Group 1 received 7 g casein per 100 g basal ration plus complete mineral supplement. Group 2 received same basal ration (7% protein) but Mg-deficient supplement. Group 3 received 14 g casein per 100 g basal ration (14% protein) plus complete mineral supplement. Group 4 received this basal ration (14% protein) but

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TABLE I.
Effect of Magnesium Deficiency on Weight Gain at Different Levels of Protein Intake.

Protein level (%)	Mineral supplement	No. rats	Avg total food consumed (g)	Avg total wt gain*	Gain (g) / Food (g)	Food efficiency ratio†
7	Complete	6	120	14.3 ± 1.7	.119	.95
7	Deficient	6	109	12.3 ± 1.3	.113	
9‡	Complete	4	112	30.5 ± 2.3	.272	.71
9‡	Deficient	4	108	21 ± 1.5	.194	
14	Complete	6	117	36 ± 2.0	.308	.23
14	Deficient	6	80	5.8 ± .7	.072	

* Mean ± stand. error of mean. † Avg total wt gain (deficient)/avg total wt gain (complete). ‡ Menaker and Kleiner(1).

Mg-deficient supplement. Basal ration contained, per 100 g, 7 g (Groups 1 and 2) or 14 g (Groups 3 and 4) purified casein(1), 78 g (Groups 1 and 2) or 71 g (Groups 3 and 4) cane sugar, 7 g corn oil, 2 g U.S.P. cod liver oil, 5 g non-nutritive cellulose fiber, and 1 g mixture of B-complex vitamins in sucrose (2). Four g of complete mineral mixture (Hawk-Oser, Osborne-Mendel) were added to each 100 g basal ration fed Groups 1 and 3. Groups 2 and 4 received an amount of mineral supplement equivalent to that of Groups 1 and 3 but lacking Mg; K_2SO_4 and Na_2CO_3 were substituted in the supplement fed Groups 2 and 4 for the corresponding Mg salts in the supplement fed Groups 1 and 3. These diets were fed 10 days.

Results. Group 3 (14% protein, complete mineral) gained far more in 10 days than Group 1 (7% protein, complete mineral), but Group 4 (14% protein, Mg-deficient) gained considerably less than Group 2 (7% protein, Mg-deficient). Thus, with complete mineral supplement, the rate of protein synthesis (as measured by weight gain) was over twice as great at the higher protein intake as at the lower protein intake. But when Mg was omitted at both levels of protein intake, the rate of protein synthesis was over twice as great at the *lower* level of protein intake as at the higher level of protein intake. At the 7% protein intake level (about 120 mg N daily), the magnesium-deficient ration allowed virtually the same small weight gain as the complete ration, thus confirming for a 10 day period the observations of Frost and Sandy (3). But at the higher level of protein intake (14%)—a moderate one for rat or man—the Mg-deficient ration permitted less than one-fifth the weight gain that the complete ration did. Table I shows the mean weight gain per rat for each group and the amount of basal ration consumed per rat for each group during the 10 day period. Results previously reported(1) for a protein intake level of about 9% are included in the table.

Anorexia appeared in Group 4 (14% protein, Mg-deficient) the fourth day, half of the rats showing marked vasodilatation of the skin of the ears by the sixth day, as first described by McCollum and Orent(4). Group 2 (7% protein, Mg-deficient) showed only a slight but statistically insignificant decrease in food intake and in weight gain.

Comments. Assumptions and limitations of gauging protein synthesis by weight-gain have been noted(1). As far less protein is synthesized on Mg-deficient rations at the 14% level of protein intake than at the 7% level, more Mg is required for normal protein metabolism at the 14% level than at the 7% level.

On a 9% protein intake, a Mg-deficient ration permits more protein synthesis than a complete ration on a 7% protein intake, because lack of protein is the chief limiting factor at the 7% level, enough Mg probably being mobilized from body stores, notably bone, for the slow rate of protein synthesis. On a 9% protein intake, the amount of Mg so mobilized is not sufficient to assure as much protein synthesis as will a complete mineral supplement. The observed results are consistent with the view that at the 9% protein level more Mg is required than at the 7% level. That the worst results are obtained with a Mg-deficient ration at the highest of the 3 protein levels indicates that more Mg is required at the 14% level than at the 7 or

9% levels. If Mg were required merely to form part of the structure of new tissue, at least as much weight would be gained on a 14% protein intake as on a 9% protein intake with Mg-deficient rations.

Comparative studies of mineral supplements, conducted on a 7% protein intake (about 120 mg N daily, the quantity employed by Frost and Sandy(3) in studying Mg-deficiency), may fail to reveal injurious effects of deficient mineral rations, because such effects may only become evident when N-intake is sufficient to assure a normal or optimal rate of protein synthesis.

Summary. Adult protein-depleted rats receiving a complete mineral supplement gain over twice as much in 10 days on a 14% level of protein intake as on a 7% (120 mg N daily) level, but those receiving a Mg-deficient ration gain over twice as much on a 7% level of protein intake as on a 14% level. These results indicate that the Mg requirement for normal protein metabolism increases during protein synthesis as protein intake is increased from 7% to 14% and that the increased Mg requirement is not merely for the structural inclusion of Mg in new tissue. A 7% level of protein intake permits only a markedly subnormal rate of protein synthesis in a previously depleted animal, but 9% and especially 14% levels of protein intake insure more normal rates of protein synthesis and reveal the markedly deleterious effects of magnesium-deficient rations on protein synthesis within 10 days.

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1. Menaker, W., and Kleiner, I. S., PROC. SOC. EXP. BIOL. AND MED., 1952, v81, 377.

2. Cannon, P. R., Frazier, L. E., and Hughes, R. H., *Metabolism,* 1952, v1, 49.

3. Frost, D. V., and Sandy, H. R., PROC. SOC. EXP. BIOL. AND MED., 1953, v83, 102.

4. McCollum, E. V., and Orent, E. R., *J. Biol. Chem.,* 1931, v92, xxx (Proceedings).

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Human Body Lice. IV. Direct Serial Passage of Typhus Rickettsiae by Oral Infection.* (20814)

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In connection with projected studies(1) of mutations in microorganisms which propagate in arthropods, methods for performing serial passages are necessary. This paper describes a simple method for serial passage of rickettsiae in lice and presents the results of its application to experimental studies of typhus.

Materials and methods. The origin and maintenance of our normal louse colony has been described(2). When lice were selected for these experiments, they were insects of the third instar, starved for 18 to 24 hours before the infective meal was offered. Experimental infections were initiated by using yolk sac propagated organisms of the Wilmington and Florida(3) strains of *Rickettsia mooseri* and the E strain(4) of *R. prowazeki.* Infection of lice was accomplished by a chicken skin membrane technic(5), and infected lice were fed daily upon normal rabbits(6).

Serial passage. Two or more infected lice

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