acid-fast bacilli. Cultures and inoculation experiments from positive animals yielded negative results.

These results as a whole appear in accord with those of the French school, who believe they have demonstrated the filtrability of this organism. Lacking as they do cultivability, or the power of producing classical tuberculosis, we feel that the nature of the acid-fast organism in the smears is uncertain as yet.

On the other hand, one of the animals inoculated with the filtrate under different conditions than the rest died between the 6th and 7th month of classical tuberculosis. The lesions involving the abdominal lymph glands, lungs and liver were widespread and unmistakable. Acid-fast bacilli were readily found and on injection again produced classical tuberculosis. A control animal inoculated with the same filtrate as the test animal but under the usual conditions died in five weeks of pneumonia but showed no evidence of tuberculosis. Attempts to repeat this rather noteworthy result are under way.

This is a preliminary report.

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Peptone Hypoglycemia.

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The effect of peptone on blood sugar has been determined by several investigators, but the results obtained have been discordant. Henderson and Underhill[1] in 1911, and Kuriyama[2] in 1917 reported hyperglycemia after intravenous injection of peptone. On the other hand, McGuigan and Ross[3] in 1915 found marked hypoglycemia, while Menten and Manning[4] in 1927 observed hyperglycemia with three samples of Witte's peptone, hypoglycemia with one sample, and no change in blood sugar with another sample.

The experiments to be described were made some time ago, but are now reported in view of Menten and Manning's paper. Our results throw light on the possible cause of the variations in blood sugar obtained after administration of peptone. The experiments recorded differ from those of previous investigators. The diet of the test rabbits was noted and kept constant in view of the relation

of food to the glycogen content of the liver and to the blood sugar level. The rations consisted of alfalfa and oats. The animals were deprived of food but not of water, 24 hours previous to injection. The injections were made subcutaneously. The material was not peptone dissolved in water with saline, but an extract of peptone prepared similarly to insulin.

Twenty grams of peptone of different makes were mixed with 1000 cc. of 70 per cent alcohol containing 0.4 per cent sulphuric acid. The mixture after standing 24 hours was filtered, and the filtrate evaporated at reduced pressure until the odor of alcohol disappeared. The aqueous residue was treated with 95 per cent alcohol, and a brown gelatinous precipitate was obtained. This was separated by filtration and dissolved in a small quantity (10 to 15 cc.) of water (pH 3) acidified with hydrochloric acid. Blood sugar was determined by the method of Schaffer and Hartmann. The injection of the slightly acidified water alone increased the blood sugar but slightly.

HYPOGLYCEMIC EFFECTS OF PEPTONE.

1. Sample: Parke and Davis peptone. Weight of rabbit, 1900 grams.

	Percent of blood sugar
Normal	0.110
1 hour after injection	0.085
3 hours after injection	0.054
5 hours after injection	0.054

2. Sample: Difco peptone. Weight of rabbit, 1550 grams.

Normal	0.092
1 hour after injection	0.098
3 hours after injection	0.079
5 hours after injection	0.072

3. Sample from bottle labelled Meat Peptone. Weight of rabbit, 2250 grams.

	Percent of blood sugar
Normal	0.130
1 hour after injection	0.092
3 hours after injection	0.060
6 hours after injection	0.060

4. Sample: Merck peptone. Weight of rabbit, 1400 grams.

Normal	0.110
1 hour after injection	0.098
3 hours after injection	0.092
5 hours after injection	0.124

The general trend of our results indicates that peptone contains a hypoglycemia-producing substance, the amount of which varies markedly with the commercial source of the peptone. The substance that lowers blood sugar is not formed as a result of the acid-alcohol treatment, for McGuigan and Ross,[3] and Menten and Manning[4] have reported hypoglycemia as the result of intravenous injection of straight peptone. The work of Henderson and Underhill,[1] Kuriyama,[2] Menten and Manning[4] points also to the presence in pep-

tone of a hyperglycemia-producing substance. The contrary findings in the literature may be expected in view of the variability in the composition of peptone. Whether a sample of peptone causes a lowering or heightening of blood sugar may depend upon the predominating presence of the substance producing hyperglycemia or hypoglycemia.

In interpreting results on blood sugar, however, we must always bear in mind the condition of the animal. A low glycogen reserve may alter very markedly the response of the rabbit to blood sugar changes. Adrenalin, which ordinarily produces hyperglycemia, may give no change in blood sugar or a hypoglycemia, depending upon the amount of glycogen in the liver. Infection also influences the blood sugar level.[5] Spontaneous infection with *Bacillus paratyphosus B* may sometimes be found.[6] Stock rabbits may develop symptoms of hypoglycemia due to liver cysts containing *Coccidia oviforme.* Degenerative changes have been observed in the liver in infected rabbits.[6, 7] This damage may affect its glycogen content as well as its function with relation to glycogenesis, glycogenolysis or gluconeogenesis. Since injections of peptone also influence the condition of the liver, it is likely that various samples of this preparation may carry different quantities of the substance producing anatomical and physiological derangement.

[1] Henderson, Y., and Underhill, F. P., *Am. J. Physiol,* 1911, xxviii, 275.

[2] Kuriyama, *J. Biol Chem.,* 1917, xxix, 127.

[3] McGuigan, H., and Ross, E. L., *J. Biol. Chem.,* 1915, xxii, 417.

[4] Menten, M. L., and Manning, H. M., *J. Biol. Chem.,* 1927, lxxxii, 255.

[5] Zeckwer, I. T., and Godell, H. I., *J. Exp. Med.,* 1925, xlii, 43. Levine, V. E., and Kolars, J. J., PROC. SOC. EXP. BIOL. AND MED., 1926, xxiv, 36.

[6] Menten, M. L., and Manning, H. M., *J. Infec. Dis.,* 1925, xxxvii, 400.

[7] Collip, J. B., *J. Biol. Chem.,* 1923, lvii, 163.

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Diet and Tissue Growth. VI. Relation of Age to Renal Injury on Diet Rich in Protein.

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In an attempt to explain the discrepancy between the results reported by different investigators on the relation between dietary protein and renal lesions, Moise and Smith[1] have postulated that