

The inhibition of the *B. paratyphosus* phage by extracts of *B. aertrycke* was evident also when *B. aertrycke* was used as a test organism.

The effect described was observed in numerous tests but the degree of inhibition was somewhat variable. Experiments are under way to determine the optimal conditions required for the reaction. In any event it is essential to observe the course of the reaction over a number of hours. This is so especially in the case of the tests with the anti-*B. paratyphosus* B phage.

It remains to be established why Prausnitz and Burnet failed in their experiment to obtain inhibition of the phage by means of bacterial extracts. Two factors that may possibly serve as an explanation are their use of anti-formin extracts or too short a period of contact of phage and extract.

Particular significance is attached to the inhibition of the anti-*B. paratyphosus* B phage by extracts of *B. aertrycke* since the 2 organisms have the same heat stable components. Experiments along this line seem to offer a general method for the investigation of the specific behavior of the bacteriophage.

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Clinical Manifestations of Variations in Blood Magnesium— Hypomagnesaemia and Hypermagnesaemia.*

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Serles and one of us reported^{1, 2} that, contrary to the usual conceptions, a large part of the magnesium ingested orally as magnesium sulphate is absorbed from the intestine and in normal animals and normal human beings about 40% of the ingested magnesium passes out in the urine in 24 hours, without causing any significant rise in plasma magnesium. The determinations of magnesium were facilitated by the introduction of a modification of Kolthoff's colorimetric method³ for determining the magnesium in the oxalated Ca-free

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¹ Hirschfelder, A. D., and Serles, E. R., *J. Clin. Invest.*, 1932, **11**, 841.

² Hirschfelder, A. D., and Serles, E. R., *J. Pharmacol. and Exp. Therap.*, 1932, **45**, 264.

³ Kolthoff, I. M., *Biochem. Z.*, 1927, **185**, 344.

plasma and in oxalated Ca-free urine, after precipitation of the phosphate by uranyl acetate. We then found that in nephrectomized or nephrotic animals, oral administration of magnesium sulphate caused the plasma magnesium to rise significantly, a single dose bringing it to about 10 mg. per 100 cc. plasma, larger doses to above the coma producing level (which is about 17 mg. per 100 cc.).

Extending these studies to human beings we have obtained similar results.

1. High blood magnesium. This was found especially in nephritics after the administration of Epsom salts. In normal individuals 20-30 gm. $\text{MgSO}_4, 7 \text{H}_2\text{O}$ by mouth does not significantly raise the blood magnesium and about 40% is excreted in the urine in 24 hours. In nephritics less of the ingested Mg is excreted in the urine, and the blood magnesium rises.

20-30 gm. $\text{MgSO}_4, 7 \text{H}_2\text{O}$ administered to 9 patients with acute and chronic glomerulo nephritis raised blood magnesium from near normal to 9.8-11.3 mg. All these patients were definitely more drowsy when at these levels. Two nephritics with blood magnesium 8.9-9.0 without administration of MgSO_4 were definitely drowsy and unresponsive.

Coma is induced in animals when the blood magnesium approaches 17 mg. Mg per 100 cc. Elevation of blood magnesium to about two-thirds of the coma-producing level thus seems to be accompanied by a tendency to somnolence.

It is probable that repeated purgative doses of Epsom salts by mouth can induce coma in nephritis, and that many cases of supposed uremic coma are really magnesium coma induced by Epsom salts. From this coma animals and probably patients can be awakened and their lives prolonged by intravenous calcium chloride.

2. Low blood magnesium (0.9 to 1.37 mg.) was found in 10 cases, all manifesting twitching or convulsions. One of these was parathyroid tetanic, two epilepsy, one cerebral injury, the others acute or chronic nephritis. Three of the latter were given Epsom salts by mouth, blood magnesium rose to 3.3 to 5.8 mg. Mg, twitchings or convulsions disappeared.

There thus seems to be a definite clinical syndrome of twitching or convulsions associated with low blood magnesium.