during the first 10 hours were approximately 430 cu. mm. in 1.0% peptone, 2400 cu. mm. in 5% and 3300 cu. mm. in 10% peptone cultures of *Esch. coli.* A marked immediate increase in oxygen-consumption was observed in 24-hour cultures in 1.0% peptone following the addition of small amounts of concentrated solutions of peptone. This suggests that the decrease in metabolic activities observed as the age of a culture increased was due in part to exhaustion of available food. This suggestion is further supported by the observation that no marked growth or oxygen-consumption was observed under the conditions of these tests when filtrates (adjusted to pH 7.2) of 24 hour or older, 1.0% peptone cultures of *Esch. coli* were lightly seeded with this test-organism. Rapid growth and marked oxygen-consumption was noted under the same conditions when 0.1 milliliter of 10% peptone was added per 0.9 milliliter of the filtrates. These results support the previous observations of Clifton[3] on the closely connected rôles played by the concentrations of foodstuffs, oxidant, and organisms in controlling the metabolic activities of bacteria and indicate that these factors must be considered in any interpretation of the different rates of metabolic activity observed during the growth-cycle of bacteria.

8593 P

Calcium Involvement in Magnesium Deficiency.*

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The existence of an intimate relationship between the metabolism of calcium and magnesium in the animal body has long been postulated. In harmony with this hypothesis the writers have accumulated considerable evidence which points to a calcium involvement in the syndrome of magnesium deficiency.

Certain aspects of such an involvement have already been pointed out by Kruse, Schmidt and McCollum[1] and by Orent, Kruse

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1 Kruse, H. D., Schmidt, M. M., and McCollum, E. V., *J. Biol. Chem.*, 1934, **106**, 553.

and McCollum[2] who observed that animals reared on a magnesium deficient diet showed a marked retention of calcium and an abnormally high percentage content of calcium in their bones.

The phases of the involvement observed by the writers are that there is an accumulation of calcium in the viscera of the body, particularly in the kidneys, and that the magnesium level of the diet at which deficiency symptoms appear is controlled by the dietary level of calcium.

A greater amount of magnesium is required to prevent the development of deficiency symptoms as the calcium of the diet is increased. This is illustrated by the performance of rats reared on diets containing the mineral composition given in Table I.

TABLE I.
Mineral Composition of High and Low Calcium Diets.

	Calcium %	Phosphorus %	Magnesium mg. %	Ca/P
High Calcium diet	1.16	0.62	5	1.85
Low Calcium diet	0.39	0.33	5	1.12

The animals reared on the high calcium diet developed all the features common to magnesium deprivation such as hyperemia, hyperirritability, and cachexia. A large percentage of these animals died from convulsive seizures. Those that survived developed a greasy unkempt appearance, became emaciated, and in other ways showed signs of malnutrition. At the same magnesium level, the rats on the low calcium diet were maintained in a normal state of health and vigor. The females that were bred went through a successful gestation period and gave birth to young of normal weight.

The accumulation of calcium in the viscera of the body of magnesium deficient rats is shown in Table II.

TABLE II.
Effect of Magnesium Deprivation on Magnesium and Calcium Content of Rat Viscera.*

	Control Animals						Deficient Animals					
	Magnesium			Calcium			Magnesium			Calcium		
Tissue	No.	Range	Mean	No.	Range	Mean	No.	Range	Mean	No.	Range	Mean
Heart	8	22.6-29.1	25.7	7	3.6- 6.1	5.15	6	21.2-26.5	23.4	5	6.1- 14.7	8.0
Muscle	17	24.6-32.6	29.4	8	4.8- 7.9	6.35	25	15.2-32.0	25.5	10	8.5- 15.2	10.1
Kidney	17	18.0-25.3	22.2	7	6.9-12.4	9.4	22	16.9-25.3	20.7	10	48 5-232	141

*Analytical figures are in mg. of element per 100 gm. of wet tissue.

From the table it appears that the magnesium content of the soft tissues is not greatly altered by the magnesium deficiency in

[2] Orent, E. R., Kruse, H. D., and McCollum, E. V., *J. Biol. Chem.*, 1934, **106**, 573.

the diet. The calcium content, on the other hand, is definitely increased. In muscle and heart, the average calcium increase is moderate, being only about 60%. In the kidneys, however, the accumulation is enormous, amounting to a 15 fold average increase. This tremendous increase of calcium in the kidneys probably indicates that metastic calcification forms part of the degenerative process in the kidneys induced by magnesium deprivation. Cramer[3] and the writers have observed the presence of multiple casts, which stain purple with hæmatoxylin and which are probably of a calcareous nature in the kidney sections of deficient rats.

8594 C

Protective Action of Specific Serum Against Experimental Vaccinia in Rabbits.

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It is well established that in certain virus diseases such as poliomyelitis immune serum exercises little or no effect on the progress of the infection, even when administered in large doses several days before the usual onset of symptoms.[1] It is also of limited prophylactic value when administered before intranasal instillation with virus.[2] The therapeutic limitations of immune serum in virus diseases generally may be explained by the fact that cytotropic viruses once established within tissue cells are inaccessible to immune bodies in the plasma. Since viruses are primarily intracellular, rather than intercellular parasites, it would appear that the effectiveness of humoral antibodies is likely to be limited largely to infections in which the virus in transported to distant susceptible tissues by the blood stream, or tissue fluids. In diseases such as poliomyelitis, in which the virus is nerve-transmitted throughout and may gain admission to susceptible tissue without necessarily passing a barrier of immune plasma[3] the prospects of establishing significant protection against infection by the natural or intranasal

3 Cramer, W., *Lancet*, 1932, **223**, 174.

1 Schultz, E. W., and Gebhardt, L. P., *J. Pediat.*, 1935, **6**, 615.

2 Schultz, E. W., and Gebhardt, L. P., *J. Pediat.*, 1935, **7**, 332.

3 Schultz, E. W., and Gebhardt, L. P., PROC. SOC. EXP. BIOL. AND MED., 1934, **31**, 728.