from erythrocytes of species susceptible to the virus, a polysaccharidelike substance which does compete with apple pectin for the virus acting as a hemagglutinin. Therefore, a search for a substance in *E. coli* which might antagonize the protective action of citrus pectin on this species seemed advisable. A number of attempts to demonstrate such a substance have been made, but none has succeeded. The testing procedure employed for this was to add the various samples to the basal medium plus citrus pectin (100 mg per 6 cc) and then to inoculate with 10^8 young cells and with 10^9 phage particles. Incubation of the mixtures for 20 hours should reveal an active preparation by failure of the bacteria to grow. This should follow logically because if the pectin were competing with a cell receptor, increasing the total concentration of the latter in the system should nullify the action of the pectin. The concentration of this receptor in the system is fixed by the number of bacteria, but it might be increased by adding extracts of the substance from the cells. Extracts were prepared from 20-hour cultures of *E. coli* by treating the cells with weak alkali, by grinding them with sand, by heating to 70° or 100°, by ultraviolet light irradiation, and by plasmolysis with ether or with chloroform. None was found able to overcome the antiviral action of citrus pectin. Needless to say, the influenza virus receptor substance extracted from human erythrocytes likewise was ineffective.

Summary. Citrus pectin was found to allow growth of *E. coli* in the presence of an excess of bacteriophage. The pectin was nontoxic to the bacteria, and was not virucidal. The action of the pectin was found not to be the prevention of attachment of the virus to the cell, and indeed, it did not inhibit the multiplication of the phage appreciably. It did, however, protect the cells from lysis, and hence in its presence a phenomenon similar to that seen with lysogenic strains was observed. Gum acacia and starch, representatives of other classes of polysaccharides, were ineffective. No substance could be demonstrated in the cells which was antagonistic to citrus pectin in the way that an influenza virus substrate in erythrocytes was antagonistic to apple pectin.

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Effect of Thyro-Parathyroidectomy upon the Blood and Plasma Volumes of the Rat.

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There have been few studies of blood and plasma volumes in myxedema or experimental hypothyroidism, none on the rat. Thompson[1] in an early study found the plasma volume in myxedematous patients to be 30% below normal, and also found that the values returned to normal upon treatment with thyroid extract. Holböll[2] likewise found the blood and plasma volume reduced in myxedema. In the most recent study, Gibson and Harris[3] studied 7 myxedematous patients. They found that

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1 Thompson, W. O., *J. Clin. Invest.*, 1926, **2**, 477.

2 Holböll, S. A., *Acta med. Scandinav.*, 1930, **73**, 538.

3 Gibson, J. G., 2nd, and Harris, A. W., *J. Clin. Invest.*, 1939, **18**, 59.

TABLE I.

Effect of Thyro-parathyroidectomy upon the Blood and Plasma Volumes of the Rat.

Final wt, g	Heart wt, g	Wintrobe hematocrit	Blood Vol. Exper., cc	Blood Vol. Norm., cc	Blood Vol. % Norm.*	Plasma Vol. Exper., cc	Plasma Vol. Norm., cc	Plasma Vol. % Norm.	Erythrocyte Vol. Exper., cc	Erythrocyte Vol. Norm., cc	Erythrocyte Vol. % Norm.
180	650	44.5	11.3	12.3	92	6.0	7.0	86	5.3	5.4	98
150	490	43.0	9.8	10.7	92	5.4	6.1	90	4.4	4.6	97
140	481	36.5	8.2	10.2	80	5.0	5.8	86	3.2	4.4	73
127	488					4.1	5.4	76			
120	510	35.0	9.4	9.1	103	5.9	5.2	113	3.5	3.9	89
159	500	41.0	9.6	11.2	86	5.5	6.4	86	4.1	4.8	86
156	553	39.0	10.5	11.1	94	6.2	6.3	98	4.3	4.8	89
174	546	37.0	10.6	12.0	88	6.5	6.8	96	4.1	5.2	79
206	620					6.9	7.6	91			
186	604	39.5	11.5	12.6	91	6.8	7.1	96	4.8	5.5	86
186	651	44.5	11.0	12.6	87	6.3	7.1	89	4.8	5.5	87
238	856	44.0	15.4	15.1	102	8.4	8.5	99	7.0	6.6	106
188	570	41.0	12.0	12.7	94	6.9	7.2	96	5.1	5.5	93
202	610	41.5	11.8	13.4	88	6.7	7.5	89	5.1	5.9	87
148	478	38.0	9.4	10.6	89	5.6	6.0	93	3.7	4.6	80
176	520	38.0	11.8	12.0	98	7.1	6.8	104	4.7	5.2	90
Mean					92			93			89
					± 1.6			± 2.13			± 2.24

* The normal blood volume, considered as 100%, has a standard deviation of the mean of 1.38%. The normal plasma volume has a standard deviation of the mean of 1.6%. The standard deviation of the differences, computed from these data, indicate that the differences are significant.

the blood volume was only 15% below normal, and attributed the lower results of earlier authors to errors in the method of blood volume determination used.

Methods. This study utilized 16 male rats, ranging in weight from 150 to 180 g at the start. Thyro-parathyroidectomy was performed, under ether anesthesia, by blunt dissection and cauterization of the thyroid bed. Following this, an interval of 3 weeks elapsed, during which the rats were undisturbed and were fed upon stock diet. At the end of 3 weeks, the rats had failed to gain weight in accordance with the normal growth curve. Blood and plasma volume determinations were performed according to the hemoglobin dye method previously described.[4] Gross postmortem examination showed no regeneration of thyroid tissue at the site of operation. The heart weights were diminished in accordance with previous experience upon thyro-parathyroidectomized rats.[5]

Results. Plasma volumes were obtained in 16 rats, and blood volumes were obtained in 14 of these. There was a mean reduction of plasma volume, erythrocyte volume and total blood volume of approximately 10%, when compared with normal standards for rats of the same weight, as previously established.[4] (Table I).

4 Lippman, R. W., PROC. SOC. EXP. BIOL. AND MED., 1947, **66**, 188.

5 Addis, T., Karnofsky, D., Lew, W., and Poo, L. J., *J. Biol. Chem.*, 1938, **124**, 33.

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Protective Action of Dietary Cholesterol in Experimental Thyrotoxicosis.*

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Experiments were made to investigate further the well known effects of hyperthyroidism on cholesterol metabolism.[1] An unexpected result of these studies was the observation that a reverse relationship also existed, *i.e.* that high dietary cholesterol modified the toxic effects of high doses of thyroid hormone, in particular, as far as survival time and body weight changes were concerned. These findings are reported in the present communication.

Experimental Procedure. Normal rats were fed *ad libitum* this laboratory's stock diet, slightly modified to contain more yeast, in order to assure an adequate supply of the vitamin B complex.† In addition, thyroid powder was mixed with the diet at concentrations corresponding to 0.4-0.8% thyroid U.S.P. One group of rats (T) received the high thyroid diet as described, a second group (T + C) was fed an identical diet, except that it contained in addition, 1% cholesterol and 0.5% bile salt. The cholesterol was dissolved in cottonseed oil, replacing a corresponding amount of the oil in the diet. The bile salt was added to increase absorption of cholesterol.[2] The rats were about 9 months (Experiment 1) and 38 days (Experiment 2) old respectively at onset of the feeding periods. Litter mates were distributed evenly between the two groups. Body weights were measured once weekly, and the survival time

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1 Peters, J. P., and van Slyke, D. D., *Quant. Clinical Chem., Interpretations I,* 2nd Edition, 1946, 497.

† Modified stock diet: 328 g whole wheat flour; 327 g ground oats; 100 g skimmed milk powder; 30 g alfalfa; 95 g yeast, Anheuser-Busch strain G; 80 g cottonseed oil; 20 g fortified cottonseed oil, containing 2500 I.U. vitamin A/g and 400 I.U. vitamin D_2/g; 5 g NaCl; 5 g $CaCO_3$.

2 Schoenheimer, R., *Biochem. Z.*, 1924, **147**, 258.