

recovered 57% of his testicular heterografts subsequent to persistence for 6 to 45 days in subcutaneous, intramuscular and intraperitoneal positions, it appears that the transplants were more rapidly eliminated from the anterior chambers than from other sites. In no case did the heterotransplanted gonads in the anterior chambers maintain secretory processes in the male accessory glands or prevent the loss of oestrus cycles in the female hosts. Castration changes in the pars distalis of the hypophysis were not retarded by testicular heterotransplants.

Sixteen vesicular glands and 12 ventral prostatic lobes from 30-day-old mice were transplanted to the eyes of normal adult male rats. After persistence for 20 days, only fibrotic nodules were recovered. Previous to the twentieth day, areas of normal-staining donor tissue occasionally could be identified, but there was no histological evidence of the ingrowth of blood vessels from the host. In no instance did these heterotransplanted accessory glands display evidence of secretion. The epithelium of the transplanted vesicular glands was low and void of secretory granules. Histological and cytological preparations indicated the loss of secretory function in the epithelial cells of the prostatic transplants.

Summary. These observations indicate that heteroplastic transplants of gonads and accessory genital glands from albino mice do not become functionally incorporated in the anterior chamber of the eyes of albino rats. It is believed that the serological and phagocytic reactions of the host produce more rapid deterioration of the heterotransplant in the anterior chamber than occurs in other transplantation sites previously studied.

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Effect of Pneumococcus Type III Specific Polysaccharide on Sedimentation of Blood Cells.

W. J. NUNGESTER AND LOUISE FORDHAM KLEIN.

From the Hygienic Laboratory, University of Michigan.

During the course of experiments with the specific polysaccharide prepared from a type III strain of pneumococcus by the method of Avery, Kendall, and Scherp,¹ it was noted that this material greatly increased the sedimentation rate of citrated human blood.

¹ Heidelberger, M., Kendall, F. E., and Scherp, H. W., *J. Exp. Med.*, 1936, **64**, 559.

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This initial finding has been confirmed, and additional observations have been made, which we desire to present in a preliminary paper.

The effect of the S III on the sedimentation rate of blood cells can be demonstrated with heparinized or defibrinated blood, as well as with citrated blood. The effect of concentration of the S III on the sedimentation rate of citrated human blood is indicated by the data in Table I. The mixtures of blood and S III were drawn up in tubes of 2 mm. bore to a height of 200 mm., and the sedimentation read as the height of the clear plasma.

TABLE I.

% concentration of S III	0	.025	.05	.075	.1	.3	1.0%
mm. sedimentation in 60 min.	3	7	21	39	74	174	174 mm.

A carbohydrate substance isolated from a mucoid variant of *B. coli* by C. Lawrence of this laboratory had a similar effect in increasing the sedimentation rate of blood cells. Gelatin acted in the same way, as did gastric mucin, but to a degree much less pronounced than did the specific polysaccharide of the pneumococcus. Glucose in concentrations up to 1% had no appreciable effect on the phenomenon in question.

When mixtures of the S III of the pneumococcus and homologous antiserum are allowed to stand for 15 minutes, and are then added to blood, the increase in sedimentation rate is markedly diminished, as is shown in Table II.

TABLE II.

	Type III Antiserum	Normal Serum	Control no S III
mm. sedimentation in 60 min.	36	74	4

This effect is type-specific, antisera of heterologous types having no effect.

When 1 mg. of the specific polysaccharide was mixed with 1 cc. of blood, the color of the mixture darkened sooner than the control. On shaking the darkened blood, the color was restored to a bright red, only to darken again before the control. The effect of the carbohydrate on the oxygen-carrying capacity of the blood is being investigated.

Pneumonia is a disease characterized by an abnormally high sedimentation rate. It is also known² that the specific polysaccharide may circulate in the blood stream during the course of the disease.

² Dochez, A. R., and Avery, O. T., *J. Exp. Med.*, 1917, **26**, 477.

Therefore it seems that the observation here reported may aid in furnishing at least part of the mechanism for the high sedimentation rate seen in pneumonia.

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Effect of Phage on Electrokinetic Potential of Susceptible Cells.*

A. P. KRUEGER AND J. H. MUNDELL.

From the Department of Bacteriology, University of California, Berkeley.

Earlier work^{1, 2} has served to establish the following essential facts concerning the phage-bacterium reaction:

A. Phage-production is conditioned by bacterial growth.

B. There is at all times a normal distribution of phage between susceptible cells and the surrounding medium providing the cells are alive, *i. e.*,

$$\frac{\text{intracellular phage per cell}}{\text{free phage}} = K$$

C. Lysis of bacteria depends upon the attainment of a critical ratio of phage to bacteria. In our experiments this threshold is approximately 100 activity units³ per bacterium.

While the above relationships are significant for the development of a rational mechanism of phage action on bacteria⁴ and have been proved to apply to more than one organism and the corresponding phage,⁵ they give no evidence as to how phage induces cellular dissolution. Apparently phage does not measurably alter the normal bacterial growth-rate or the rate of cellular metabolism;^{4, 6, 7} it may or may not bring about swelling of susceptible bacteria just before lysis begins. Bronfenbrenner⁸ has found that phage produces hydrolytic cleavage of bacterial proteins although

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¹ Krueger, A. P., and Northrop, J. H., *J. Gen. Physiol.*, 1930, **14**, 223.

² Krueger, A. P., *J. Gen. Physiol.*, 1931, **14**, 493.

³ Krueger, A. P., *J. Gen. Physiol.*, 1930, **13**, 557.

⁴ Krueger, A. P., *Physiol. Reviews*, 1936, **16**, 129.

⁵ Clifton, C. E., and Morrow, G., *J. Bact.*, 1936, **31**, 441.

⁶ Eaton, M. D., *J. Bact.*, 1931, **21**, 143.

⁷ Hallauer, C., *Centralbl. f. Bakt., I. Orig.*, 1933, **130**, 194.

⁸ Bronfenbrenner, J., Muckenfuss, R. S., and Hetler, D. M., *Am. J. Path.*, 1927, **3**, 562.