For a further substantiation of this, an analysis of covariance of the logarithms of body weight and glutathione content was conducted, the results being presented in Table II.

It should be noted that the F values of the logarithms of body weight and of the logarithms of glutathione content are 15.14 and 60.41 respectively, the corresponding 1% point being 3.98, indicating highly significant differences between the means of the breeds with respect to these 2 variables. The difference in glutathione shows a considerably higher probability of significance than does body weight. Furthermore, a high correlation exists between the two, the fact that the correlation for the total is less than for either between or within breeds, being undoubtedly due to the discarding of fourth place decimals.

However, the significant fact apparent from the table is that while the regression of glutathione on body weight is only 0.902 within breeds, it is 1.829 between means of breeds. This indicates that for every unit of change in the logarithms of body weight within breeds, an increase of only 0.902 obtains in the logarithm of glutathione, while for every unit increase of logarithm of body weight between breeds, a change of 1.829 occurs in the logarithm of glutathione content. The inevitable conclusion is that each of the breeds has its characteristic glutathione content as well as a characteristic rate of change of this factor, which is correlated with the definitive adult size of the 4 breeds studied.

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Streptococcus Anticoagulant.*

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It was shown by Neter and Witebsky[1] that many bacterial species which produce no demonstrable fibrinolysin in veal-infusion broth do produce fibrinolytic factors if grown in the same medium plus 0.4-2% glucose. They found that in this glucose broth many fibrin-

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[1] Neter, E., and Witebsky, E., PROC. SOC. EXP. BIOL. AND MED.,1936, **34**, 549, 858.

olytic bacteria often produce 2 fibrinolysins; *S. hemolyticus,* for example, producing: (a) the highly specific Tillett-Garner fibrinolytic enzyme and (b) a hitherto undescribed, relatively non-specific lytic factor. The new lytic factor prevents the coagulation of human plasma, a property rarely demonstrable with the Tillett-Garner enzyme.

We have repeated their work, using the isolated-fibrin technic[2] in place of their relatively crude plasma-clot technic. Confirming their results, we have found that many apparently non-fibrinolytic strains of *S. hemolyticus* will produce anticoagulants if grown in veal-infusion broth plus 0.4% glucose. Demonstrably fibrinolytic strains produce this anticoagulant in addition to a normal amount of the routine Tillett-Garner fibrinolysin. Unlike the fibrinolysin, the anticoagulant is not specific for human fibrin; but will also prevent the clotting of isolated fibrinogen-thrombin-complex from rabbit, sheep, cow and domestic swine. The anticoagulant is not neutralized with concentrations of commercial streptococcal antiserum sufficient to neutralize the specific fibrinolysin. The anticoagulant and fibrinolysin are apparently independent variables in different streptococcal strains.

Chemical differences between the anticoagulant and the routine fibrinolysin are demonstrable by the enzyme-concentration technic of Tillett and Garner. From 24-hour glucose-broth cultures of relatively high anticoagulative titers, purified fibrinolysin is obtained by alcohol (75%, ice-cold) precipitation. Even in a 10-fold concentration this purified fibrinolysin is without demonstrable anticoagulating effects. The anticoagulant remains in solution in the supernatant alcohol, from which it can be recovered by evaporation. The anticoagulant thus recovered is thermostable. It resists heating to 100°C. for 30 minutes. The purified fibrinolysin is destroyed quantitatively if heated to 60° C. for 30 minutes.

There is as yet no evidence that the streptococcal anticoagulant produced in glucose broth is of clinical interest. The fibrinolysin of Tillett and Garner, however, has been demonstrated in clinical lesions.[3]

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[2] Tillett, W. S., and Garner, R. L., *J. Exp. Med.*, 1933, **58**, 495.

[3] Neter, E., and Witebsky, E., *J. Bact.*, 1936, **31**, 77.