cautions were taken. Also, successive rows throughout the experimental dish were similarly affected while the region toward the cathode remained alive in all plants.

The extent of the dead region increased with greater current density; also with greater duration of exposure. Larger pieces were more susceptible than small, *i. e.*, the percentage dead region/plant size was greater. No difference was detected in quantity of effect between plants oriented in opposite directions; that is, apex and base were equally susceptible when turned toward the anode. Regeneration did not, of course, occur toward the anode, but was not absent elsewhere. As a result new growths were more frequently seen on plants turned with apex toward the cathode.

This report is based on 116 plants which were subjected to direct currents of approximately ½ to 6δ for a period of time up to 350 hours. The dead region comprised 5 to 63% of the length of the thallus. Large plants (about 6 cm. long) showed about 20% more injury than small ones (1 cm. or less) for the same duration and density of current.

Perhaps some light may be thrown by these findings on those cases cited[1, 2, 3] in which the polarity of growth is affected by electricity. (It is not intended to infer, however, that toxic action at one pole is necessarily responsible there.)

8350 C

Evidence for the Presence of a Diffusible Organic Substance in Blood Which Accelerates Blood Clotting.

CLARENCE E. LARSON AND DAVID M. GREENBERG.

From the Division of Biochemistry, University of California Medical School, Berkeley.

Dialysis of blood plasma, it has been presumed, removes only one component essential to the blood clotting mechanism, namely, calcium. However, during the course of certain experiments in which plasma was very thoroughly dialyzed, the authors observed that when the plasma proteins were redissolved in a saline solution containing an adequate amount of calcium, no clot was formed for 24 or 48 hours. If to this artificial plasma there is added a small portion of the ultrafiltrate of whole blood or serum, the formation of a firm clot is induced within an hour or less.

This observation seems to indicate the presence of a dialyzable substance in the blood other than calcium ion which is concerned in the blood clotting process. The substance appears to be an organic compound since the power of accelerating blood clotting is lost if the serum ultrafiltrates are charred.

Either citrated or oxalated plasma may be used to demonstrate this phenomenon. However, a thorough dialysis of the plasma is required, ordinary dialysis against tap water not being sufficient. In the present experiments the dialysis was carried out in a rocking dialyzer[1] at the temperature of 5° against running distilled water. After about 2 to 3 days of dialysis, an artificial plasma is prepared by adding sodium chloride, sodium bicarbonate, sodium phosphate, magnesium chloride, and glucose in the amounts required to produce the concentration found in blood. The pH is adjusted to 7.4 by bubbling in CO_2. Calcium is not added until it is desired to initiate a clotting experiment. The plasma proteins disperse completely in the above menstrum, giving a solution which is no more turbid than the original plasma.

If properly dialyzed, such an artificial plasma shows no sign of clotting for 24 hours or longer after calcium has been added. However, on the addition of serum ultrafiltrate, a firm clot is formed in from 30 minutes to 1 hour. The amount of ultrafiltrate required is small, as little as 0.1 cc. in 5 cc. of artificial plasma being sufficient to reduce the clotting time to about 2 hours. Less than this proportion gives indefinite results.

This phenomenon is not limited to any particular animal species. It was demonstrated by the authors on plasma from human, dog, sheep, beef, and chicken blood. Neither is the substance species specific since an ultrafiltrate from dog serum will accelerate the clotting of dialyzed human plasma and vice versa.

The results of tests to characterize the clot accelerating substance in the ultrafiltrate are given in Table I.

TABLE I.
Experiments on the Accelerating Effect of Serum Ultrafiltrates on Blood Clotting.

Experimental Procedure	Clotting Produced
Addition of untreated serum ultrafiltrates	+
After boiling ultrafiltrate for 45 minutes	+
" charring ultrafiltrate	—
" adsorption with permutit	+
" extensive bacterial contamination	+

+ indicates acceleration of clotting.
— indicates no acceleration of clotting.

[1] Kunitz, M., and Simms, H. S., *J. Gen. Physiol.*, 1928, **11**, 641.

From the table it is seen that of the procedures tried, only heating of the ultrafiltrate to charring destroyed the accelerating effect on the clotting process.

The substances known to influence blood clotting which might be removed by dialysis are sulfhydryl compounds[2] and perhaps cephalin, the latter possibly by adsorption on the walls of the dialyzing membrane. Tests made to determine if these materials were in any way concerned gave negative results. Addition of sulfhydryl compounds in the form of cysteine and thioglycollic acid and of an ether extract of brain to supply cephalin did not produce a reduction in the clotting time of the artificial plasma.

8351 C

Fibrinolytic Titer of Scarlatinal Streptococci.*

FRIEDA H. FRASER AND R. R. MADISON. (Introduced by W. H. Manwaring.)

From the Connaught Laboratories, University of Toronto, and the Laboratory of Bacteriology and Experimental Pathology, Stanford University, California.

Applying the Tillett-Garner plasma-clot technic[1] with rate of thrombolysis as the quantitative index, Dack, Woolpert and Hoyne[2] found a slight correlation between thrombolytic titer and clinical severity of scarlet fever. At the time of the publication of their paper a similar study of scarlatinal streptococci was in progress in our laboratories We had selected the more delicate isolated fibrin-clot technic, however, with serial-dilution methods of determining lytic titer.

The 60 strains of *S. scarlatinae* used in this study were originally isolated in The Connaught Laboratories, University of Toronto. With 43 of these strains Dick-toxin titrations have been made by one of us (F). The fibrinolytic titrations were made in Stanford University (M). Both series of titrations are summarized in Table I.

From Table I it is seen that there is a slight correlation between toxin-titer and fibrinolysin-titer with scarlatinal streptococci. The

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[2] Carr, L. J., and Foote, F. S., *Arch. Surg.*, 1934, **29**, 227.

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[1] Tillett, W. S., and Garner, R. L., *J. Exp. Med.*, 1933, **58**, 485.

[2] Dack, G. M., Woolpert, O. C., and Hoyne, A. L., PROC. SOC. EXP. BIOL. AND MED., 1935, **32**, 1431.