The results of a considerable number of experiments clearly show that the products of injury liberated in an area of acute inflammation are *per se* capable of inducing in a normal dog a prompt leukocytosis to a degree reasonably comparable with that seen in the animal serving as source of the exudative material. Furthermore, some preliminary observations indicate that exudates from animals with marked leukopenia tend to contain a minimal amount of what might now be appropriately termed *leukocytosis-promoting factor*.

The effect on the leukocyte level of a dog manifested by the intravascular injection of an exudate transcends, as a rule, the maximum rise occurring during the rhythmical leukocytic variations. During the period of an experiment, *i.e.,* 6 to 8 hours, the maximum increase in leukocytes of several normal dogs averaged 23.8 per cent. This therefore roughly indicates that there is, as a result of a single injection of exudate, a threefold increase in the number of circulating leukocytes.

To extend the observations, dog serum, sterile broth, large doses of leukotaxine, a broth culture of an exudate and finally a culture of killed staphylococcus *aureus* were injected into the circulating blood of normal animals and of dogs which on other days had received exudates. A leukocytosis invariably failed to develop in these experiments.

For the following reasons it is improbable that the leukocytosis-promoting effect of exudates can be directly referred to the irritant *per se* or any of its derivatives:

(1) Exudates obtained by a variety of unrelated irritants produced in the blood stream an essentially similar effect on the level of circulating leukocytes. Turpentine incubated in serum for varying lengths of time failed to induce a leukocytosis when introduced into the blood stream. These observations, however, do not preclude the possibility that derivatives of turpentine formed in the exudate may still not be responsible for the cellular response. On the other hand, the variety of irritants employed, as well as the fact that a number of unrelated irritating substances fail when injected into the circulation to increase promptly the leukocytic level, suggest that the irritant *per se* bears no direct relation to the leukocytosis-promoting effect of exudates.

(2) The introduction into the circulating blood stream of exudative material obtained as a result of physical injury (*e.g.,* in the form of a severe burn) induces a state of leukocytosis. Such observations clearly indicate that it is unnecessary to refer the leukocytosis-promoting effect of exudate to either the presence of a chemical irritant or any of its derivatives.

The leukocytosis-promoting factor of exudate is thermolabile. Heating the exudate at 60° C. for several hours inactivates it. It is in large part indiffusible, failing to dialyze through a Cellophane membrane. The effect of the factor seems to be primarily on the bone marrow, producing an outpouring of granulocytes into the circulation. Histamine, adenosine, nucleic acid, all fail to reproduce the same pattern of reaction as does an exudate. The details of all these observations will form the subject of a separate communication to be published elsewhere. Further studies are now in progress in an endeavor to identify the nature of the leukocytosis-promoting factor which seems to offer an explanation for the mechanism of leukocytosis accompanying inflammation.

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GLYCOGEN IN SWEET CORN

IT has previously been shown that certain tissue extracts exercise specific effects on the crystallization patterns of cupric chloride, and that the specific oat pattern is dependent on the presence of a polysaccharide in the extract.[1] It has since been found that there is a corresponding polysaccharide in sweet corn, which is essential for the production of the specific corn pattern. This polysaccharide is apparently glycogen.

Though glycogen has been found in a number of lower members of the plant kingdom—yeasts, bacteria, fungi, etc.—it has not previously been reported in any of the higher plants. The designation of a polysaccharide from a plant source as glycogen is rendered uncertain because certain dextrins have properties very similar to those of glycogen. The only striking difference between glycogen and some of the erythrodextrins is the fact that an aqueous solution of glycogen is opalescent, whereas dextrin solutions are usually clear. Hence it is insufficient to show that a polysaccharide exhibits all the usual characteristics of glycogen, but in addition it must be shown specifically that it is not a dextrin or a mixture of dextrins.

The corn polysaccharide has all the properties commonly associated with glycogen. Its aqueous solution is opalescent, it is resistant to the action of hot alkali, and with acids it is hydrolyzed quantitatively to glucose. Its specific optical rotation is + 188° with sodium D light. When iodine is added to its aqueous solution a red-brown color is produced that fades when warmed and reappears when cooled. An erythrodextrin can be prepared whose properties differ essentially from these only in the lack of opalescence of its solution.

The glycogen and dextrin can be easily differentiated, however, by their effect on the cupric chloride crystal patterns. That of the corn glycogen is totally indis-

[1] Morris and Morris, *Jour. Phys. Chem.,* 43: 623, 1939.

tinguishable from those of the animal glycogens, whereas the dextrin pattern is entirely different.

The action of malt diastase on corn glycogen has been studied, since Glock[2] has shown that glycogen is hydrolyzed by the diastases at a much slower rate than are the starches. The corn glycogen has been compared with starches, with the erythrodextrin mentioned above and with glycogen from other sources. Table 1 shows the fraction of each polysaccharide hydrolyzed after one hour under identical conditions. The amount of hydrolysis was measured by determination of the reduction of the solution by Hanes method[3] calculated as maltose.

TABLE 1

Polysaccharide	Source	Per cent. hydrolyzed
Glycogen	*Cysticercus fasciolaris*[4]	10
Glycogen	Oyster	15
Glycogen	Yeast	31
Glycogen	Sweet corn	32
Erythrodextrin	Corn starch	50
Starch	Rice	60
Starch	Corn	65

It is evident that the corn glycogen is hydrolyzed at a rate much lower than that of the starches, higher than that of the animal glycogens, but nearly identical with that of yeast glycogen.

PREPARATION

The corn is soaked for a day or two, extracted with water, and the extract boiled and filtered. Two volumes of glacial acetic acid are added,[5] and the precipitated starch either centrifuged or allowed to settle. The acetic acid concentration of the supernatant solution is increased to 75 per cent.,[6] whereupon the glycogen precipitates. The starch should be redissolved and reprecipitated several times to increase the yield of glycogen, but the separations eventually become less efficient, since both starch and glycogen apparently combine with the acid to form more soluble compounds. Purification of the glycogen can be accomplished by the usual methods.

On one occasion 3.9 gm of glycogen were obtained from 30 gm of Golden Bantam sweet corn. The quantity in one or two kinds of field corn investigated was negligible.

SUMMARY

A polysaccharide apparently identical with glycogen has been extracted from sweet corn.

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SCIENTIFIC APPARATUS AND LABORATORY METHODS

A METHOD FOR THE CONTINUOUS RECORDING OF GASTRIC pH IN SITU

A METHOD has been developed for the determination and the continuous recording of gastric pH *in situ*. To obtain accurate results it was found necessary to keep the silver-silver chloride glass electrode isolated from the gastric mucosa. This was accomplished by a glass or bakelite guard or by a small balloon attached above the electrode. The wire from this electrode was shielded and encased in a Levine tube. In order to mix the gastric contents and to obtain samples another Levine tube was bound to that containing the electrode lead in such a way that the tip of this second tube extended a few centimeters beyond the electrode. With the exception of the opening adjacent to the electrode all other openings of this second tube were covered with balloon rubber. Contact with the saline silver-silver chloride reference electrode was made either on the skin or by means of a thread placed in the aspirating Levine tube. The electrode leads were connected through a Beckman pH meter to a recording potentiometer.

In order to insure accurate recording, certain precautions had to be observed. The exact position of the glass electrode in the stomach was determined by fluoroscopic observation. The gastric contents were constantly mixed by a continuous pump which alternately withdrew and reinjected 25–50 cc of gastric contents at 30-second intervals. By these technical refinements the wide variations reported by Eyerly and Breuhaus[1] between the values obtained by the use of the gastric electrode and those determined on the aspirated speci-

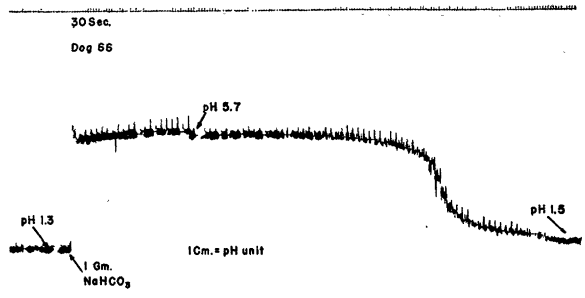

FIG. 1. Changes in gastric pH following administration of 1 gram of sodium bicarbonate in a dog of 17 kilograms, anesthetized with morphine and chloralose.

[2] G. E. Glock, *Biochem. Jour.*, 30: 1386, 1936.
[3] C. S. Hanes, *Biochem. Jour.*, 23: 99, 1929.
[4] The cysticercus glycogen was very kindly furnished by Dr. L. Frank Salisbury, of the Department of Chemistry, Yale University.
[5] *Cf.*, "Methods of Analysis of the Association of Official Agricultural Chemists," Washington, 1935, p. 357.
[6] *Cf.* Bell and Young, *Biochem. Jour.*, 28: 882, 1934.
[1] J. B. Eyerly and H. C. Breuhaus, *Am. Jour. Dig. Diseases*, 6: 187, May, 1939.