While carotene showed some fluctuation from time to time, there were no changes in this pigment which could be definitely related to the irradiation.

From the purely physical data represented by spectrophotometric curves, the psychophysical specification of the color stimulus received by the eye of a normal observer was computed[3] for I. C. I. Illuminant C using data standardized by the International Commission on Illumination in 1931.

	Relative brightness	Dominant wave-length	Excitation purity
	Per cent.	mμ	Per cent.
Before exposure	48.6	580	20.2
11 hours after exposure	31.8	590	20.5
48 hours after exposure	31.8	592	20.9
4 days after exposure	36.9	587	20.7
19 days after exposure	37.4	585	20.7
4 months after exposure	42.6	580	20.5
9½ months after exposure	43.6	583	20.1

In this color language "relative brightness" is a measure of the quantity of light reflected by the skin, while "dominant wave-length" and "purity" describe the quality of this light. Thus, hyperemia caused the skin to be much darker and noticeably more red, but it produced only a very slightly more pure or saturated color. At the end of four days the skin was less red but still much darker than normal. This is ascribable not so much to melanin as to the venous stagnation previously referred to. After four months the color of the skin had returned to its original quality, but its brightness was reduced by melanin. The gradual recession of this substance is shown by the increased brightness of the nine and a half months' data. Even at this time the skin was darker than before exposure.

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PRESENCE OF A LEUKOCYTOSIS-PROMOTING FACTOR IN INFLAMMATORY EXUDATES[1]

THE recent studies of the writer have indicated the presence of a crystalline nitrogeneous substance liberated in areas of acute inflammation, capable *per se* of increasing capillary permeability and of inducing prompt leukocytic migration at the site of injury.[2, 3, 4] This substance has been termed leukotaxine. Its liberation and recovery from injured tissue offer a reasonable explanation for two of the basic initial sequences in the development of the acute inflammatory reaction. Leukotaxine has been shown to have no manifest physiological property in common with histamine.[2, 4, 5]

Leukotaxine, injected either subcutaneously or intravenously, fails to induce an increase in the leukocytic level of the circulation in both the dog and the rabbit. Repeated injections of 30 to 50 milligrams of the material over an interval of several days leave the number of circulating leukocytes essentially unaltered. The independence of the chemotactic factor (leukotaxine) from that concerned with leukocytosis is not wholly surprising when it is recalled that certain inflammatory processes characterized by marked leukocytic infiltration can even be accompanied by distinct leukopenia.

Having apparently eliminated leukotaxine as the factor responsible for the state of leukocytosis accompanying inflammation, studies were undertaken in an endeavor to determine whether the active principle might not be liberated in the exudate as a result of tissue injury. Ponder and MacLeod[6] recently expressed the opinion that in the blood stream of rabbits with peritonitis the shift to the left of polymorphonuclear counts is probably referable to the absorption of breakdown products of the cells appearing first in the exudate.

Inflammatory exudates were obtained by a variety of methods. In the majority of instances the exudative material resulted from an intrapleural injection of 1.5 cc of turpentine in dogs. Several of the exudates studied, however, were derived either after the intrapleural injection of 0.1 cc 10 per cent. croton oil in olive oil, the combined irritating action of several substances (*e.g.*, magnesium carbonate, staphylococcus *aureus* toxin and a mixture of aleuronat and colibactragen), or finally by physical injury. The latter consisted in recovering exudative material from a severe burn induced under nembutal anesthesia by scalding the limb of a dog in water heated to about 90° C. The exudate was obtained by removing the edematous subcutaneous tissue, and gently expressing the oozing and abundant exudative fluid content.

From 15 to about 25 cc of whole or cell-free exudate was injected by intracardiac puncture into the circulating blood stream of a normal dog. The results of eighteen such experiments indicate that there is a conspicuous rise in the leukocytic counts several hours after the introduction of an exudate into the blood. The increase in the number of circulating leukocytes averages 70 per cent.

[3] A. C. Hardy, "Handbook of Colorimetry," Technology Press, Cambridge, 1936.

[1] Aided by grants from the Milton Fund of Harvard University, from the International Cancer Foundation and from the Academy of Arts and Sciences.

[2] V. Menkin, *Jour. Exp. Med.*, 64: 485, 1936.

[3] *Ibid.*, *Jour. Exp. Med.*, 67: 129, 1938.

[4] *Ibid.*, *Physiol. Rev.*, 18: 366, 1938; *Proc. Soc. Exp. Biol. and Med.*, 40: 103, 1939.

[5] V. Menkin and M. A. Kadish, *Am. Jour. Physiol.*, 124: 524, 1938.

[6] E. Ponder and J. MacLeod, *Jour. Exp. Med.*, 67: 839, 1938.

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.