treated with 2,4-D at 1,000 ppm appeared normal in every respect. The yield of sweet-potato roots for the treatments that received activated carbon were as follows for the test conducted at Stoneville, Mississippi: Ck, 319 bu; 1,000 ppm, 324 bu; 2,000 ppm, 298 bu; 3,000 ppm, 246 bu; 4,000 ppm, 234 bu. The yields were not definitely affected except in the manner in which 2,4-D influenced plant survival. The weeds were controlled by cultivation in this test. At the same time, this concentration of 2,4-D controlled annual weeds, such as crabgrass (*Digitaria sanguinalis*), pigweed (*Amaranthus retroflexus*), bindweed (*Convolvulus* sp.), spurge (*Euphorbia* sp.), and others, at State College, Mississippi, and in tests on other crops at Stoneville, Mississippi.

TABLE 1

EFFECT OF ACTIVATED CARBON ON THE SURVIVAL OF SWEET POTATOES AT STATE COLLEGE, PLANTED ON LEEPER CLAY SOIL PREVIOUSLY SPRAYED WITH VARYING RATES OF 2,4-D

Free-acid equivalent of 2,4-D		Survival of original planting (%)*	
Concentration of solution (ppm)	Lbs/acre	Activated carbon treatment	Untreated
0	0.0	100.0	93.0
1,000	1.3	95.0	2.5
4,000	5.2	32.0	0.0

* Average of 4 replications; 10 plants per plot; planted May 16, 1947; survival readings taken June 30, 1947.

TABLE 2

EFFECT OF ACTIVATED CARBON ON THE SURVIVAL OF SWEET POTATOES AT STONEVILLE, PLANTED ON SARPY SANDY LOAM PREVIOUSLY SPRAYED WITH VARYING RATES OF 2,4-D

Free-acid equivalent of 2,4-D		Survival of original planting (%)*	
Concentration of solution (ppm)	Lbs/acre	Activated carbon treatment	Untreated
0	0.0	70.0	75.0
1,000	1.3	78.0	5.0
2,000	2.6	70.0	1.7
3,000	3.9	62.5	0.0
4,000	5.2	28.3	0.0

* Average of 3 replications; 20 plants per plot; planted May 27, 1947; survival readings taken July 2, 1947.

The sweet potato plants that survived the heavy (4,000 ppm) treatment were dark green, and some of the leaves showed abnormalities.

The above data indicate that activated carbon may be used to protect certain crop plants against the effect of 2,4-D when the latter has been applied to the soil as a pre-emergence herbicide.

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Studies on Radiosensitivity of Cells[1]

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It has been commonly believed that the sensitivity of mammalian cells to ionizing radiations is proportional to cell activity in terms of mitotic proliferation—the more active the proliferation, the more sensitivity. An increased erythropoiesis can be initiated in the laboratory animal by phlebotomy or by the hemolytic action of acetyl phenylhydrazine on the circulating erythrocytes. These procedures made possible an experiment designed to compare the vulnerability to irradiation of the highly sensitive erythrocyte precursors existing in the normal hemopoietic tissue of rabbits with those in hemopoietic tissue of rabbits which have an induced hyperplasia of erythrocyte precursors (Table 1). The effect of 800 r

TABLE 1

PREPARATION AND TREATMENT OF ANIMALS

Phenylhydrazine Experiment			
Group	Phenylhydrazine subcutaneously	X-ray	No. of animals
I	None	None	10
II	*ca* 35–40 mg	800 r	28
III	None	800 r	16
IV	*ca* 35–40 mg	None	16
Blood Withdrawal Experiment			
Group	Blood removed	X-ray	No. of animals
V	None	None	10
VI	*ca* 90 cc	800 r	15
VII	None	800 r	15
VIII	*ca* 90 cc	None	10

whole-body X irradiation upon animals that have a previously induced regenerative anemia was compared with the effect of this same dose on normal animals of comparable age and weight. Determinations of the erythrocytes and reticulocytes/cu mm and hemoglobin in grams/100 ml are recorded in Figs. 1, 2, and 3. The normal irradiated animals developed an anemia that reached a maximum at 14 days. The animals in which a regenerative anemia had been produced prior to irradiation with

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800 r developed no further anemia. The recovery time in both groups was comparable (23 days). The mean reticulocyte value of the phenylhydrazine-injected, X-rayed animals was reduced below the normal control

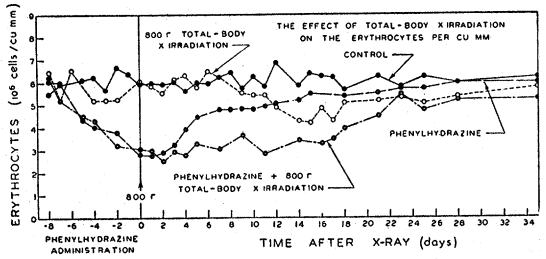

FIG. 1

value on the 6th postirradiation day only. The phlebotomized group of animals responded to irradiation in a manner comparable to the animals treated with phenylhydrazine and X-ray.

The histologic studies made on appropriately sacrificed animals revealed that erythropoietic tissue in the bone

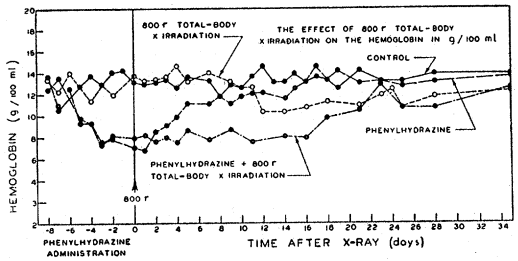

FIG. 2

marrow and the spleen was essentially completely destroyed by 3 days in normal animals receiving 800 r. A relatively slow recovery ensued beginning on the 7th to the 9th day. On the other hand, the erythropoietic tissue in the bone marrow and spleen was only partially

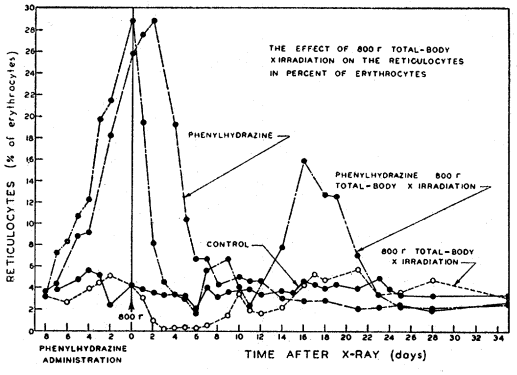

FIG. 3

destroyed in animals irradiated with 800 r after a regenerative anemia was produced by phenylhydrazine or phlebotomy (Fig. 4). Sufficient viable erythropoietic tissue remained to permit an essentially normal production of erythrocytes.

These data would tend to indicate that erythroblast vulnerability to irradiation injury is not enhanced by increased mitotic activity and proliferation. In fact, the hyperplastic erythroid tissue sustained less histologic injury than the normal, and the production of erythrocytes was maintained. Since the erythroblasts in hyperplastic and in normal tissue received the same dose of radiation, the completeness of destruction should have been comparable in the two experimental setups unless differences

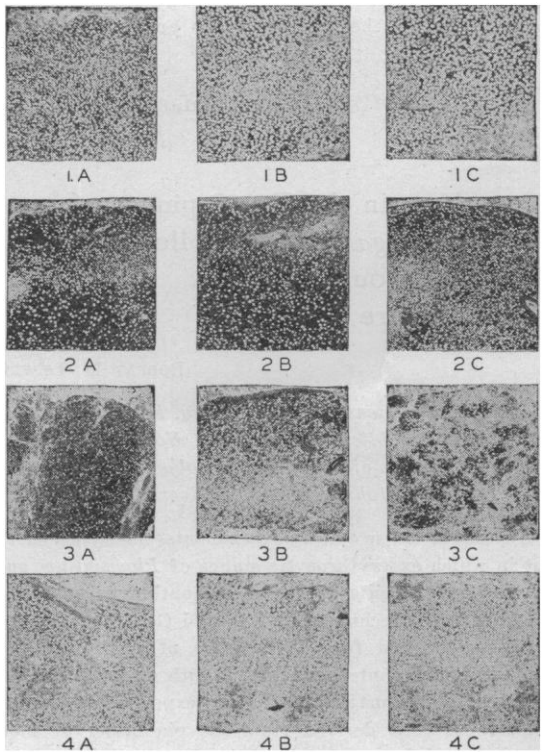

FIG. 4. The hematologic effects of 800 r on normal and hyperplastic bone marrow of the rabbit: 1A, B, C—range of normal control marrow; 2A, B, C—bone marrow after phenylhydrazine-induced hyperplasia 3, 4, and 6 days, respectively, after phenylhydrazine withdrawal; 3A, B, C—bone marrow of phenylhydrazine-induced hyperplasia at 1, 3, and 5 days, respectively, after 800 r and 3, 4, and 6 days, respectively, after phenylhydrazine withdrawal; 4A, B, C—bone marrow of normal rabbits exposed to 800 r at 1, 3, and 5 days after 800 r. (Mag. 16 ×.)

in sensitivity to radiation existed. One factor which may have caused this apparent difference in sensitivity is that a larger number of basophilic and polychromatophilic erythroblasts was present in the hyperplastic tissue than in the normal tissue at the time of irradiation. It was largely these cells that survived and were immediately capable of mitotic proliferation. Actually, any attempt to explain differences in radiosensitivity of cells involves factors concerned in the metabolism of cells which we do not understand at the present time.

It has been reported that the reduction of blood flow

to a lymph node, and thus a reduction in oxygen supply to the node, reduces the sensitivity of the lymphocytes therein to irradiation (*1*). Reduced oxygen tension, however, does not "stimulate" lymphocyte production. The production of anemia in the experimental animal by phlebotomy or phenylhydrazine administration by virtue of reducing the oxygen supply to the bone marrow produces a stimulus, an optimum condition, for the proliferation of erythroblasts. The mechanism for the reduced radiosensitivity in the lymphatic tissue with O_2 deprivation is probably different from that operating in erythropoietic tissue in the presence of an anemia.

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Alterations in the Development of *Plasmodium gallinaceum* Following Passage Through Tissue Culture

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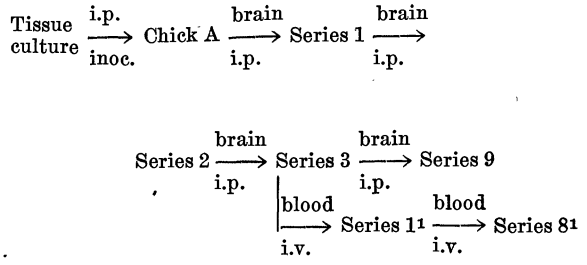
A series of tissue culture experiments[2] have been carried out in which exoerythrocytic stages of *Plasmodium gallinaceum* have been maintained in continuous culture by the roller tube technique of Gey and Gey (*1*). The cultures were tested for the presence of parasites by inoculating chicks intraperitoneally with material from the cultures. The majority of the experiments involved colonies of cells derived from the pia mater of blood-infected, quinine-treated chicks. Some of the cultures, however, had been derived from heart muscle of such birds. Each of the cell strains was subcultured at intervals which varied with the rate of growth of the tissue-cultured host cells. Microscopical examination of such cultures failed in most instances to reveal extensive development of parasites. Nevertheless, young chicks receiving inoculations from cultures which had been maintained for four or five subcultures, and which were as much as 70 days old, became infected with *P. gallinaceum*.

The primary purpose of this preliminary paper is to emphasize the peculiar character of the resulting infection in chicks, since this has been exclusively of an exoerythrocytic nature. Chicks infected as indicated above ranged from 7 to 32 days old. They invariably developed an overwhelming exoerythrocytic infection which terminated fatally 10–17 days after their inoculation with material from culture. No pigmented erythrocytic stages developed in any of the birds, although, in several instances, at the time of death a low percentage (1–3%) of the erythrocytes were parasitized with minute, unpigmented, uninuclear forms. With the exception of a more rapid onset and an apparently greater severity, this infection is almost identical with that encountered in blood-infected, quinine-treated chicks (*3, 4*).

In a number of instances the parasites obtained from chicks infected from culture have been maintained by serial passages in other chicks. The nature of this exoerythrocytic infection has remained unchanged in one experiment through 9 serial passages (note diagram below) accomplished by means of intraperitoneal inoculation (i.p.) of brain suspended in saline. The infection in birds 2–35 days old continues to be acute, with exoerythrocytic parasites readily demonstrable in the capillary endothelium of the brain as early as 4 days after inoculation. Death from exoerythrocytic parasitism has occurred as early as 6 days after inoculation with infected brain material.



In contrast to the above, serial passage of the infection by intravenous inoculation of blood from such birds resulted in a somewhat different picture of parasitism. For example, blood taken from chicks of the previously mentioned series (see diagram) was used to initiate a blood inoculation series. Parasitism of the birds of the first and second inoculations of this series was again almost exclusively of the exoerythrocytic type. However, after the third blood passage pigmented erythrocytic stages were found. Continued blood passage seemed to increase the number of the pigmented blood stages. Nevertheless, even at the end of the 8 serial blood passages, the infection continued to be preponderantly exoerythrocytic in most of the infected birds.

Since completion of this work, a report of similar alteration in the development of *P. gallinaceum* has been made in which passage through chick embryos, rather than tissue cultures, was a contributing factor (*2*).

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[1] The author wishes to express his appreciation for the assistance and advice which G. F. Otto, of the Department of Parasitology, and G. O. Gey, of the Division of Cellular Physiology, have given during the course of this investigation.

[2] To be reported on at a later date.