conventional pattern of association or society organization and functions, it will attempt to provide "fluid mechanisms"—stripped of all formalism or meaningless detail—for the most effective interchange of information and cooperation in current and vital services at the least possible cost in time, effort and expense.

At an executive committee meeting held in the Engineering Societies Building on March 25, Robert B. Colgate, vice-president of Colgate-Palmolive-Peet Company was elected chairman of the executive committee and Mr. H. W. Graham, general metallurgist, Jones and Laughlin Steel Corporation vice-chairman. The executive committee will serve as a committee of the National Research Council by appointment of Chairman Bush during the formative and development period of the institute. Later it is expected that the institute will be incorporated as a membership non-profit organization under the laws of the State of New York. In the meantime the Division of Engineering and Industrial Research of the National Research Council has offered its facilities, the services of its staff, and its technical resources and contacts during the experimental period. Maurice Holland, director of the division will serve as executive officer of the institute.

MAURICE HOLLAND,
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NEW YORK,
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SPECIAL ARTICLES

SUBLETHAL EFFECTS OF LONG WAVE-LENGTH ULTRA-VIOLET

IN work previously reported on the effect of long wave-length (3,130 and 3,660 Å) ultra-violet radiations upon protozoans[1] large dosages were found to have no immediate visible effects and the animals so irradiated divided when fed and conjugated when starved following generous feeding. In some cases, however, division seemed retarded and the possibility of slight sublethal effects was suggested, but control of the test material was inadequate for accurate work. Subsequent studies of the effects of short wave-length ultra-violet rays upon cleavage of the eggs of the sea urchin (*Strongylocentrotus purpuratus*) and upon excystment of *Colpoda duodenaria* cysts indicated that these were more sensitive and reliable test materials. The effect of long wave-length ultra-violet radiations upon cleavage and excystment of these forms was therefore studied.

The apparatus was similar to that previously employed.[1] The methods for handling Colpoda cysts were similar to those described by Taylor, Brown and Strickland,[2] by whom the materials used here were generously supplied. The excystment studies reported here were made at $20 \pm 0.3°$ C. The methods for handling sea urchin eggs will be described in detail elsewhere. The temperature of the room in which the work on the sea urchin eggs was carried out was $15 \pm 1°$ C.

In a representative set of experiments after a dosage of 400 ergs/mm² at λ3,025Å, which is at the long wave-length end of the lethal spectrum, the 8-celled stage in cleavage of the sea urchin egg was reached about one hour later than in control cultures. While such small doses with λ3,130Å usually had no noticeable effect upon the cleavage rate, a dosage of 3,200 ergs/mm² caused approximately an hour delay in the appearance of the 8-celled stage. With a dosage of 12,600 ergs/mm² the retardation was 2 hours. While in all the above cases a delay was still discernible at the time blastulation and gastrulation occurred, plutei normal in appearance were formed from all the eggs, retardation being no longer observable.

Following irradiation of eggs with λ3,660Å slight retardation was noticeable only after a dosage of 40,000 ergs/mm², but even after a dosage of 74,500 ergs/mm² the retardation did not become conspicuous enough to measure accurately. No retardation was observable at the time of blastulation or thereafter, observations being made until the pluteus stage was reached.

In no case were eggs irradiated at these wave-lengths with less than the retarding dosage observed to cleave more rapidly than controls, the cleavage rate in such cases being equivalent to that of the controls.

Excystment time of Colpoda cysts (time elapsed from the moment the cysts are placed in an excysting medium to the exit of the animals from the cysts) was increased by irradiation with λ3,025Å, thus in a representative set of experiments a dose of about 3,000 ergs/mm² caused an increase in time of 50 per cent. excystment of about ¾ hour, a dose of 8,000 ergs/mm² an increase of 3¾ hours (time of 50 per cent. excystment of controls: 132.3 ± 5.9 min. in the experiments reported). A dose of 3,000 ergs/mm² at λ3,130Å, however, failed to increase excystment time, and ten times this dose caused an increase of only about 1/3 hour. Prolonged irradiation with λ3,660 even after dosages of 100,000 ergs/mm² had been given produced no increase in excystment time, the irradiated animals excysting at the same time as the controls.

[1] Giese and Leighton, SCIENCE, 81: 53, 1935.
[2] Taylor, Brown and Strickland, *Jour. Cell. and Comp. Physiol.*, 9: 105, 1936.

In no case in which conditions were properly controlled was there evidence of a decrease in excystment time when cysts were irradiated with dosages below the threshold dose causing increased time of excystment.

The results indicate that (1) while radiations of λ3,130Å failed to produce visible effects or kill protozoans after prolonged exposures, when the dosage is sufficiently great, slight retardation of certain cell processes may occur. This can be demonstrated if the test material is sufficiently delicate. (2) The fairly large dosages of radiations of λ3,660Å had exceedingly slight or no retarding effects upon even the more sensitive cell functions here tested.

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THE PRODUCTION OF LYMPHOMATOSIS IN MICE OF KNOWN GENETIC CONSTITUTION[1]

LYMPHOMATOSIS, with or without a leukemic blood picture, has been observed in mice after painting the skin with 1:2:5:6-dibenzanthracene[2] and following the subcutaneous injection of sodium 1:2:5:6:-dibenzanthracene-9:10-endo-α:β-*succinate*.[3] An atypical leukemia followed the intrasplenic injection of 3:4-benzpyrene in a mouse of the S strain.[4] The chemical production of leukoses has been criticized because of the high spontaneous incidence of mouse leukemia and the alleged failure to adequately control the genetic factors.[5] We wish to record the appearance of lymphomatosis in mice of known genetic constitution that followed painting with methylcholanthrene.

The mice used were of the dilute brown strain developed by Little. They were obtained from the Roscoe B. Jackson Memorial Laboratory, where the strain has been followed for more than twenty-five generations. Leukemia has occurred rarely and never before the animals were eighteen months old.[6] Breeding females have a very high incidence of spontaneous mammary cancer.

Forty-eight mice were used. They were not allowed to breed. On November 1, 1937, when the animals were four to six weeks old painting was commenced. The material used was 0.5 per cent. methylcholanthrene in commercial benzene. It was applied with a No. 6 camel's hair brush. The site was changed with each painting so that the same area was not painted twice during the same month. Contiguous regions were not painted in succession. The order was: head, left hind leg, right hind leg, left foreleg, right foreleg, sacral region, abdomen, interscapular region, anterior thorax. Three mice died of infection before the fifty-third experimental day.

When the animals had received their tenth painting (sixty-ninth day) a subcutaneous inguinal mass was found in a female mouse. Three days later bilateral inguinal and axillary lymphadenopathy was present. The animal died on the seventy-fifth day of the experiment. At autopsy the axillary, inguinal, abdominal and tracheo-bronchial lymph nodes were greatly enlarged, attaining a diameter of more than 1 cm. The liver was pale. The spleen was large and gray.

Microscopically the architecture of the lymph nodes was obliterated. The nodes were filled with atypical lymphoid cells which were closely packed together. The cells were larger than normal lymphocytes. They had scanty basophilic cytoplasm and large, round, vesicular nuclei with prominent nucleoli. Mitotic figures were numerous. Capsular infiltration was noted. The normal structure of the spleen was lost. The pulp was replaced by cells similar to those found in the lymph nodes. The liver showed marked leukemic infiltration, particularly in the peri-portal areas.

Nine more mice, four females and five males, have subsequently developed the same picture. The disease is first noted as a cervical, axillary or inguinal lymphadenopathy which is bilaterally symmetrical in distribution. The mice die within two or three weeks of the onset and present more or less generalized lymphoid enlargement. Some of the nodes have measured more than 2 cm in diameter. Subcutaneous edema, ascites and hydrothorax have been found. The fluid is usually clear. Leukemic infiltration has been seen in lung, skin, coagulating gland, bladder, kidney and skeletal muscle. The myeloid cells of the bone marrow are replaced by lymphoid cells.

Blood studies have been done on two mice. The leucocytes numbered 90,000 and 139,000, respectively. A secondary anemia was present. The white cells were almost all of the lymphoid series. Many immature forms were seen.

Five skin tumors have been produced thus far (one hundred and third day), one of which is malignant. None of these animals have shown any evidence of lymphadenopathy.

No tumors have appeared in fifty control mice.

Although the experiment has not been completed, the production of lymphomatosis with a leukemic

[1] From the Department of Surgery, the University of Rochester School of Medicine and Dentistry. This investigation was aided by grants from the International Cancer Research Foundation and from Mr. Simon Stein.

[2] I. H. Perry and L. L. Ginzton, *Am. Jour. Cancer*, 29: 680, 1937.

[3] H. Burrows and J. W. Cook, *Am. Jour. Cancer*, 27: 267, 1936.

[4] W. A. Barnes and J. Furth, *Am. Jour. Cancer*, 30: 75, 1937.

[5] M. N. Richter and E. C. MacDowell, *Physiol. Rev.*, 15: 509, 1935.

[6] C. C. Little, personal communication.