SPECIAL ARTICLES

THE PRODUCTION OF MUTATIONS BY ULTRA-VIOLET LIGHT

THE discovery by Muller that x-rays produce mutations led to the suggestion that similar forms of radiation, occurring in nature, might be the cause of natural mutations. Undoubtedly, part of the natural mutation rate is due to the radiation in question, but, as shown by the calculations of Muller and Mott-Smith, the entire rate could not be accounted for in this way. The problem was therefore raised as to what other agencies might cause natural mutations. One such possible agency is ultra-violet light, since it is known that ultra-violet light causes chemical activation. Moreover, it seems probable from the studies of Gurowitch and others that growing and dividing cells give off "mitogenetic" rays, which are in the ultra-violet region of the spectrum. It therefore seemed desirable to test the effect of ultra-violet light on the mutation rate.

Previous experiments which I did on Drosophila indicated that ultra-violet light had a slight positive effect on the mutation rate but in many of these experiments I treated the adults and it was difficult to get the rays to the reproductive cells on account of the high screening effect of the superficial tissues. I therefore decided to use the developing eggs and, in particular, eggs in which the "pole cells" were formed; that is, the germ tract cells at the time that they form a polar cap at the amicropilar end of the egg. Only the polar cap was treated, the rest of the egg having been screened (by a cover glass). A quartz mercury arc lamp was used as a source of the light. It was run at 50 volts. The eggs were at a distance of 150 cm from the lamp, and they were given 3 to 4 minutes treatment. Lethals were looked for in the X-chromosomes of males that developed from the treated eggs, and Muller's Cl B method was used for the detection of the lethals. In case any mutations were produced by the ultra-violet light at the polar cap stage, then these should appear in "bundles"; that is, in a fairly large proportion of the sperm cells. Moreover, they should be "reduplications"; that is, the same mutation multiplied. For, assume that there are 10 pole cells in the polar cap at the time of treatment and that one of the cells is struck in the right way and a mutation produced in its X-chromosome. Then roughly one tenth of the sperm cells of the adult should contain the mutation, and the male in question should transmit the mutation to about one tenth of his daughters.

Some tests already made show just such an effect. The data are as follows. From 108 males treated in the polar cap stage I got 8 cases of "reduplicated" lethals. From 110 males in the controls I got 1 reduplicated lethal.

A certain number of lethals turned up in the controls, but these were apparently lethals that occurred after the polar cap stage (that is, after treatment), and represented the natural mutation rate. For they were either not reduplicated at all, or only to a small extent, just as would be expected if they occurred at a later stage in development. The treated lot also contained these "natural" mutations. As a criterion for a lethal that was produced in a cell at the polar cap stage, I use the number of cells present in the polar cap (10 to 20) at the time of treatment. In other words, I regard as induced lethals any that are reduplicated in about 5 to 10 per cent. (or more) of the reproductive cells, and which are shown by linkage tests to be the same lethal.

On *a priori* grounds it would be very unlikely that a lethal should occur, apart from treatment, at just the polar cap stage, especially in view of the small number of cells at this stage, and the short length of time that it lasts (less than 1 hour). It is therefore very surprising that there should have been a reduplicated lethal in the controls. It is possible that the male which yielded this lethal belonged with the treated lot or that some stray light (reflected from the walls of the room) got around the sides of the plate glass that I had in front of my controls, and that it happened to strike one of the control eggs.

I wish to thank the American Academy of Science for a grant of $500 to defray the expenses of an assistant for the above experiment. I wish also to thank Miss Frances Ward for her capable assistance.

THE RICE INSTITUTE EDGAR ALTENBURG

THE IMMUNOLOGICAL RELATIONSHIP OF EASTERN AND WESTERN STRAINS OF EQUINE ENCEPHALOMYELITIS VIRUS

THE epizootic of equine encephalomyelitis which invaded sections of Delaware, Maryland and Virginia during July, August and September, 1933, has presented characteristics judged to be closely comparable with those seen in the disease as it has occurred in the West, the only apparent material difference being in a more acute course and perhaps greater mortality. Epizootiologically, the diseases have much in common and the syndromes do not differ, except as above noted.

The anatomical changes observed at autopsy have been confined largely to the central nervous system and have not been uniformly different from those seen in cases of the western disease.

The histological alterations appear to differ only in degree, the eastern disease exhibiting a more intense small cell infiltration, and a more marked extravasation of erythrocytes and fluid into the perivascular and pericellular spaces.

The writers have isolated eight strains of a filterable