examination of such transfers the following conclusions were derived.

(1) The L.D.50 for nuclei is 0·00015 per cent nitrogen mustard.

(2) The L.D.50 for cytoplasm is 0·0015 per cent nitrogen mustard.

(3) The cytoplasm is independently damaged, that is, by a direct action of the drug.

(4) The nuclei are probably damaged independently of the cytoplasm, that is, by a direct action of the drug.

(5) If the cytoplasm is lethally damaged, cell division never occurs.

(6) If the nuclei are lethally damaged, nuclear division usually occurs unless the nuclei are present in very badly damaged cytoplasm. Division of the nuclei is not necessarily followed by cytoplasmic division. If damaged nuclei are in normal cytoplasm, cytoplasmic division normally follows nuclear division; but if the cytoplasm is also damaged the probability of cytoplasmic division varies inversely as the amount of damage.

(7) Division of the amœbæ is delayed to an increasing degree with exposure to increasing concentrations of nitrogen mustard. The first division after treatment is little delayed unless lethal damage has occurred. The second division is much more sensitive than the first, and the delay increases more rapidly with increase in concentration.

(8) Other abnormalities observed included: unequal division; prolonged prophase with death of amœba before completing division; failure of nuclear reconstruction after mitosis; division into more than two cells (up to seven), some of which may be anucleate.

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X-Rays

In view of the results recorded in the previous communication it was decided to study the site of action of X-rays by a similar technique. Most experiments were made with a 400-kVp. machine, providing a dose-rate of 3,000 r. per minute. Some results were obtained using a 10-kV. machine giving a dose-rate of 27,300 r. per minute. Using the technique of nuclear transfer, the following conclusions were reached with *Amœba proteus*.

(1) The L.D.50 for nuclei is 120,000 r.

(2) The L.D.50 for cytoplasm is 290,000 r.

(3) The cytoplasm is independently damaged, that is, by a direct action of the radiation.

(4) Cytoplasmic damage is of two types: reversible damage already maximal at less than 100,000 r.; and lethal damage becoming prominent above 280,000 r.

(5) The nuclei are probably damaged independently of the cytoplasm.

(6) If the cytoplasm is lethally damaged, cell division never occurs.

(7) If the nuclei are lethally damaged, nuclear division seldom occurs. If division of a lethally damaged nucleus does occur, it is normally followed by cytoplasmic division.

(8) Division of the cytoplasm is delayed and is normally preceded by nuclear division. The delay reaches a maximum at 120,000 r. This delay is prominent for the first division, and small for the second division.

The main differences observed between the action of X-rays and of nitrogen mustard were:

(*a*) The nuclei are ten times more sensitive to nitrogen mustard than is cytoplasm, compared with a ratio of 2·5 for X-rays.

(*b*) The slopes of the dosage-mortality curves both for nuclei and for cytoplasm are steep for X-irradiation and relatively flat for nitrogen mustard. Consequently, a dose of X-rays which is just lethal for all nuclei will not cause any lethal damage to cytoplasms, whereas an exposure to nitrogen mustard which is just lethal to all nuclei will also cause lethal damage to some cytoplasms.

(*c*) Whereas with X-rays delay in division is prominent for the first division and small for the second, with nitrogen mustard the delay is small for the first division and prominent for the second.

(*d*) Lethal damage to nuclei by X-rays usually prevents nuclear division immediately, whereas with nitrogen mustard nuclear division is usually possible after lethal damage.

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'AZIDINE' TRANSFORMATIONS

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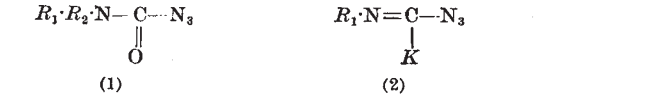
THE carbamylazides, as a group, were classified by Bertho[1] as resistant to the Curtius rearrangement. However, other work[2] showed that while some members of this group (1, R_1 or R_2 = H, R_2 or R_1 = alkyl/aryl) failed to become rearranged, others (1, R_1 or R_2 = C_6H_5) did so.



(1) (2)

Hurd[3] explained this anomaly by ascribing a hydroxyurea structure (2, K = OH) to the carbamylazides which resisted rearrangement. Hurd's formula contains the azidine system, a characteristic of which[4] is ready cyclization to substituted tetrazoles, in the presence of bases. (By analogy with the nomenclature change of amide → amidine for the structural change —$CONH_2$ → —$C(=NH)$—NH_2, it is suggested that, for the structural change —CON_3 → —$C(=NH)N_3$, the nomenclature change should be from azide → azidine. This latter term does not appear to have been used previously in the literature.) The expected cyclization product from carbamylazide (2, R_1 = H, K = OH) would be 5-hydroxytetrazole (3). Under a wide variety of reaction conditions, and with a large range of basic reagents, we have found