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Table 3.	DEOXYRIBONUCLEIC ACID AND RIBONUCLEIC ACID SYNTHESIS					
	IN NORMOBLASTIC AND MEGALOBLASTIC MARROWS					
Grain counts in 24-hr. cultures						

Tahel	Normal marrows		Pernicious anæmia	
Labor	Normo- blasts	Myelo- cytes	Megalo- blasts	Myelo- cytes
Formate carbon-14 (DNA)	120	98	170	100
$\begin{array}{c} \text{Adenine carbon-14} \\ \text{(DNA + RNA)} \end{array}$	245	185	603	295
Adenine minus formate = RNA	125	87	433	195
DNA/RNA	0.96	1.10	0.39	0.51

anæmia marrows than in myelocytes or normoblasts from normal marrows (inter-marrow variations of grain counts over myelocytes or erythroblasts is \pm 10 per cent).

These observations complement earlier findings by Davidson et al.1 and indicate that in pernicious anæmia marrows deoxyribonucleic acid synthesis is slightly increased in megaloblasts but not in the myelocytes. Synthesis of ribonucleic acid, however, is greatly increased in myelocytes and even more so in megaloblasts of pernicious anæmia marrows. Since in short-term (6-20 hr.) cultures no marked difference was noted between normal and pernicious anæmia serum in the medium, it is suggested that the megaloblastic change takes more than 20 hr. to develop. This is in agreement with earlier observations on the formation of megaloblasts in normal marrows cultured in pernicious anæmia serum⁴.

The underlying biochemical defect in the megaloblasts is far from clear. Determinations based on deoxyribonucleic/ribonucleic acid ratios may be misleading, because a greatly increased ribonucleic acid and a slightly less increased deoxyribonucleic acid content may give the erroneous impression of deficiency in deoxyribonucleic acid.

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Effect of Ultra-violet Light on Mouse Skin over a Wide Range of Intensities

WHETHER the effect on the living skin of a constant dose of ultra-violet radiation is the same if given with high intensity in a short pulse as if given with low intensity over a longer period of time has not apparently been recorded. We have therefore examined the tissue injury caused in mouse skin by ultra-violet light of wave-length about 300 mµ when the intensity of the incident light was varied from 10^{14} to $10^{21} hv/cm.^2$ sec.

A high-pressure mercury lamp with appropriate filters (2 mm. Jena WG 7 + 10 mm. NiSO₄.6H₂O,



Fig. 1. The relation between light intensity $(\lambda \sim 300 \text{ m}\mu)$ and minimum dose to cause tissue injury in mouse skin

500 gm./l.) served as source of light at the lower intensities. To obtain different intensities, working distances of about 10, 12, 35, 40 and 140 cm. were used with exposure times varying from 1 min. up to 120 min. The light pulse of high intensity was obtained from a photolysis flash lamp with maximum emission at about 300 mµ¹ provided with the same filters. The working distance was 4.5 cm., giving a constant average light-intensity of about 1.3×10^{21} hv/cm.² sec. or about 860 watts/cm.²; suitable exposure times were 50-150 µsec. The intensities and doses were determined by means of chemical actinometry (both uranyloxalate and potassium ferrioxalate).

The mice, a commercial albino stock, were shaved with a barium sulphide paste on the back. They were fastened rigidly without anæsthesia by means of black rubber sheets to plane wooden boards and irradiated on the back through 20 mm. circular openings in the sheets. Each animal was normally irradiated at two positions, both placed over the spine, with about 25 mm. between their centres. A total of about 100 animals were used in the experiments reported here.

The degree of tissue damage was estimated by the leakage into the irradiated areas of intravenously injected Evans blue. A volume of 0.3 ml. of the dye solution (0.5 per cent in saline) was injected intravenously 20 hr. after irradiation. The animals were killed 15 min. after injection. The degree of blueing, estimated by visual colorimetry of the dried skin, was plotted as a function of the irradiation dose. From these curves the minimum dose causing blueing was estimated and is plotted against intensity in Fig. 1. It is evident that this minimum dose is virtually constant over a 107-fold change in intensity.

The intensity of the blueing increases with the dose of radiation up to a certain point. The blueing then gradually becomes weaker, probably due to a decrease in blood supply as the damage is increased. This latter limb of the curve is markedly influenced by the light intensity. At the flash intensity the decrease in blueing is evident at much lower doses than in the case of the lower intensities.

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