of this material may be to assist in the binding of cells together. The material for this work was obtained from normal adult Wistar rats. It was fixed by the method described by Barnes and Davis[8] and embedded in 'Araldite' as described by me[9].

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PATHOLOGY

Porphyrin Fluorescence of Experimentally produced Squamous Cell Carcinoma

Ulcerated squamous cell carcinomas of the human skin frequently show a red fluorescence when examined under ultra-violet radiation[1-3]. This red fluorescence is usually restricted to small areas of the necrotic surface. Hitherto the substance or substances responsible for this phenomena have not been identified. This is probably due to the fact that very small amounts of material are available for study in any single case. Recently, Ghadially[4] observed that experimentally produced squamous cell carcinomas of the skin of many animals show a similar red fluorescence. It seemed to us that by pooling material from a number of tumours, sufficient might be obtained for a chemical analysis. Six rabbits with large ulcerated carcinomas produced by repeated painting of the skin with a 2 per cent solution w/w of 9 : 10 dimethyl 1 : 2 benzanthracene in a mixture of equal parts of lanolin and paraffin were available for study. After many attempts, we were ultimately able to collect one large (5 gm.) pooled sample for analysis. Another large sample was obtained from a single rabbit which died as a result of a very large fungating carcinoma of the ear. Here about 5 gm. of red fluorescent debris had collected on the carcinoma and within the ear over a prolonged period of time. The results obtained in each case were essentially similar.

Red fluorescence has been attributed to the presence of porphyrins in a chloroma[5], a fungating adenocarcinoma of the breast[3] and a hepatic adenoma[6] of man. Therefore it seemed reasonable to suppose that similar material might be associated with the red fluorescent squamous carcinomas.

An ethyl acetate (75 ml.) – acetic acid (25 ml.) extract of red fluorescent debris (5 gm.) from the rabbit carcinoma was examined for protoporphyrin, coproporphyrin, and uroporphyrin by the methods of Schwartz and Wikoff[7] and Dresel and Falk[8]. The absorption spectrum of a fraction corresponding to protoporphyrin was measured in ethyl acetate solution with a Unicam spectrophotometer *SP* 500 in the range 350–800 mμ (in 5-mμ steps) in 1-cm. cells versus an ethyl acetate blank. Table 1 gives wave-lengths and optical density values of the maxima and minima exhibited by this porphyrin solution, and, for comparison, the corresponding values are quoted for the spectrum of an authentic sample of protoporphyrin (Light and Co.) in ethyl acetate. These spectra were closely similar, and it may be concluded that protoporphyrin occurs in the carcinoma debris.

Table 1

Material from carcinoma debris			Authentic protoporphyrin		
400 mμ	max.	1·750	400 mμ	max.	2·520
460	min.	0·025	465	min.	0·030
505	max.	0·135	505	max.	0·197
525	min.	0·048	525	min.	0·058
540	max.	0·077	540	max.	0·123
565	min.	0·019	560	min.	0·019
585	max.	0·052	580	max.	0·073
615	min.	0·008	615	min.	0·007
635	max.	0·027	635	max.	0·049

The fraction which should contain coproporphyrin fluoresced red in ultra-violet light, but the porphyrin content was so low that only a very feeble Soret band was detected at 400 mμ in the most concentrated extract we could obtain.

Finally, the fraction which should contain uroporphyrin according to Dresel and Falk failed to fluoresce in ultra-violet light.

Thus the material responsible for the fluorescence of rabbit ear carcinoma seems to consist mainly of protoporphyrin with perhaps a trace of coproporphyrin.

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Phenylketonuria in Infant Monkeys

We had previously established[1] that the characteristic aberrations found in human phenylketonuria, namely, high plasma phenylalanine, phenylketones in the urine, and characteristic odour were observed in adolescent monkeys fed excessive amounts of L-phenylalanine in their diet. From the results of behavioural tests utilizing discrimination and maze performance, as well as poor adaptability to the test situation, these monkeys appeared to be retarded in their learning ability. It seemed important to determine whether phenylketonuria could be produced in infant monkeys when the brain is apparently more sensitive to chemical insult. Long-term observation could then be made throughout the period of development and it might be possible to correlate the biochemical changes with behavioural patterns.

Four infant macaques born in this laboratory were separated from their mothers at birth and wore given a high phenylalanine milk diet from the second day of life. Whole cow's milk or powdered milk formula containing 0·25 gm. L-phenylalanine per 100 ml. in final dilution was fed for the first two weeks. Later,