

failed to show cycasin (Table 2 A). Prolonged boiling did not reveal any cycasin as it had already been destroyed.

Further indirect confirmation of the foregoing observations, on the varying content of cycasin and the effect on it of boiling and storing, emerged from our long-term experiments on the feeding of various preparations of cycad nuts to different groups of monkeys. Two of three animals fed on the nut, after it had been intensely boiled, died of jaundice and hepato-toxicity while only one out of five monkeys fed on unwashed cycad suffered this fate (unpublished observations).

This investigation was supported in part by a grant from the National Multiple Sclerosis Society of America.

DARAB K. DASTUR
RAM S. PALEKAR

Indian Council of Medical Research,
Neuropathology Unit,
Post-Graduate Research Laboratories,
J. J. Group of Hospitals, Bombay 8.

¹ Palekar, R. S., and Dastur, D. K., *Nature*, **206**, 1363 (1965).

² Riggs, N. V., and Korsch, B. H., *Second Conf. Identification of Toxic Elements of Cycads*, National Institutes of Health, Aug. 1962; mimeograph, Appendix 6, 103.

³ Matsumoto, H., and Strong, F. M., *Arch. Biochem. Biophys.*, **101**, 299 (1963).

⁴ Nishida, K., Kobayashi, A., and Nagahama, T., *Bull. Agr. Chem. Soc., Japan*, **19**, 77 (1955).

⁵ Nishida, K., Kobayashi, A., and Nagahama, T., *Bull. Agr. Chem. Soc., Japan*, **19**, 172 (1955).

⁶ Dastur, D. K., *Proc. Third Conf. Toxicity of Cycads, Fed. Proc.*, **23**, 1368 (1964).

⁷ Keresztesy, J. C., *Second Conf. Identification of Toxic Elements of Cycads*, National Institutes of Health, Aug. 1962; mimeograph, 6.

⁸ Armstrong, E. F., and Armstrong, K. F., *The Glycosides: Monographs on Biochemistry* (Longmans Green and Co., London, 1931).

⁹ Matsumoto, H., *Second Conf. Identification of Toxic Elements of Cycads*, National Institutes of Health, Aug. 1962, mimeograph, 46.

Red Fluorescence in Costo-vertebral Spot of Hamsters

DURING the course of experimental production of melanomata in hamsters^{1,2} and the study of red fluorescence in tumours^{3,4}, we discovered, by chance, that the costo-vertebral spot of some of our normal hamsters showed a well-marked red fluorescence under ultra-violet radiation. Red fluorescence has been observed in the Harderian gland of hamsters and some other rodents⁵, but its significance is obscure. Red fluorescence is also seen in many tumours^{3,4}. In all these cases the red fluorescence is due to the presence of porphyrins.

Fifty males and fifty females from our colony of hamsters, in which the exact date of birth was known, were shaved in the region of costo-vertebral spots and examined under ultra-violet radiation. This was done in the autumn (23.10.64) when the animals were between 5 and 18 months old. Only one male showed red fluorescence in one of its costo-vertebral spots. Twenty-one males and twelve females from the same group of animals were shaved and re-examined in the following summer (19.5.65). (The remaining animals were not available because they had been used for other experiments.) The age of the animals used in this experiment ranged from 11 to 15 months. Of the females, only one animal showed a weak reddish fluorescence in one of the costo-vertebral spots, but sixteen males showed fluorescence in both spots, two in one spot, and three showed no red fluorescence. Even in males showing red fluorescence in both spots, one spot was usually more strongly fluorescent than the other.

Histological examination of cryostat prepared sections under ultra-violet radiation showed that the red fluorescence is mainly located in the necks of the hair follicles above the large sebaceous-type gland that occurs in this region. At times a faint patchy red fluorescence was also seen in the adjacent sebaceous glands themselves.

Red fluorescence in biological material is due to the presence of porphyrins. It would therefore appear that the male hamster in the summer season produces a secretion

containing porphyrins from the glands in the costo-vertebral spot. It has been suggested that the secretion from the spot of the male hamster serves to mark territory⁶, but that from the female has no such function. It is intriguing to speculate whether this difference is due to the presence of porphyrins. As yet, little is known about the fluorescence of the secretion of other territory-marking animals.

O. ILLMAN
F. N. GHADIALLY

Department of Pathology,
University of Sheffield.

¹ Ghadially, F. N., and Barker, J. F., *J. Path. Bact.*, **70**, 263 (1960).

² Illman, O., and Ghadially, F. N., *Brit. J. Cancer*, **14**, 483 (1960).

³ Ghadially, F. N., *J. Path. Bact.*, **80**, 345 (1960).

⁴ Ghadially, F. N., Neish, W. J. P., and Dawkins, H. C., *J. Path. Bact.*, **85**, 77 (1963).

⁵ Figge, F. H. J., *Cancer Res.*, **4**, 465 (1944).

⁶ Lochbrunner, A., *Zool. Jahrb. Abt. allg. Zool. u. Physiologie d. Tiere*, **66**, 389 (1956).

HAEMATOLOGY

Effect of Ferritin on Globin Synthesis

In certain hypersideraemic anaemias the erythroid precursor cells in the bone marrow contain iron deposits. The iron overloaded mitochondria of these sideroblasts are not capable of synthesizing haem at a normal rate¹. Iron-containing deposits are demonstrable not only in mitochondria, but also in other parts of the cytoplasm of these cells. It is, therefore, possible that iron overload also affects globin synthesis directly. In some of these hypersideraemic anaemias pyridoxine has been found to have a therapeutic effect which is likely to be due to the mobilization of the mitochondrial iron deposits². In view of the amelioration of the anaemia of thalassaemia with pyridoxine³, a direct influence of this vitamin on globin synthesis might also be assumed.

The effect of ferritin on globin synthesis was investigated in a cell-free system meant to imitate the conditions in sideroblasts. Reticulocytes, obtained from phenylhydrazine-treated rabbits, were lysed by short exposure to hypotonic tris chloride buffer containing magnesium chloride (ref. 3). The stroma, mitochondria, and intact leucocytes were removed by centrifugation at 15,000g for 15 min. Ribosomes and the enzyme fraction were then obtained and processed according to the method of Allen and Schweet⁴ with the following exceptions: sRNA was not separated from the enzyme fraction; valine-1-¹⁴C was used and the incubation period was 40 min. To some of the incubation mixtures (Table 1) commercial, 2x crystallized, cadmium-free ferritin was added which had been dialysed against 0.05 M tris chloride buffer, pH 7.5. Experiments were also done with the addition of buffered pyridoxal, pyridoxine, pyridoxamine, or deoxyypyridoxine in concentrations from 10 µg to 1 mg per tube.

After incubation the ribosomes and, when present, most of the ferritin were removed by centrifugation at 100,000g for 2 h. The supernate was freed of unincorporated valine-1-¹⁴C by dialysis against running tap water for 36 h. Aliquots of the dialysed samples were then dissolved in hydroxide of hyamine and added to naphthalene-dioxane scintillation fluid⁵. Radioactivity was deter-

Table 1. THE EFFECT OF FERRITIN ON GLOBIN SYNTHESIS WITH VARIATIONS IN THE COMPOSITION OF THE INCUBATION MIXTURE

Tube content	Without ferritin (c.p.m./tube)	With 10 mg ferritin (c.p.m./tube)
Standard mixture*	1,703	473
Standard mixture, double enzyme	3,268	795
Standard mixture, double ¹⁴ C-valine	1,797	997
Standard mixture, double enzyme†	1,315	584
Standard mixture, double GSH	1,726	1,676

* 3 mg ribosomes; 5 mg enzyme; 1 µmole ATP; 120 µg pyruvate kinase; 10 µmoles phosphoenolpyruvate; 20 µmoles GSH; 50 µmoles potassium chloride; 5 µmoles magnesium chloride; 0.25 µmoles GTP; 0.05 ml. of amino-acid mixture (3); 10 µg D,L-valine-1-¹⁴C; 50 µmoles tris chloride buffer, pH 7.5; total volume 1.45 ml.

† 2 µmoles ATP; 240 µg pyruvate kinase; 20 µmoles phosphoenolpyruvate.