ANALYSIS OF FAECAL PORPHYRINS IN THE PORPHYRIAS*

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Although quantitative estimations of faecal porphyrins in South African patients have been reported, these have depended simply on the extraction of an ethereal solution of faecal porphyrin with O·1N and 1·5N HCl to give copro- and protoporphyrin fractions. This abstract reports a more detailed study of the faecal porphyrins of 7 patients with porphyria and of 2 normal subjects.

The normal subjects showed no evidence of disordered porphyrin metabolism. The porphyric patients included 3 with South African genetic, 3 with acquired and 1 with Swedish genetic porphyria.** Only 1 patient (H.B.) was studied during an acute attack of porphyria.

A report of the analytical methods used is in the press; these include spectroscopy, column and paper chromatography, electrophoresis and a study of crystalline porphyrin esters.

Table I contrasts the total faecal porphyrin as measured by the method of Holti *et al.*[2] with the pattern of porphyrin excretion obtained by our chromatographic analysis. The small amounts of uroporphyrin found may be due to incomplete recovery from the faeces. As expected, protoporphyrin exceeded coproporphyrin in the normal subjects; in the 3 South African genetic porphyrics reported here coproporphyrin exceeded protoporphyrin, but the reverse has also been found, which is more in keeping with the results of analysis by the Holti method. The acquired porphyrics differed considerably from the others; an appreciable amount of the total porphyrin had more than 4 carboxyl groups; uroporphyrin was present in 2 of these patients. The percentage excreted as protoporphyrin was very low. Despite an increased total faecal porphyrin, G.S. was included in this group because of the low level of faecal protoporphyrin and the presence of a considerable amount of porphyrins with 4-8 carboxyl groups.

In all patients a dicarboxylic porphyrin resembling protoporphyrin spectroscopically was found; crystalline protoporphyrin was prepared from one normal patient (F.B.), the Swedish genetic porphyric (M.E.), and all 3

TABLE I. RESULTS OF FAECAL PORPHYRIN ANALYSIS

	Total faecal porphyrin µg./G.	*Carboxyl groups—approximate % of each present*				
		8 Uro	*7-5*	*4 Copro*	*3*	*2 Proto*
Normals						
F.B.	45	0	0	25	<5	70
J.K.	75	0	0	25	5	70
Swedish genetic:						
M.E. (remission) ..	96	0	5	10	0	85
South African genetic:						
A.V. (remission) ..	712	0	<5	55	15	25
H.S. (remission) ..	787	0	5	50	5	40
H.B. (acute) ..	2,257	0	5	45	10	35
Acquired:						
E.C.	130	<5	30	55	5	5
J.T.	280	0	25	60	10	5
G.S.	960	<5	15	60	15	10

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** South African genetic: protocoproporphyria or porphyria variegata. Acquired: urocoproporphyria or symptomatic porphyria. Swedish genetic: pyrolloporphyria or acute intermittent porphyria.[1]

South African genetic porphyrics. These specimens were recrystallized to melt in the range 221° – 224° C. The infrared spectra of protoporphyrin from South African genetic porphyrics H.S. and H.B. agreed precisely with that of protoporphyrin IX from human blood. Faeces of the acquired porphyrics yielded too little protoporphyrin to permit crystallization. A dicarboxylic porphyrin resembling mesoporphyrin was found in varying amounts in the faecal specimens studied.

In normal subjects and in the Swedish genetic porphyric, 70% of the faecal coproporphyrin was isomer I and the remainder isomer III. In both the South African genetic and the acquired porphyrics faecal coproporphyrin was predominantly isomer III, 75 - 80% in the South African genetic and 70% in the acquired. Isomer II was not found. The faecal coproporhyrin from South African genetic patient H.B. was fractionally crystallized to yield coproporhyrins I and III, which gave infrared spectra identical to those of the reference materials.

Possible mechanisms were discussed. It was suggested that in acquired porphyria there is an increased tendency for porphyrinogen to undergo spontaneous oxidation to porphyrin.

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