Melanin Hyperpigmentation in South African Bantu Patients with Toxic Psychosis

THE PROBABLE ROLE OF IRON OVERLOAD

ASHLEY H. ROBINS, M.B. CH.B. UNIV. CAPE TOWN, M.D. UNIV. RAND, D.P.M. R.C.P. LOND., R.C.S. ENG.,
Department of Psychiatry, Johannesburg Hospital and the University of the Witwatersrand, Johannesburg

SUMMARY

Skin melanin concentrations were measured by reflectance spectrophotometry in 3 Bantu male groups of 46 toxic-psychosis patients, 36 schizophrenics, and 21 normal subjects; and in 2 White male groups of 25 alcoholic patients with psychiatric disorders, and 27 normal subjects. The Bantu patients with toxic psychosis had a significantly higher skin melanin content than the schizophrenics and the normals. The group of White alcoholics had a tendency (often not significant) to be more pigmented than the normals, but, proportionately, to a much smaller degree than the corresponding Bantu groups. The marked hyperpigmentation of the Bantu toxic-psychosis patients is explained on the basis of ascorbic acid depletion caused by iron overload after the ingestion of home-brewed alcoholic beverages of high iron content. Ascorbic acid deficit would lead to a reduction of epidermal sulphydryl groups, with the resultant release of tyrosinase inhibition and, thereby, an increased melanogenesis. The same hypothesis is offered to account for the melanin hyperpigmentation of haemochromatosis.


Hyperpigmentation was noted in White non-schizophrenic psychiatric patients drawn from diverse diagnostic categories. The majority suffered from long-standing conditions (e.g. chronic alcoholism, mental subnormality, and dementia) that had predisposed them to dietary insufficiency for prolonged periods: hypovitaminosis was, therefore, incriminated as the most likely explanation for the melanosis. The present study aimed to be more specific and to measure skin melanin concentrations in a diagnostically homogeneous group of patients in whom nutritional factors were clearly delineated. South African Bantu† males with toxic psychosis were selected for this purpose.

This so-called toxic psychosis is a common mental disorder among Bantu psychiatric patients. It presents as an acute and relatively short-lived psychotic illness which is characterized predominantly by features of a confusional state. The psychosis is related to the excessive ingestion of alcoholic drinks frequently accompanied by a heavy intake of dagga (cannabis, marijuana). The alcoholic liquors are usually potent concoctions of home-brewed beverages; they are often obtained from shebeens where they tend to be adulterated with toxic ingredients which impart a greater ‘kick’.

Bantu patients with toxic psychosis were compared with schizophrenic patients and normal subjects, on the basis of skin melanin content. In addition, White patients admitted to a mental hospital, with psychiatric disorders associated with alcohol, were compared with normals. These alcoholic White subjects were comparable with the Bantu patients as regards the common aetiology of the mental state. Thus, pigmentary changes in the Bantu group could be compared with changes in a corresponding White group.

METHOD AND MATERIALS

Reflectance Spectrophotometry

Skin melanin concentrations were measured by reflectance spectrophotometry. The latter is recognized as the best available method for obtaining objective and reproducible measurements of pigmentary variation on a continuous scale of reflectance. Its accuracy in determining skin colour has been established by a study of monozygotic and dizygotic twins.

The instrument used in the present study was the portable EEL Reflectance Spectrophotometer. It was recently used by Wassermann and Heyl in a quantitative investigation of South African racial pigmentation. The practical application of the reflectance spectrophotometer followed essentially the method described by Weiner and Lourie. Reflectance readings were obtained at all 9 Ilford filters (601 - 609); these filters 'sample' the entire visual spectrum from the blue (601) to the red (609) with these dominant wavelengths: 601 - 425 μm; 602 - 465 μm; 603 - 485 μm; 604 - 515 μm; 605 - 545 μm; 606 - 575 μm; 607 - 595 μm; 608 - 655 μm; and 609 - 685 μm. The reflectance value at any one wavelength is inversely proportional to the melanin content; however, readings from filters 608 and 609 represent more accurate indices because they are valid over a very wide range of melanin concentration, and they are relatively unaffected by the skin blood supply.

Testing Procedure

The groups of subjects tested will be discussed below. Subjects were included in this study only if they were of sound physical health. Particular care was taken to exclude those showing even minimal clinical evidence of nutritional disorder. The groups to be compared were tested during the same time of year, to avoid seasonal fluctuations in skin pigmentation.

Skin reflectances were taken from unexposed and exposed areas. The unexposed area selected was the medial aspect of the upper arm; the exposed area selected was the hairless region on the dorsomedial aspect of the hypothenar eminence, about halfway between the head and base of the fifth metacarpal. Readings were obtained

*Date received: 29 March 1972. Based on part of an M.D. thesis accepted by the University of the Witwatersrand.
†Referred to also as Bantu-speaking South African Negroes.
from the unexposed and exposed areas on both sides. As the reflectance varied with different limb positions, the subject was always tested with the upper extremity abducted to 90°.

Description of Subjects

All the patients tested in this study were at Sterkfontein Hospital, Krugersdorp. The following groups (all male) were included:

A. Bantu groups. Subjects in these groups approximated one another in their mean ages. The South African Bantu-speaking tribes do not constitute a homogeneous population, but vary according to the degree of Khoisan (Bushman-Hottentot) admixture. As the Khoisan peoples are of lighter skin colour, the different tribal groups could be expected to show pigmentary differences according to the Khoisan genetic influence. The extent of the latter among the various Bantu-speaking tribes has been determined by Jenkins et al. who used the Gm alleles as genetic markers. On the basis of their figures, the 3 groups of subjects tested below showed an over-all comparability in terms of percentage Khoisan admixture.

(i) Toxic psychotics. There were 46 patients with toxic psychosis. All gave an unequivocal history of excessive ingestion of various alcoholic beverages immediately before hospital admission; many of them also admitted to concurrent dagga smoking. These patients were tested very soon after admission; when they had received no or negligible treatment with psychotropic drugs. They were followed-up to ensure that there had been no change in diagnosis.

(ii) Schizophrenics. There were 36 schizophrenic patients, most of whom presented in the acute phase of the illness. All were tested very soon after admission; thus they had received no or negligible psychotropic medication. The diagnosis of schizophrenia was established by at least 2 psychiatrists, and the patients were followed-up to confirm that the diagnosis remained unaltered.

(iii) Normal subjects. There were 21 normal subjects who had never suffered from any form of psychiatric illness. They were drawn from the hospital staff.

B. White groups. Subjects in these 2 groups were similar with regard to their mean ages. They were born in South Africa.

(i) Alcoholics. There were 25 patients in this group. They had been admitted to Sterkfontein Hospital under the Mental Disorders Act (1916) on account of psychiatric disorders due to excessive and prolonged alcoholic intake. They had long-standing histories of heavy drinking. Before admission, many of these patients had exhibited violent and aggressive behaviour in a state of clouded consciousness; others presented with delirium tremens, alcoholic hallucinosis, alcoholic paranoid state, and Korsakoff’s psychosis. These patients were tested very soon after admission; they had received no or negligible treatment with psychotropic agents.

(ii) Normal subjects. There were 27 subjects in this group who were drawn mainly from the nursing personnel. They had never suffered any form of psychiatric illness.

RESULTS

Skin reflectance readings show an inverse relationship to the skin melanin concentration. The readings from the right and left unexposed areas were averaged, as were those from the right and left exposed areas.

The significance of differences between sample means was determined by the Student t-test. The statistical data were analysed by the University of the Witwatersrand IBM Computer System 360, Model 50.

A. Bantu Groups

(i) Toxic psychotics versus schizophrenics (Table I). The patients with toxic psychosis have significantly lower reflectances at all wavelengths in the unexposed areas, and at all wavelengths in the exposed areas except 685 μm, where the difference just falls short of the 5% significance level (t=1.94), than the schizophrenics.

(ii) Toxic psychotics versus normal subjects (Table I). The patients with toxic psychosis, generally, show highly significant lower reflectances than do the normals, in both unexposed and exposed areas. The only non-significant difference is at 425 μm in the exposed areas.

(iii) Schizophrenics versus normal subjects (Table I). There are no significant differences between these 2 groups at either the unexposed or the exposed areas.

B. White Groups

Alcoholics versus normal subjects (Table II). The alcoholics generally tend to have lower reflectances than the normals. However, the differences are significant only at 545 μm in the unexposed areas, and at wavelengths 545 μm - 655 μm in the exposed areas.

Over-all Findings

1. Bantu patients with toxic psychosis have significantly higher skin melanin concentrations, at unexposed and exposed areas, than both schizophrenic patients and normal subjects. There are no significant differences between the schizophrenic and the normal groups.

2. Alcoholic White patients generally tend to have higher skin melanin concentrations than the normal subjects, but these differences attain significance at only relatively few wavelengths.

DISCUSSION

This study, like a previous one, has failed to confirm the hypothesis of Greiner and Nicolson that schizophrenics have increased melanosis. The Bantu male schizophrenic group did not differ from normals in skin melanin content. However, patients with toxic psychosis were significantly more pigmented than both the schizophrenic and the normal subjects.

All the patients with toxic psychosis gave a definite history of excessive and often prolonged intake of alcoholic beverages before admission; in many cases this was combined with heavy dagga-smoking. These activities may have caused dietary insufficiency and a resultant hyperpigmentation. Certain vitamins are implicated in melanogenesis: deficiencies of vitamin A, nicotinamide, and ascorbic acid can produce increased melanin pigmentation. None of these patients exhibited any clinical stigmata of
TABLE I. SKIN REFLECTANCE READINGS IN BANTU MALE GROUPS: 46 TOXIC PSYCHOTICS (MEAN AGE 25,5); 36 SCHIZOPHRENICS (MEAN AGE 28,3); AND 21 NORMALS (MEAN AGE 27,8)

<table>
<thead>
<tr>
<th>Wavelength (µm)</th>
<th>425</th>
<th>465</th>
<th>485</th>
<th>515</th>
<th>545</th>
<th>575</th>
<th>595</th>
<th>655</th>
<th>685</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arm²</td>
<td>Toxic psychotics</td>
<td>Mean¹</td>
<td>13,2</td>
<td>14,1</td>
<td>12,9</td>
<td>14,3</td>
<td>13,8</td>
<td>15,7</td>
<td>22,0</td>
</tr>
<tr>
<td></td>
<td>SEM</td>
<td>0,35</td>
<td>0,38</td>
<td>0,36</td>
<td>0,40</td>
<td>0,40</td>
<td>0,45</td>
<td>0,66</td>
<td>0,81</td>
</tr>
<tr>
<td></td>
<td>Schizophrenics</td>
<td>Mean</td>
<td>14,8</td>
<td>16,3</td>
<td>15,0</td>
<td>16,7</td>
<td>16,3</td>
<td>18,5</td>
<td>25,8</td>
</tr>
<tr>
<td></td>
<td>SEM</td>
<td>0,45</td>
<td>0,51</td>
<td>0,52</td>
<td>0,60</td>
<td>0,59</td>
<td>0,70</td>
<td>0,84</td>
<td>1,00</td>
</tr>
<tr>
<td></td>
<td>Normals</td>
<td>Mean</td>
<td>15,1</td>
<td>17,5</td>
<td>16,6</td>
<td>17,4</td>
<td>17,7</td>
<td>20,1</td>
<td>27,1</td>
</tr>
<tr>
<td></td>
<td>SEM</td>
<td>0,40</td>
<td>0,56</td>
<td>0,61</td>
<td>0,69</td>
<td>0,68</td>
<td>0,83</td>
<td>1,02</td>
<td>1,19</td>
</tr>
<tr>
<td>Toxic psychotics versus schizophrenics</td>
<td>P &lt; 0,01 &lt; 0,01 &lt; 0,01 &lt; 0,01 &lt; 0,001 &lt; 0,01 &lt; 0,001 &lt; 0,01 &lt; 0,01</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Toxic psychotics versus normals</td>
<td>P &lt; 0,001 VHS &lt; 0,001 VHS VHS VHS &lt; 0,01 &lt; 0,01</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schizophrenics versus normals</td>
<td>P NS NS NS NS NS NS NS NS NS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hand³</td>
<td>Toxic psychotics</td>
<td>Mean</td>
<td>13,7</td>
<td>14,6</td>
<td>13,7</td>
<td>14,5</td>
<td>14,0</td>
<td>15,9</td>
<td>21,8</td>
</tr>
<tr>
<td></td>
<td>SEM</td>
<td>0,26</td>
<td>0,30</td>
<td>0,35</td>
<td>0,30</td>
<td>0,31</td>
<td>0,39</td>
<td>0,54</td>
<td>0,70</td>
</tr>
<tr>
<td></td>
<td>Schizophrenics</td>
<td>Mean</td>
<td>14,7</td>
<td>15,8</td>
<td>14,8</td>
<td>16,0</td>
<td>15,5</td>
<td>17,8</td>
<td>24,1</td>
</tr>
<tr>
<td></td>
<td>SEM</td>
<td>0,32</td>
<td>0,35</td>
<td>0,36</td>
<td>0,36</td>
<td>0,38</td>
<td>0,46</td>
<td>0,61</td>
<td>0,76</td>
</tr>
<tr>
<td></td>
<td>Normals</td>
<td>Mean</td>
<td>14,2</td>
<td>16,7</td>
<td>15,6</td>
<td>16,5</td>
<td>16,3</td>
<td>19,1</td>
<td>25,9</td>
</tr>
<tr>
<td></td>
<td>SEM</td>
<td>0,34</td>
<td>0,43</td>
<td>0,37</td>
<td>0,52</td>
<td>0,44</td>
<td>0,61</td>
<td>0,78</td>
<td>0,82</td>
</tr>
</tbody>
</table>

1. These wavelengths represent Ilford filters 601 - 609.
2. This term refers to the unexposed areas tested.
3. This term refers to the exposed areas tested.
4. The mean is that of averaged right-left reflectances.
5. Very highly significant, i.e. where P<0,0001.

TABLE II. SKIN REFLECTANCE READINGS IN WHITE MALE GROUPS: 25 ALCOHOLICS (MEAN AGE 43,6) AND 27 NORMALS (MEAN AGE 49,3)

<table>
<thead>
<tr>
<th>Wavelength (µm)</th>
<th>425</th>
<th>465</th>
<th>485</th>
<th>515</th>
<th>545</th>
<th>575</th>
<th>595</th>
<th>655</th>
<th>685</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arm</td>
<td>Alcoholics</td>
<td>Mean</td>
<td>34,8</td>
<td>40,8</td>
<td>41,1</td>
<td>41,2</td>
<td>37,8</td>
<td>41,5</td>
<td>55,2</td>
</tr>
<tr>
<td></td>
<td>SEM</td>
<td>1,08</td>
<td>1,12</td>
<td>1,15</td>
<td>1,13</td>
<td>1,02</td>
<td>0,97</td>
<td>0,94</td>
<td>0,66</td>
</tr>
<tr>
<td></td>
<td>Normals</td>
<td>Mean</td>
<td>36,3</td>
<td>41,4</td>
<td>43,1</td>
<td>43,4</td>
<td>40,4</td>
<td>43,8</td>
<td>56,5</td>
</tr>
<tr>
<td></td>
<td>SEM</td>
<td>0,78</td>
<td>0,86</td>
<td>0,83</td>
<td>0,82</td>
<td>0,78</td>
<td>0,65</td>
<td>0,66</td>
<td>0,58</td>
</tr>
<tr>
<td>Alcoholics versus normals</td>
<td>P NS NS NS NS &lt; 0,05 NS NS NS NS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hand</td>
<td>Alcoholics</td>
<td>Mean</td>
<td>27,1</td>
<td>32,5</td>
<td>32,6</td>
<td>33,0</td>
<td>30,0</td>
<td>33,7</td>
<td>45,4</td>
</tr>
<tr>
<td></td>
<td>SEM</td>
<td>0,82</td>
<td>0,96</td>
<td>0,94</td>
<td>0,89</td>
<td>0,83</td>
<td>0,84</td>
<td>0,91</td>
<td>0,80</td>
</tr>
<tr>
<td></td>
<td>Normals</td>
<td>Mean</td>
<td>27,9</td>
<td>32,5</td>
<td>34,1</td>
<td>34,4</td>
<td>33,0</td>
<td>36,5</td>
<td>48,3</td>
</tr>
<tr>
<td></td>
<td>SEM</td>
<td>0,72</td>
<td>0,89</td>
<td>0,84</td>
<td>0,86</td>
<td>0,79</td>
<td>0,75</td>
<td>0,75</td>
<td>0,48</td>
</tr>
<tr>
<td>Alcoholics versus normals</td>
<td>P NS NS NS NS &lt; 0,05 &lt; 0,05 &lt; 0,05 &lt; 0,05 NS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

deficiency disease. The hyperpigmentation (detected by reflectance spectrophotometry), may therefore have been indicative of subclinical hypovitaminosis.

Skin melanin concentrations were determined in White male patients with psychiatric disorders following on alcohol abuse. These patients would also have been susceptible to hypovitaminosis from prolonged drinking. The White alcoholics tended to have higher skin melanin concentrations than normal subjects, but these differences attained significance (P<0,05) at only a small number of wavelengths (Table II); in contrast, the Bantu toxic-psychosis patients showed a highly significant increase in skin melanin (P<0,001 at the majority of wavelengths) over normals (Table I). This disproportionately greater pigmenitary difference in the Bantu comparisons (Fig. 1) suggested the operation of an additional factor within
the toxic psychosis group. It is pertinent that Laura Longmore, in her social anthropological study of Johannesburg Bantu, observed that even fair-complexioned drinkers could rapidly develop skin darkening after the ingestion of adulterated alcoholic concoctions.

Analysis of the various types of beverages drunk by the Bantu showed that the home-brewed liquors are high in mean iron concentration. These brews are prepared in rusted tins and drums from which iron is extracted during fermentation. Over 80% of the iron in the drink is in the ionized form and it is absorbed to the same degree as a simple ferric salt. It has been estimated that many Bantu males derive 50 - 100 mg of iron a day from their alcohol intake alone; the net daily gain by absorption is about 2 - 4 mg which, accumulated over years, explains the high incidence of siderosis by middle age.

Dietary iron overload in the South African Bantu produces a state of ascorbic acid deficiency, and severe cases of siderosis may be causally associated with both scurvy and osteoporosis. Schulz and Swanepoel, on the basis of in vitro data, hypothesized that the massive ferric deposits in the tissues (especially in the duodenal and jejunal mucosa) brought about an irreversible oxidative catabolism of ascorbic acid. Subsequently, Lynch et al demonstrated that, before and after ascorbic acid loading, siderotic Bantu subjects, whether clinically scorbutic or not, had a lower ascorbic acid excretion, a higher excretion of oxalic acid (the oxidation end-product) and a more rapid plasma clearance of ascorbic acid than did non-siderotic controls. The same pattern of metabolic disturbance emerged in White subjects with iron overload due to idiopathic haemochromatosis and transfusional siderosis.

Ascorbic acid has an important function in maintaining optimal sulphhydril (thiol) groups in body tissues. In the skin sulphhydril groups exert an inhibitory effect on melanogenesis by chelating the copper required for the enzymatic activity of tyrosinase, although more complicated biochemical mechanisms have been invoked. Rothman et al reported the presence of a sulphhydryl-containing compound in human epidermis which inhibited melanin biosynthesis from tyrosine and dopa. Halprin and Ohkawara cited the sulphhydryl-containing compound, reduced glutathione, as the specific inhibitor of melanogenesis; they found Negro skin to have less reduced glutathione and glutathione reductase than Caucasian skin. Seiji et al demonstrated an inverse relationship between sulphhydril group content and melanin formation in melanoma tissue.

In the presence of ascorbic acid, melanin cannot be formed by the action of tyrosinase on tyrosine or dopa until all the ascorbic acid has been oxidized. Large doses of ascorbic acid have decreased Addisonian pigmentation. In addition to maintaining sulphhydril groups, ascorbic acid (being a reducing agent) can convert melanin to a lighter-coloured product, called reduced melanin.

Ascorbic acid deficiency would, therefore, deplete the concentration of epidermal sulphhydril groups and thereby release tyrosinase from sulphhydril inhibition, with consequent hyperpigmentation. Thus, the melanosis of the Bantu toxic-psychosis group may be related to an ascorbic acid deficit induced by dietary iron overload. The characteristic feature of all the patients in this group was their excessive consumption of intoxicating alcoholic drinks (usually home brews of high iron content) before admission. Many of these patients also gave a long history of heavy drinking. In the Bantu, drinking bouts are reflected by increased concentrations of iron in the stools. It seems reasonable to speculate that the intense drinking period which precipitated the toxic psychosis, was para-

![Fig. 1. Skin reflectance curves in toxic psychotics, alcoholics and normals.](image-url)
led by increasing iron ingestion. The latter could have overwhelmed the mucosal controlling mechanism of the gut; the surplus absorption of iron could then have created the potential for an accelerated oxidative catalolism of ascorbic acid. Studies of leucocyte ascorbic acid showed its tendency to vary inversely with the serum iron concentration: the lowest levels were generally associated with the highest beer-drinkers. Furthermore, these heavy drinkers and the patients with toxic psychosis rarely ate fruit; therefore, they had in addition a superimposed dietary inadequacy of ascorbic acid.

The alcoholic White subjects, like the Bantu patients with toxic psychosis, gave a history of excessive alcohol consumption followed by psychiatric disorder. The White patients showed proportionately a much smaller trend towards hyperpigmentation than did the Bantu patients (Fig. 1). On the assumption that both the White and Bantu groups of patients had been subject to a poor nutritional background, this discrepancy may be ascribed to the relatively low iron ingestion by the White alcoholics. However, even among the latter, there are several relevant factors: alcohol itself potentiates the absorption of ferric iron by the gut; some subjects with chronic pancreatitis and liver disease have an increased iron absorption; and most European and American wines contain iron (although in considerably lesser amounts than in the Bantu drinks). Despite all these influences, it has been acknowledged that severe dietary siderosis is a rarity among heavy drinkers of all population groups other than the South African Bantu.

The other condition which may well be associated with the ingestion of Bantu home-brewed intoxicating liquors and skin hyperpigmentation is the symptomatic form of porphyria cutanea tarda. There is evidence to link iron overload with cutaneous porphyria; this has been discussed by Epstein and Redeker who induced remissions of the porphyria by repeated phlebotomies. Another pertinent aspect is that a certain porphyrinogenic fungus may contaminate the alcoholic beverages, with the possible causation of symptomatic porphyria. However, the patients with toxic psychosis tested in the present study showed no signs of cutaneous porphyria. Furthermore, tests for urinary porphyrins, done on such patients, were consistently negative.

The hyperpigmentation noted in the toxic psychosis group was present in both unexposed and exposed areas, unexposed areas showing, generally, a more significant pigmenitary increase (Table I). In cutaneous porphyria, hyperpigmentation has been specifically described in the light-exposed areas.

Lamont et al. noted that Bantu patients with severe siderosis developed a grey-black pigmentation of the facial skin which they called the 'coal-heaver's' look. Hyperpigmentation is one of the commonest signs in haemochromatosis, and occurs in about 90% of cases at the time of diagnosis; hence the term 'bronzed diabetes'. Although this abnormal pigmentation has been attributed to the pigments melanin and haemosiderin, recent studies have suggested that it is due exclusively to melanin. One case report discussed a patient with both haemochromatosis and vitiligo: the vitiliginous areas remained pure white despite their large dermal iron deposits.

The aetiology of the melanosis in haemochromatosis is obscure. Suggestions have related the hyperpigmentation to adrenal hypofunction (there is little direct evidence to support this) or to enhanced melanogenesis through the increased oxidation of tyrosine by the iron deposits. Another explanation is that the latter bind epidermal sulphhydryl groups and thereby release tyrosine inhibition. These theories are weakened by the finding that in haemochromatosis, haemosiderin occurs in the connective tissue of the corium where it is usually concentrated around sweat gland acini. No haemosiderin was present in the epidermis, although the basal cells contained increased melanin. It is also relevant that Wassermann found the iron content of hair melanoprotein in Bantu samples to be of the same order as in Caucasian material, and lower than in Cape Coloured samples, a surprising observation in view of the prevalence of Bantu siderosis.

It is postulated that the melanin hyperpigmentation of haemochromatosis has the same aetiology as that hypothesized to account for the melanosis in Bantu toxic psychosis patients. Haemochromatosis sufferers show a depletion of ascorbic acid, similar to that in Bantu siderotics. As discussed above, this ascorbic acid deficit could produce increased melanogenesis by decreasing the epidermal sulphhydryl groups.

I wish to thank Professor I. A. Hurst of the University of the Witwatersrand for his encouragement in this research project; the Commissioner for Mental Health and Dr N. van der Westhuizen for their permission to test patients at Sterkfontein Hospital, Krugersdorp; Dr E. D. Freed (formerly Psychiater, Sterkfontein Hospital) for making the initial psychiatric assessment of the Bantu patients and particularly for his detailed analysis of diet and toxic factors in the patients with toxic psychosis. I should also like to thank Mr Brian Cohen for computing my statistical data and Miss E. J. Walker for drawing the graph.

REFERENCES