Nutrition in the Australian Aborigines—Effects of the fortification of white flour

M. Kamien*, Joan M. Woodhill†, Silvia Nobile‡, Pat Cameron** and Pam Rosevear††

From The Department of Medicine, University of Western Australia, The Division of Nutrition and Dietetics, Prince Henry Hospital, Little Bay, New South Wales and The Vitamin Laboratories of Roche Products Pty. Ltd., Dee Why, New South Wales

Summary: The nutritional status of 66 part Aborigines was re-examined in 1974—with particular reference to blood levels of haemoglobin and vitamins—after white bread fortified with iron and the vitamins B₁ and PP (niacin) had been available for six and a half months to the population of Bourke, New South Wales. The results found in 1971 and 1974 are compared. A significant improvement from deficient to acceptable blood levels of vitamins B₁ and B₆ was found in 44% and 52% of the subjects respectively. This is attributed to the consumption of fortified bread since the levels of the other vitamins had remained either unchanged or worsened. The biochemical improvement in vitamin B₆ is attributed to the sparing effect of vitamin PP on vitamin B₁ requirement because the conversion of tryptophan to niacin is impaired in vitamin B₆ deficiency. Iron deficiency anaemia in children had decreased by 50% but this could have been due to many other factors besides the iron which had been added to the bread. Clinically there was a marked decrease in angular stomatitis and skin xerosis which could be related to the biochemical improvement of the two B-vitamins and a decrease in active trachoma and supplicative otitis media probably due to intensive treatment received since 1971. The results of this study and the extent of biochemical vitamin B₁ and B₆ deficiency found in other groups, indicate that fortification of bread may be of benefit to the community as a whole.

Australia has been regarded by its legislators as a land of plenty. In recent years no national nutrition survey sampling the population as a whole has been conducted but evidence from current studies is accumulating that there are socio-economic groups in the white Australian population who are consuming an inadequate diet. Many of these people would be amongst that 5·5 to 7·7% of the population which was concluded by Henderson to be “in need”. The existence of nutritional problems amongst Aboriginal people is not new. More than 20 years ago Wilson reported inadequate diets in Aborigines particularly in regard to calcium, vitamin A and vitamin C in Western Australia, and in the Northern Territory. More recently, serum folate levels of less than 2·7 ng per ml were found in 58% of 52 Aborigines tested in Western Australia. Jose and Welch showed that growth retarded Aboriginal children in Queensland had deficiencies in serum folate, carotene, calcium, iron and vitamin C. Using an ascorbic acid saturation test, Stuart and Connellan found evidence suggesting that 51% of 65 Aboriginal children under the age of 15 months in Queensland had poor vitamin C status and that the breast milk of 80% of the mothers tested had less than 3 mg vitamin C per 100 ml, and this tended to be associated with poor vitamin C excretion in...
their babies. Nobile\textsuperscript{11} reported that a considerable number of Aboriginal children and particularly their mothers in Western New South Wales had multiple biochemical deficiencies of vitamins. In a random sample of the Aboriginal population of Bourke studied by the authors in 1971\textsuperscript{12}, 45% of 85 individuals of all age groups had four or more low blood vitamin levels. In particular children below the age of three and women of child bearing age had low blood vitamin levels.\textsuperscript{12} All these investigators emphasized the need to improve the nutritional status of the Aborigines.

This paper reports the results of a follow-up study of the nutritional status of 66 Aborigines after white bread fortified with iron and the vitamins B\textsubscript{1} and PP (niacin) had been available for six and a half months to the population of Bourke, New South Wales. Clinical features and blood levels of haemoglobin and vitamins of the 66 subjects are compared.

Population and Survey Methods
Sixty-six part Aborigines from all age groups as shown in Table 1 were re-examined. They comprised 52 of the 91 subjects previously studied\textsuperscript{12} who were still resident in Bourke and 14 of the 17 members of two families who had agreed to take part in an extensive dietary study in 1972. These two families were selected as examples of the best dietary habits and living conditions of the Aborigines in the town and the reserve of Bourke.\textsuperscript{13} The first investigations were conducted during August-October, 1971 and in April 1972 and the follow-up study in April, 1974. Individual anthropometric, haematological, clinical and biochemical data were collected using the methods described by Kamien \textit{et al.}\textsuperscript{13} The significance of the difference between the two studies was evaluated statistically by means of the Student T-test.

Nutrition Education and Nutrition Measures
Following the initial studies in 1971\textsuperscript{12} and 1972\textsuperscript{13} efforts were made by various people, including a community health nursing sister, to help the Aborigines to become more food conscious. A report of the dietary findings was provided to the mothers of the two families with recommendations as to how to improve their family diet in general and in particular their own diet which was the least satisfactory.\textsuperscript{13}

The nutritional study which was conducted in April 1972 on the two families considered to represent the best dietary habits of those living in the town and on the reserve was based on weighing individual food intakes for six consecutive days, on vitamin analysis in the food and blood and on medical examination. Seventeen people aged 2–39 years were surveyed. The study showed that bread provided a mean of 33\% of calories (range 19–44\%) and 30\% of protein (range 12–43\%) for the town family and 30\% of calories (range 22–35\%) and 25\% of protein (range 14–32\%) of the family on the reserve.\textsuperscript{13} Since bread provided the highest source of calories and the second highest source of protein, it seemed the ideal food to fortify. The local baker agreed—as a public health measure—to add iron and vitamins to the white flour used in bread. The level of fortification permitted by the regulations under the Pure Food Act of New South Wales (1908, 1971)\textsuperscript{14} was chosen to provide 1·1 mg vitamin B\textsubscript{1}, 11·0 mg vitamin PP (niacin) and 10·0 mg iron per reference quantity, i.e. per 8 ounces of fresh bread. Iron was added in the form of ferrous sulphate. Vitamin B\textsubscript{1}, had to be omitted because the yellow colour which it imparted to the bread was not acceptable to some people in Bourke. Fortified bread became available for consumption in August 1973. It raised the contents of vitamin B\textsubscript{1} from 0·09 mg to 0·43 mg, of vitamin PP from 1·8 mg to 6·3 mg and of iron from 1·6 mg to 3·0 mg per 100 g of fresh bread. These are average figures (+5\%) found on direct analyses of several bread samples. Fortified flour was made available also to one of the two families for making damper and fried scones.

On 1st March, 1974 following a press report (\textit{National Times} No. 160, February 25th, 1974)\textsuperscript{15} claiming that the inhabitants of Bourke were being used as "guinea pigs" and that the iron supplementation of bread could cause haemochromatosis and Cooley's anaemia, the baker decided to cease fortifying the bread.

In order to evaluate the effect of this nutritional measure, the 66 subjects described in Table 1 were examined at the end of April 1974, eight weeks after flour fortification ceased.

Economic and Dietary Situation in Bourke in April, 1974
The first survey in October, 1971 had been performed at a time of full employment. The follow-up study in April, 1974 took place during a period of hardship for the Aboriginal people. There had been a major flood in the area and unemployment relief grants had ceased. Most subjects were now living on social service benefits. The average weekly per caput income of the total population from all

\begin{table}[h]
\centering
\begin{tabular}{|l|l|l|}
\hline
\textbf{Age in 1971} & \textbf{Sex} & \textbf{No. of subjects re-examined} \\
\hline
\textbf{(years)} & & \\
\hline
< 1 & M & 5 \\
& F & 5 \\
1–2 & M & 7 \\
& F & 6 \\
3–9 & M & 4 \\
& F & 8 \\
10–14 & M & 6 \\
& F & 4 \\
15–29 & M & 4 \\
& F & 5 \\
30–54 & M & 3 \\
& F & 6 \\
> 55 & M & 2 \\
& F & 1 \\
\hline
\end{tabular}
\end{table}
TABLE 2
Most commonly found physical signs in 1971 (Pre-fortification) and in 1974 (Post-fortification). Children's anthropometry

<table>
<thead>
<tr>
<th>Age group in 1971</th>
<th>0-2</th>
<th>3-9</th>
<th>10-14</th>
<th>15-29</th>
<th>30-54</th>
<th>55+</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number tested</td>
<td>23</td>
<td>12</td>
<td>10</td>
<td>10</td>
<td>8</td>
<td>3</td>
<td>66</td>
</tr>
</tbody>
</table>

**Physical signs**
- Active trachoma
  - 1971: 10
  - 1974: 5
- Severe dental caries
  - 1971: 5
  - 1974: 4
- Gingivitis
  - 1971: 1
  - 1974: 1
- Spongy bleeding gums
  - 1971: 1
  - 1974: 1
- Angular stomatitis
  - 1971: 3
  - 1974: 0
- Hepatomegaly > 2 cm
  - 1971: 4
  - 1974: 1
- Skin xerosis
  - 1971: 8
  - 1974: 1
- Suppurative otitis media
  - 1971: 10
  - 1974: 3
- Upper respiratory tract infection
  - 1971: 15
  - 1974: 18

**Anthropometry**
- < 10th Percentile for height
  - 1971: 16
  - 1974: 19
- < 10th Percentile for weight
  - 1971: 11
  - 1974: 16

TABLE 3
Haematological parameters found in 1971 (Pre-fortification) and in 1974 (Post-fortification)

<table>
<thead>
<tr>
<th>Age group in 1971</th>
<th>0-2</th>
<th>3-9</th>
<th>10-14</th>
<th>15-29</th>
<th>30-54</th>
<th>55+</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number tested</td>
<td>19</td>
<td>10</td>
<td>7</td>
<td>9</td>
<td>9</td>
<td>3</td>
<td>57</td>
</tr>
</tbody>
</table>

**Haemoglobin ≤ 11 g/100 ml**
- Pre-fortification 1971
  - 7
- Post-fortification 1974
  - 3

**Microcytic hypochromic blood film**
- Pre-fortification 1971
  - 6
- Post-fortification 1974
  - 4

**White cell count > 11,000/mm³**
- Pre-fortification 1971
  - 9
- Post-fortification 1974
  - 8

**Mean Haemoglobin g %± 1 SD**
- Pre-fortification 1971
  - 11.2±1.3
- Post-fortification 1974
  - 11.8±0.9
sources had fallen from eight to just under seven dollars per week. At the same time the cost of food had risen by over 25%.

Despite the current economic difficulties of Aborigines in Bourke, the general standard of living had marginally improved due to a programme of community development. Housing had become more plentiful and overcrowding, though still severe, had diminished. Aboriginal children were still suffering from illness episodes but, mainly due to better delivery of health care, the degree of severity had diminished from 20% of all hospital admissions to the Bourke District Hospital in 1971 to 13% in 1974. The average hospital stay had also decreased from 7.8 to 5.5 days over the same period of time.

A qualitative survey of food intake was conducted by two of the authors (M. K. and P. C.) at the time of the 1974 survey. Less butter and margarine were being consumed due to the difficulty of storing these items without refrigeration during the summer months. Distribution of school milk had also ceased at the end of the 1973 school year. The children of one of the surveyed families had increased their intake of oranges. Apart from this, there was no apparent change in the dietary habits of those who participated in this survey in 1974 from their recorded dietary patterns in 1971 and 1972.

**Results**

**Clinical Examination**

The data of the most commonly found physical signs in 1971/72 and in 1974 are summarised in Table 2. The most marked changes were the decrease in angular stomatitis, skin xerosis, active trachoma and suppurative otitis media. Table 2 compares also percentiles of height and weight of the children in the two studies.

**Haematology**

Haematological results in the various age groups are shown in Table 3. Frequency distribution of haemoglobin values of the whole group and the 35 children under 10 years of age is illustrated in Figure 2.

There was a statistically significant improvement ($P < 0.05$) in haemoglobin levels in young children, a decrease in the number of levels below 11 g% and a decrease of a microcytic hypochromic blood film in 50% of the subjects. All children who were discovered in 1971 to be iron deficient and to be infested with Giardia lamblia (which was noted to be associated with iron deficiency anaemia) had been treated.
Biochemical Levels of Vitamins*

Figure 1 shows the frequency distribution according to plasma levels of vitamin A, its provitamin β-carotene and the vitamins E, C and B<sub>1</sub> (folic acid) in the 66 subjects and the 35 children sampled in 1971/72 (pre-fortification) and in 1974 (post-fortification). The means ± 1 SD and the significance of the differences are indicated.

*The standards adopted for the evaluation of blood vitamin levels of the 66 subjects examined in 1971 and 1974 are the same as those reported and discussed by Nobile<sup>1</sup> and are shown in Table 4.

Figure 2 likewise illustrates haemoglobin values and the results of the functional tests in the erythrocytes used to assess status of the vitamins B<sub>1</sub>, B<sub>2</sub> and B<sub>6</sub> in the same individuals.

Figure 3 represents the number of individuals and of the children whose haemoglobin and blood vitamin levels in 1974 had either improved from low to adequate, remained unchanged (low or adequate) or deteriorated from adequate to low.

These data show a highly significant (P < 0.005) deterioration in the plasma levels of vitamin A and its provitamin A in the whole group and the children, and no significant
change in the plasma levels of vitamins E, C and B₆ (folic acid). With respect to vitamin B₂, the results of the erythrocyte glutathione reductase test (FAD* effect) in 1974 compared to 1971 showed no significant change in the total population and the 35 children. The number of subjects with grossly abnormal vitamin B₂ values had decreased but there was a significant increase of deficiency amongst the women of child bearing age. The 1974 (post-fortification) results for vitamins B₁ and B₆, which were measured by the transketolase test (TPP⁺% effect) and the EGOT⁺ test (P-5'-P % effect) respectively, indicate a highly significant biochemical improvement \( (P < 0.005) \) in the children and in the whole group.

The improvement in vitamin B₁ and particularly in vitamin B₆ status is evidenced by the reduced number of low levels (Fig. 2—vitamin B₁: 33% in 1971, 14.5% in 1974; vitamin B₆: 86% in 1971, 36% in 1974), by the individual improvement noted in 1974 (Fig. 3—vitamin B₁: 44%, vitamin B₆: 52%) and by the decrease of the group means from marginal (B₁) and deficient (B₆) into the respective adequate ranges (Fig. 2).

**Profile of Women of Child Bearing Age. Pregnancy and Lactation**

There were nine women of child bearing age amongst the 66 subjects, their state of pregnancy and lactation, their haemoglobin and blood levels of vitamins and their intakes of iron, folic acid and oral contraceptives are shown in Table 4.

Pregnancy, lactation and the intake of oral contraceptives was associated with marked biochemical deficiency of several vitamins particularly of the vitamins C, B₆ (folic acid) and B₂. Fortification of flour did not appear to
### TABLE 4
Profile of 9 women of child bearing age. Pregnancy and lactation

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Condition</th>
<th>Year of survey</th>
<th>Number of pregnancies</th>
<th>Age (yrs)</th>
<th>HB (%)</th>
<th>HA %</th>
<th>Pro-A, mg%</th>
<th>E, mg%</th>
<th>C, mg%</th>
<th>B12, mg%</th>
<th>B1, mg%</th>
<th>B2, mg%</th>
<th>EGR</th>
<th>FAD %</th>
<th>P-5-P %</th>
<th>Medical comments</th>
<th>Medications</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Non pregnant</td>
<td>1971</td>
<td>0</td>
<td>15</td>
<td>14-8</td>
<td>109</td>
<td>0.47</td>
<td>0.73</td>
<td>1.1</td>
<td>358</td>
<td>10</td>
<td>12</td>
<td>77</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
</tr>
<tr>
<td>1.</td>
<td>Non pregnant</td>
<td>1974</td>
<td>0</td>
<td>17</td>
<td>14-1</td>
<td>150</td>
<td>0.72</td>
<td>0.42</td>
<td>5.1</td>
<td>283</td>
<td>13</td>
<td>36</td>
<td>49</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
</tr>
<tr>
<td>2.</td>
<td>Non pregnant</td>
<td>1971</td>
<td>0</td>
<td>17</td>
<td>15-5</td>
<td>185</td>
<td>0.77</td>
<td>0.52</td>
<td>2.1</td>
<td>242</td>
<td>12</td>
<td>7</td>
<td>84</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
</tr>
<tr>
<td>2.</td>
<td>Post-partum 3/52</td>
<td>1974</td>
<td>1</td>
<td>19</td>
<td>13-2</td>
<td>77</td>
<td>0.84</td>
<td>0.25</td>
<td>8.5</td>
<td>378</td>
<td>29</td>
<td>27</td>
<td>140</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
</tr>
<tr>
<td>3.</td>
<td>Pregnant 18/52</td>
<td>1971</td>
<td>2</td>
<td>20</td>
<td>11-5</td>
<td>99</td>
<td>0.98</td>
<td>0.31</td>
<td>32-4</td>
<td>187</td>
<td>31</td>
<td>6</td>
<td>131</td>
<td>nil</td>
<td>nil</td>
<td>Follic acid 5 mg</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Non pregnant</td>
<td>1974</td>
<td>2</td>
<td>12-2</td>
<td>162</td>
<td>0.72</td>
<td>0.23</td>
<td>2.3</td>
<td>88</td>
<td>23</td>
<td>24</td>
<td>77</td>
<td>nil</td>
<td>nil</td>
<td>Contraceptives</td>
<td>nil</td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Non pregnant</td>
<td>1971</td>
<td>5</td>
<td>23</td>
<td>13-0</td>
<td>226</td>
<td>0.69</td>
<td>0.12</td>
<td>1.8</td>
<td>203</td>
<td>17</td>
<td>8</td>
<td>89</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
</tr>
<tr>
<td>4.</td>
<td>Pregnant 9/12</td>
<td>1971</td>
<td>6</td>
<td>25</td>
<td>11-2</td>
<td>110</td>
<td>1.40</td>
<td>0.06</td>
<td>1.5</td>
<td>128</td>
<td>21</td>
<td>27</td>
<td>109</td>
<td>nil</td>
<td>nil</td>
<td>Folic acid 5 mg</td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Post-partum 1/52</td>
<td>1974</td>
<td>5</td>
<td>29</td>
<td>11-0</td>
<td>230</td>
<td>1.17</td>
<td>0.29</td>
<td>16.5</td>
<td>36</td>
<td>21</td>
<td>224</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
<td>Contraceptives</td>
<td>nil</td>
</tr>
<tr>
<td>6.</td>
<td>Non pregnant</td>
<td>1971</td>
<td>10</td>
<td>30</td>
<td>15-5</td>
<td>224</td>
<td>0.74</td>
<td>0.07</td>
<td>2.0</td>
<td>160</td>
<td>23</td>
<td>3</td>
<td>155</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
</tr>
<tr>
<td>6.</td>
<td>Non pregnant</td>
<td>1974</td>
<td>10</td>
<td>13-5</td>
<td>140</td>
<td>1.22</td>
<td>0.14</td>
<td>2.2</td>
<td>104</td>
<td>25</td>
<td>33</td>
<td>132</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
<td>nil</td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>Breast feeding 7/12</td>
<td>1971</td>
<td>9</td>
<td>34</td>
<td>13-7</td>
<td>197</td>
<td>1.76</td>
<td>0.21</td>
<td>4.8</td>
<td>408</td>
<td>15</td>
<td>14</td>
<td>93</td>
<td>nil</td>
<td>nil</td>
<td>Recurrent UTI, catars, slightly overweight</td>
<td>nil</td>
</tr>
<tr>
<td>7.</td>
<td>Breast feeding</td>
<td>1974</td>
<td>11</td>
<td>37</td>
<td>13-8</td>
<td>131</td>
<td>1.28</td>
<td>0.15</td>
<td>2.3</td>
<td>88</td>
<td>18</td>
<td>23</td>
<td>86</td>
<td>nil</td>
<td>nil</td>
<td>Contraceptives</td>
<td>nil</td>
</tr>
<tr>
<td>8.</td>
<td>Non pregnant</td>
<td>1971</td>
<td>15</td>
<td>39</td>
<td>14-0</td>
<td>144</td>
<td>0.88</td>
<td>0.12</td>
<td>3.5</td>
<td>654</td>
<td>21</td>
<td>13</td>
<td>33</td>
<td>nil</td>
<td>nil</td>
<td>Nasolabial seborrhoea, moderate gingival decrease</td>
<td>nil</td>
</tr>
<tr>
<td>8.</td>
<td>Non pregnant</td>
<td>1974</td>
<td>15</td>
<td>42</td>
<td>12-2</td>
<td>150</td>
<td>0.88</td>
<td>0.12</td>
<td>3.5</td>
<td>654</td>
<td>21</td>
<td>13</td>
<td>33</td>
<td>nil</td>
<td>nil</td>
<td>Nasolabial seborrhoea, moderate gingival decrease</td>
<td>nil</td>
</tr>
<tr>
<td>9.</td>
<td>Breast feeding 15/12</td>
<td>1971</td>
<td>11</td>
<td>39</td>
<td>14-5</td>
<td>144</td>
<td>1.17</td>
<td>0.50</td>
<td>2.0</td>
<td>336</td>
<td>25</td>
<td>13</td>
<td>115</td>
<td>nil</td>
<td>nil</td>
<td>Severe periodontal disease, catars, gingivitis</td>
<td>As above</td>
</tr>
<tr>
<td>9.</td>
<td>Non pregnant</td>
<td>1974</td>
<td>11</td>
<td>42</td>
<td>12-2</td>
<td>79</td>
<td>0.96</td>
<td>0.25</td>
<td>8.8</td>
<td>180</td>
<td>15</td>
<td>21</td>
<td>64</td>
<td>nil</td>
<td>nil</td>
<td>As above</td>
<td>nil</td>
</tr>
</tbody>
</table>

Pro-A β-Carotene, Vit B, Folic acid - not tested, TKA Transketolase, TPP Thiamine pyrophosphate, EGR Erythrocyte glutathione reductase, FAD Flavin adenine dinucleotide, EGOT Erythrocyte glutamate oxaloacetate transaminase, P-5-P Pyridoxal-5-phosphate
have had much effect on the vitamins B₁ and B₆ status of these women.

Discussion

Butter, margarine and milk are the main sources of vitamin A in the Aboriginal diet.¹²,¹³ The decreased consumption of these foods may well be responsible for the significant increase of biochemical vitamin A and provitamin A deficiency in the children and in the whole group in the 1974 survey.

With respect to plasma levels of vitamin C there was no marked change in either group but amongst the children re-examined there were 5 who were receiving rose hip syrup and they had increased their plasma level from 0·25 mg ± 0·26/100 ml in 1971 to 0·59 mg ± 0·24/100 ml in 1974.

Direct nutrition education had achieved few changes in the dietary habits of the Aborigines. The parents of the family who lived in the town did begin to purchase oranges regularly for their children. This was reflected in a mean rise of plasma vitamin C from 0·30 mg ± 0·08/100 ml to 0·68 mg ± 0·17/100 ml in the six children re-examined. No change was found in the blood levels of the parents who had multiple deficiencies.

The family who lived in the reserve had used fortified flour for making damper and fried scones until it was lost in the floods. The five children of this family had maintained their adequate vitamin B₁ status and significantly improved to normal their vitamin B₁ levels. The father was not re-tested but no improvement was found in the mother who meanwhile had had two pregnancies. (Table 4, Case 7).

The first study (in 1971)¹² was originally intended as one of the baseline measurements of health in the Bourke Aboriginal population. It was only after the baker in Bourke had agreed to fortify his bread, as a public health measure, that the main author realised that a follow-up survey would yield some data on the effects of adding nutrients to bread. Consequently, a short-coming in this study is the two to two and a half year interval between the baseline measurements and the instituting of bread fortification. Although a dietary enquiry showed that no qualitative or quantitative difference in dietary habits had occurred in that period, it is still only an assumption that the baseline blood vitamin levels at the beginning of the fortification of bread were similar to those of 1971–72. However, the marked biochemical improvement in the blood vitamin B₁ and B₆ levels, but not in any of the other blood vitamin levels, suggests—in the absence of other known variables—that this was due to the consumption of fortified bread.

In 1972 the analysed vitamin B₁ intakes of the two families averaged 0·56 mg per day (overall individual 6 day range 0·2–1·0 mg). This corresponded to an average intake of 0·36 mg vitamin B₁ per 1000 calories because their average caloric intake was only 1530. The dietary allowance is 0·4 mg vitamin B₁ per 1000 calories.¹⁶ The main source of vitamin B₁ was bread.¹³ Biochemical deficiency of vitamin B₁ was also found in 42% of the 90 Aborigines studied in 1971.¹²

Fortification of bread raised the contents of vitamins B₁ and PP (niacin) fourfold and that of iron twofold. The biochemical vitamin B₁ improvement in 52% of the 66 subjects in the 1974 post-fortification study is not surprising. Vitamin PP was not measured in blood. Why the improvement in vitamin B₆ status? Vitamin B₆ was not added to the flour but the increased intake of vitamin PP (niacin) from fortified bread must have had a sparing effect on vitamin B₆ requirement. Vitamin B₆ in the form of pyridoxal phosphate acts as coenzyme of the kynureninase in the production of niacin from tryptophan. This conversion is impaired in vitamin B₆ deficiency as was demonstrated in experiments with animals and humans fed a vitamin B₆ deficient diet.¹⁷ In 1972, the analysed dietary vitamin B₆ intakes of the two Aboriginal families¹³ were very low on American standards.¹⁸

The intake of dietary niacin of one of the two mothers who consumed less than 2000 calories was below 13 mg¹⁹ and that of the 13 children ranged from 4·0–6·0 mg/1000 calories (the allowance is 6·6 mg), and were close to or below 7·5 mg dietary niacin/day which is the quantity associated with the production of pellagra.²⁰ Cases of chronic pellagra had been seen in Bourke.¹² The National Health and
Medical Research Council\textsuperscript{16} does not specify any allowance for vitamin B\textsubscript{6} and no data are available on the vitamin B\textsubscript{6} content of Australian foods.\textsuperscript{21} The Australian allowance for niacin is calculated in terms of niacin equivalents by means of the formula:

\[
\text{dietary niacin} - (0.16 \times \text{dietary protein in grams})
\]

expressed in milligrams per 1000 calories.\textsuperscript{16}

Such evaluation is based on the assumption that a constant fraction of the protein intake is tryptophan and that this is fully available as niacin precursor in a 60 mg to 1 mg ratio. In the recent edition of the Recommended Dietary Allowances, the National Research Council (NRC) of the United States of America has recognised that the allowance in niacin equivalents is misleading because the contribution of tryptophan is “variable and unpredictable”.\textsuperscript{19}

It is perplexing however that the role of vitamin B\textsubscript{6} in the utilisation of tryptophan as vitamin PP precursor is not considered in the compilation of dietary allowances.\textsuperscript{16, 18, 19}

A commonly used biochemical index of vitamin B\textsubscript{6} deficiency is the presence of xanthurenic acid in the urine after tryptophan load.\textsuperscript{17} This is at the same time an index of impaired conversion of tryptophan to nicotinic acid which occurs at the hydroxykynurenine stage. In vitamin B\textsubscript{6} deficiency this compound forms xanthurenic acid instead of being converted to hydroxyanthranilic acid, an intermediate metabolite of nicotinic acid.\textsuperscript{17} Wachstein\textsuperscript{22} reports that xanthurenic acid excretion in pyridoxine deficient rats could be depressed by the administration of nicotinic acid. Another example of the metabolic interrelationship between the two vitamins is the EGOT test used in this study. It determines transaminase activity in the erythrocytes in the formation of glutamate and oxaloacetate, the effect of added coenzyme (pyridoxal phosphate = vitamin B\textsubscript{6}) onto the system and the rate of conversion of oxaloacetate to malate, which is nicotinamide adenine dinucleotide (NADH) dependent, and is measured by the decrease of NADH absorption at 334 nm.

Hypochromic anaemia has been also found in humans fed a vitamin B\textsubscript{6} deficient diet.\textsuperscript{17} It may be speculated that the decrease in hypochromic blood films in 50% of the children in the 1974 post-fortification study (Table 3) was due to the sparing effect of increased vitamin PP (nicotinic acid) intake on vitamin B\textsubscript{6} requirements. The twofold increased iron supply from fortified bread may have been responsible for the improvement of haemoglobin levels in the children. However it is also known that an improvement in haemoglobin levels occurs in most Aboriginal children with increased age and decreased bowel infections and infestations.\textsuperscript{23}

Improvement of clinical features such as active trachoma and otitis media was probably due to the intensive treatment with antibiotics and antiseptics received over the previous two years. The decrease in angular stomatitis and skin xerosis is not fully understood although it may be speculated that the general improvement in the vitamins B\textsubscript{1} and B\textsubscript{6} status may have been a factor since cutaneous lesions have been also associated with vitamin B\textsubscript{6} deficiency.

Bread fortification was not sufficient to improve the nutritional status of the women under the stress of pregnancy and lactation nor that of some of the women who were taking oral contraceptives. These findings reinforce the recommendation made in the previous paper that pregnant Aboriginal women be prescribed a multi-vitamin supplement in addition to iron and folic acid and that these supplements be continued for the duration of breast feeding\textsuperscript{12} and supplied also to women on contraceptives.\textsuperscript{19}

\textbf{Recommendations}

We pointed out in a previous paper\textsuperscript{12} that “it is naive to believe that direct nutrition education would have quick or even any results in view of the abundant literature available on the difficulties of changing attitudes and practice on target populations”.

The outcome of several programmes of nutrition education (some independent of the authors) has had little effect on improving the diet of the Aboriginal people of Bourke. We do not wish to imply that fortification is preferable to nutrition education but nutrition education is a tardy measure to remedy an acute public health problem. We consider the nutritional
status of the Aboriginal population of Australia as poor. This has been repeatedly demonstrated over the last 20 years. Our recommendations are based on the fact that fortification of a staple food is under these conditions the only quick and practical means to ensure that the target population obtains sufficient of the needed nutrients. The results of this study suggest that the provision of a food with improved nutritional value had a significant effect on the overall nutritional status of the 66 Aborigines tested.

Had not the sensationalist and inaccurate report of the National Times forced the local baker to cease bread fortification—which had been devised entirely for the benefit of the Bourke population—fortified bread would be still available in Bourke as it is in other countries such as America, Canada, Denmark and the United Kingdom for the benefit of those sections of the community that are most in need.

It is stressed that the authors are aware that a good diet is better than artificial supplements. Until it is shown that the diet of those Aboriginal groups in the community known to suffer socio-economic hardship meets such criteria, it is recommended that fortification of bread be re-introduced in Bourke and also made available to other Aboriginal communities. It is also considered that fortification of bread be more balanced to include other essential nutrients such as vitamins B₁ and B₆ which are also lost in the processing of cereals and are lacking in the Aboriginal diet.

This would require a change in attitude from rejection to acceptance of the yellow tinge imparted to bread by vitamin B₂. This acceptance would need to be based on the colour being seen as an index of fortification and not of contamination.

Whereas the majority of Australian children are probably not in need of school milk, our results indicate that some populations of Australian school children are in need. It is recommended that where there is a sizeable Aboriginal population the school milk scheme should continue.

All these recommendations are aimed at the improvement of the nutritional status of Aboriginal communities. It is important that they have the endorsement of the Aboriginal communities concerned, as they do in Bourke. It is also important that they are accepted by the white population living in the same area who may also derive some benefit from these nutritional measures.

The 1974 edition of the Recommended Dietary Allowances of the Food and Nutrition Board of the National Research Council of the United States of America cited above states that “there is little evidence that small surpluses of nutrients are detrimental; deficits, even small ones, will, on the other hand, lead to deficiencies over a long period of time”.

Acknowledgements

This study could not have been performed without the collaboration of the local baker, Mr. Morrell, and the Aboriginal population of Bourke. Special thanks are due to Mr. W. Byers and Pastor W. Reid, field officers of the Bourke Aboriginal Advancement Association, and to Mrs. P. Baker, Matron of the Bourke District Hospital, for making the organisational arrangements for the 1974 survey.

References

The Prevalence of Chronic Gastritis in Patients with Gastric Ulcer

Wye-Poh Fung, Swee-Kok Lee and Cho-Yook Tye

From the Departments of Medicine (Medical Unit 2) and Social Medicine, University of Singapore, and the Department of Pathology, General Hospital, Singapore.

Summary: Multiple gastric biopsies were performed with a duodenofiberscope in 58 Chinese subjects. All 58 cases had a gastric ulcer crater, demonstrated by endoscopy, or barium meal, or both. Histological sections of the gastric biopsies showed chronic atrophic gastritis in 12 (20.6%), chronic gastritis in 31 (53.4%), and acute-on-chronic gastritis in 11 (19%). Only two cases or 3.4% had normal gastric mucosa, while 54 cases or 93% had some form of chronic gastritis, on the gastric biopsy. The gastritis was "associated with intestinal metaplasia in 16 cases (27.6%), while two cases had histological evidence of gastric atypia. No significant difference was found in the mean basal and peak acid outputs among the three groups of gastric ulcer cases with chronic atrophic gastritis, chronic gastritis, and acute-on-chronic gastritis. The high frequency of chronic gastritis (93%) occurring in patients with gastric ulcer in the present series suggests that chronic gastritis may be an important aetiological factor in the pathogenesis of gastric ulceration.

In a critical analysis of various theories of the pathogenesis of peptic ulceration, Grossman\(^1\) in 1951 stated that "Many factors that play a role in peptic ulcer are recognised and their effects can be explained; others are recognised but their effects cannot be explained; and certainly still others remain to be recognised". In a review of the pathogenesis of peptic ulcer in 1974, it was stated that Grossman’s statement remains true even today.\(^2\) Although many factors have been found to be involved in the pathogenesis of peptic ulceration, no single cause has been found which is capable of causing peptic ulcer by itself. In the past, most authors have stressed on the importance of gastric acid, since acid appears to be present in every case of peptic ulceration. While it is largely true that acid must be present in the stomach before peptic ulceration can occur, it is also true that many