NOTES BY
TOMANGO LTD.
on the
Prevention of Scurvy
Amongst Native Workers;
Oranges and Orange Juice.

MAY, 1936.
ORANGE JUICE FOR USE AS AN ANTI-SCORBUTIC

SUPPLY.

We can supply large quantities of orange juice on a contract basis, packed in casks of 40 to 100 gallons capacity.

QUALITY.

The juice we supply is natural orange juice, uncooked or heated, in any way, rose-head extracted in our hygienically equipped factory; all the natural fruit cells of the orange are present; it is preserved with sulphur dioxide, suitably sweetened with cane sugar, and makes a very pleasing drink.

VITAMIN C.

The orange juice we supply is tested for Vitamin C, and the quantity present is stated at the time of delivery.

PRICE.

In 40-gallon Casks and upwards—

- Johannesburg and Reef ... 3/- per gallon, delivered nearest railway station.
- Elsewhere ... ... ... 2/6 per gallon, f.o.r. Factory.

Lesser Quantities—

- All Places ... ... ... 3/- per gallon, f.o.r. Factory.

Large Quantities and Contracts—

Prices on application.

Containers (Casks or jars and crates)—

Charged but returnable, railage forward.

They must be consigned by the original receiver from the receiving station and described as "Empty Returns."

Prices are subject to market fluctuation.

TOTS PER GALLON.

One gallon of sweetened orange juice will provide 64 tots of 2½ fluid ounces each.

PRICE PER TOT.

On the basis of 3/- per gallon, delivered, the price of one tot of 2½ fluid ounces is 0.562 pence.

A tot of 2½ fluid ounces per boy per day is recommended as a preventive of scurvy.

TOMANGO LIMITED.
NOTES BY

TOMANGO LTD.

on the

Prevention of Scurvy

AMONGST NATIVE WORKERS;

ORANGES AND ORANGE JUICE.

MAY. 1936
PREFACE.

The object of this brochure is to draw attention to the merits of orange juice for the prevention of scurvy in native labourers.

The brochure discusses Vitamin C deficiency and the nature, distribution and application of this Vitamin for the prevention of scurvy.

The discussion is deliberately confined to the nutritional and economic aspects of Vitamin C avitaminosis in native labourers, and not with the native in his own environment.

The subject of nutrition and food supply of the native labourer is not discussed, and, although we fully realise its importance, we do not wish to include a review in this brochure on so wide and involved a subject.

The brochure is addressed to officials concerned with the welfare of the native labourer, and we trust our review will prove of some small service to them.

The citrus grower will, we think, find information of value to him, and, we feel, will be interested in the possible outlet for orange juice as a source of antiscorbutic ration for native labourers.

We are fully conscious of the incompleteness of our review, and ask the indulgence of our readers for sins of commission and omission. It is because we are interested in oranges and Vitamin C deficiency that we have issued this brochure on a subject which we believe is of national importance.

P.O. Merebank, 
Durban. 
May, 1936. 

TOMANGO LIMITED.
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NOTES BY TOMANGO LTD., ON
THE PREVENTION OF SCURVY
AMONGST NATIVE WORKERS;
ORANGES AND ORANGE JUICE.

In March, 1931, we issued a brochure entitled "Notes by Tomango Limited on Diets for Native Workers, Scurvy, Oranges and Orange Juice."

The publication was favourably received, and the demand soon exhausted all copies.

The present brochure discusses more modern work, and offers information of a practical value, with a review of the more important literature up to the beginning of 1936.

The Bantu population of the Union of South Africa at the 1921 full Union Census is 4,697,813 (1), and the estimated mean population for 1934 is 5,761,900 (1).

On the basis of the estimated figures for 1934, and assuming a ratio of 1:1, males to females, the male population would be 2,880,950.

During the year 1934, the value of gold produced in the Union, including the gold premium valued at £27,795,410, was £1,383,106,305 (1). £ 1,383,106,305

For the same year, 252,036 native workers were employed in the gold mining industry (2). Of the 252,036 natives employed, 47,691 were recruited from the Protectorates, 57,693 from Portuguese Territory, and 1,903 from north of latitude 22° South.

As only 144,749 Union natives were employed, it would appear that approximately only 5% of the male Bantu population were available as suitable for employment in the gold mining industry. Is this unsuitability due to rejection following medical examination? To malnutrition? To vitamin deficiency? Apparently, such is the case, for it is stated in the 1934 Annual Report of the Department of Public Health, Union of South Africa:

"Owing to the poor health and physique of the natives in our own reserves, half of those brought to the examining doctors

(1) Year Book No. 16 (1933–34), Union of South Africa.
(2) S.A. Mining Year Book (1935–36), Johannesburg.
by mine recruiters have to be turned down. For that reason, we have to import some 100,000 natives from our neighbours on the East, and we are now arranging to import labour from northern tropical areas. Our present requirements could all be met from our own native reserves if ill-health, due to entirely preventable causes, were removed.

What are the preventable causes?

In the same official report quoted we read:

"The food in large portions of the native territories consists at certain times almost exclusively of mealie-meal. In times of drought, there is frequently a grave shortage of even this unsatisfactory form of food. Deaths, particularly of children, then occur from frank shortage of calories. But at all times the bulk of our Bantu population subsists largely on one of the most unsatisfactory forms of starch. Mealie-meal is deficient in practically all the important constituents of a properly balanced dietary; protein, vitamins, particularly B, C and D, and minerals such as calcium and phosphorus. The virtual absence of Vitamin B₂ and C is responsible for the sub-pellagric and sub-scorbutic state of such a large proportion of the population of the territories. Outbreaks of pellagra and scurvy are common as soon as these people are made to do hard work. The sub-scorbutic state of mine recruits is fully appreciated on the Witwatersrand. On the mines, experience has proved the necessity of allowing new boys a period of loafing while they are permitted to eat as much as they like of the nourishing food adequately provided with protein and vitamins. These facts indicate clearly that even when mealie-meal is plentiful, the natives are still being starved of the essential constituents of food. This starvation results in much morbidity and consequent mortality amongst them."

Scurvy in natives is again discussed in the 1933 Official Report, and it is stated:

"This disease continues to cause trouble in spite of provision throughout the mines of anti-scorbutic substances to supplement the mealie-meal on which the natives largely subsist. During the year, 273 cases were admitted to mine hospitals for this condition. The number is large when it is borne in mind that only severe cases are treated primarily for this condition. Many accident cases, for instance, develop scurvy, but are not recorded as such for statistical purposes. Florid scurvy develops in recent recruits who are put on hard work underground. The reason for this is that they arrive on the mines in a sub-scorbutic state due to their subsistence in the territories almost exclusively on mealie-meal, which is devoid of Vitamin C.

Owing to the easy life at home, scurvy may not become manifest there, but it quickly shows itself on the mines if the boy is immediately put on hard work without a preliminary feeding
upon the good mine diet. Even in older boys the condition will develop if they deliberately discard the anti-scorbutic substances provided. This is now obviated on many mines by the grinding up of the raw vegetables to a pulp which is stirred into the stew at the end of the cooking process.”

Further reference to scurvy in natives is made in the Annual Report for 1934 of the South African Institute for Medical Research, where it is stated:

“Scurvy has been the most definite deficiency disease noticeable on the mines for many years, and that this disease is of particular importance to South Africa . . . .”

Scurvy, then, is one of the important factors limiting the employment of the native in industries, and is nationally of grave importance. Scurvy in South Africa will be found discussed in papers by MacVicar (1906) (1), Birt (1907), MacRae (1908), Turner (1911), Hewetson (1911), Nightingale (1912), Dyke (1918), Orenstein (1921), Donaldson (1921), Delf (1921), Stammers (1925), Spencer (1926), Williams (1928), Maynard (1928), Park-Ross (1931), S. Ross (1931).

The paper by Dr. S. Ross (1931), of the Victoria Hospital, Lovedale, Cape Province, refers to a series of 208 cases of scurvy, of whom 9 succumbed and 199 recovered. Dr. S. Ross stated that scurvy, open or latent, is fairly common amongst children. Lemon juice was used for treatment.

As it is now proposed to recruit native labour from north of latitude 22° South, we may enquire as to the condition of natives drawn from these parts. In 1910, Fleming, Macaulay and Clark issued a “Report on the prevalence and prevention of scurvy and pneumonia in Southern Rhodesia and amongst native labourers” ; this is cited by McCollum and Simmonds (1929) ; Dry (1933) published a paper on “Avitaminosis in natives of Rhodesia. Treatment of epidemic scurvy by Citrus juices,” and states that he found 60% suffering from scurvy in a large group of labourers in Northern Rhodesia.

In an editorial article in Nature for October 20th, 1934, on “The Native Problem and Research in Africa,” it is stated:

“The degree of physical disability among Kenya natives is illustrated by two recent instances. Among 16,754 men called up for enlistment as carriers, 10,912 were rejected on medical grounds and a further 17% fell out on the line of march to Nairobi ; and in a railway job employing 14,000 men the death rate was 35.4 per thousand, and admissions to hospital were 5,331. While lack of sanitation and ignorance are largely responsible for these conditions, the fundamental cause is generally held to be malnutrition.”

(1) Consult the list of references quoted on pages 41–50 for full title of paper and reference.
Numerous outbreaks of pellagra are also known to occur in areas north of latitude 22° South, as well as in South Africa. Pellagra in South Africa is discussed in papers by Drummond (1913), Mitchell (1914), and Cluver (1929), and for other centres in Africa by Stannus (1919), Shelley (1930), and Corkill (1934). Aykroyd (1933) contributes a useful article on pellagra and states inter alia that in the United States, during 1930, no less than 7,086 deaths occurred from pellagra, and the death rate 5.9 per 100,000; in Roumania, during 1932, a total of 55,013 cases were recorded, with 1,656 deaths and a death rate of 9.0 per 100,000.

Maize is held by some investigators to be the primary cause of pellagra, and due to the presence of toxin in maize or deficiency of amino-acid. The most generally accepted view is that pellagra is due to vitamin deficiency—one of the vitamins of the B2 complex.

Pellagra, Poverty and Maize certainly form an unholy trinity, and with Scurvy are decidedly the nutritional factors retarding the physical development and consequently economic value of our Bantu.

Diseases due to vitamin deficiency are then also likely to prove factors limiting in time the supply of suitable native labour from north of latitude 22° South.

Scurvy.

Scurvy is a disease due entirely to the absence of an adequate supply of Vitamin C, or to use its modern name, Ascorbic Acid.

The symptoms of scurvy are soreness, with bleeding of the gums, and frequently showing “sponginess,” with “raspberry” like swellings; loosening of the teeth; haemorrhages under the thick fibrous membranes covering the surface of bone and in almost every part of the body; dizziness on standing; great depression of spirits; marked lassitude and fatigue; weakness and almost complete powerlessness of the legs; anaemia and other signs which are more within the province of the medical practitioner to recognise. The period of development of scurvy is probably four to eight months.

The deficiency of Vitamin C in nutritional anaemia is discussed by Davidson and Leitch (1934), who state that in experimental scurvy a progressive anaemia results, and quote Mettier, Minot and Townsend (1930) as regards the occurrence of a similar condition in man. Dosage with orange juice results in a reticulocytic rise and a rapid increase in the blood level; Mettier and Chew (1932) find retarded maturation of erythrogenic cells of the bone marrow. Piney (1931) says that in scurvy “there is a failure of hyperplasia of the myeloid tissue rather than atrophy of that normally present”; Hadfield and Garrod (1934) state that “Vitamin C is also required for the maturation of the normoblast: scurvy is therefore accompanied by anaemia, and, since lymphocyte
formation is also impeded by lymphocytopenia.” Vitamin C may be considered “anti-infective” in the limited sense that this deficiency leads to pathological changes paving the way for secondary infection.

Harris (1935) discusses papers by Rinehart and colleagues on the relationship between Vitamin C deficiency to rheumatic conditions. “The suggestion is made that a sub-clinical degree of scurvy may constitute the rheumatic tendency, in which the added factor of infection causes the development of rheumatic fever, and, possibly, the closely allied condition of rheumatoid arthritis.”

Bondurant (1934), in discussing adult scurvy, records marked diuresis with complete disappearance of oedema, follows promptly the use of anti-scorbutics such as the juice of *Citrus* fruits.

We have noticed in natives on a liberal supply of orange juice a marked diuresis, so much so as to offer difficulty in titrating the urine for Vitamin C. Vitamins, in relation to infection, with a review of the literature, is discussed by Clausen (1934), and he states *inter alia*:—

“So far as the reviewer is aware, no definite studies have been made of the use of diets high in Vitamin C, but not in other vitamins, for the treatment of tuberculosis. This is remarkable, in view of the evidence supplied by the experimental work with scurvy and tuberculosis in animals”; and also, “A deficiency in the diet of Vitamins A and C appears quite definitely to lower resistance to infection.”

Hanke (1932) reports the use of orange juice in the treatment of gingivitis and caries in children. The experiment was conducted on a large scale and involved 323 children for 2 years as a test group, and 99 children for 1 year as a control group, and ranging in age from 10 to 17 years. All were on a good diet. The test group of 323 children received daily 16 fl. ozs. of orange juice and the juice of one lemon. The control group received no supplement to their diet. The children on orange juice were all cured of gingivitis not associated with calculus, and caries was stopped in nearly 50% of cases and retarded in 16%. Hanke (1933) details further experiences with the use of orange juice for gingivitis and caries in his book on “Diet and Dental Health” (1933). Hanke considers we should take 1 pint of orange juice per day.

Our knowledge of scurvy has been based largely on a study of the disease in guinea-pigs, as they are susceptible to the disease and offer a more readily controlled means of study of the disease in man. It is just over forty years ago since Theobald Smith (1895) made the first observations on a haemorrhagic disease of guinea pigs, which has since come to be recognised as a condition of scurvy. The most fundamental work on scurvy dates from the investigations
of Holst (1907, 1912) and his colleagues, working at the University of Christiania, who made a thorough and systematic study of scurvy in guinea pigs, and concluded that its etiology, symptoms and methods of prevention and cure were analogous with the human disease. Our definite knowledge of scurvy as a vitamin deficiency disease may be said to date from the work of these investigators, and modern research has confirmed the earlier work.

Although always associated with malnutrition, scurvy is not necessarily a disease associated with poverty, as amongst even well-to-do people, the frequent use of over-cooked food and improperly selected diets results in a condition of ill-health, which modern knowledge is now associating with sub-scurvy.

Frank scurvy is usually easily recognised, but the recognition of "sub-scurvy," "latent scurvy," "incipient scurvy," "early scurvy" and such like conditions, to use the terms employed by those who have described them, offers uncertainty and difficulty.

In the case of Europeans, such conditions have been detected by a method introduced by Gothlin (1931), which consists of establishing the Vitamin C standard and requirements of physically healthy individuals by testing the strength of their cutaneous capillaries. This is determined by counting the number of petechiae appearing within a circle of 60 mm. diameter drawn on the front of the elbow, after compression of the upper arm for 15 minutes at a pressure of 50 mm. of mercury. A sphygmomanometer is used for determining the pressure. Six petechiae or more was taken as evidence of capillary fragility, an important cause of which is held to be inadequate supplies of Vitamin C. Persons on a deliberately scorbutic diet gave tests indicating diminished capillary resistance and a corresponding increase of petechiae. Their resistance could be restored to normal with sufficient supplies of Vitamin C amounting to from 0.7 to 1.0 ml. of orange juice per day per kilo of body weight. Gedda (1932) and Green (1934) have used the method.

The urinary excretion of Vitamin C affords useful information respecting the conditions of the person on his usual diet and on one fortified with anti-scorbutics. It depends on an estimate of the "state of vitamin saturation" by determining the output of Vitamin C following large test doses of orange juice or Vitamin C.

The examination of urine for Vitamin C commences with van der Walle (1922), who reported the absence of Vitamin C from urine; Euler and Klussman (1933) examined normal, febrile and diabetic urines with negative results; van Eekelen Emmerie, Josephy and Wolff (1933) found Vitamin C in urine, and stated:—

"People using much fruit, produce more reducing substance (Vit. C). Intake of much decitrated lemon juice gives rise to a greater output of the reducing substance." The same investigators
later (1933) reported the presence of 25 mgms. of Vitamin C excreted daily on a normal diet and still later (1934) reported the average daily excretion of Vitamin C was 15 to 25 mgms. on ordinary diet, and liberal intake of orange juice raised this level; Harris, Ray and Ward (1933) found that "In a number of normal individuals the amount of Vitamin C excreted in the urine was surprisingly constant, generally in the neighbourhood of 30 to 33 mgms. per day, or roughly 0.02 to 0.03 mgms. per ml. of urine (this concentration being naturally lowered in diuresis, and being sometimes rather higher in the more concentrated early morning urine). If a normal individual is given a single very large dose of Vitamin C, e.g., 600 ml. of orange juice, 30 times the reputed daily requirement, the concentration of Vitamin C in the urine sharply rises, reaching in about 3 hours a maximum as high, e.g., 8 or 10 times the "normal," it then rapidly drops again, and within a day or so reaches the "normal" resting level of about 33 mgms. excreted per day where it remains remarkably steady, although the subject may be restricted, e.g., for a week or more, to a Vitamin C free diet. This "normal" daily loss of about 33 mgms. is somewhat greater than the reputed daily minimum requirement for man (viz.: 1 oz. of orange or lemon juice)."

Hess and Benjamin (1934) found that children consuming normal amounts of fruit and vegetables and 1 oz. of orange juice daily, did not excrete appreciable amounts of Vitamin C, but the daily intake of 1 pint of orange juice caused high excretion after the fourth day and consider that, under ordinary nutritive conditions, Vitamin C is not excreted in appreciable amounts in human urine; van Eekelen, Emmerie, Josephy and Wolff (1934) report that human urine contains a little more than 1 mgm. per cent. and the diurnal output averaging 15 to 25 mgms.; Johnson and Zilva (1934) find that the "urinary excretion of Vitamin C under normal conditions of existence is variable. The output of Vitamin C in the urine is conditioned by the amount stored in the body and the quantity consumed in the diet. When the store of Vitamin C is complete, a more or less constant level of urinary excretion can be achieved, and the level varies with the magnitude of the daily quantity of Vitamin C taken. It is possible to exist on diets containing sufficient Vitamin C to insure good health and freedom from scurvy even when the store of Vitamin C in the body is maintained low." Tauber and Kleiner (1935) find 22 to 46 mgms. of Vitamin C per 100 ml. of urine; Ahmad (1936) records figures for the Vitamin C content of urine and confirms the findings of Harris and colleagues (1933); Shinohara and Padis (1936) find 3.2 to 26.8 mgms. Vitamin C per 100 ml. of urine.

All the results discussed for Vitamin C and its excretion in urine refer to Europeans, and we know of no published figures for the urinary Vitamin C excretion of the native. In our laboratory we have recently examined the urines of three healthy natives and two scorbutics.
In Table I appears the results, showing the Urinary Excretion of Vitamin C (Ascorbic Acid) for Healthy and Scorbatic Natives and for Healthy Europeans.

**TABLE I.**
**TABLE SHOWING THE URINARY EXCRETION OF VITAMIN C (ASCORBIC ACID) FOR HEALTHY AND SCORBATIC NATIVES AND FOR HEALTHY EUROPEANS.**

<table>
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<tr>
<th>Subject</th>
<th>Total volume of urine voided during 24 hrs. (ml.)</th>
<th>Weight of Vit. C in urine voided (mgms.)</th>
<th>Concentration of Vit. C in urine (mgms. per 1 ml.)</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>&quot;James&quot;</td>
<td>1,689 (1st day)</td>
<td>19.05</td>
<td>0.0112</td>
<td>Taken 1.75 litre orange juice in 3 days equals 525 mgms. of ascorbic acid.</td>
</tr>
<tr>
<td>(healthy native)</td>
<td>1,685 (2nd day)</td>
<td>9.43</td>
<td>0.0056</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1,890 (3rd day)</td>
<td>18.90</td>
<td>0.0100</td>
<td></td>
</tr>
<tr>
<td>&quot;Pete&quot;</td>
<td>1,246 (1st day)</td>
<td>15.84</td>
<td>0.0127</td>
<td>Taken 0.5 litres of orange juice equals 150 mgms. of ascorbic acid.</td>
</tr>
<tr>
<td>(healthy native)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&quot;Absalom&quot;</td>
<td>565 (1st day)</td>
<td>7.08</td>
<td>0.0125</td>
<td>Taken 3 litres orange juice in 3 days equals 900 mgms. of ascorbic acid.</td>
</tr>
<tr>
<td>(healthy native)</td>
<td>665 (2nd day)</td>
<td>22.41</td>
<td>0.0337</td>
<td></td>
</tr>
<tr>
<td></td>
<td>300 (3rd day)</td>
<td>12.43</td>
<td>0.0414</td>
<td></td>
</tr>
<tr>
<td>&quot;Matsheilsa&quot;</td>
<td>1,650 (1st day)</td>
<td>16.50</td>
<td>0.0100</td>
<td>Taken 20.5 litres orange juice in 20 days equals 6,150 mgms. of ascorbic acid.</td>
</tr>
<tr>
<td>(frank scurvy)</td>
<td>2,155 (2nd day) Diuresis.</td>
<td>7.60</td>
<td>0.0035</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Urines too low in ascorbic acid for accurate titration.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&quot;Mitsbango&quot;</td>
<td>2,597 (1st day) Diuresis.</td>
<td>13.18</td>
<td>0.0051</td>
<td>Taken 4.5 litres orange juice in 9 days equals 2,150 mgms. of ascorbic acid.</td>
</tr>
<tr>
<td>(mild scurvy)</td>
<td>2,030 (2nd day) Diuresis.</td>
<td>6.34</td>
<td>0.0031</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Urines too low in ascorbic acid for accurate titration.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>European &quot;A&quot;</td>
<td>1,200 (1st day)</td>
<td>24.27</td>
<td>0.0202</td>
<td>Taken 0.25 litres of orange juice equals 75 mgms. of ascorbic acid.</td>
</tr>
<tr>
<td>(healthy)</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>European &quot;B&quot;</td>
<td>925 (1st day)</td>
<td>11.06</td>
<td>0.0120</td>
<td>Taken 5 litres orange juice in 20 days equals 1,500 mgms. of ascorbic acid.</td>
</tr>
<tr>
<td>(healthy)</td>
<td>1,985 (2nd day) Diuresis.</td>
<td>20.54</td>
<td>0.0103</td>
<td></td>
</tr>
<tr>
<td></td>
<td>574 (6am/3rd week)</td>
<td>14.20</td>
<td>0.0247</td>
<td></td>
</tr>
<tr>
<td>European &quot;C&quot;</td>
<td>1,075 (1st day)</td>
<td>13.39</td>
<td>0.0124</td>
<td>Taken 0.25 litres of orange juice equals 75 mgms. of ascorbic acid.</td>
</tr>
<tr>
<td>(healthy)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
All subjects received preserved and sweetened orange juice containing 30 mgms. of ascorbic acid (Vitamin C) per 100 ml. of juice. All three healthy natives and the three Europeans were on their ordinary diets, supplemented by the quantity of orange juice indicated in the "remarks" column. The scorbutic natives were on the diet of the hospital at which they were located, plus the orange juice ration as indicated in the "remarks" column.

The figures submitted are hardly sufficient to afford information permitting of drawing definite conclusions. They do indicate, however, that the three healthy natives excreted about 15 mgms. of ascorbic acid (Vitamin C) per 24 hours, or less than half of the "normal" resting level of 33 mgms. per 24 hours reported for Europeans by Harris, Ray and Ward (1933).

These three natives were employed on light work and most likely would break down under hard work.

The urinary excretion of two of the three Europeans are also below the "normal" resting level of 33 mgms. per 24 hours. European "A" would be about "normal" and his diet is known to be more frugivorous as compared with "B" and "C."

The native case of "frank scurvy" (Matshelisa) was, before commencing to take orange juice in a most hopeless condition. His mouth was bad and showed "raspberry" swellings; he had severe pains and stiffness of his joints, particularly the knees; he was unable to walk; anaemic and very depressed. He received 1 litre (approximately 1 pint 15 fl. ozs.) per day, divided into three doses taken every 4 or 5 hours, of sweetened and preserved orange juice. On the fourth day of this treatment, he got out of bed and walked about and already the condition of his mouth was improved and his spirits more cheerful. After 20 days of this treatment, he left hospital; he could walk readily; his mouth was in good order and almost all the pain and stiffness of his joints had disappeared. The last estimation of Vitamin C in his urine was made on the 13th day, and this indicated 13.7 mgms. for his early morning urine. After taking orange juice, marked diuresis was a feature of this case, and most of the urines voided consequently so low in Vitamin C as to make analysis inaccurate. This could possibly have been avoided by restricting his fluid intake. He was taking unlimited amounts of "Marewu" as part of the hospital diet. The Vitamin C content of native beverages is apparently low, as Levy and Fox (1935) report 2 to 5 mgms. of Vitamin C per 1,000 ml. for eleven samples of Kaffir Beer, from compounds of the Witwatersrand Gold Mines.

The native case of "mild scurvy" (Mitshbango) did not exhibit any very marked signs of scurvy, except for pain in his joints and a mild gingivitis. The figures for urinary output of Vitamin C (ascorbic acid) however, show definitely he was sub-scorbutic, and, unless promptly treated, would soon have developed into a
case of "frank scurvy." He responded quickly to treatment with sweetened and preserved orange juice and left hospital on the ninth day of treatment. He had been on 0.5 litres (approximately 17 fl. ozs.) per day, divided into two doses.

The Vitamin C (ascorbic acid) content of the urines were determined by titration, using 0.05% strength phenol indo 2 : 6 dichlorophenol indicator, standardised against pure ascorbic acid and the ascorbic acid solution standardised against N/100 Iodine solution. Usually, 50 ml. of urine were used for the test, acidified with 1 ml. of 2N/Sulphuric acid and titrated until the red endpoint remains constant for one minute. Analyses were made soon after the urines were voided, or, if they were left for any length of time were acidified by adding 1 to 5 ml. of 2N/Sulphuric acid (depending on the volume of the urine). It is necessary to acidify urines as a rapid loss of Vitamin C occurs on standing, particularly should the urine be on or near the alkaline side.

The actual titration occupies a few minutes and the cost of reagents used is small. The titration method for estimating Vitamin C (ascorbic acid) has been confirmed by biological tests. The method is applicable to foodstuffs; blood; etc. The well-known biological test methods such as the Hojer (1924, 1926) method, involves the use of 15 guinea pigs for a period of 14 days, a basal diet, decalcification of the lower jaw, embedding and cutting sections by means of the freezing microtome, staining and a study of the histological structure of the roots of the incisors. The loss in weight and degree of protection method of Zilva—see Bracewell, Hoyle and Zilva (1930)—involves the use of a basal diet, six guinea pigs for each dose of material under test and a period of 60 to 90 days. The indicator method is described by Tillmans and his colleagues (1932), Harris and Ray (1933), and other workers.

Information as to the urinary output of Vitamin C by natives of different races, of both sexes and of all ages, is badly needed. In respect of applicants for work in industries, the Vitamin C content of the urine could be determined by means of the indicator method, which can be quickly and cheaply accomplished, and the condition of the Native determined by his "state of Vitamin C saturation." In the case of natives on a "period of loafing," prior to employment on hard work on the mines, application of the test and dosage of large quantities of orange juice would shorten this period of non-economic idleness and define the condition of the subject as regards his scorbatic state. The relatively small cost of the orange juice would be more than balanced by shortening the "period of loafing" and, by the certainty that the boy is unlikely to break down later as a result of being in a sub-scorbutic state.

The Vitamin C excretion test could be applied in native hospitals, hostels, etc.
VITAMIN C.

The work of Hopkins (1912) made clear our knowledge of the part vitamins play in diet.

Within recent years, Vitamin C has been isolated in pure form and is now available commercially. The compound consists of white micro-crystals of rectangular plates, readily soluble in cold water, but somewhat unstable in solution. The compound is optically active and its formula is $\text{C}_6\text{H}_8\text{O}_6$.

The isolation of Vitamin C is due largely to the work of an Hungarian chemist, Albert Szent-Györgi. In 1928, he was studying any possible relationship between the brown pigmentation of the skin (a characteristic of persons suffering from Addison’s disease) and the brown darkening of the surface of the skin of a cut apple or potato.

During the course of this work, he isolated from the adrenals a body which he considered to be a “hexuronic” acid. He found the same body in oranges and cabbages, and from the latter isolated 250 mgms. from 5 kilo of cabbage. Later work showed that the body was not a “uronic” acid, and that it possessed marked anti-scorbutic properties. Szent-Györgyi re-named the body, Ascorbic Acid. H. E. Armstrong (1933) thinks this should have been called “Anti-scorbutic Acid.”

Ascorbic acid, or Vitamin C, is now extracted on a commercial basis from plant sources, and is also prepared synthetically, the synthesis having been accomplished by Herbert, Haworth, and colleagues in England, and by Reichstein and colleagues on the Continent. Vitamin C is the first vitamin to be built up synthetically by the organic chemist; although Vitamin D had previously been produced by the irradiation of ergosterol.

The identity of Vitamin C of plant and animal origin with the isolated and with the synthetic product has been confirmed by a large number of workers. The Vitamin C, or ascorbic acid, present in orange juice is identical with the pure and isolated product, and exists in orange juice in the “free” form and immediately available for utilisation in the animal body.

The physiologically active compound is the levorotatory form and is adopted as the Vitamin C standard of the International Conference of Vitamin standardisation held in London during June, 1934. The unit is defined as the Vitamin C activity of 0.05 mgms. of L-ascorbic acid. The 1931 Conference adopted lemon juice as the international standard, and the 1934 unit is equal to the average Vitamin C activity of 0.1 gram of freshly expressed lemon juice. It is about one-tenth of the daily dose necessary to prevent the development of gross macroscopic lesions in young guinea-pigs maintained on a scurvy-producing diet.

A small supply of the International Vitamin C standard obtained by courtesy of the Medical Research Council, London
is kept in our laboratory and is employed as a standard of reference for such tests as we have occasion to conduct.

Vitamin C, or ascorbic acid, is readily oxidisable and precautions are required for the storing of the product and of its solution and use in water. Ahmad (1933) reports that even 10 minutes after the preparation of a solution in water, its antiscorbutic value was lowered. The oxidation is catalysed by traces of metals, and Kellie and Zilva (1933) find that ordinary laboratory-distilled water contains sufficient quantities of metals—copper and iron—to irreversibly oxidise the product; Barron, De Meio and Klemperer (1936) report 46 micrograms of copper will act as a marked catalyst of oxidation. Mawson (1935) reports ascorbic acid is relatively stable in glass-distilled water. Copper and, to a less extent, iron, and especially a mixture of the two metals, rapidly oxidise ascorbic acid. Lemon juice contains no protective mechanism other than the low pH. Hirst and Zilva (1933) state that the anti-scorbutic value of dehydroascorbic acid—the oxidation product of ascorbic acid—is very nearly the same as for ascorbic acid. Fox and Levy (1936) confirm the anti-scorbutic value of dehydroascorbic acid for guinea-pigs.

Vitamin C, or ascorbic acid, can be used per os or intravenously for the prevention and cure of scurvy. Parsons (1933) treated a 9 months old infant with mild scurvy and on a diet which was not altered, except that 40 mgms. of ascorbic acid daily per os for 8 days and 20 mgms. for a further 6 days was used. At the end of the period of 14 days, the cure was complete, and 440 mgms. of ascorbic acid were used. Recovery time was approximately the same as would have been expected with routine treatment giving fresh orange juice.

Schultzer (1933), in a case of scurvy, and with gastric achylia, used 600 mgms. of ascorbic acid intravenously over a period of 20 days and effected a complete cure; Bauke (1934) describes a case of severe scurvy with ulcerative gingivitis, severe anaemia, widespread effusions and pyrexia treated successfully intravenously using 100 mgms. ascorbic acid per day.

**DOSAGE OF VITAMIN C.**

A. Mavrogordato, cited Delf (1921), suggested 0.5 ml. of orange juice per kilo of body weight as an estimate of the daily need of man. A man of 65 kilo (143 lbs.) would therefore require 32.5 ml. (a little more than 1 fl. oz.); Gothlin (1931) reported that the capillary resistance of scorbutic persons could be restored to normal by taking 0.7 to 1.0 ml. of orange juice per day per kilo of body weight. A man of 65 kilo (143 lbs.) would, therefore, require 45.5 to 65 ml. (about 1½ to 2 fl. ozs.). “Vitamins,” Medical Research Council, London, Special Report Series No. 167 (1932) states:—
To protect a guinea-pig from the ordinary symptoms of scurvy, a daily dose of 1.5 ml. of orange juice is needed, whilst 3.0 ml. are required to prevent degeneration of the dental pulp. Assuming that the dose required to preserve normal capillary resistance is of the same order as that required to prevent degeneration of the dental pulp, it would follow that an average adult would require 14 to 20 times as much as a guinea-pig. This is equivalent to 21 to 30 ml. (about 1 fl. oz.) of orange juice per day.

Göthlin (1934) states that “the daily minimum requirements of man to be given \textit{per os} and protecting a person weighing 60 kilo (approx. 132 lbs.) against the slightest objectively ascertainable prescorbutic alterations—those in the capillaries—is 19 to 27 mgms.” Carr (1934) states: “40 to 80 mgms. is the daily human requirement”; Harris, Ray and Ward (1933) say that the “normal” daily loss of ascorbic acid of 33 mgms. is somewhat greater than the reputed minimum daily requirements for man—1 oz. of orange juice or lemon juice—20 mgms. of ascorbic acid.

The dosages recommended by various investigators are summarised in Table 2.
TABLE 2.

**TABLE SHOWING THE MINIMUM DAILY PROTECTIVE DIETARY REQUIREMENTS OF MAN FOR VITAMIN C (ASCORBIC ACID).**

<table>
<thead>
<tr>
<th>Observer.</th>
<th>Year</th>
<th>Basis of Recommendation.</th>
<th>Ascorbic Acid (mgms.)</th>
<th>Orange Juice equivalent quantity. (1)</th>
<th>Remarks.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Government Notice No. 2241.</td>
<td>1920</td>
<td>Germinated peas or beans (3 ozs. wet weight).</td>
<td>23.0</td>
<td>2½ fl. ozs.</td>
<td></td>
</tr>
<tr>
<td>Mavrogordo</td>
<td>1921</td>
<td>———</td>
<td>9.8</td>
<td>32.5 ml.</td>
<td>(1 fl. oz.).</td>
</tr>
<tr>
<td>Gothlin.</td>
<td>1931</td>
<td>Capillary resistance test.</td>
<td>12.6–18</td>
<td>42–60 ml.</td>
<td>(1½–2 fl. ozs.).</td>
</tr>
<tr>
<td>Medical Research Council.</td>
<td>1932</td>
<td>Protective dose, guinea-pigs.</td>
<td>6.3–9.0</td>
<td>21–30 ml.</td>
<td>(1–2 fl. ozs.).</td>
</tr>
<tr>
<td>Harris, Ray &amp; Ward.</td>
<td>1933</td>
<td>Urinary excretion.</td>
<td>33.0</td>
<td>110 ml.</td>
<td>(4 fl. ozs.).</td>
</tr>
<tr>
<td>Hanke.</td>
<td>1933</td>
<td>Gingivitis and caries.</td>
<td>170.0</td>
<td>568 ml.</td>
<td>(1 pint—20 fl. ozs.).</td>
</tr>
<tr>
<td>Gothlin.</td>
<td>1934</td>
<td>Capillary resistance test.</td>
<td>19–27</td>
<td>61–90 ml.</td>
<td>(2–3 fl. ozs.).</td>
</tr>
<tr>
<td>Carr.</td>
<td>1934</td>
<td>———</td>
<td>40–80</td>
<td>133–267 ml.</td>
<td>(4½–8½ fl. oz.)</td>
</tr>
<tr>
<td>&quot;Tomango&quot;</td>
<td>1936</td>
<td>(2)</td>
<td>23.0 or 33.0</td>
<td>2½ fl. ozs. or 4 fl. ozs.</td>
<td></td>
</tr>
</tbody>
</table>

(1) Preserved orange juice containing a minimum of 30 mgms. of ascorbic acid (Vitamin C) per 100 ml. of juice.

(2) The dosage of 2½ fl. ozs. is recommended on the basis of the Gothlin (1934) test, and is the mean of his dosage, i.e., 19 to 27 mgms. ascorbic acid per day. On the basis of the Harris, Ray and Ward (1933) figure of 33 mgms. of ascorbic acid excreted per day, the dosage of orange juice would be 4 fl. oz. These dosages refer to sweetened orange juice, i.e., orange juice to which pure cane sugar has been added so as to increase the palatability of the juice.

Some uncertainty exists as to correct protective doses for Europeans, and no data at all is available for the requirements of the Bantu. In connection with the minimum doses recommended in the table above, a caution appears in the 1934 Annual Report of the South African Institute for Medical Research, Johannesburg, in which it is stated, inter alia:—"It is clear that the organism
finds it difficult to accumulate any store of Vitamin C if presented with a slight surplus only above actual requirements; quite considerable quantities must be given over and above that required to protect against scurvy."

In view of the known facts that our Bantu population is subscorbutic, the use of orange juice in doses so small as 2 1/2 fl. ozs. of sweetened juice can result only in beneficial effects, and would decidedly help to check the sub-scorbutic state developing into frank scurvy.

So far back as 1921, Dr. W. Watkins-Pitchford, in his editorial preface to Dr. M. Delf’s well-known publication on “Studies in Experimental Scurvy—with special reference to the Antiscorbutic Properties of some South African Foodstuffs” : South African Institute for Medical Research, Publication No. 14 (1921), wrote :—"This menace (of scurvy) is not so much that of declared and recognised scurvy as of the 'pre-scorbutic' or latent scurvy condition . . . . Not the least important conclusion to be drawn from the observations of all the leading workers in vitamin research is the pre-eminent value of the orange as a preventive and curative of scurvy, whether latent or declared."

Modern research on scurvy and the methods of dealing with this insidious malady all confirms the merits of orange juice as the most effective weapon for stamping out scurvy—latent or open.

South Africa, with its large and rapidly expanding Citrus fruits industry, has the remedy at hand!

Hypervitaminosis of Vitamin C is not known to occur. Demole (1934) reports the use of 2.5 grams per kilo of body-weight in one dose was tolerated by guinea-pigs, and, as the minimum curative and prophylactic dose is 0.5 to 1 mgm. daily, it is evident that the animals tolerate 500-1,000 times the therapeutic dose daily without ill effects.

Histological examination of the various organs (pancreas, kidney, liver, heart and lungs) revealed no definite change from the normal. He also reported the use of 0.1 gram per kilo of body-weight injected subcutaneously into a dog, resulted in the excretion of 75% of the Vitamin C during the following 9 hours.

**GERMINATED BEANS AND PEAS FOR THE PREVENTION OF SCURVY.**

Provision was made some 16 years ago for the inclusion of Vitamin C in the diet of native labourers. Delf (1921) says that it was largely owing to the efforts of Dr. A. J. Orenstein that the diet, as compared with the ration gazetted in 1911, for example, was improved. Details of the modified diet appear in Government
Notice No. 2241 dated December 8th, 1920, in which is laid down a "Minimum Ration Scale for Native Labourers" and provision for the supply of Vitamin C secured by the use of germinated beans or peas. The regulation reads:—"Beans or peas, at least half of which to be germinated, 3 oz. per day," and, "if, during the months of July, August, September and October, an orange or other approved substitute be issued every second day, the vegetable ration may be reduced to 3 ozs." (it is 5 ozs. of vegetables per day). The use of germinated seeds goes back some 150 years, as Charles Curtis, formerly Surgeon of the Medea Frigate, in "An Account of the Diseases of India as they appeared in the English Fleet (1807)" quotes a Mr. Young, of the Navy (1782) as regards the merits of germinated seed, and remarks:—"Beans and pease, and barley and other seeds, brought under the malting or vegetating process . . . . eaten in this state without any sort of preparation . . . . cannot fail to supply precisely what is wanted for the cure of scurvy." A fuller account of Curtis' experience appears in the publication on "Vitamins," Special Report Series No. 167 (1932) issued by the Medical Research Council, London. Chick and Hume (1917) describe the modus operandi for the germination of peas and beans:—

(1) The dry seeds must be whole, retaining the original seed coat, not milled or decorticated.

(2) They must be soaked in water for several hours; the time necessarily depends on the temperature—24 hours at 50°F. to 60°F., and 12 hours at 90°F.

(3) The water must be drained away and the peas, etc., allowed to remain in the moist condition with access of air. They then germinate and the small rootlets grow out. This germination will take 48 hours at 50°F. to 60°F., and 12 hours to 24 hours at 90°F.

(4) It is important that the germinated pulses and beans should be cooked and eaten as soon as possible after germination, and not allowed to become dry again; in that case, the antiscorbutic properties acquired during the process of germination will again be destroyed.

(5) The pulses and beans should be boiled rapidly for not more than half-an-hour.

Delf (1921) writes:—"In calculating the possible increase in vitamin value from the addition of germinated pulses to a dietary, allowance must be made for:—

(a) the proportion of seeds which will not swell when cooked;
(b) the proportion which will not germinate;
(c) the loss in value to be expected from the method of cooking employed."

Peas and beans damaged by weevils will reduce the percentage likely to germinate.
The Vitamin C (ascorbic acid) content of germinated peas, determined by modern methods involving chemical titration of the ascorbic acid, is given by Harris and Kay (1933), who found no Vitamin C in the ungerminated peas; soaked for 48 hours and germinated, 0.69 mgm. of ascorbic acid per gram; soaked for 72 hours and germinated, 0.82 mgm. per gram; and soaked for 96 hours and germinated, 0.86 mgm. per gram. Ray (1934) records for peas, soaked 48 hours and germinated, 0.23 mgm. of ascorbic acid per gram; soaked for 60 hours and germinated, 0.43 mgm. per gram; and soaked for 90 hours and germinated, 0.50 mgm. per gram; and states, *inter alia*, "The rate of production of ascorbic acid in pea seedlings germinated naturally ....... was found to rise for two days and then reach a constant concentration of 0.5 mgm. ascorbic acid per gram of the wet weight of the seedlings."

Griaznoff et al (1934) states:—"About 100 germinated peas was calculated as the daily anti-scorbutic dose for man." We find 100 peas, of the kind in actual use on the gold mines of the Rand, weighed 20 grams; soaked overnight and drained, weighed 40 grams; and, on the basis of 0.5 mgm. of ascorbic acid per gram wet weight, the ascorbic acid content is 20 mgms. Calculating on the gazetted ration basis of 3 ozs. of peas or beans per day, of which half is to be germinated, 1½ ozs. of peas germinated and on the wet weight would theoretically yield 42.0 mgms. of ascorbic acid. It must be borne in mind, however, that the reported figures for the ascorbic acid content of germinated peas of 0.5 mgm. per gram wet weight are based on laboratory extraction of all the Vitamin C present, and this is accomplished by grinding with sand and extraction with solvents such as trichloracetic acid. They represent the theoretical figures, which in actual utilisation, all of the Vitamin C in the germinated peas is not likely to be accomplished by the digestive processes of man. Then again, in mine kitchens, it is unlikely that the germination and cooking would be so effective as to avoid any loss of Vitamin C. It might be safer to assume that half of the theoretical quantity present is actually used by man, and this would represent about 20 to 25 mgms. of ascorbic acid for 1½ ozs. of dry peas.

**VEGETABLES FOR THE PREVENTION OF SCURVY.**

Government Notice No. 2241, December 8th, 1920, states:—"Fresh vegetables, exclusive of carrot tops and maize husks, 5 ozs. per day. In order to encourage the consumption of raw vegetables by natives, where possible it is recommended that vegetables such as cabbage, carrots, leeks or any other vegetable which the native would eat raw, be minced up fine and stirred raw into the stew or beans immediately prior to using."

Goldsmith (1929) describes the procedure adopted in the
compound of one of the larger gold mines, a compound of 2,200 natives, and states that the vegetables are cut by machines into pieces 1 to 2 inches long by \( \frac{1}{4} \) inch thick, and finally minced.

Kohmann, Eddy and Gurin (1931) state that a considerable loss of Vitamin C occurs in shredded carrots and report that the protective dose for guinea-pigs of whole carrots is 15 grams; shredded and held 1 hour, 18 grams; and shredded and held 3 hours, 20 grams. Ahmad (1935) reports results for an Indian vegetable called “Karela” (a kind of vegetable marrow) which is rich in Vitamin C. Cutting up or shredding the vegetable profoundly affected the anti-scorbutic value. The fresh “karela” contained 52 mgms. per 100 grams; shredded and after ten minutes it contained 46.3 mgms. per 100 grams; after 20 minutes, 43.1 mgms. per 100 grams, and after 2 hours, 17.8 mgms. per 100 grams. Approximately 67% of the Vitamin C content of the “karela” was destroyed during two hours when left exposed to the air after being cut and shredded. McHenry and Graham (1935) find that “when shredded turnip is allowed to stand in the open air for a short time, the juice obtained from the pulp is inactive . . . . . this inactivation of pulp is practically complete in 30 minutes, and even in 15 minutes the pulp loses 75% of its activity. With some vegetables, an increase, and with others a loss, of Vitamin C occurs on cooking.” McHenry and Graham (1935), studying the anti-scorbutic value of raw and cooked Ontario (Canada) foodstuffs, find higher values for the Vitamin C content of cooked cauliflower, carrots, parsnips, beets and potatoes, and attribute this not to cellular disintegration and more complete extraction of the Vitamin C, but to the liberation of “bound” ascorbic acid, perhaps existing as an “ester.” It is not certain as to whether the “bound” or “ester” ascorbic acid is biologically active. van Eekelen (1935) was not able to confirm these observations, and considers that the increase on cooking is not a real increase, and attributes the apparent increase “to the destruction of an oxidase present in some vegetables which changes the reduced form of ascorbic acid into the reversibly oxidised state when the vegetables are ground up.” Ahmad (1935) considers the “bound” or “ester” idea plausible for some work he has done on cabbage. McHenry and Graham (1935) report for raw cabbage 15 mgms., and cooked 13 mgms.; raw old potatoes 1.5 mgms. and cooked 4.1 mgms.; raw spinach 18 mgms. and cooked 13 mgms.; raw onions 8.9 mgms. and cooked 3.1 mgms.; raw carrots 1.2 mgms. and cooked 2.6 mgms.; raw peas 14.0 mgms. and cooked 8.1 mgms.; and raw turnips 35 mgms. and cooked 18 mgms.; all figures on 100 grams of tissue. Birch, Harris and Ray (1933) record for cabbage, 1.0 mgms.; potatoes, 0.15 mgm.; and carrots, 0.028 mgm. per 1 gram of tissue: Sandor (1934) finds 4.79 mgms. for Hungarian onions; 2.02 mgms. for Holland onions, and 5.20 mgms. for Spanish onions; all figures on 100 gram of onions.
The calculated Vitamin C value of vegetables rationed at 5 ozs. per day per person and for ascorbic acid values determined by the modern indicator titration method, will be found summarised in Table 3.

TABLE 3.
TABLE SHOWING THE VITAMIN C (ASCORBIC ACID) CONTENT OF FIVE OUNCES OF RAW AND COOKED VEGETABLES, BASED ON FIGURES REPORTED FOR CANADIAN VEGETABLES.

<table>
<thead>
<tr>
<th>VEGETABLE</th>
<th>RAW (mgms. of ascorbic acid in 5 ozs.)</th>
<th>COOKED (1) (mgms. of ascorbic acid in 5 ozs.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cabbage</td>
<td>21.3</td>
<td>17.0</td>
</tr>
<tr>
<td>Potato</td>
<td>2.1</td>
<td>5.8</td>
</tr>
<tr>
<td>Carrot</td>
<td>1.7</td>
<td>3.7</td>
</tr>
<tr>
<td>Spinach</td>
<td>25.5</td>
<td>17.5</td>
</tr>
<tr>
<td>Onions</td>
<td>12.8</td>
<td>4.4</td>
</tr>
<tr>
<td>Peas</td>
<td>19.9</td>
<td>11.5</td>
</tr>
<tr>
<td>Turnips</td>
<td>49.7</td>
<td>25.5</td>
</tr>
</tbody>
</table>

Results for South African vegetables are reported by Delf (1921) using guinea-pigs as test animals: “the anti-scorbutic value of raw sweet-potato is about equivalent to that of an equal weight of raw vegetable marrow (young); but that mature vegetable marrows have about twice the value of an equal weight of pumpkin, and about one-third that of sweet-potato. It is not possible, however, to predict what effect the influence of cooking would have on these relative values.”

We know of no data showing the Vitamin C content of the cooked food supplied to the native labourers on the gold mines. Information is wanted to show the actual Vitamin C content of the food as eaten by the native.

LUCERNE FOR THE PREVENTION OF SCURVY.

Fodder plants, such as lucerne (alfalfa) among other leafy plants, are reported high in vitamins.

Douglass (1931–32) reports 52 units of Vitamin A per gram for alfalfa hay cured in diffused light, and only 27 units of Vitamin A for hay cured in direct sunlight; Virtanen, Lundmark and Peltola (1932) report the presence of Vitamin C in purple clover, and found 3 gram daily doses protected guinea-pigs from scurvy; Douglass, Tobiska and Vail (1933) report green alfalfa rich and

(1) McHenry and Graham (1935) say that “The plant tissues were heated by procedures similar to those used in preparing them for table use.”
the dried hay low in Vitamin C. Hauge (1934) confirms the richness in Vitamin A for alfalfa. Scheunert and Schieblich (1934) report 60 units of Vitamin A for fresh alfalfa. Levy and Fox (1935) report that the concentration of Vitamin C in the average leaf of lucerne is about four times that found in Citrus juice. They find for leaves of young plants 3.2 to 3.8 mgms.; for leaves and small stalks, 2.7 to 3.3 mgms.; for shoots, 3.0 to 3.8 mgms.; for leaves of plants at the flowering stage, 2.2 to 3.1 mgms.; and for the leaves only of another specimen, 4.5 mgms.; small stalks, 1.8 mgms.; and fibrous stalks, 0.7 mgm.; all figures per gram of fresh lucerne. Lucerne, when cooked, retained 90% of its original ascorbic acid. Lucerne, oven-dried for 6 minutes at 250°F., gave over 7 mgms. per gram, and lucerne hay gave 0.9 to 1.6 mgms. per gram of hay. Fox and Wilson discuss the merits of lucerne as an addition to the diet of native labourers in Laboratory Report No. 3 of an Enquiry into Native Diets (1), issued by the South African Institute for Medical Research, Johannesburg, and, as the subject is of considerable interest, we reproduce herewith the author's conclusions:

"SOME SUGGESTIONS REGARDING THE VALUE AND METHODS OF USING LUCERNE AS PART OF THE RATION FOR NATIVE MINE LABOURERS.

Value as a Food.

This depends principally upon the fact that uncooked lucerne is an exceptionally good source of the anti-scorbutic vitamin, being about five times as rich as the same weight of orange juice. In addition, it contains valuable amounts of the other vitamins, particularly Vitamin A. Quite apart from the vitamins, however, lucerne is rich in mineral salts and protein.

Lucerne is usually obtainable on the Witwatersrand during six to eight months of the year. Growth during the winter is very slow, but it should be possible to arrange for adequate winter supplies by sowing down larger areas. Not only is it a very hardy plant, but it can be repeatedly cropped and lasts for several seasons. Small amounts are already being grown on some mines for feeding cattle, but it is suggested that these areas might be developed to provide a part, at any rate, of the amount that would be needed for feeding the native labour force.

Method of Feeding.

The best method is to strip the leaves and tender shoots from the plant, mince and then add to the stew just before issue.

(1) The publication is undated.
If the utmost value is to be obtained from the ration, there are, however, a number of points to which it is essential that attention should be paid.

(a) The lucerne should be cut when young, certainly before it has begun to flower; this is because it is the more tender and therefore more palatable, as well as much more easy to chop up; in addition, the protein and mineral constituents are much more abundant in the young plant.

(b) It is also most desirable to use the lucerne as fresh as possible, since the anti-scorbutic properties begin to diminish as soon as the plant is cut, whilst destruction becomes more rapid during stripping, and particularly during mincing. Every effort should therefore be made to avoid using wilted plants kept overnight, supplies of the really fresh material being obtained daily if at all possible. The least possible delay should elapse between mincing and time of issue in the ration.

(c) The lucerne should first be washed thoroughly in water to remove grit, etc. If it is young and tender, the coarse part of the stem, say one-third of the plant, can be chopped off and the rest minced up. With older plants, it is first necessary to strip off the leaves and shoots, the stalks being thrown away.

Mincing is then carried out, preferably by means of a mechanical mincing machine. Young lucerne is readily minced in this way, but the older plants tend to clog the machine; this difficulty can be overcome by adding other vegetables such as potatoes or carrots during mincing.

(d) The minced lucerne should be added to the pot just as the stew in that particular pot is to be issued. It is added to one pot first, and, when that is finished, it is added to the next and so on. This ensures that the lucerne shall not be in the boiling liquid for more than a few minutes.

If only inferior lucerne is available, it may be cooked along with the other vegetables, but such a course leads to a considerable loss of vitamin.

(e) Even a tablespoonful of the chopped material per boy is a useful addition to the ration. An ounce per day may be regarded as a fully protective ration against scurvy, even without the addition of other vegetables. This amount has actually been tried out and raised no unfavourable comments from the boys. Such a quantity adds much other valuable material to the stew. It is suggested that the lucerne be introduced gradually, starting with, say, one-quarter of an ounce, and increasing to about one ounce.”

Delf (1921) wrote that dietary improvements should be made “with due regard to the habits and tastes of the natives concerned.”
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