

✓ A STUDY OF VITAMIN A AND CAROTENOIDS AND OF
VITAMIN C IN THE BLOOD SERUM OF LEPROSY
PATIENTS AND OF THEIR HEALTHY
HOUSEMATES

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I. VITAMIN A AND CAROTENOIDS

Insufficiency or deficiency of vitamin A may be considered one of several factors which weaken the body and lower its resistance against leprosy. It is reported in the literature that lack of vitamin A causes rat leprosy to spread rapidly, and that administration of this vitamin prevents the appearance of symptoms. In studying this question in leprosy patients we must bear in mind the principle of cause and effect: Is vitamin A deficiency a primary or a secondary factor in leprosy?

The investigation here reported aimed at answering this question by determining the levels of vitamin A and of the carotenoids in the blood sera of ambulant patients living in various sections of Djakarta. At the same time we were able to compare these findings with those of their healthy housemates.

FINDINGS IN THE DIFFERENT TYPES AND STAGES OF LEPROSY

All of the 344 patients studied belonged to the same class of society, and their diet was that of the ordinary people of Djakarta. Sex and age were not taken into consideration when comparing the different stages and types of the disease. Only 25 of the patients were younger than 15 years. Our material may therefore be considered homogeneous.

The findings are given in Table 1, where it will be seen that there were 122 lepromatous cases and 54 of the "mixed" class, making a total of 176 lepromatous, and 168 of the neural type. These cases are arranged in the table according to type and stage (degree of advancement) of the disease as provided by the Cairo congress. A single average is given for all stages of the mixed (LN) condition, because the clinical differences between the stages (for instance between L1-N1 and L1-N2, or L2-N1 and L2-N2), were slight, which led us to suppose that the differences in concentrations of the substances studied would also be small.

TABLE 1. Concentrations of vitamin A and carotenoids in the different types and stages of leprosy, per 10 ml. of blood serum.

Grade	Cases	Vitamin A, I. U.	Carotenoids, γ
<i>Lepromatous type</i>			
L1	13	7.2 ± 0.70	4 ± 0.61
L2	76	6.0 ± 0.40	3 ± 0.15
L3	33	5.0 ± 0.72	3 ± 0.33
<i>Neural type</i>			
N1	32	8 ± 0.62	5 ± 0.58
N2	123	8 ± 0.37	5 ± 0.25
N3	13	7 ± 1.02	4 ± 0.51
<i>Mixed cases</i>			
LN	54	7.0 ± 0.47	3 ± 0.26

The figures show that the content of vitamin A and carotenoids varied with the different stages of both types of leprosy, the more advanced the stage the lower the concentrations. This is seen more clearly in the lepromatous type than in the neural type. This seems to justify the conclusion that the decrease of either of these substances is caused by and correlated with the advancement of the disease.

On comparing the figures of the most advanced stages of the two types (L3 and N3), it was found that the difference is not statistically significant, although the arithmetical difference indicates that the concentrations of both vitamin A and carotenoids in the N3 cases were higher than in the L3 cases.

The distribution of the different concentrations of vitamin A in the various stages of lepromatous and neural leprosy is shown in Table 2.

As before, it is shown that L3 cases had lower vitamin A values than L1 and L2 cases. Six per cent of the patients in the first of these groups had no vitamin A in the blood serum, whereas it was found in all cases of the L1 and L2 grades (89 in total).

The differences found when comparing the figures of the vitamin A content for the various stages of the lepromatous

TABLE 2. *Distribution of the different concentrations of vitamin A in lepromatous and neural leprosy, by grades.*

Grade	Cases examined	Concentrations, I. U.					
		0-3	3.1-6	6.1-9	9.1-12	12.1-15	15+
<i>Lepromatous type</i>							
L1	13	-----	5 (38%)	5 (38%)	3 (23%)	-----	-----
L2	76	16 (21%)	27 (36%)	15 (20%)	14 (18%)	3 (4%)	1 (1%)
L3	33	12 (36%)	12 (36%)	2 (6%)	4 (12%)	3 (9%)	-----
<i>Neural type</i>							
N1	32	-----	8 (25%)	11 (34%)	9 (28%)	2 (6%)	2 (6%)
N2	123	5 (4%)	37 (30%)	35 (28%)	18 (15%)	22 (18%)	6 (5%)
N3	13	1 (8%)	4 (31%)	4 (31%)	3 (23%)	-----	1 (8%)

type are not statistically significant; this is probably due to the limited numbers of cases, which renders the standard deviation too great. We are convinced, however, that the degree of advancement of the disease in this type strongly influences the decrease of vitamin A in the blood serum of the patient, i.e., that the decrease is caused by the disease itself.

It is evident that this change is not as distinct in neural leprosy as in the lepromatous type. For instance, in the 9.1 to 12 column, N2 grade, we find 15 per cent, whereas the N3 grade unexpectedly shows 23 per cent. It may therefore be concluded that the stages of neural leprosy have no decisive influence on the vitamin A content of the blood serum.

No computation for mixed leprosy has been made because of the many possible combinations, which renders evaluation well-nigh impossible.

The distribution of the different concentrations of the carotenoids is shown in Table 3. Here again, in the lepromatous cases, are lower figures in the L3 group. In the 9.1 to 12 column, L1 is 23 per cent, while L3 falls to 3 per cent. In this case, also, it may be possible that the decrease was caused by the disease. With regard to the neural cases, the figures for carotenoids in

the N3 grade reach only the 6.1 to 9 column, whereas those for the N2 and N1 grades attain higher values.

TABLE 3. *Distribution of the different concentrations of the carotenoids in lepromatous and neural leprosy, by grades.*

Grade	Cases examined	Concentrations, γ					
		0-3	3.1-6	6.1-9	9.1-12	12.1-15	15+
<i>Lepromatous type</i>							
L1	13	3 (23%)	7 (54%)	-----	3 (23%)	-----	-----
L2	76	42 (55%)	29 (38%)	4 (5%)	1 (1%)	-----	-----
L3	33	17 (51%)	14 (42%)	1 (3%)	1 (3%)	-----	-----
<i>Neural type</i>							
N1	32	5 (16%)	16 (50%)	7 (22%)	2 (6%)	1 (3%)	1 (3%)
N2	123	32 (26%)	60 (49%)	21 (17%)	7 (6%)	2 (2%)	1 (1%)
N3	13	3 (23%)	8 (62%)	2 (15%)	-----	-----	-----

My conclusion is that it may well be that there exists a relation between the stage of the disease and the concentrations of vitamin A and carotenoids in the blood serum, i.e., the more serious the disease the lower the concentrations. This is demonstrated more clearly in the lepromatous type than in the neural type.

When comparing the values for these substances in the lepromatous type (including mixed cases) and the neural type (including tuberculoid cases), we obtain the following averages:

$$\begin{array}{l}
 176 \text{ L cases: Vit. A, } 6.3 \pm 0.27 \\
 168 \text{ N cases: Vit. A, } 8.3 \pm 0.30
 \end{array}
 \left. \vphantom{\begin{array}{l} 176 \text{ L cases: Vit. A, } 6.3 \pm 0.27 \\ 168 \text{ N cases: Vit. A, } 8.3 \pm 0.30 \end{array}} \right\} \text{difference} = 2 \pm 0.40$$

$$\begin{array}{l}
 176 \text{ L cases: Carot., } 3.5 \pm 0.13 \\
 168 \text{ N cases: Carot., } 5.3 \pm 0.22
 \end{array}
 \left. \vphantom{\begin{array}{l} 176 \text{ L cases: Carot., } 3.5 \pm 0.13 \\ 168 \text{ N cases: Carot., } 5.3 \pm 0.22 \end{array}} \right\} \text{difference} = 1.8 \pm 0.25$$

These figures almost warrant the conclusion that the decrease of vitamin A and of carotenoids is more marked in the

lepomatous type than in the neural type. This is what we expected to find, considering that the lepomatous type is the more serious one.

The average figure for vitamin A for all types and stages in 7.3 ± 0.20 I.U., for carotenoids 4.4 ± 0.13 .

FINDINGS IN HEALTHY HOUSEMATES AND THE RELATED PATIENTS

Out of the total of 344 patients examined, the findings in 204 could be compared with those in one or more of the 329 housemates. This lot of the patients and their housemates formed two groups for comparative study.

The average figure for vitamin A of housemates was 8.3 ± 0.20 I.U., and that for carotenoids was 5.8 ± 0.16 .

95 L cases: Vit. A., = 6.4 ± 0.40	} difference = 1.9 ± 0.5
152 Mates: Vit. A., = 8.3 ± 0.30	
109 N cases: Vit. A., = 8.5 ± 0.42	} difference = 0.2 ± 0.5
177 Mates: Vit. A., = 8.3 ± 0.28	
95 L cases: Carot., = 3.4 ± 0.22	} difference = 2.4 ± 0.33
152 Mates: Carot., = 5.9 ± 0.25	
109 N cases: Carot., = 5.5 ± 0.29	} difference = 0.3 ± 0.37
177 Mates: Carot., = 5.8 ± 0.23	

The following conclusions may be drawn: Sufferers from lepomatous leprosy had less vitamin A and carotenoids than their healthy housemates. In patients of the neural type, these substances were in about the same concentration as in their housemates. Whereas neural leprosy does not seem to affect vitamin A, this is not so in the lepomatous form. Taking into account the characteristics of the two types, the explanation may be as follows: The neural type affects the skin and the nervous system, and the lepomatous type the skin and the deeper parts of the body, as it seeks its way along the blood vessels. The leprosy bacillus penetrates the inner organs, as evidenced by postmortem findings in patients with advanced lepomatous leprosy, in whom lepromas are found in the liver, spleen and at times elsewhere. As the liver is the storeroom of vitamin A, it is hardly surprising that the distribution of this vitamin is disturbed by the disease. The same phenomenon can be

observed in pneumonia patients whose livers are reported to contain sufficient quantities of vitamin A, although it is insufficient in the blood serum. It has been suggested that the vitamin reserve in the liver is not large enough for the distribution, due to a blockade of the reticuloendothelial system, or because the liver absorbs most of the vitamin A; it might also be possible that vitamin A cannot be dissolved in the blood serum because of lack of blood lipoids.

FINDINGS IN HOUSEMATES OF PATIENTS AND NONCONTACTS

Is there any evidence that there exists a relationship between the decrease of vitamin A and the susceptibility for infection with leprosy? It is mentioned in the literature that decrease of vitamin A in the body renders it more susceptible to infection. Menken (11) writes: "Decrease of the quantity of vitamin A in the body causes the function of abnormal epithelium in different systems of the body (eyes, respiratory tract, urinary tract) which constitute a nesting place for bacteria, as evidenced by the many ensuing infections." These systems become less resistant because the action of antitoxin is hindered, according to Greene (cited by Sie Boen Lian).

We were not able to use other healthy individuals for further investigations along this line, but have compared our findings in the healthy housemates of leprosy patients with data reported in the literature, taking care that the standards of living in both groups were the same.

Sie Boen Lian (13) examined 30 healthy individuals in the Eye Outpatient Clinic, Djakarta, and found the quantity of vitamin A to be 10.5 L.U.B. and of carotenoids 42.8 γ per 100 ml. of blood serum, i.e., 6.72 I.U. of vitamin A and 4.28 γ of carotenoids for every 10 ml. In patients with xerophthalmia the average quantity of vitamin A was 2.78 L.U.B., and carotenoids 24.5 γ for every 100 ml. of blood serum, i.e., 1.78 I.U. of vitamin A and 2.45 γ of carotenoids per 10 ml.

De Haas and Meulemans (4) examined 210 nurses of the Central Hospital, Djakarta, and found the concentration of vitamin A to vary from 70 to 110 I.U., and of carotenoids from 50 γ to 70 γ , per 100 ml. of serum.

Donath and Gorter (1) examined the inhabitants of several villages in West and East Java where xerophthalmia prevailed. The results, per 100 ml. of serum, were as follows:

	<i>Vit. A, I.U.</i>	<i>Carotenoids, γ</i>
Gresik and surrounding villages	22.3 \pm 1.12	33.1 \pm 2.31
Segalaharang (Purwakarta)	26.5 \pm 3.71	26.0 \pm 2.11
Warungkondang (Tjiandjur)	59.4 \pm 2.58	29.1 \pm 1.94

Van Veen *et al.* (14) obtained the following figures per 10 ml. of blood serum in twelve assistants of the laboratory of the Eykman Institute, Djakarta: vitamin A, 9.4 ± 1.7 I.U., carotenoids, 8.3 ± 2.4 γ .

On comparing the quantities of vitamin A and carotenoids found in the healthy housemates of leprosy patients, 8.3 ± 0.20 I.U. and 5.8 ± 0.16 γ , respectively, with the figures of the authors cited, it will be seen that the difference for vitamin A is 1.1 ± 0.5 , which is not statistically significant.

The average quantities found by Donath and Gorter in Gresik and Segalaherang were lower than those that we found, even in the leprosy patients themselves. However, this is not surprising as there were many sufferers from hemeralopia and xerophthalmia among the cases examined by Donath and Gorter.

It may be concluded that the quantities of vitamin A in housemates of leprosy patients are the same as, or at any rate not lower than, those of other Indonesians of the same social standing. There seems to be no evidence, therefore, for assuming that there exists a correlation between the decrease of the vitamin A content of the serum and the susceptibility to infection with leprosy. Moreover, the vitamin A content of the leprosy patients themselves (7.3 ± 0.20) also shows that they were not suffering from xerophthalmia or hemeralopia; these figures come from a district where ordinarily there is no sign of avitaminosis A.

SUMMARY AND CONCLUSIONS

Investigations were made of the concentrations of vitamin A and carotenoids in the blood serum of 344 ambulant leprosy patients in Djakarta, and of 329 of their healthy housemates.

The average concentration of vitamin A in 176 patients with lepromatous leprosy (including mixed cases), in 10 ml. of serum, was 6.3 ± 0.27 I. U. The average concentration of carotenoids was 3.5 ± 0.13 γ . In 168 patients with neural leprosy (including tuberculoid cases), the average vitamin A content was 8.3 ± 0.30 I.U., and that of carotenoids was 5.3 ± 0.22 γ . The average vitamin A content of patients of the lepromatous type was lower than that of the neural type.

When comparing the vitamin A content of the patients with that of their healthy housemates, it was found to be significantly lower in cases of the lepromatous type, but not in those of neural leprosy.

The vitamin A content of the serum of the housemates was found to be normal when compared with that of other groups of

Indonesians who did not suffer from a disease which lowers the vitamin content (vitamin A 8.3 ± 0.20 I.U., carotenoids 5.8 ± 0.16 γ).

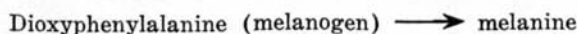
The following conclusions are drawn:

1. The content of vitamin A and carotenoids in the blood serum of patients with lepromatous leprosy is somewhat lower than in normal individuals. In patients with the neural type this content is normal.
2. There is no indication of a diminution of vitamin A which might enhance the susceptibility to infection with leprosy.
3. Possibly the decrease of vitamin A is caused by the disease.

II. VITAMIN C

This section of the present paper deals with my investigation of the vitamin C content of the blood of leprosy patients and of healthy inmates of their houses. The investigation had for its objectives: (1) to ascertain the vitamin C content of the blood of the patients and to find out if there is any correlation between lack of the vitamin and the communicability of leprosy, and (2) to throw light on the question of whether vitamin C has an influence on the depigmentation of the skin lesions of neural leprosy.

It has been pointed out in the literature that ascorbic acid has an important bearing in the formation of pigments in the skin, the production of which in the cells can be represented as follows:



In this "Dopa reaction" ascorbic acid acts as a stabiliser, i. e., the reaction takes place not only through the influence of one of the ferments but also through the elimination of oxygen, and ascorbic acid acts as a reducing agent. When there is an excess of ascorbic acid this reaction will progress slowly, on the other hand insufficiency of ascorbic acid will hasten the formation of melanine.

This investigation involved 98 patients, together with 95 of their healthy housemates, at least one of the latter being investigated with each patient except, for one reason or another, in 5 instances. Because of the small number of persons available for this investigation, differentiation is only made between the lepromatous and neural types of leprosy. The different stages of the advancement of the disease are not taken into consideration. The mixed (LN) cases are included in the lepromatous

type, and the tuberculoid (Nt) cases are included in the neural type.

The average figures obtained for the vitamin C concentration from the blood of the persons examined are shown in Table 4.

TABLE 4. *Vitamin C concentration of the blood in lepromatous and neural cases and their housemates; averages, mgm. per liter.*

Type of leprosy	Number of patients	Vitamin C concentration	Number of housemates	Vitamin C concentration
L	38	$7.2 \pm 0.54/a$	38	$9.8 \pm 0.57/b$
N	60	$10.3 \pm 0.45/a$	57	$10.2 \pm 0.37/c$

a The difference between lepromatous and neural cases is 3.1 ± 0.7 .

b The difference between the lepromatous cases and their housemates is 2.6 ± 0.7 .

c The average figure for the two groups of healthy housemates shown in this column is 10.00 ± 0.3 .

Comparing the findings in the lepromatous and neural type groups, the average figure of the former is clearly lower than that of the latter. On the other hand, the average for the neural cases is practically equal to that of the associated group of healthy housemates.

The reason for the lower concentration in the lepromatous type is probably the same as that which has been pointed out with respect to the findings of vitamin A, namely, that the livers may possibly have been affected by leprosy. It is a well-known fact that the liver is a storehouse for vitamin C in the body.

At first glance, the figures in the last column of Table 4 suggest that there is a great difference between the vitamin C concentrations of the housemates of the patients with lepromatous leprosy and those of the neural-type patients. Actually, however, this difference is not significant. It therefore seems that there is no connection between the vitamin C concentration in the blood and the liability of contracting leprosy of either type.

In connection with that question, i.e., whether there is a relationship between vitamin C concentration and the liability of contracting leprosy, reference is made to the results of an investigation by Donath and Gorter on the vitamin C content in several groups of the people of West and East Java (1), in places where the frequency of leprosy is well-known (8). The data, together with the average for the healthy housemates of leprosy patients in Djakarta, are shown in Table 5. What is

significant in the data there shown is that there is no clear indication of an existing connection between the vitamin C content of the inhabitants of a region and the frequency of leprosy there.

TABLE 5. *Vitamin C concentration of the blood in the normal populations of certain localities of Java.*

Name of town or village	Number of persons examined	Vitamin C concentration (averages)	Leprosy frequency per mil
Gresik:			
Bendjeng	20	14.3 \pm 2.1	3.0
Tjermee	20	7.9 \pm 1.3	2.1
Pantjeng	19	6.5 \pm 0.95	1.7
Sedaju	20	11.5 \pm 1.25	1.2
Manjar	22	16.0 \pm 0.7	0.6
Bungah	26	10.9 \pm 2.85	0.5
Karangbunangun	20	13.0 \pm 3.1	0.3
Kalitengah	24	11.0 \pm 1.0	0.1
Djakarta:	95/a	10.0 \pm 3.0	2.0
Tjiandjur:			
Warungkondang	148	12.1 \pm 1.06	Few patients

a The healthy housemates of leprosy patients, of the present investigation.

VITAMIN C AND THE FORMATION OF SKIN PIGMENTS

For a study of the bearing of vitamin C on the formation of pigments in the skin of leprosy patients, those of the neural type are obviously the proper subjects. These patients have as their symptoms macules with abnormal pigmentation, hypo- or hyperchromic. In total, 60 patients of this type were examined, and according to the stages of the disease they may be differentiated as follows: Ns1, 14 cases; Ns2, 36 cases; Ns3, 4 cases; Nt (tuberculoid), 5 cases; Na2, 1 case. The last one of these patients lacked macular lesions, presenting only anesthesia and thickened nerves. Of the 54 patients of the Ns1, Ns2 and Ns3 groups, the spots or patches were either without pigment or showed diminution from the normal. There were no patients with flat patches showing excessive pigmentation and loss of sensibility (hyperpigmented maculoanesthetic), a condition

which is often found in cases of the neural type. In the 5 patients with the tuberculoid form of the disease, the patches varied in color from pink to dark brown.

It is shown in Table 4 that the vitamin C concentration of the blood of the patients of the neural type was, on the average, 10.3 mgm. per liter, which is practically the same as that of the healthy inmates (10.2 mgm. per liter). From this it may be concluded that there is no connection between the vitamin C content and the change of color in the skin. This is the more evident from the findings in the cases of the lepromatous type, with their low vitamin C content (± 7 mgm. per liter), for in that form of the disease there are, in general, no changes in the skin pigments. There is, therefore, no reason to see a causative relationship in the vitamin C content in the blood with pigmentary changes in the skin.

The loss of pigment in neural leprosy is probably the result of disease of the nerves in the affected areas. The answer to the question whether the change of color in the skin is accompanied by decrease of the vitamin C content of the blood may possibly be found upon further investigation of the concentration of that vitamin in patients of either type presenting patches or infiltrations of a brownish color.

SUMMARY AND CONCLUSIONS

The purpose of this investigation of the concentration of vitamin C in the blood of leprosy patients and their healthy housemates, like that of vitamin A, was to determine whether decrease of vitamin C is the result of the disease, and whether there is a connection between it and the liability to contract the infection. A second objective was to determine whether the influence of the ascorbic acid in the "Dopa reaction" in the changing of color in the skin (hypo- or hyperpigmentation) of patients of the neural type with maculoanesthetic lesions has a connection with the vitamin C content of the blood.

The number of patients investigated was 98, of whom 38 had the lepromatous type of the disease or were mixed cases, while the other 60 were of the neural type, including 5 tuberculoid cases. The number of healthy housemates of patients examined was 95, they being associates of 93 of the patients; 38 of them were associates of patients with the lepromatous type of the disease, and 57 were associates of patients with the neural type.

The average vitamin C concentration of the lepromatous cases was 7.2 ± 0.54 , while that of the neural cases was 10.3

± 0.45 . The latter figure is practically the same as that of the healthy housemates of neural cases, which is 10.2 ± 0.37 . The figure for the lepromatous cases is quite different, it being significantly lower than that of their healthy associates, which was 9.8 ± 0.57 . The average figure for the healthy housemates of the patients of both types was 10.0 ± 0.30 , not very different from the values obtained in other Indonesians generally.

Comparing the findings for vitamin C concentrations which have been obtained in village communities in Java with the prevalence of leprosy in those places, there is seen no significant correlation.

With respect to the question of a correlation between the vitamin C concentrations and the pigmentary changes in the neural cases, all but one of which had anesthetic macular lesions with more or less hypopigmentation, no correlation is seen. The vitamin C concentration of these people did not differ materially from the ordinary average.

The following conclusions are drawn:

1. In lepromatous leprosy there is a decrease of vitamin C, which is probably not a primary factor with respect to infection, but rather the result of the disease.
2. In the data for healthy housemates of leprosy patients, no indication has been found of a correlation between the decrease of vitamin C and the liability of contracting leprosy.
3. The ascorbic acid in the blood of the patients of the neural type has probably no direct bearing on the matter of hypopigmentation of the skin lesions.

RESÚMEN

Este es un trabajo sobre las concentraciones de vitamina A y los carotenoides, y la vitamina C, en la sangre de pacientes leprosos y de sujetos saludables (cohabitantes) en Djakarta. Los hallazgos también se comparan con aquellos reportados por otros autores en habitantes de otras aldeas en Java.

I. Se determinó la vitamina A y los carotenoides en 344 casos, 176 de los cuales eran del tipo lepromatoso (incluso 54 casos "mixtos") y 168 eran del tipo neural (incluso algunos del tipo tuberculoide). Los valores promedios para concentración de vitamina A fueron 6.3 ± 0.27 I.U. en casos lepromatosos, y 8.3 ± 0.30 I. U. en casos neurales, valores dentro límites normales. Los carotenoides fueron 3.5 ± 0.13 γ y 5.3 ± 0.22 γ respectivamente. Los valores promedios en 329 cohabitantes saludables fueron para la vitamina A 8.3 ± 0.20 I.U. y para los

carotenoides $5.8 \pm 0.16 \gamma$, valores dentro límites normales si se les compara con otros grupos en Indonesia. Las diferencias entre los dos tipos de lepra, que son significantes, se achacan probablemente a los efectos de la enfermedad en su forma lepromatosa. Se concluye que no hay evidencia de desminución de la vitamina A en los contactos, que pudiera predisponerlos a la infección.

II. Las determinaciones de la vitamina C se practicaron en 98 leprosos, 38 lepromatosos y 60 neurales, y en 95 cohabitantes saludables. El promedio en el grupo lepromatoso fué más bajo que en el grupo neural, 7.2 ± 0.54 comparado con 10.3 ± 0.45 . La última cifra está de acuerdo con la de los cohabitantes normales de 10.0 ± 0.30 , que a su vez compara con los valores normales en otros grupos Indoneeses. Aquí también la diferencia se adscribe a los efectos de la enfermedad y no a una condición primaria relacionada con la susceptibilidad a la infección. No hay relación entre los valores obtenidos en otras comunidades y la prevalencia de la lepra en esas comunidades. Como el valor obtenido en casos neurales no es menos que el normal, se concluye que no hay relación directa entre el nivel del ácido ascorbico en la sangre y la depigmentación cutánea en tales pacientes.

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