THE ROLE OF VITAMIN A IN NUTRITION

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For over twenty years it has been generally accepted that vitamin A is in some way peculiarly essential for the process of growth. This belief is based on the observation that when a young animal is fed on a diet from which vitamin A is absent it ceases, after a time, to put on weight, and eventually dies. This arrest of weight increase has been termed "cessation of growth." In 1928 Green and Mellanby¹ expressed the opinion that the term "growth-promoting," as applied to vitamin A, was a misnomer. They pointed out that when growth ceases owing to the absence of vitamin A from the diet, it often means that the animal is definitely ill, in the sense of its having developed some infective condition. They described the manifold infective conditions they had found in rats suffering from this deficiency, and suggested the term "anti-infective vitamin" as a more accurate definition of vitamin A.

The true significance of the results of Green and Mellanby does not appear to have been grasped either by the clinician or by the biochemist, the one tending to ascribe too wide a power to vitamin A as an anti-infective agent, the other failing to recognize the bearing of the findings with respect to the assay of vitamin A. On the clinical side the tendency has been to regard vitamin A as anti-infective in a generalized sense, and, presumably, as able to combat all types of bacterial infection—an assumption for which satisfactory experimental proof has yet to be given. Harris² gives evidence which indicates that infections occurring in the absence of vitamin A are of special type, localized in origin, and associated with keratinization of mucous membrane. He suggests the term "anti-keratinizing factor" for vitamin A. He found that where infection had occurred keratinization of epithelium could, without exception, be detected, but he could not definitely demonstrate an earlier stage of keratinization without infection. Arons and Van der Rijst,³ however, who carried out post-mortem examinations on 276 rats, state that keratinization without infection is often found. Mendel⁴ considers it probable that the influence of the vitamin is felt indirectly in helping to preserve the physiologic integrity of various epithelial structures, and thus to maintain the "first line of defence" against the invasion of bacteria. A similar view had previously been expressed by Cramer.⁵

On the other hand, the biochemists have not realized that in such findings as those of Green and Mellanby lies the explanation of many of the difficulties associated with the curative method of assaying vitamin A. In this method the young animals are given a diet free from this constituent until they "cease to grow," the vitamin A content of a test substance being then measured by the weight increase which follows administration of the substance. It seems obvious that the weight response of such animals to vitamin A dosing must be largely determined by the nature and severity of the pathological conditions present.

Continuance of Growth in Vitamin A Deficiency

Recent investigations at this institute have made it clear that the anti-infective, anti-keratinizing, or health-preserving function of vitamin A is indeed all-important, and that its claim to "growth-promoting" activity is no greater than that of any other food constituent that is essential for optimum physiological development. Green and Mellanby drew the inference from their work that the term "growth-promoting" was not an accurate definition of the function of vitamin A, but they still made

use of the expression "when growth ceases owing to the absence of vitamin A." Our experiments have shown that growth does not cease in vitamin A deficiency when the animal ceases to increase in weight. This has been proved, not only by direct measurements of body length on the live animal, but by measurements of the limb bones post mortem. Growth in the sense of increase in length continues, in spite of the fall in weight due to the pathological condition, although the rate of growth may be diminished, owing to the reduced food consumption of the sick animal. The so-called "cessation of growth" in avitaminosis A is thus merely an instance of the commonplace clinical observation that the pathological condition is very frequently accompanied by loss in weight, the pathological condition in this particular case being due to lack of vitamin A.

We have shown, further, that the various types of discrepancy reported in vitamin A assays by the curative method may be explained by the diversity and varying severity of the pathological conditions which have developed in the animals during the preliminary period of depletion in vitamin A. It seems clear that no physiological test can give satisfactory results unless it is carried out on prophylactic lines.

The biochemical aspects of this work have been discussed in detail in two recent publications,⁶ but certain of the results obtained seem to be of considerable interest also from the clinical standpoint. In this paper it is intended, therefore, to deal more fully with the occurrence of pathological conditions in vitamin A deficiency, and to emphasize the possible bearing of the findings on questions connected with human nutrition.

The main points to which it is desired to draw attention are: (1) the early occurrence of pathological conditions in young rats deprived of vitamin A; (2) the nature of the conditions observed, and in particular the very high incidence of gastro-intestinal affections both in young and in adult animals; (3) the persistence of the pathological conditions once they have been established, in spite of subsequent dosing with vitamin A; and (4) the parallelism between the findings in rats and various reported cases of disease in human beings.

Early Occurrence of Pathological Conditions in Young Rats

In carrying out a curative test for vitamin A it is assumed that the initial growth of the animal during the preliminary period on the basal diet devoid of vitamin A is due to the reserve stores of the vitamin present in the body, and that the characteristic "cessation of growth" indicates complete exhaustion of these reserves. As already pointed out there is in reality no cessation of growth, and all our results suggest that the preliminary period of "depletion of the reserve stores" is simply the time taken for the outward manifestation of symptoms of disease that has been gradually developing. Under our experimental conditions the length of the "depletion" period as estimated by the weight curves generally lasts as long as four and a half to six weeks, but in rats killed after only three weeks on the diet, macroscopic evidence of disease can be detected in a large proportion of the animals. Mouriquand *et al.* have found it possible in the case of rats to detect ulceration of the cornea microscopically twenty days after beginning a vitamin A deficient diet, and twenty days before the onset of clinically recognizable symptoms of xerophthalmia. This result is significant, and it becomes even more so if it is realized that xerophthalmia, in spite of the undue share of attention it has received, is neither an invariable nor the most dangerous result of vitamin A deficiency in rats. We have found the earliest macroscopic signs of disease in the epithelial lining of the digestive tract. In rats killed before reaching a moribund state from lack of vitamin A,

inflammation of the duodenum, of the small intestine, and of the caecum is frequent, and the glandular portion of the stomach shows pittings, haemorrhagic points, and even ulceration. In Table I are given the findings for seventy such rats, which were killed after three to seven weeks on the deficient diet.*

TABLE I

	Weeks on Diet				
	3	4	5	6	7
Number of rats examined	19	18	20	8	5
Number classed as "normal"	3	6	0	0	0
Post-mortem findings:					
Stomach: Ulceration (glandular part) ...	2	2	5	3	2
Pitting, haemorrhagic points, etc.	5	5	12	7	5
Duodenum mucosa inflamed	8	9	12	2	3
Small intestine inflamed	4	3	6	1	2
Caecum: Inflammation	5	5	15	5	5
Ulceration	3		3	2	1
Bladder wall thickened				1	2

It will be seen that of thirty-seven rats killed after three and four weeks on the diet, 75 per cent. showed evidence of disease even at this early stage, while of those killed at five weeks or later, none was classed as normal on macroscopic examination. Close examination of the weight curves of vitamin A deficient rats shows that while there may not be a marked falling off for four weeks or more, divergence from the normal course tends to show as early as thirteen to fourteen days. It is probable, therefore, that microscopical examination might reveal the presence of diseased tissues at an even earlier stage, and the results suggest that any reserve stores of vitamin A that may be present in the young rat at weaning are rapidly exhausted.

Nature of Pathological Conditions in Young Rats

In Table II are given the results of the post-mortem examination of sixty-four young rats (that is, rats which were placed on vitamin A free diet at weaning) which, in one of our experiments, were allowed to die or become

TABLE II

	Weeks on Diet							Total
	5	6	7	8	9	10	11	
Number of rats examined	2	6	15	21	15	4	1	61
Post-mortem findings:								%
Tongue abscess		1	10	12	11	2		36 = 56.3
Lung infection (broncho-pneumonia or abscesses)		1	4	3		1		9 = 14.1
Stomach: Keratosis		2	11	13	13	3		42 = 65.6
Ulceration (non glandular)		2	5	6	5	3		21 = 32.8
Ulceration 'glandular)	1	2	5	11	10	2		31 = 48.4
Pitting, haemorrhagic points	1	2	9	10	7	2		31 = 48.4
Duodenum mucosa much injected	1	1	7	6	4	2	1	22 = 34.4
Small intestine inflamed... ..	1	2	5	4	7	2	1	22 = 31.3
Gastro-intestinal dilatation	1	1						
Caecum: Inflammation		5	12	17	15	3	1	53 = 82.8
Ulceration			3	7	4		1	15 = 23.4
Pyelitis		2	7	14	4	1	1	29 = 45.3
Bladder: Keratosis, inflammation, etc.		1	10	12	4	3	1	31 = 48.4

* The vitamin A free diet, of which the details are given by Richards and Simpson,⁶ contained ample amounts of vitamins B, C, and D.

moribund through lack of vitamin A.* It will be seen that the prevalence of gastro-intestinal trouble, already noted in young rats killed in the earlier stages of the deficiency, becomes still more pronounced in the later stages. In addition, cases of tongue abscess, lung infection, and pronounced kidney disease, which were absent in the early stages, now occur with considerable frequency.

In general, the pathological findings are similar to those reported by Green and Mellanby, except as regards the very high incidence of disease in the stomach and caecum. These authors found that, although infection of the alimentary tract was common, in most cases it appeared as a terminal event. Acute inflammation of the small or large intestine was found in 21 per cent. of their rats, the intestine being filled with a copious haemorrhagic exudate. They state that many of these cases arose during an epidemic of enteritis in the rat room, the vitamin A deficient animals proving very susceptible to the infection. We have found such enteritis common among the deficient rats, although there was no epidemic, the control rats which received vitamin A remaining entirely unaffected. We regard inflammation of the intestines, in particular of the caecum, as one of the commonest results of the avitaminosis A. It will be noted from Table II that inflammation of the caecum was found in nearly 83 per cent. of the cases, and that ulceration was present in 23 per cent. It is of interest, therefore, to recall that Cramer⁸ observed in his vitamin A deficient rats marked invasion of the mucous glands of the caecum by bacteria, with profound atrophy of the intestinal villi; and that Tilden and Miller⁹ have found gastro-intestinal lesions to be the chief manifestation of similar deficiency in the monkey.

With regard to the stomach, Green and Mellanby do not mention any abnormal findings, except in the cases of intestinal inflammation, when there were usually signs of recent haemorrhage into the pyloric portion of the stomach. We found that in fifty-two out of the total of sixty-four rats included in Table II—that is, in 81 per cent.—definite keratosis or ulceration of the stomach, or both, occurred, and that in most of the remaining cases haemorrhagic points were present in the glandular part. The keratosis was liable to occur in any portion of the fore-stomach, but was frequently most marked near the opening of the oesophagus. In a number of cases it appeared as a ridge round a large eroded area, which in some cases had perforated. In the glandular stomach ulceration was generally confined to the pyloric portion, which often contained three or more large ulcers. McCarrison¹⁰ describes ulceration and epithelial new growths found in the stomach of rats on poor diets. He ascribes these to vitamin A deficiency, since this is a principal fault of the diets used, but he does not seem to have proved it by the use of synthetic diets with no known deficiency except vitamin A. Manville¹¹ found that rats on a vitamin A deficient diet developed gastric ulcers and peptic erosions, and that the percentage occurrence approached 100 as the deficiency became more severe. The work of Fujimaki *et al.*¹² indicates that the epithelial hypertrophy and keratinization in the fore-stomach of rats on a vitamin A deficient diet are related to a disorder of fat metabolism, since the proliferation is slight or absent when the diet is deficient in fat as well as vitamin A. Manville gives no details of the diet used in his experiments, but our vitamin A deficient diet contains approximately 1 part hardened cotton-seed oil to 6 parts dry substance.

* In our macroscopic examination attention was confined to tongue, lungs, stomach, duodenum, small intestine, caecum, liver, kidney, and bladder; nasal sinuses and middle ear were not examined, nor was the exact percentage incidence of xerophthalmia noted. Xerophthalmia occurred frequently, but by no means invariably.

Pathological Conditions in Adult Rats

An experiment was carried out on sixteen older rats (eight male, eight female) past the stage of rapid growth. These rats were kept on normal diet until the age of 5½ to 6 months, all of them having thus equal opportunity for "storage" of vitamin A. Their survival periods, after transference to the vitamin A free diet, varied from 158 to 320 days—a difference which serves to indicate the variability of pathological condition that may arise in animals suffering from lack of this substance. Although the pathological conditions found in the adult rats were similar in type to those found in the young rats, the percentage incidence of the various infective conditions differed considerably in the two groups, as is shown by the following summary of the chief post-mortem findings: tongue abscess, 0; abscess on roof of mouth, 4; pus in neck glands or bronchial tubes, 4; lungs affected, 10; fore-stomach: keratinization 10, ulceration 7; glandular stomach: ulceration 11; duodenum: inflammation 11, ulceration 5; small intestine: inflammation 10; caecum: inflammation 12, ulceration 4; kidneys: pyelitis, 3; bladder: cystitis, keratinization of wall, etc., 4.

It will be seen that in these older animals there were no cases of tongue abscess, such as occurred in 56 per cent. of the young rats which died of avitaminosis A (Table II), although in a few cases a small abscess was found on the roof of the mouth, and sometimes pus was present in either the neck glands or bronchial tubes. The incidence of lung infection was much higher (62 per cent. as against 14 per cent.), as might be expected in view of the well-known difficulty of keeping older rats free from this type of disease. On the other hand, pyelitis was less common, only three cases occurring among the sixteen rats. The occurrence of stomach and intestinal trouble, of which the incidence was already high in the young animals, became still more marked in the adults. In every case the stomach lining was affected, either keratinization or ulceration being present. Keratinization was less severe than in the young animals, but ulceration, especially in the pylorus, was much more pronounced. In ten cases the condition was severe, three to sixteen ulcers being easily detectable. Occasionally bands of erosion were present on the remaining part of the glandular stomach. Inflammation of the small intestine was present in over 60 per cent. of the rats, and was very intense in half of these cases. Inflammation of the caecum, accompanied in some cases by ulceration, was noted in all but four of the animals. In two cases the caecum was highly distorted, apparently due to perforation by abscesses and subsequent healing. In one female rat the bladder was completely filled by a crystalline stone 7 to 8 mm. in diameter. In the adult rats, as in the younger animals, diarrhoea was not infrequent in its occurrence, particularly in the later stages of the deficiency.

Persistence of Pathological Conditions

The pathological conditions induced by a period of vitamin A free diet may be very persistent. Macy *et al.*¹³ pointed out that if the preliminary period in a curative test were too prolonged, advanced pathological conditions might be established which could not be ameliorated by vitamin adjuvants. They found that generalized infections resulting in the localization of pus in the upper part of the digestive and respiratory tracts could not be cured by feeding methods, regardless of the richness of the diet in vitamin A. Various workers have made reference to the subnormal growth response shown by many of the animals in curative tests, but few, apart from Macy, seem to have realized the importance of pathological condition in determining the response. Post-

mortem examination at the end of the five weeks' test period throws much light on this frequent failure of the rats to attain normal growth performance when vitamin A is given after a period of "depletion." In our work comparison of the post-mortem findings with the course of the weight curves has shown that subnormal "growth" is invariably associated with the presence of some pathological condition which has not yielded to treatment with vitamin A. A number of typical examples are given in the following table. In these cases the doses of vitamin A given may be regarded as adequate, in that they proved sufficient for recovery and the attainment of satisfactory weight increase in other animals in which apparently the pathological conditions present had been less severe.

TABLE III

No. Rat	Dose of Vitamin A	Weight Response*	Main Post-mortem Findings at End of Five Weeks' Dosing with Vitamin A
2591	0.46 mg. vit. conc.	Continuously subnormal	Chronic inflammation of caecum
2592	"	"	Caecum ulcerated
2507	0.23 mg. vit. conc.	"	Chronic inflammation of caecum; healed perforated ulcer
2576	0.68 mg. vit. conc.	"	Several healed ulcers in stomach
2610	0.6 mg. vit. conc.	"	Lower end of intestine inflamed
2484	4.3 mg. cod-liver oil	"	Caecum ulcerated
2593	0.46 mg. vit. conc.	Good at first; falling off later	Pyelitis. Haemorrhagic pits in stomach
2520	"	"	Caecum—ulcer, and patch of chronic inflammation
2736	"	"	Pneumonia
2439	0.68 mg. vit. conc.	"	Large ulcer in caecum
2332	6.5 mg. cod-liver oil	"	Keratoses of stomach
2418	"	"	Keratoses of stomach. Lymphatics much enlarged. Lower end of ileum congested
2497	4.3 mg. cod liver oil	"	Chronic inflammation of caecum
2524	0.46 mg.	"	Chronic inflammation of caecum. Lymphatics enlarged
2477	"	"	Chronic inflammation of caecum
2480	2.2 mg. cod-liver oil	Fairly good at first; falling off later	Entire caecum chronically inflamed and distorted
2456	6.5 mg. cod-liver oil	Practically normal	Chronic inflammation of stomach
2541	"	"	Caecum—three ulcers and inflammation
2494	"	"	Pyelitis in left kidney

* The weight curves of these rats are shown in Fig. 6 of the paper by Richards and Simpson.⁶

Even where weight response has been good throughout the test period, such conditions as inflammation of the stomach, ulceration and inflammation of the caecum, and pyelitis may still persist, as may be seen from the last three examples in the above table. There is little doubt that if these animals had been allowed to live, their pathological condition would ultimately have manifested itself in subnormal growth. We have evidence which shows that the pathological conditions due to vitamin A deficiency may persist even after several months on normal diet. Five rats which had appeared to be in good condition at the end of a curative test were placed on normal diet for periods varying from three to eight and a half months, but their growth remained subnormal. On being killed and examined pathologically the conditions found included pleurisy, lung abscesses, ulcers in caecum, stomach, and duodenum, and peritonitis round the caecum.

Vitamin A and Human Nutrition

With regard to the problem of vitamin A and human nutrition, much valuable information is contained in the section of the Medical Research Council Report¹⁴ on Vitamins, entitled "Vitamins and Human Diets" (1932, Chapter X). As far as vitamin A is concerned, many instances are given (p. 231) in which xerophthalmia

and hemeralopia in human beings have apparently been associated with deficiency of vitamin A in the diet, and have yielded promptly to treatment with cod-liver oil or other rich source of the vitamin. These examples certainly furnish strong grounds for the assumption that as regards eye trouble rats and human beings are affected very similarly by lack of vitamin A. It is probable, therefore, that in other respects also the results found in rats may have their counterpart in man. Various observations actually reported seem to be highly significant in this direction. Thus it has been found that in Bombay respiratory infections provide by far the commonest cause of death in a population where cases of xerophthalmia occur (Report of the Medical Officer of Health for Bombay City, 1920-29). Further, Bloch,¹⁵ in describing an outbreak of xerophthalmia, calls attention "to the frequency with which other diseases, such as diarrhoea, bronchitis, pneumonia, pyuria, discharges from ears and nose, and catarrhs of all sorts, were found associated in these patients with xerophthalmia, showing a lowered resistance to infection resulting from their malnutrition." When considered in conjunction with our findings in rats suffering from vitamin A deficiency this list is striking. With the exception of discharges from the ears, which may have escaped our notice, each of the conditions mentioned has occurred again and again in our vitamin A deficient rats. Of much interest also is the association of ulcerative colitis with corneal and conjunctival inflammation, which was noted in two patients by Crohn and Rosenberg.¹⁶ A similar association of xerophthalmia and intestinal infection was described by Schick and Wagner,¹⁷ while Berglund *et al.*¹⁸ speak of cases of anaemia in China, coupled with diarrhoea, where the diarrhoea responded dramatically to treatment with cod-liver oil.

There is thus a distinct similarity in type between the diseases found in vitamin A deficiency in rats and those found associated with xerophthalmia in human beings. Moreover, there is some evidence of similarity between the two species also in regard to the persistence of the pathological conditions accompanying xerophthalmia. Thus Bloch¹⁹ reported that infant mortality continued high in children who had recovered from xerophthalmia, barely two-thirds reaching the age of 8 years. Various authors have reported the presence of kidney and bladder stones in rats on a diet low in vitamin A. Of considerable interest, therefore, is a suggestion made by De Langen²⁰ that the occurrence of bladder stones and also of keratomalacia in Java, where the average diet contains sufficient vitamin A, may generally be referred back to early childhood, the only period when there is a serious danger of a shortage of the vitamin. Intestinal disease is frequent in occurrence, and in such cases the infant receives only pap food, without any milk, so that little vitamin A is present. De Langen was struck with the bad nutrition of infants with keratomalacia or bladder stones. The patients with keratomalacia had all suffered from intestinal disorders for a long time, and in other cases urinary complaints could be traced back to a period of continued diarrhoea in childhood.

These indications of parallelism between the human species and the rat are too suggestive to be ignored. It is evident that the necessity of ensuring an ample supply of vitamin A in infancy and early childhood is urgent, and it is precisely during this period that the intake of this substance is most liable to be insufficient. In view of the frequency of infantile diarrhoea and other intestinal disorders it would be wise to bear in mind the possibility that deficiency of the vitamin may have been a contributory factor in their production. The most serious danger, however, lies in the possibility that irreparable damage to the tissues may be caused by deficiency

in this early period of life. Our results have indicated the difficulty of eradicating the pathological sequelae of avitaminosis A in young rats; Bloch's mortality data indicate a similar persistence of diseased conditions in children known to have suffered from xerophthalmia. Thus it may well be, as pointed out in the Medical Research Council's report with regard to vitamins in general, that the damage caused by insufficiency of vitamin A in early youth cannot afterwards be made good by an adequate supply of the vitamin, and that much chronic ill-health in later life may have its origin in this early deficiency.

The high incidence of gastric and intestinal trouble in adult rats deprived of vitamin A is another suggestive fact not to be overlooked in considering the frequency of the occurrence of gastric and duodenal ulcers in adult human beings. Crohn and Rosenberg urged the importance of giving a complete diet in ulcerative colitis, and Harris²¹ believes that a large percentage of cases of gastric and duodenal ulcers could be cured by a diet rich in every vitamin. Our findings would seem to indicate that so far as vitamin A is concerned his suggestion may have ample justification, and there is little doubt that the provision of an ample supply of this vitamin, both in childhood and in adult life, would aid in the production and maintenance of that perfect health and optimal growth which is the ultimate aim of the science of nutrition.

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In sending out a leaflet compiled from a report issued by the London County Council, the council of the National Association for the Prevention of Tuberculosis, Tavistock House North, Tavistock Square, W.C.1, wishes to draw the attention of local authorities and others to the facilities offered at Burrow Hill Sanatorium Colony. This institution is solely devoted to the care of male adolescents aged 13 to 19, and is staffed and equipped for treatment, education, and technical training. The work of this combined school and sanatorium is something new in the way of institutional care and treatment of the tuberculous. Since the inception of the scheme in 1929 excellent results have been obtained, not only in regard to successful treatment, but also in regard to rehabilitation, no less than 60 per cent. of trained youths having been placed in employment.