Role of Vitamin A in Health and Dentition*

ADELLE DAVIS, *Consulting Nutritionist*

Oakland, California

Perhaps there is no field of dentistry where diet plays such an important part as in that of orthodontics. Work on experimental animals (1) has shown that when they are given adequate diets, the jaws are wide and well rounded, the arch is as it should be and the occlusion is normal. It is only on inadequate diets, usually those low in vitamins A and D, that the arch is narrow, the jaws badly formed, with resulting malocclusion, and the teeth are malposed. The same dietary factors which lead to malformation of the jaws, likewise cause poor structure of teeth which lack resistance to decay. It is not only that the orthodontist must give a diet which will prevent caries and pyorrhea from developing later in already susceptible ground, but he must bring about normal formation of a growing bone in order that the teeth, once moved, may be held in place.

The orthodontist has been made to feel that he must widen his knowledge of diet, and hundreds of articles have been published in the journals that he may do so. While many of the articles have covered all the factors known to affect the health of the teeth, they have been written in such summary fashion that the orthodontist feels he lacks sufficient background to recommend diets. In others, one writer will approach the solution of dental health from one point of view, such as alkaline ash foods, while another will make no mention of alkaline ash, but will put his entire emphasis on vitamin C. Thus, the orthodontist, regardless of how conscientiously he may strive to grasp the subject, is left in a fog of bewilderment. Just what is it, he asks himself, that we must emphasize in recommending diets to our patients? It is for this reason, in an attempt to give rather full information of one factor involved, that the present article deals solely with the role of vitamin A in dental and physical health.

Comparatively little research has been done showing the specific effect of vitamin A on dentition. Howe and Wolbach (2), working with rats on diets deficient in vitamin A, found cavities at the base of the tongue and

*Read before the Northern California Component of the Edward H. Angle Society of Orthodontia.

in the pharynx, filled with a yellow cheesy material, and similar abcess-like cavities in the submaxillary glands. Microscopic examination revealed that the cavities were, in reality, cysts lined with a stratified keratinizing epithelium and the cheesy content was a mixture of desquamated keratinizing cells and leukocytes. The salivary glands and accessary salivary glands were likewise affected, and frequently showed retention of keratinized epithelial cells. This has been confirmed repeatedly by other experimentors. Mori (3) found that the salivary glands of rats on vitamin A deficient diets either secreted very little or not at all, the secreting cells having become greatly shrunken or completely closed.

Microscopic studies (Howe) (4) of the incisors of 22 rats showed changes in the formation of enamel, irregularity in the growth of the odontoblasts and uneven deposits of dentin. The odontoblasts not infrequently laid down bone instead of dentin, resulting in a less dense and resistant calcification of the tooth structure. Osteodentin has also been reported by Marshall (5) and McCollum and his coworkers (6) in the teeth of rats on vitamin A deficient diets.

Howe repeated his work on guinea-pigs on diets lacking in vitamin A and found the same tooth changes which occur in rats, although they were more prompt and more marked in appearance; yet his animals did not develop eye disease. Howe has also observed in his laboratory that bone has been laid down instead of dentin in a section of human tooth.

Gies found that vitamin A deficiency in rats led to less firm interprismatic cement making the tooth both more brittle and less resistant to the attack of bacteria.

Eddy, (7) making vitamin C assays of foods on scorbutic guinea-pigs, whose basal diets were supposedly adequate in vitamin A, found that when greens were used as an antiscorbutic, a much more rapid tooth regeneration took place than when either orange or tomato juice was used. It seems possible that the tooth growth required vitamin A in excess of the amount necessary to normal growth, and the greens supplied this extra amount.

Although the Mellanbys (8) place almost their entire emphasis on vitamin D as the controlling factor in the building of firm structure of teeth, they state that much better teeth are produced when cod liver oil is used as an antirachitic than either sunshine or irradiated ergosterol. Neither sunshine nor irradiated ergosterol contains vitamin A, while cod liver oil is one of the richest known sources.

Marshall (9) states that when rats are placed on diets deficient in vitamin A, one of two dental conditions develops: either the teeth become

carious or they show relatively heavy deposits of calculus. His work was done on more than 200 animals given diets adequate in all other respects except for vitamin A, irradiated lard being used to furnish the vitamin D. (4). The vitamin A deficient animals lost weight to that of one-half the normal animals and in many, the characteristic inflammatory conditions of the eye developed. Microscopic examination showed the presence of not only relatively large pulp stones, but, in other instances, of infected and abcessed pulps. The pulp stones were found in teeth which did not show caries as well as in those which were carious. Some of the experiments showed such a degree of degeneration that the caries involved the cementum, and in other instances, the molars were decayed to the pulp line, while the pulps had undergone degenerative change, areas showing intense inflammation. More calculus appeared in the mouths, which showed little decay, although there were accumulations of such deposits in the gingival areas, with gingival recession, even when the teeth were decayed. In other mouths there was no decay, but these showed pulps which were greatly inflamed, and blood vessels distended to an unusual size. In his experiments, caries developed only after the animals reached maturity, and not in the young. He did not find it possible to arrest caries when an animal, previously on a vitamin A deficient diet, was given vitamin A later.

The bones of the animals fed on vitamin A deficient diets, (4) compared to normal animals, showed marked differences. For example, a normal animal 49 days old showed the same degree of ossification as was found in animals 120 days old on diets low in vitamin A. It is also significant to remember that Mellanby (1) found that poor bone structure was produced by feeding inadequate diets early in life, a condition which later led to paradontosis, and that this condition could not completely be overcome by feeding adequate diets later.

The practical application of any research is, in the final analysis, its test of accomplishment. Hence, as would be expected, all of the diets used successfully in arresting caries in human subjects, as reported by Hanke (10), Drain and Boyd (11), Walter Crandall (12), Mellanby (1), Bunting (13), Price (14), and others, have contained abundant supplies of vitamin A. Here, of particular interest, is work reported by Price (14), in which special "activators" were given in capsule form to more than 200 individuals, and these were compared with other individuals not given capsules. Twenty individuals between the ages of 10 and 20 showed only three new areas of caries during the experimental period, while a similar twenty persons of the same age group, not given capsules, showed 141 cavities during that time.

In all groups, there was 10 times as much decay in the patients not treated as in those given capsules. The capsules contained a high vitamin A cod liver oil, together with butter fat particularly rich in vitamin A. Price makes no mention of checking the diet otherwise. Hence, in this experiment, high vitamin A (together with some additional vitamin D) seems to be the controlling factor.

Vitamin A is a substance essential to growth of the young and to normal nutrition and health of all ages. Its chemical nature is unknown, although it is thought to be an alcohol, for which the empirical formula C20H30O has been suggested. (15). When withheld from the diet of a standardized experimental animal, fed on purified foodstuffs, adequate in all other respects except for vitamin A, growth gradually ceases until the amount stored in the animal's body is used up, a general susceptibility to infection sets in, and a gradual decline results, leading to death. A characteristic eye disease, known as xerophthalmia, opthalmia, or keratomalacia, usually develops before death, although this is not always the case.

In human beings, as in animals, low vitamin A results in lack of growth and a widespread weakening of the body and of its ability to resist infections. A complete lack manifests itself in opthalmia. Livingston's men in Africa suffered from this disease (16) because of a very limited variety of foods. It has been found not infrequently in Russia in nursing infants during religious fasts of the mothers. Over 400 cases (17) were reported among the children of China, cured by adding either cod liver oil or chicken livers to the diet. The most noted outbreak of opthalmia was in Denmark during the war, when the high price of butter for export led to the use of skim milk in feeding infants. The disease disappeared when cod liver oil or butter fat was added to the diet. While xerophthalmia is undoubtedly a bacterial infection, just as is tooth decay, it is significant that without any local treatment, the disease heals quickly when vitamin A is added to the diet.

While the development of eye disease results from a dire deficiency of vitamin A, it is only one of the consequences, and perhaps not the most important one. There are undoubtedly a great many people, even in prosperous communities, who suffer from more or less lack of vigor and lowered resistance to infections, because they live on foods low in vitamin A. The fact that one has never seen a case of opthalmia by no means indicates that he has not encountered numerous cases of vitamin A deficiency.

Let us look again to experimental animals, this time on a diet not completely lacking in vitamin A, yet abnormally low in this factor. The growth of the animal is slow or stunted, and changes in the mucous membranes

throughout the body soon manifest themselves. The epithelial cells cease their normal secretions, leaving the cell abnormally dry and no longer self-cleansing. Whether or not these secretions have bacteriacidal action or merely act in washing the surface is unknown. The cells are displaced by squamous epithelial cells, resulting in a loss of cilia in the respiratory tract, which normally act in cleansing the surface by expulsion of foreign particles. The logical roughness and stickiness of the horny cells undoubtedly hold bacteria which would normally be expelled. Thus it is that a diet rich in vitamin A helps to preserve one of the first lines of defense against bacterial invasion.

The animals show poor appetite and digestion is disturbed. In a study of the effect of low vitamin A on monkeys (18), 9 out of 11 animals developed colitis, showed microscopic epithelial lesions, digestive disorders, and inflammation of the alimentary tract. Rats, at this time, cough, sneeze, have a nasal discharge, and develop bronchial and lung infections. Their feet become sore, their ears and tails scabby, and abnormal growths may appear on their bodies, as well as skin eruptions and infections. Studies (19) show that the blood count, both red and white, decreases 20%, and the blood pressure may be abnormally low (20). A change of temperature in the room often causes the A-deficient animal to die of pneumonia. The span of life is often shortened by one-half, and there is a general lack of vigor and general health. A diet low in vitamin A affects reproduction, and in other cases where reproduction is not interfered with, lactation is impossible. This shows specifically that it is an essential to adult as well as to early life.

Autopsy shows pus in the middle ear, mastoids, sinus, kidney, pancreas, genito-urinary tract, larynx, bladder, and pus pockets at the base of the tongue and in the salivary glands, as already mentioned. The infection differs with each individual animal, depending on the amount of vitamin A in the mother's diet, and in its own. The amount of the vitamin stored depends on the capacity of the individual, and the time in life to which it was subjected to the dietary insult. A deficiency coming in early life leads to eye infection, while one coming in adolescence leads to infections of the lungs. An animal may have no more than one focus of infection, but in experimental work done on thousands of subjects, each focus appears repeatedly. The above symptoms appear in animals of widely different types, such as the fowl, rat, mouse, guinea-pig, rabbit, monkey, pig, and dog, which indicates beyond all doubt the possibility that a diet low in vitamin A may lead to similar infections in human beings.

It seems useless to summarize the above infections which appear in man. That opthalmia is caused by a deficiency of vitamin A has been proven. Most common among these infections are colds, which are accepted by all too many people as an inevitable part of the winter regime. Poor appetite, disturbed digestion, low blood count and low blood pressure, acne and other skin infections, mastoid, sinus and kidney infections, are met with almost daily. Not infrequently is found the inability to reproduce, and too often, mothers are unable to nurse their infants.

Tuberculosis and bronchial infections are treated by the thousands, often without the slightest increase in vitamin A intake in the diet. During the past week, I have had two tuberculosis patients referred to me, both under excellent medical care, and both being given large doses of irradiated ergosterol (visterol) as a substitute for cod liver oil. I don't believe that it can be overstressed that irradiated ergosterol, while it may be beneficial to cases of tuberculosis particularly where the bone is infected, *does not* contain vitamin A, and therefore, is *not* a substitute for cod liver oil. It may also be significant to remember that the dentist sees larger calculus deposits in the teeth of tuberculosis patients than in those of any other one class of patients. A keen awareness of these symptoms will help the orthodontist to recognize patients with vitamin A deficiencies while in his office, and enable him to recommend diets high in this vitamin.

Comparing the life span of experimental animals with that of human beings, one is impressed by the fact that similar infections appear at relatively the same time. Like the babies in Denmark, the young animal develops opthalmia, and shows a tendency, if the deficiency comes later in life, to break down with lung trouble at the same time pulmonary tuberculosis so often develops in the adolescent boy or girl. "Colds" in animals appear throughout life, though most frequently met with in early life, as in children. Kidney and gall-bladder infections come later in life, as in human adults.

The fact that vitamin A is stored in the body plays an important part in preserving the health over a period of nutritional scarcity. This vitamin is stored in the liver in relatively large amounts and over relatively long periods. For example, two groups of young animals (15) were fed during early life, one on a diet fairly rich in vitamin A, and the other only meagerly, though both were well within normal limits. Each was then subjected to a period when the diet was deficient, and again small amounts were added to their diets. At this time, they were killed, and examined for signs of infection. Despite the fact that through a greater period of their lives, they were treated identically, the group whose diet contained the smaller amount

of vitamin A early in life showed many times more infection than the other. Comparing the time of such experiments to that in human life, Dr. Sherman is led to the conclusion that the high incidence of disease in children between the ages of ten and twelve may quite possibly be due to diets low in vitamin A before they are three years old.

The capacity of each individual to store vitamin A is shown by the fact that young animals, put on deficient diets at the age of four weeks, show different growth curves and survival periods on the same diets. By experiments, in which animals of known nutritional history were killed, dissected, and their tissues fed to young rats, it was shown that the kidneys and lungs contained 40 times as much vitamin A as the muscles, and the liver 200 to 400 times as much, depending on the previous diet. Moderate differences of vitamin A in the food, yet such as are well within the range of variation encountered in human experience, showed large differences in the concentration of this vitamin in the liver, and quite distinct differences in the lung tissues. The latter is of particular interest because it has shown that an abundance of vitamin A in the food makes the lung more resistant to disease.

Age has an important influence on the maximum body storage of vitamin A, the largest store being found at about the beginning of adult life. In rats, this maximum storage is achieved by feeding a ration of 4% of the basal diet cod liver oil, which supplies about *eighty times* more vitamin A than is considered necessary for adequate nutrition.

Bloch (21), reporting on eighty-six children with xerophthalmia, writes that fifteen developed pneumonia, twelve, bronchitis, thirteen, infection in the middle ear, twenty-seven, pyelitis, and fourteen, infections of the skin. It has been pointed out repeatedly in articles in dental journals that these children, though subjected to gross vitamin A deficiencies when a few months of age, have teeth which are no worse than those of other children not subjected to such a deficiency (22). Hess (23) pointed out, contrary to popular opinion, that there was very little calcification of the teeth at birth, and even during the first few months of life. It must also be remembered that these children were given cod liver oil in relatively large quantities and over a long period of time, in an attempt to cure the keratomalacia. Mellanby (4) shows a cross section of the tooth of a puppy which was given cod liver oil for *one month* in order to keep it alive until the end of the experiment. During that *one month,* one third of the dentin showed new growth of excellent structure. Judging from this, and other experiments identical with it,

is it not possible that the cod liver oil may have more than compensated for the vitamin A deficiency suffered by the children reported by Bloch?

In the presence of sunshine, a pigment called carotene (and perhaps other pigments) (24) is synthesized by growing plants. It is a brilliant yellow in color and is found not only in yellow fruits and vegetables, but in even greater quantities in green vegetables and algae which grow in the water. When carotene is eaten by a living animal, it is transformed into vitamin A, apparently by the liver, which stores it in excess, and very probably regulates its distribution to other parts of the body. Animals are unable to synthesize either carotene or vitamin A, though large amounts may be obtained from the cow as butter fat, and from the hen in the yolk of the egg. The term vitamin A covers both its precursor, carotene, and vitamin A proper, as changed by the living body.

Vitamin A is soluble in fat, is unhurt by alkalis, and is only slightly affected by acids. Both carotene and vitamin A are hurt by oxidation, and therefore by cooking, (unless oxygen is excluded, as in commercial canning) since heat acts as a catalyzer. It is often surprising that successful clinical results are achieved by raw food faddists and yet one only has to remember that vitamins A, B, C, and proteins are all more or less hurt by cooking. It follows that all cooking should be done in as short a time as possible, and with the greatest exclusion of oxygen.

A unit of vitamin A, used in American laboratories, and adopted by the United States Pharmacopoecia, is that amount which, when fed to a standard experimental animal, a rat, promotes an average three-gram gain per week over an eight-week period. (For a more complete knowledge of the number of units found in all foods, the reader is referred to page 365, Chemistry of Food and Nutrition, H. C. Shermen, Fourth Edition, Macmillan Co., 1932.) Although exact requirement is unknown, it is thought that no less than 3,000 units are needed daily as a preventive dose, and at least 10,000 units daily for a curative dose.

Escarole, spinach, chard, alfalfa, (wisely added to a cereal now on the market) for babies and other leaves equally green, carrots, and apricots, are among the richest food sources, containing about 60 units per gram. Artichokes, green string beans, Brussels sprouts, green peas, lettuce, red and green peppers, tomatoes, pumpkin, green or yellow squash, prunes, yellow peaches, dried peas, contain one to four units per gram, depending on the color. Fruits, if dried with sulphur, are also a good source. White cabbage, cauliflower, eggplant, bleached celery, lima beans, navy beans, potatoes,

onions, beets, turnips, cereals (except yellow corn), nut and vegetable oils, contain very little or negligible amounts. While liver may be one of the richest sources, its value depends on the diet of the animal from which it came, making it not a dependable source. Kidney is fairly rich, while muscle meats contain little. Egg yolk and dairy products, cheese, cream, butter, and whole milk, are more dependable, and are most excellent sources. Evaporated, condensed, and dried whole milk retain vitamin A.

Breast milk is usually much richer than cow's milk, depending again upon the diet of the mother. Colostrum has been found to be exceedingly rich. The first act in prevention, for the orthodontist is to do whatever he can toward influencing his patients to nurse their children rather than to feed them artificially. It is well known that artificially-fed babies, like experimental animals, develop infection of the upper respiratory tract, and are likely to have poor tooth structure. Daniels (25) reported an interesting observation showing an adverse effect of vitamin A deficiency on absorptive power of the intestine. An ether extract of a three weeks fecal excretion of an artificially-fed baby was prepared. An extract of the excreta of a breast-fed baby of approximately the same age was likewise made. Both extracts were tested on rats on low vitamin A diets. The animals fed the extract from the artificially-fed baby recovered, while the others died. It appears that the artificially-fed baby did not absorb its vitamin A.

A study was made (26) of 2,815 babies, two thirds of whom were nursed for eight months. The infant mortality rate of these babies was much lower than that of the country as a whole, while the morbidity of these children compared to children nursed a shorter time, shows the most marked difference.

An excerpt from the report is as follows: "The greatest difference is found, as would be expected, in diseases of the digestive tract . . . Respiratory disease shows the next highest variation. Note especially the prevalence of pneumonia and bronchitis among the infants with limited infectious diseases, especially whooping cough, measles, and chickenpox . . . " Undoubtedly, the larger amount of vitamin A in breast milk, and the superior power of absorption of equivalent amounts on the part of the breast-fed plays a part in such freedom from infections.

Cod liver oil varies with the locality from which it comes, Icelandic oils and those from Newfoundland being much richer than Norwegian oils. Oils may vary from 3,000 to 10,000 units per tablespoonful, all being ex-

cellent sources. Oil from halibut liver recently has been put on the market, and has been found to be many times richer in vitamin A than cod liver oil. Pure carotene, in a solution of oil, is now available, and is excellent to use in vitamin A deficiencies, although it is *not* a substitute for cod liver oil, since it contains no vitamin D. Concentrated fish oils in tablets and capsules, which are palatable and contain very few calories, are now available on the market, and are probably excellent sources if the covering cannot be permeated by oxygen.

Recently, I asked a class in nutrition to list what they had eaten during the day and to estimate their intake of vitamin A. None on the class had eaten more 2,000 units many having consumed far less than this amount, with the exception of one young woman, who had been sent to the office a few days before to have diet instruction in the treatment of a severe pyorrhea. She had obtained more than 11,000 units, and this did not count the halibut liver oil capsules she was taking.

The following are some of the richest sources of vitamin A, given in approximately equivalent values, each amount yielding about 3,000 units: 5 halves of dried apricots*, 1½ tablespoonfuls of liver, 1 small carrot, ¼ cup cooked or canned spinach, 4 thin squares of butter, 1 teaspoonful high grade cod liver oil**, 2 ounces or 2/3 package cream cheese, 4 ounces American cheese, 3 medium eggs, 1½ cups uncooked prunes* or dried peaches*, 3 large tomatoes, 6 medium bananas, 1 1/3 quarts whole milk, 2½ cups canned peas, 3 large green peppers.

Recently a study (27) was made of the cost of vitamin A, and it was found that among families with the same amount of money to spend on food, some were buying three times as much vitamin A as others. By using cheese and eggs as substitutes for muscle meats, a more frequent appearance of liver on the menu (cow's or lamb's rather than calves' liver, which is sometimes prohibitive in price), the use of more milk, be it whole, powdered, condensed or evaporated instead of fresh, particularly to replace tea or coffee, many times more vitamin A can be purchased for the same amount of money. Spinach, carrots, broccoli, sweet potatoes, and other colored vegetables never cost more than cauliflower, turnips, white potatoes, celery or onions. Storage or summer eggs and butter have been reported to be richer in vitamin A than winter products, and may be bought during the

*Morgan, A. F. and Field, A.—Journal of Biol. Chem. Volume 88, No. 1, August, 1930. The effect of Drying and of Sulphur Dioxide upon the Vitamin A. Other figures computed from reference (15) (Content of Fruits).

**Figures from Council Accepted Commercial Products.

winter for less money. By substituting colored fruits for desserts instead of colorless ones, or, still better, instead of cakes and pastries, the budget stays within bounds and greater health insurance is purchased.

Patients can easily be taught these simple rules by which to select their menus. And, in addition, in such conditions as pregnancy, lactation, rapid growth, and particularly in the presence of infections such as tuberculosis, sinus, pyorrhea, susceptibility to caries, calculus deposits, skin infections and colds, a fish oil should be given, not only to children, but *to adults* as well. Even as a preventive measure, it is not out of place for the normal person to take fish oil, the equivalent of one tablespoonful of cod liver oil a day.

A patient will never stay on a diet he dislikes unless it is absolutely necessary. For this reason, individuals finding the taste of oil unpleasant, should always be given a concentrate in capsule or tablet form. In cases of obesity, fat allergy, or other cases where oil is not desirable, with a consequent exclusion of butter, cream and cheese, a diet low in vitamin A is yielded, and such a form should always be given. Almost daily a patient asks, "How long must I take cod liver oil?" The only answer which can be given sincerely is, "As long as you are interested in optimum health!"

At last, here is a substance, vitamin A, where the old saying, so often erroneous, holds true, that if a little is good, more is better. Although the body uses vitamin A less economically when it is generously supplied, to get too much seems to be an impossibility. An abundant supply of vitamin A today means a store of health for the future.

Unfortunately millions of people lie in the borderland between recognized sickness and positive health. The physician does not see them, for they are not ill; hence he has no chance to aid them. The dentist does, for they come into his office one or two times annually.

The orthodontist has even a greater opportunity to better the condition of his patients, for they come to him, perhaps weekly, over a period of years, beginning at an early age, when not only the teeth and dental structure, but the body as a whole, is rapidly growing. It is not only his opportunity, but almost his duty, to help them over the border to *optimum* health.

Wakefield Building

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