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ACCESSORY FOOD FACTORS (VITAMINES)

IN THE FEEDING OF INFANTS

BY

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ACCESSORY FOOD FACTORS (VITAMINES) IN THE FEEDING OF INFANTS.[1]

IT is obviously a matter of great difficulty for a laboratory worker to open up a discussion concerned with the feeding of children. The task is not rendered easier by my consciousness of the exceptional difficulty in arguing from one species of animal to another when dealing with questions of diet and metabolism. Every worker on the subject of vitamines has this difficulty always before him. We must also recognise that much of the accessory food factor story has not been extended to children or tried beyond the laboratory walls. It is just for these reasons, however, that a discussion in which the laboratory worker and the clinician meet face to face is of great value. The physiologist may throw new light on the points under discussion, and, still more important, the clinician may assist the investigator in evaluating his experimental results and so prevent the exaggeration of one fact at the expense of another.

We have to consider the part played in the nutrition of infants by the three known accessory food factors: (1) antiscorbutic; (2) fat-soluble A; (3) water-soluble B or antineuritic. From the point of view of the children of this country we can probably eliminate the water-soluble B factor. Although infantile beri-beri exists in rice-eating countries and probably in other places where one-sided and limited diets are in use, the extensive distribution of this factor in food, together with its well-recognised resistance to heat, would appear

[1] Opening Remarks at the Discussion at the Section for the Study of Disease in Children of the Royal Society of Medicine on Feb. 27th, 1920 (v. THE LANCET, March 13th, p. 604).

to make disease due to its deficiency unlikely to occur in Western countries.

Problems Concerning the Antiscorbutic Factor.

Of the antiscorbutic factor I shall say but little. I suggest, however, that there are certain points in connexion with this factor which urgently require consideration. It would be interesting if we could have authoritative statements on the following points: (1) Is the scurvy problem among children of this country one involving real danger? We know that the classical scurvy symptoms are rare, but it is not equally certain that milder symptoms indicating a deficiency short of the production of scurvy are not common. (2) Is it essential that children feeding on dry milk compounds alone should take orange or swede juice?

The second problem involves an answer to two questions: (1) How much of the antiscorbutic factor is destroyed in the drying of milk? (2) How susceptible is the child as compared with the experimental animals? Speaking without any particular knowledge on this point, I should think it probable that, although an infant feeding entirely on whole dried milk may not require extra antiscorbutic, yet when cereals are added to the diet additional antiscorbutic factor will be essential.

The Relation between the Fat-soluble A Factor and the Antirachitic Factor.

I propose to deal with what is possibly the fat-soluble A accessory factor. You may remember that the identification of the fat-soluble factor depended on work by Macollum in which the growth of rats was studied. The antirachitic factor, on the other hand, has been so called because of the effect of certain substances in preventing the development of rickets in dogs. Whether they are identical is unsettled, but one point of difference is so striking that it is essential to bear it in mind. The fat-soluble factor is, according to the rat-feeding experiment, absent from all vegetable fats. In the rickets experiments, however, it seems definite that the amount of antirachitic factor is fairly high in some vegetable fats, such, for instance, as peanut, cotton-seed, and coconut oils, and is only present to a small extent in palm-kernel, linseed, and babassu oils.

A second point worthy of emphasis is that fat-soluble A does not appear to be as necessary for growth in the

case of puppies as it is in rats. It is true that all the diets of the pupples must have contained a trace of fat-soluble A, but the growth was often just as good when the diet was very deficient in this factor as when containing it in abundance. This fact, previously described by me, has been extended to children recently by Hess and Unger. On a diet of dried separated milk, sugar, cotton-seed oil, autolysed yeast, orange juice, and cereal, sustained over many months, they found the growth of children practically normal. The question arises as to whether, in speaking of the fat-soluble factor in relation to child nutrition, we should still continue to call it a "growth factor." For the growth of puppies and children its influence is probably smaller than that of the other elements of the diet.

It seems most likely that both these differences—that is, (1) the difference in distribution of the fat-soluble factor and the antirachitic accessory factor in vegetable fats; (2) the importance of the fat-soluble factor for the growth of rats and its probable unimportance in the growth per se of puppies and children—can be explained by variations in the general and intermediate metabolism of these animals. These points will, no doubt, be ultimately solved by further experimental work.

In the meantime I would beg those who are extending the results of experimental research on rickets to children not to mix up the two types of work—namely, the growth experiments on rats and the rickets experiments on puppies. In giving cotton-seed oil to children, as recently carried out by Hess and Unger, they had in their minds undoubtedly the generally accepted fact that this oil contained no fat-soluble A. On the other hand, so far as my experiments have shown, cotton-seed oil is to be classed as one of the better vegetable oils in preventing rickets, and, taken in conjunction with the other conditions of diet as described by Hess and Unger, it does not appear to me surprising that rachitic symptoms did not develop. I shall touch again on their experiments later.

From my earlier work, a short account of which has been already published, I consider that it is undoubted that something of the nature of a vitamine, distributed in varying quantities in fats, plays an important part in the development of rickets. I do not wish now to deal further with this particular point. In view, however, of the erroneous and exaggerated views that are widespread as to the action of the antirachitic accessory factor in diet, it is essential that an effort should be made to bring such substances away from their atmosphere of mysticism, and place them side by side with other elements of the diet. If we can

in some way link up their action with the known dietetic substances, so that we can discuss them in terms that are familiar, good progress will have been made along these lines. I propose, therefore, in the remaining time at my disposal, to describe some of the more recent results of my experimental work carried out with this end in view.

The Relation of the Antirachitic Factor to Age.

It is a well-recognised clinical fact that active rickets is a rare disease in children over 2 years of age. On the other hand, arguing on the basis of my experimental results obtained with puppies, it is equally certain that, after this age, the diet of a child is often of a more rickets-producing nature—that is to say, it is frequently composed more of bread and other cereals and less of milk. The only deduction that is possible from these facts is that after a time a child becomes more independent, from the rickets point of view, of its diet. I wish to emphasise that it is only the rickets point of view I am discussing. No further deduction from this result must be made as regards resistance to infection or any other pathological condition produced by diet, and more particularly the antirachitic factor of diet at various ages. The fact that the antirachitic accessory factor is of less importance in the older child is also brought out strikingly in puppies. It is obvious for the following well-defined reasons. (1) After the puppy has arrived at a certain age I have been unable to produce rickets by feeding it on rickets-producing diets. (2) On definite diets puppies develop rickets, but if not too severe, and the general condition does not become too bad, recovery at the growing ends of the bones, as indicated by radiographic examination, often takes place, although the animal may remain on the same diet and under the same conditions.

To take the first of these facts, a puppy put on to a rickets-producing diet of separated milk, white bread, meat, orange juice, and linseed oil at the age of 5 months was photographed (Fig. 1) and radiographed (Fig. 3) at the age of 10½ months and was found to be free from rickets. Fig. 2 is a radiogram taken at the age of 5 months, before beginning special diet. The dieting has now been continued over 11 months in all, and the dog is still well and healthy and shows no signs of rickets. The earlier treatment of this particular dog I shall refer to later, when I deal with the effect of exercise and confinement on the development of rickets.

As regards the second of the above facts, photographs show an instance of self-cure in a puppy when the diet and other conditions were kept as far as possible the same as when the rickets developed. Figs. 4, 5, and 6 represent the changes taking place in the wrist of a puppy. The diet in this case consisted of separated milk, bread, orange juice, yeast, meat, and linseed oil, and you will see that on March 28th, after 3½ months of this diet, rickets had developed. It will be seen, however, from Fig. 5 that, one month after the first radiograph, the healing process had started and renewed calcification is evident between the epiphyses and diaphyses of the distal ends of the radius and ulna. In Fig. 6 the healing process had continued to a further stage. Here, then, we have evidence of a self-curative process taking place in a puppy as it grows older quite comparable to the self-cure which is well recognised as taking place in children.

The above facts can be taken as evidence that the antirachitic accessory factor is more necessary in the diet the younger the animal—that is to say, until some essential process or secretion has developed in the young animal. Until this process has evolved (of its nature I have no idea at the present time) rickets will more readily develop. After the necessary establishment of the new process in the young animal or child, it is a matter of some difficulty to produce rickets. Whether all new calcification processes proceed normally after this event, even in the presence of deficient diets, still remains unknown, but further experiments will soon clear up this point. I should expect that a deficient diet will continue to act in a detrimental way to calcification processes, but possibly not to such a profound extent as in the younger animal and child. It is probable that the legs will continue to bend owing to poor calcification of the periosteal bone of the shafts even after there is good evidence of a healing process going on at the epiphyseal ends. The recovery and calcification of the periosteal bone of the shaft appears to be a much slower process than that at the growing ends.

The Relation of the Antirachitic Accessory Factor to the Energy-bearing Portions of Diet.

In a paper already published I laid stress on the fact that, although rickets is primarily a deficiency disease of a dietetic nature, other metabolic conditions must obviously be involved, because it is impossible to regard the accessory food factors as independent of

the general metabolism and of the other elements of the diet. I emphasised the unity of a perfect diet because we are becoming more and more aware that the removal or deficiency of one element in the diet involves much more than the absence of function or defective function carried out by this one element. It also means that the other parts of the diet cannot do their task efficiently because of the constant interplay between the chemical changes in the body and their dependence on each other. During the past 18 months I have been endeavouring to prove that the same dependence and inter-relationship holds between the vitamines and the proteins, fats and carbohydrates, and, although this part of the work is still in progress, sufficient evidence has been obtained to allow the conclusion that their interaction in metabolism is of great importance so far as the development of rickets is concerned. I have explained in my earlier work how the tendency to rickets is increased by more rapid growth, using the weight of the animal as the indication of growth. I am fully aware that increase in weight is not a true indication of rate of growth, because it obviously includes other factors, such as the laying on of fat. For the present, however, I shall confine myself to the relation of the development of rickets to the increase of weight. In many of the experiments the only variable part of the diet was bread, given generally to the animals *ad lib.* Therefore it is evident that, other things being equal, the greater the amount of bread eaten the greater will be the tendency to rickets. This generality is probably true, so long as the increased bread intake results in an increased *storage* of foodstuff, retained by the animal as body tissue. The same condition probably holds with oatmeal and other cereals.

In order to illustrate this point I shall show you an experiment carried out on three puppies of the same litter. The diet in each case consisted of separated milk, white bread, orange juice, meat, and palm-kernel oil. In Fig. 7 you see the relative rates of growth of these animals. In the course of growth 221, the least rapidly growing, gained 1700 g., 223 gained 2150 g., and 220, the most rapidly growing, gained 2850 g. during the same period of 66 days. Throughout the experiment, except that the meat was raised from 5 to 10 g. per diem a few days earlier in 220 than 223 and 221, the only variable was the amount of bread eaten. All other conditions, such as housing, were identical. If, therefore, any great difference regarding the development of rickets occurred it can probably be ascribed to the variable bread intake. The radiographs of these three puppies after about nine weeks of the diet can be seen in Figs. 8, 9, and 10. It will be noticed that 221, the least

rapidly growing, has very slight rickets, 220, the most rapidly growing, very severe rachitic changes, and intermediate between these two is 223, whose rate of growth is also midway between 221 and 220. Figs. 11, 12, and 13 are photographs of these three puppies, 221, 223, and 220. It will be seen that the bending and external appearances of their legs are in keeping with the radiographs of Figs. 8, 9, and 10. Let me again repeat that

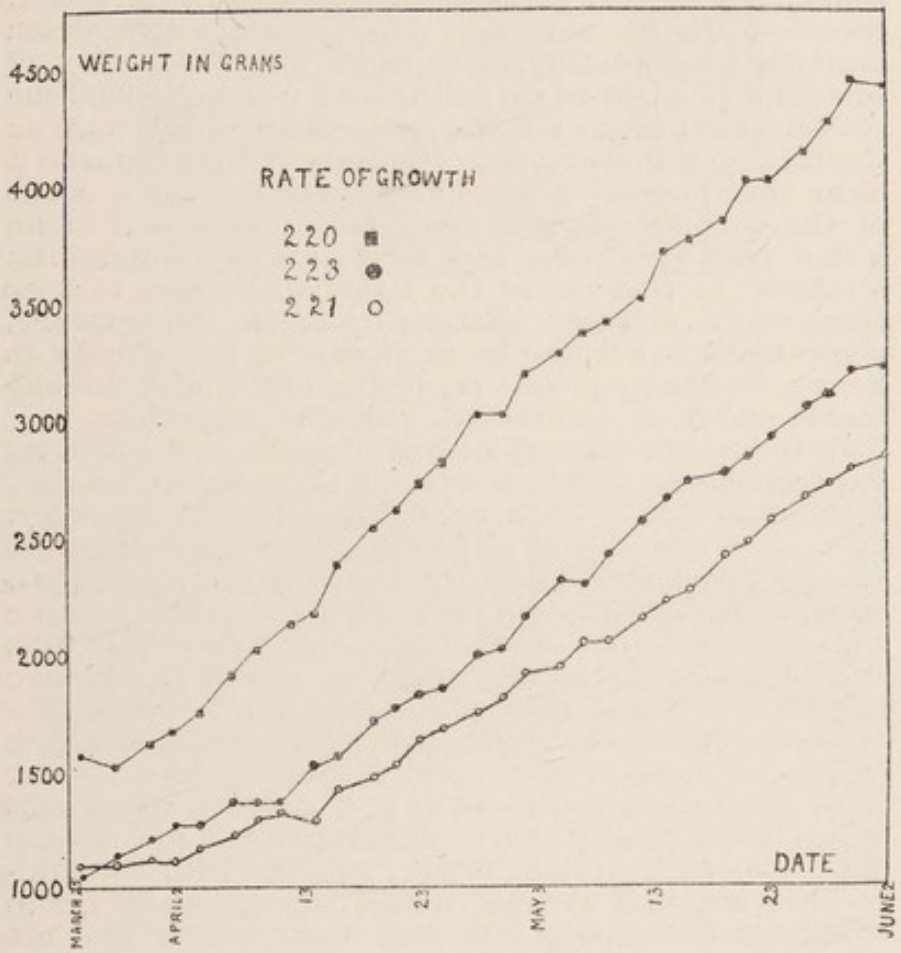

FIG. 7.—Relative rates of growth of three puppies of the same litter on diets differing only in quantity of bread eaten.

I am only using the word growth in the sense of putting on weight. It appears then that puppies, living under similar conditions, show a greater tendency to develop rickets when the diet contains a larger amount of cereal, all other elements of the diet being kept constant. The question arises as to what special element of the bread is responsible for this greater tendency to rickets. This point awaits

complete solution, but evidence is accumulating which indicates that the carbohydrate moiety is the offending substance. There is some evidence, on the other hand, that protein has an antagonistic action to the development of rickets. In my earlier paper I referred to the effect of meat in making a slightly rickets-producing diet into a safe one. There is also an indication that casein has the same effect. F. G. Hopkins has shown that ordinary commercial casein, as used in these experiments, may have a large amount of fat-soluble A accessory factor associated with it. Whether, therefore, the protein effect depends on its own action remains to be decided. If the antirachitic effect of protein is established we will be able to comprehend one reason why milk is a better preventive of rickets than the corresponding amount of butter. I wish to make it clear that increasing the protein alone in the absence of the antirachitic accessory factor does not make a diet safe. My view is that it aids the antirachitic vitamine, so that less of the latter will suffice to keep the growth normal. Carbohydrate, on the contrary, especially when it results in storage of fat, appears to have a decidedly rickets-producing effect, and thereby makes additional antirachitic vitamine imperative.

It is possible that Hess and Unger's recent feeding experiments on children referred to above, where they found that 180 g. of a dried separated milk powder, 30 c.cm. of cotton-seed oil, 30 g. of sucrose, 15 c.cm. of orange juice, and 30 c.cm. of autolysed yeast, and, later, additional cereal per diem, did not produce rickets, may have as one part of the explanation the high protein intake of these children. I have already explained that cotton-seed oil is one of the better vegetable oils as regards its antirachitic action in puppies. 180 g. of a dried separated milk powder must have contained about 65 g. of protein, an amount which is abnormally high for children from 5 to 9 months old. This high protein intake, together with the antirachitic factor of the cotton-seed oil, appear to me to explain satisfactorily the fact that rickets did not develop in these children. As regards their observation that the children grew fairly well, in spite of the great deficiency of the fat-soluble accessory factor, this is additional evidence that it is not impossible to argue from my experimental results on puppies to children, as I have previously commented on the good growth that can be obtained in puppies under the same condition.

I should further like to point out that the whole subject of the relationship of accessory food factors to the energy-bearing portion of the diet is one of great importance, and it is necessary that investigations

FIG. 1.—Photograph of a puppy, 10½ months old, which has been on a rickets-producing diet for the last five months without developing rickets.

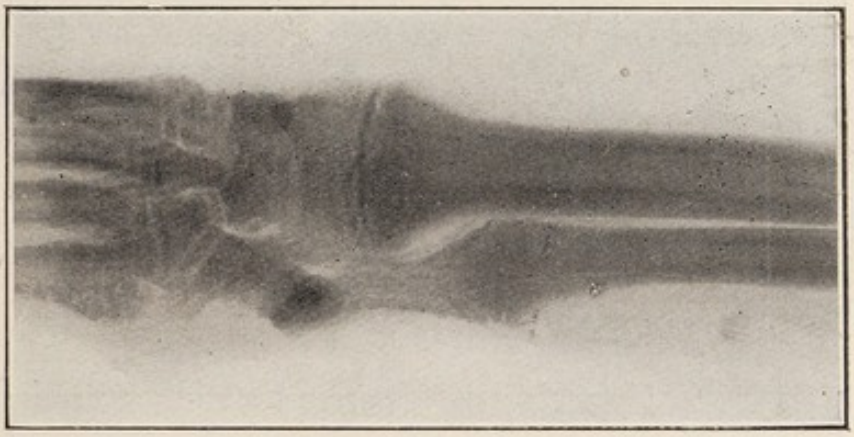

FIG. 2.—Radiogram of puppy in Fig. 1 at the beginning of experimental feeding, showing healthy wrist-joint.

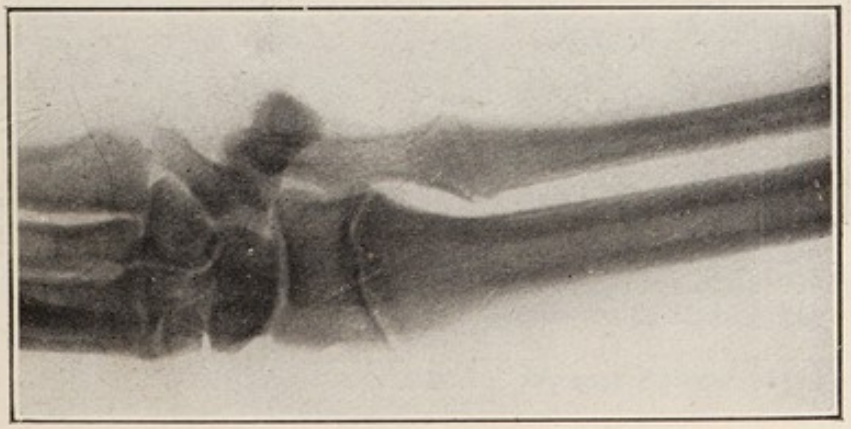

FIG. 3.—Radiogram of puppy in Fig. 1, taken at same time as the photograph, showing healthy wrist-joint.

FIG. 4.—Rickets in wrist-joint of a puppy after 3½ months of rickets-producing diet in spite of complete freedom.

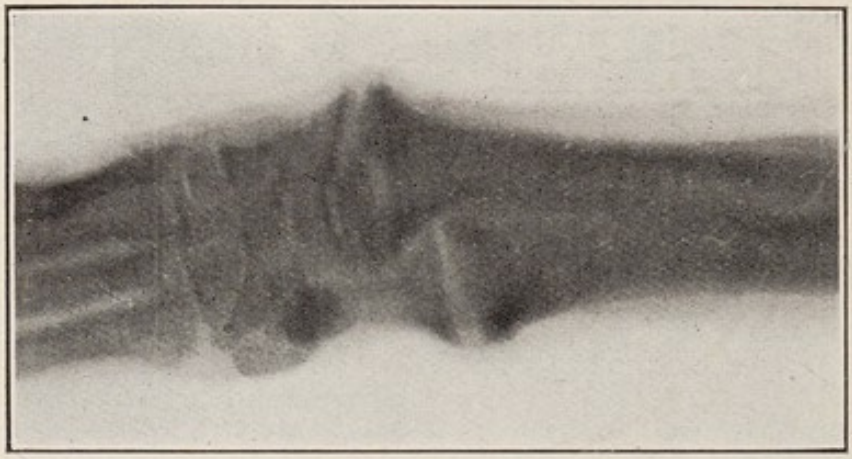

FIG. 5.—Wrist-joint of same puppy as Fig. 4 one month later, undergoing self cure. Puppy on same diet.

FIG 6. –Same puppy as Fig. 5 one month later, showing healing process still further advanced.

FIG. 8 (221).—Very slight rickets only in wrist-joint of slow-growing puppy. (Cf. Fig. 11.)

FIG. 9 (223).—Rickety changes intermediate in severity between Figs. 8 and 10, corresponding to intermediate rate of growth. (Cf. Fig. 12.)

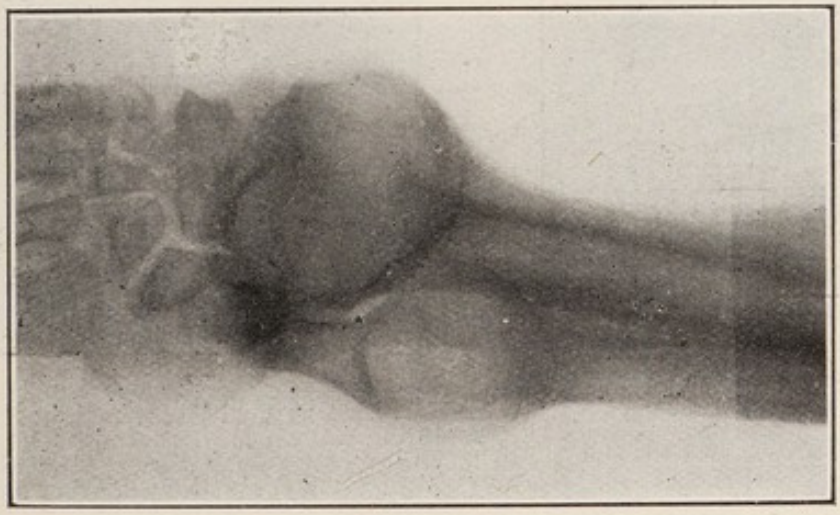

FIG. 10 (220).—Severe rickets in wrist-joint of most rapidly growing puppy. (Cf. Fig. 13.)

FIG. 11 (221).—Slight rickets. Wrist-joint shown in Fig. 8.

FIG. 12 (223).—Rickets intermediate in severity between Figs. 11 and 13. Joint shown in Fig. 9.

FIG. 13. (220.)—Severe rickets. Joint shown in Fig. 10.

FIG. 14.—A typical "black house," Island of Lewis, in the Hebrides. The sanitary conditions are very bad, but rickets is almost unknown in the island. Note smoke of peat fire issuing from door.

on scurvy and beri-beri should also be undertaken in this connexion. It will be generally agreed that neither scurvy, beri-beri, nor rickets will develop as the result of starvation only. Therefore we must consider whether there is not a positive side also to these dietetic diseases. We know in the case of scurvy and beri-beri that administration of the suitable vitamine will effect a cure, but is it not also probable that the diseases are brought on by some other element of the diet, since they do not develop as the result of starvation? There is some direct evidence that this is the case in avian polyneuritis, and the offending element in the diet is probably carbohydrate. Results tending to prove this have been obtained by Abderhalden, Braddon and Cooper, and Funk. So, also, in the case of rickets, it may be ultimately established that it is excess of carbohydrate in an unbalanced diet which is largely responsible for the development of the disease, and that it is the special function of the antirachitic accessory factor to prevent the abnormality. The fact that the laboratory results obtained on deficiency disease have not been entirely confirmed in practice may be the result of the omission to link up accessory food factors with carbohydrates and the other energy-bearing substances in the diet.

The Effect of Exercise on the Development of Rickets.

I have previously pointed out that, although I considered Finlay's results showing the effect of exercise on rickets to be important, I do not think exercise the prime factor in the ætiology of rickets, but only subsidiary to diet. It is, however, obvious that the ultimate explanation of rickets must embrace this also. Whatever inhibitory effect the constant running about has in the case of puppies, we must, when considering the disease in children, discount a large part of this effect. The reason for this is that rickets develops in many children when only 6 months to 1 year old, and it is difficult to see how running about can play a large part at this age. The exercise obtained by children at this period of their life consists of small movements which make up their general liveliness, and I think it will be agreed that the activity of a child depends, more than anything else, on the adequacy of its diet. Surely the size and number of rooms in the house where the child may carry on its movements are of subsidiary importance to its diet. If exercise and muscular contraction are the explanation of the ætiology of rickets, then there is

clearly a simple method of eliminating the disease from Vienna, and the apostles of this gospel ought now to be preaching the glad tidings and observing its effect there. It appears to me absurd to think that the widespread and intense nature of rickets in Vienna and elsewhere in recent times has arisen primarily because of any lack of exercise or because of the more defective hygienic conditions that may have developed within the last few years.

My experiments on exercise have been made along various lines and the results obtained may be briefly stated: (1) Confinement on an adequate diet will not produce rickets. (2) Freedom in the daytime will not prevent rickets when the diet is inadequate. (3) Confinement will not prevent the cure of rickets, when the diet is good. On the other hand, complete freedom and the possibility of constant running about, will oppose to some extent the rickets-producing effect of a deficient diet and may carry it over to the safe side in small, slowly-growing dogs.

Fig. 1 is a photograph of a dog previously referred to, which was confined from the second to the fifth month of its life. During this period its diet consisted of 250 c.cm. whole milk, 20 g. meat, 5–10 c.cm. cod-liver oil, 5 c.cm. orange-juice, and white bread. In spite of the absence of the opportunity for exercise, it was a beautiful dog, showing no signs of rickets, as can be seen from the radiograph (Fig. 2) taken after the period of confinement.

The brother of this puppy was allowed complete freedom during the daytime (with a special type of muzzle on) and developed slight rickets, which can be seen in Fig. 4. It was eating a rickets-producing diet during this period, consisting of 175–250 c.cm. separated milk, white bread ad lib., 5–10 g. meat, 10 c.cm. linseed oil, and 5 c.cm. orange-juice. This animal was an instance of self-cure and, while living under the same conditions, calcification of the growing ends of the bones was resumed. This point is seen in the radiographs (Figs. 5 and 6), and has been previously commented on. The rate of growth of this puppy was not very good—it increased 2730 g. in 19 weeks while eating the diet.

Here, then, we have instances of two puppies (terriers) and members of the same family, the one remaining normal in spite of lack of exercise and the other developing rickets with full opportunity for exercise. The dominant factor in each case was the diet. The first dog had an abundance of antirachitic factor in its diet, which included whole milk and cod-liver oil. The second on separated milk and linseed oil was getting little or no antirachitic factor. I wish to repeat, however, that muscular contraction has some inhibitory

action on the development of rickets and must be considered in the general scheme.

The points I have discussed, more particularly the relation of the antirachitic accessory factor to the energy-bearing portions of the diet and to exercise, indicate plainly that rickets must be considered as a problem of general metabolism. As a working hypothesis it seems possible to formulate a general scheme in which each of the various elements already discussed can find a place. Any condition which induces a laying on of tissue seems to necessitate a greater intake of antirachitic accessory factor to prevent rickets. On the other hand, conditions which stimulate the metabolism and increase the heat loss relatively to the energy of the stored food work together with the antirachitic accessory factor, and make a less amount necessary for normal growth. For instance, excessive carbohydrates in the diet often brings about a condition of laying on of fat associated with lethargy. Confinement works in the same direction. The diet under these conditions must have more antirachitic accessory factor, otherwise rickets develops. Proteins and exercise, on the other hand, are stimulants to the metabolism, and, when the diet has a relatively high protein content, and the animal is active, less antirachitic accessory factor is necessary.

It is upon this hypothesis that my present investigations on rickets are being continued, and while it is freely admitted that it is not based on complete experimental evidence, a general conception is always useful to an investigator, and can do no harm so long as it is regarded by others in its true light. The generalisation may not explain all conditions under which rickets develops, but we are in the satisfactory position of knowing that its proof or disproof can be easily tested by further experiment.

The Importance of the Antirachitic Accessory Factor in the Feeding of Children.

Up to the present I have dealt entirely with the effect of the antirachitic vitamine in the case of puppies, and it may be of interest to attempt to supply some evidence in support of the animal results, which show that, in the case of children also, this substance is of great importance. I shall deal with two investigations[2]

[2] These instances were kindly brought to my notice by Dr. H. Scurfield, medical officer of health for Sheffield.

carried out at a time when the presence of the anti-rachitic accessory factor in food was undreamt of.

In examining school children at Leeds Dr. William Hall was much interested in the great difference in general nutrition, and more especially in the teeth of Jewish and Gentile children, the financial position and housing of whose families were comparable. The results of his investigations were described at a health congress in Leeds in 1902, and his general observations and conclusions are so concordant with my experimental results that I should like to record them. The table here given represents a few of these results.

	Rickets.	Bad or backward teeth.
Good district Gentile school ...	8%	..... 38%
,, ,, Jewish ,, ...	5%	..... 11%
Poor ,, Gentile ,, ...	50%	..... 60%
,, ,, Jewish ,, ...	7%	..... 25%
Country school (Ripon Cathedral School)	11%	..... 33%

The great difference between these two classes of people, more especially in the poor schools, is very striking. It will be further observed that the condition of the poorest Jewish children is better than that of the country children.

Hall then proceeded to investigate the cause of the above-described differences, and finally decided that only the dieting could be held responsible. From his analysis of the diets, which he found very different in the two classes of the community examined, I take the following points: 1. The Jewish families used large quantities of oil in cooking, even in making bread. Fish were generally fried in oil. Potatoes were not boiled in water. If boiled it was usually in milk. Otherwise they were cooked in oil. In making broth, butter and oil were added to it. 2. The normal beverage was cocoa made with milk. This was usually drunk three times a day except on the days when meat was eaten. 3. An analysis of the eggs eaten by the Jewish families showed the large numbers consumed. 4. Fruit and vegetables were used abundantly. 5. The Jewish mothers combined to buy large quantities of the cheaper fish in the market. Herrings were commonly eaten.

Dr. Hall's conclusions as to the inferior physique of the Gentile families were (1) that it did not depend on heredity or on city life; (2) that it was purely dietetic. My researches on animals are in complete agreement with the outcome of these investigations. The diet is certainly the key to the physical defects so common among urban and city inhabitants. It is also remarkable how closely the diets of the Jewish community as

observed by Hall follow the animal experimental results. Nearly all the substances mentioned by Hall contain an abundance of antirachitic accessory factor.

The next subject connected with diet and child nutrition to which I wish to refer concerns the inhabitants of the Island of Lewis in the Hebrides. The facts I shall mention are taken from the Carnegie Report of the Physical Welfare of Mothers and Children of Scotland. Many of the inhabitants of these islands live in what are called "black houses." A photograph of a typical "black house" is shown in Fig. 14. They are constructed of turf and stones with a thatched roof. There is often no chimney to the house, and since the peat fires are kept constantly burning and there is no exit for smoke except through the door, the condition of the atmosphere of the house can be well imagined. The walls are often as much as 5 feet thick. Cattle often live under the same roof, the byre adjoining the house, and it is sometimes necessary to pass through the byre to enter or leave the building. Chickens have full run of the house and go on to the beds, tables, and dressers. Altogether the hygienic conditions are dreadful. What about the children? The statements are made in the report: "The children are not taken out until they can walk"; "never taken out until they can walk, except possibly for a few minutes on a fine day in the summer time." It is quite clear, therefore, that, if bad hygiene and lack of exercise are responsible for rickets and ill-health, then the infants of Lewis ought to succumb at a great rate. What are the facts? The main fact is that the death-rate of these infants under 1 year is about the lowest in the British Isles, and has occasionally fallen to as low a figure as 40 per 1000. This death-rate compares very favourably with the infantile mortality of 100 to 300 per 1000 usually found in the towns of this country, in spite of the efforts made to improve the general hygiene. It is remarked that rickets is almost unknown in the island, and "the most striking fact in the adult population is their beautiful teeth—a testimony to the absence of rickets in infancy." Parenthetically, I regret that lack of time has prevented me from showing on the lantern some of the beautiful specimens of my wife's work on teeth—work that has shown that the formation of perfect and imperfect teeth and jaws is affected by diet in a similar way to the development of the normal and rachitic condition of bones.

If we now examine the diet of these people we find results in close agreement with expectations based on the above described experimental work on puppies. In the first place, practically all the children are breast-

fed. Again, an analysis of the foods eaten shows that the staple articles of diet are fish, oatmeal, and eggs. It is true that milk is scarce, except in the summer, but against this we have breast-feeding affording the opportunity to childreh to get milk at the most important period of their lives. As regards fish, we find that the liver of fish—that is, the best source of the antirachitic accessory factor—is regarded as the favourite dish. It is described as being mixed with oatmeal and milk and cooked in cods' heads, each member of the family being provided with a cod's head. It is almost certain that the breast-feeding of the children, together with the high protein and antirachitic accessory factor content of the adult diet, are responsible for the absence of rickets, the formation of beautiful teeth, and the very low infant mortality found in these islands, and that this is the case in spite of the dreadful hygienic condition of many of the houses. For the benefit of those interested in the antiscorbutic factor I may mention that vegetables are almost unknown in these parts.

Evidence is accumulating from various sources showing the effect of the diet of the mother on the accessory food factor content of milk, so that it is likely that the milk of the Lewis women is particularly rich in the antirachitic accessory factor. I do not wish, however, to discuss this point now.

If we follow up the mortality and health history of the Lewis children after the age of 1, additional facts of importance can be observed. From 1 to 5 the death-rate among the children is high, when considered in relation to the infantile mortality under 1. The housing conditions are such that epidemics spread rapidly and fatally, and phthisis in 1914 caused a death-rate double that on the mainland. When the children begin to go to school they fall off rapidly until they attain an age when they can look after themselves. This is because they generally have to go to school before their parents are out of bed, and so often do not get a real meal until midday. When the children have arrived at the age when they can fend for themselves and satisfy their appetite by seizing food, if need be, they develop well and ultimately become the fine stock so well recognised as coming from these islands.

The Relative Importance of Diet and Housing at Different Ages.

I have dwelt on this particular point because it seems to afford an opportunity of realising to some extent the relative importance of diet and housing

at the different ages. It is difficult to avoid the conclusion that diet is everything to infants under 1, and so long as this is good, bad hygienic conditions are of small significance. After the first year, however, when the child becomes more susceptible to measles, bronchopneumonia, and other infections, then clearly the housing and hygienic factors, in addition to the diet, are of great importance. If these suggestions could be definitely proved and accepted, we should have gone a long way towards the solution of the problem of race decadence. It is reasonable to accept as facts that where there is low infant mortality there is an almost complete absence of rickets and the teeth of the people are good. Also that the production of rickets depends on a *relative* insufficiency in the diet of the antirachitic accessory factor, the best sources of which are fish oils, animal fats except lard, milk, and eggs. It is also probable that anything which stimulates metabolism, such as high protein in the diet and exercise, aids the antirachitic accessory factor; while excess of carbohydrate, especially if it leads to a laying on of fat, works in an antagonistic way to the vitamine and makes it necessary to have a greater amount in the diet to ensure normal growth of bone, formation of sound teeth and jaws, and good general health.

My investigation on rickets is still in progress and the results remain incomplete. The mode of production of the disease is obviously complex, otherwise it would have long been solved; but by considering and altering each factor in turn the animal experimental method will certainly in time clear up all the points of difficulty.

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