THE RELATIVE EFFECTS OF DIET UPON THE BODY WEIGHT,
SKELETAL DEVELOPMENT, AND RATE OF ERUPTION
OF THE INCISOR TEETH OF ALBINO RATS

by

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THE RELATIVE EFFECTS OF DIET UPON THE BODY WEIGHT,
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INTRODUCTION

Although considerable study has been given in the past
to the effects of diet upon the body weight, skeletal de­
velopment and tooth development of animal organisms, much
of the early work done was complicated by the existence of
more than one dietary deficiency, and evidence concerning
the relationship between these factors has often been con­
flicting or lacking. What is usually accepted as normal
growth and skeletal development for the albino rat is given
in considerable detail by Donaldson. This information
gathered together in his book entitled, "The Rat", is based
upon studies of adequately nourished rats and no mention of
dietary relationships are given. That body development is
directly dependent upon diet is well known and many experi­
ments establishing this relationship have been carried out.
In the literature review which follows only those studies
dealing with the effects of one uncomplicated dietary de­
ficiency on the body weight, skeletal development, and tooth
development of the animal body will be considered, correlations
between these factors being included wherever they have been
noted.
Review of the Literature

Variations in the Protein Content of the Diet

It is known generally that a diet lacking in any one of the essential amino acids, namely, lysine, cystine, tryptophane, and histidine, has a retarding effect on the body weight of the animal. This fact has been brought to light largely through the work of Osborne and Mendel in 1911, 1912, 1914, 1915, and 1916, Hart, McCollum, Steenbock, and Humphrey in 1911, McCollum, Simmonds, and Parsons in 1919, Sure in 1920, Rose and Cox in 1924, and Sherman and Woods in 1925.

The level at which an adequate protein (one containing sufficient amounts of all the essential amino acids) is fed in the diet has also been shown to have a marked effect upon the body weight of the animal. At a protein level of 18% Mendel and Cannon in 1926 and 1927 found that male and female rats gained, respectively, an average of 1.77 gms. and 1.09 gms. daily, whereas when this level was increased to 35% the respective gains in weight were 5.00 and 3.00 gms. daily. Similar observations were noted by Anderson and Smith in 1922 who reported excellent growth in rats fed a diet containing 22% protein. Osborne and Mendel in the same year obtained good growth in rats
on diets containing as high as 90% protein. Contrary to these results as to the beneficial effects on growth obtained with such high levels of protein, Slonaker in 1931 secured the best gains in weight in young rats with a protein level of 14%, levels above 22% resulting in a progressive retardation of body weight. Among other investigators who have studied the influence of the plane of protein intake on growth (body weight) are McGollum and Davis in 1915, and McCollum, Simmonds, and Pitz in 1916.

The effect of varying the protein content of the diet on the skeletal development of the body has not been studied so thoroughly as has been the effect of such variations on the body weight. Much of the early work, such as that of Schulz in 1912 with puppies and Bruning in 1914 with rats, was complicated by more than a mere protein deficiency, hence it is excluded here. Probably the most comprehensive study on the effect of a protein deficiency on the skeleton was that reported in 1927 by Winters, Smith, and Mendel. They kept groups of male rats at a stationary body weight for a period of forty days after weaning by feeding diets adequate in all respects but lacking either in a sufficient amount of an adequate protein (casein) or in a sufficient amount of the essential amino acid, lysine. Measurements were made of the trunk, tail, and leg bone lengths at the death of the animals and these measurements compared with normal animals of
the same age and weight. It was found that animals on the low protein and low lysine diets gained only 5 mm. and 9 mm. in body length, respectively, during the time in which the normal animals gained 46 mm. In other words, the low protein and low lysine animals made 10.8 and 19.5% of the normal gain in length, respectively, in the forty day test period. But compared to normally growing rats of the same body weight the length of the body of the animals on the restricted diets had increased though the weight had remained constant. The normal rat weighing 45 gms. measured 120 cm. as compared to 125 and 129 cm. for rats whose weight had been held at 45 gms. by feeding diets low in protein and lysine, respectively. It was also shown that the tails and leg bones of the stunted rats were longer than those of normal rats of the same body weight but were not as long as those of normal rats of the same age. Therefore, persistent though not normal growth of the skeleton had occurred on diets lacking either in sufficient protein or sufficient lysine. Mendel and Judson in 1916 also noted that the skeleton grows persistently but not normally in spite of a stationary body weight in mice maintained on diets low in protein.

The effect of a diet high in protein on the tooth development, as measured by the rate of eruption of the incisor, was determined in 1931 by Downs. He found that the rate of eruption of the lower incisor of the normal rat which was 2.78 mm. per week, was accelerated to 3.21 mm.
per week by feeding a high protein diet. No correlations between the rate of eruption of the rat incisor and the body weight or skeletal development were noted.

Variations in the Caloric Content of the Diet

The phenomenon of retarded body weight due to a lack of calories in the diet has been observed universally and regularly. By restricting the amount of food consumed, a loss in body weight occurs immediately and if such a procedure is continued for any length of time, death ensues. This fact has been noted by many investigators among whom have been Waters in 1908 and 1909 with steers, Aron in 1911 with puppies, Jackson in 1915 with young rats, Stewart in 1918 and 1919 with newborn rats, and Evans and Bishop in 1922 with adult rats. The latter investigators found that on an optimum standard diet the albino rat at the end of one year reached a body weight of about 330 gms; whereas on a consumption of \( \frac{2}{5} \) the normal ration the weight was reduced to 140 gms., and on \( \frac{1}{2} \) the normal ration the weight was only 60 to 85 gms. at the end of one year.

It is interesting to observe the changes occurring in the skeleton when the animal is maintained on a diet restricted in calories. Waters in 1908 and 1909 kept a large number of steers at stationary body weights by the underfeeding of a presumably adequate diet for varying
periods up to one year. The form of the stunted animals was distinctly different from normal controls, the length of the fore leg, length of the body, and height at the withers having increased greatly during this time. Similar observations were reported by Aron in 1911 on puppies held at nearly constant weight by limitation of their energy intake. In this case there was almost a normal increase in both length and height with a resulting distortion of bodily contour. Unfortunately in neither of these series of experiments were the animals compared to normal controls of the same body weight, a comparison which brings out the alterations in body form much more clearly. Jackson in 1915 working with young rats noted that underfeeding resulted in a slight increase in the body and tail lengths, though the body weight had remained stationary, but that the changes in general body proportions as compared to normal controls of the same body weight otherwise were not marked. These findings were confirmed by Stewart in 1916 and by Thompson and Mendel in 1918. However, as pointed out by Winters, Smith, and Mendel in 1927, limiting the calories by underfeeding of an adequate diet, as was done in the above mentioned experiments, results not only in a caloric deficiency but may also involve a deficiency of protein, salts, and vitamins. Hence, they conducted a study in which male rats were kept at a stationary body weight for a period of forty days after weaning by a diet adequate in every respect except that the
calories only were limited. It was found that the stunted animals made only 8.7% of the normal gain in body length, the actual gains in body length being 4 and 46 mm. in the stunted and normal animals of the same age respectively. But, compared to normally growing rats of the same weight, the length of the body of the stunted group had increased though the weight had remained constant. The humerus and radius made 36% of the normal gain in length and the femur and tibia 41% and 39% respectively. But as was the case with the body length, the leg bones of the body were longer than those of normal animals of the same body weight. More recently McCay, Crowell, and Maynard in 1935 retarded the growth of rats by feeding a low caloric diet for periods varying from 766 to 911 days, at which times the animals were given a normal diet. They found that the animals grew after the period of restricted feeding but that the ultimate body size (body length and length of the long bones) was considerably below normal.

It appears, therefore, that although the body weight of experimental animals can be retarded easily by interference with the caloric content of the diet, such an interference has little effect upon the persistent growth of the skeleton. The combination of a cessation in body weight and the persistent growth of the skeleton results in a distortion of the body form which is distinctly different from normal controls.
Variations in the Calcium, Phosphorus, and Anti-Rachitic Vitamin Content of the Diet

It is difficult to consider the effect of a calcium, phosphorus, and anti-rachitic factor deficiency on the weight of the body alone since most of the work done with these deficiencies has obviously been concerned with their effects on the skeleton, the body weight having been of secondary interest. However in 1921 Sherman, et al did study the effects of a diet deficient in calcium and fat soluble vitamins on growth in body weight alone. They obtained normal growth but not normal reproduction in rats fed a diet containing 4/5 bread and 1/5 milk. When the same diet was substituted with 4/5 whole wheat in place of the bread, reproduction was normal but the young grew at a slower rate. A normal growth rate in the young was secured by using a higher percentage of milk (1/3) and a lower percentage of wheat (2/3), thereby increasing the calcium and fat-soluble vitamin content of the diet appreciably. Toepfer and Sherman in 1936 found that rats on Sherman's diet A which is only slightly over one-half as rich in calcium (.20%) and fat soluble vitamin as diet B, grew at a rate slightly below the average but within the normal range. However, when the calcium content was increased to 0.64%, there was a definite increase in rate of gain in body weight. With a diet that was much lower in calcium (.094%) but adequate in all other respects, Campbell, Bessey, and Sherman in 1935 reported that rats gained normally in body weight.
but that the young of such rats showed about three-fourths the normal growth and size and failed to reproduce themselves successfully. In a study concerning the effect of different levels of calcium in the diet on the growth of rats, Sherman and Booher in 1931, and Whitcher, Booher, and Sherman in 1936 reported that the gain in body weight of young growing rats was normal regardless of the amount of calcium in the diet, the amount of calcium ranging from .16% to 1.04%. The amount of phosphorus in the diet was .43% at all levels, thus the Ca:P ratio varied widely throughout the experiment, yet this factor apparently was of no significance as far as the body weight of the rat was concerned. However, a diet completely lacking in calcium was capable of keeping the weaning weight of rats at a constant level according to the findings of Winters, Smith, and Mendel in 1927, Smith and Schultz in 1930, Swanson and Smith in 1934, Clarke and Smith in 1935, Clarke, Bessin, and Smith in 1936, and Epright and Smith in 1937. Bethke, Steenbock, and Nelson in 1923 found that the retarded growth of rats resulting from feeding a diet containing only .05% of calcium could be raised to normal values by additions of either calcium or cod liver oil to such a diet. Similarly, Fairhall in 1928 succeeded in retarding the growth of rats by feeding a diet low in calcium but noted that irradiating such rats produced a normal growth rate. It appears, therefore, that
the growth rate of rats is retarded if the amount of calcium in the diet is at a very low level, otherwise it remains normal.

The fact that a diet low in calcium and fat soluble vitamins produces not only growth disturbances but also deformities in the skeleton was shown in 1921 by McCollum and co-workers. Later they produced evidence that some substance or substances in cod liver oil caused calcium to be deposited in rachitic bones in the same fashion in which deposition occurred in the healing of rickets in man. Their work indicated that two factors were concerned in the production of rickets (skeletal deformities) namely, a "fourth vitamin" (now designated as Vitamin D) closely associated with vitamin A, and a dietary deficiency of either calcium or phosphorus.

In an effort to arrive at a fuller understanding of the chemistry of normal growth and development, Sherman and MacLeod in 1925 conducted an extensive investigation on the calcium content of the body in relation to age, growth, and food. They found that the average percentage of calcium in the normal rat body (raised on diet B) increased from 0.25% at birth to about 1.0% and 1.2% in eight months in males and females respectively, the increases being most rapid up to ninety days of age after which time the percentage of calcium in the body increased more slowly up to one year and then remained fairly constant throughout the life of the animal. In comparison with the increase in body weight occurring dur-
ing this time it was found that the amount of calcium in the body increased much more rapidly. At adult size, rats had multiplied their birth weights seventy times and the weights of calcium contained in their bodies at birth three hundred sixty times. Therefore, normal skeletal development, as measured by deposition of calcium in the body, proceeds at a faster rate from birth to one year than does the increase in body weight during this time.

A similar study showing the relation of the phosphorus content of the normal body to age, growth, and food was conducted in 1926 by Sherman and Quinn. It was found that the average phosphorus content of the normal rat (raised on diet B) body increases from .34% at birth to 0.68% and 0.74% in eight months in males and females, respectively. The amount of phosphorus in the body increased more rapidly than did the body weight but not as rapidly as did the amount of calcium in the body. Thus, the average adult rat showed about seventy times the body weight with which he was born, about one hundred fifty times as much phosphorus and about three hundred sixty times as much calcium. Again it is evident that skeletal development in the rat, as measured by deposition of calcium and phosphorus in the body, proceeds at a faster rate during the first year of life than does the increase in body weight.

In these studies (50, 51) Sherman and MacLeod, and
Sherman and Quinn found that rats raised on diet A (which contains slightly more than 1/2 as much calcium and fat soluble vitamin as diet B) contained somewhat less calcium and phosphorus (both in weight and percentage of body weight) at all ages studied than parallel animals raised on the better diet B. Evidently the deposition of calcium phosphate in the growing bones on diet A was slightly retarded or at least did not proceed so rapidly as it did on diet B. Additions of cod liver oil to diet A made no appreciable difference in the percentage of calcium or phosphorus in the rat body but additions of calcium as calcium lactate increased the percentages of calcium and phosphorus up to the normal values. However, although the animals which received additions of calcium had the same percentage of calcium in their bodies as the average normals, they contained less total weight in calcium since they were below the average normal weight. As previously stated Toepfer and Sherman found that increasing the calcium content of diet A to 0.64% resulted in a better growth rate. At the same time these investigators also noted slightly greater body lengths in animals consuming diet A with the added calcium. Males grew in one hundred eighty days to an average length of 23.4 cm. on the supplemented diet as compared with 22.4 cm. on diet A alone, while females showed an average length of 20.5 cm. on the diet with the added calcium as compared to 19.5 cm. on diet A alone. Quinn, King, and Dimit in 1929, however,
reported no consistent difference in the body trunk form of rats on diet A and the better diet B. The younger animals on diet A generally showed longer leg bones relative to the body weight than was found in the case of diet B animals of the same age, but the differences were negligible for animals of the same weight.

In a study of the effects of a very low calcium diet on body weight and calcium deposition in young growing rats, Sherman and MacLeod reported that rats placed at twenty-eight days on a low calcium diet (flour and salt) survived without an increase in body weight for eighty days and showed a slight decrease in the amount of calcium in the bodies at this time as compared to the amount they had had at twenty-eight days. Investigating this observation further they found that if rats were allowed to make the normal gain in body weight and to attain a normal calcium content, they actually lost calcium from their bodies when placed on the above low calcium regimen. Therefore, the amount of calcium ingested is a primary factor in maintaining the normal calcification of the system. This fact was brought out in a slightly different manner by Fairhall in 1928. He found, as previously stated, that rats fed a low calcium diet ceased to gain in weight, but when such rats were irradiated, growth was normal. However, upon analysis the irradiated rats were found to have about 50% less calcium than normal rats. Non-
irradiated rats had about 75% less calcium than normal controls. Therefore, although vitamin D aided to some extent in calcification of the body, an ample supply of calcium was needed for optimal skeletal development. Concerning the effect of a low calcium diet on calcification, Campbell, Bessey, and Sherman in 1935 found that although rats subsisting on a diet containing only 0.094% calcium showed normal growth in body weight and the physical appearance of good health, they were shown to have on analysis only about three-fourth to four-fifths the amount of calcium in the bodies of normal rats. It was also reported that the offspring of such rats showed only three-fourths the normal growth and size and failed to reproduce themselves successfully. Sherman and Booher have reported that up to a certain level of calcium in the diet (0.50%) calcium is deposited in the system in a direct relationship with the amount ingested, the body weight remaining normal and being unaffected by such graded doses. Whitcher, Booher, and Sherman found that percentages of calcium from 0.55% to 1.04% produced no distinct increase in the rate of calcification, rate of growth, or length of the body of rats, and that increasing the phosphorus content of such diets from 0.43% to 0.73% likewise was ineffective. The effect of a diet low only in inorganic salts (principally calcium and phosphorus) on the growth of the skeleton was studied extensively in 1927 by Winters, Smith and Mendel. It was found that animals whose body weights had remained
stationary for a period of forty days by feeding a diet restricted in inorganic salts gained only 4 mm. in length which was only 8.7% of the normal gain shown by normal animals of the same age. However, when the stunted animals were compared to normal animals of the same body weight (45 gms.), they were found to have made a slight increase in body length. Similarly, the bones of the stunted animals were 10% to 20% longer than the bones of normal animals of the same body weight but were only .36% to .39% as long as those of normal animals of the same age.

It seems, therefore, that although the body weight of animals appears normal on diets containing a fairly low amount of calcium, (.094%) the skeletal development is markedly retarded by diets which contain these low amounts of calcium. When the diet is lacking in calcium altogether, both the body weight and the skeletal development are markedly retarded, though on such diets persistent but not normal growth of the skeleton occurs.

The role played by calcium, phosphorus, and antirachitic vitamin in the formation and calcification of the teeth of experimental animals has been shown by many investigators. Mellanby working with dogs found that (a) diminishing the calcium as well as fat soluble vitamins in the food resulted in the formation of badly formed teeth; (b) diminishing the calcium in the food, when the fat-soluble vitamin con-
tent of the food was high, reduced the amount of calcification, especially that of the dentine; (c) increasing the calcium in the food and diminishing the fat soluble vitamins, also resulted in badly formed teeth; and (d) increasing the calcium and fat soluble vitamins resulted in perfectly formed and regularly arranged teeth. Later, she in 1924 investigated the effects of irradiation on the formation of the teeth and stated that when the diet was only "moderately defective" exposure to ultra violet light had a decidedly beneficial effect on the calcification and structure of the teeth. She also found that if the diet was very low in calcium, thus resulting in poorly calcified teeth, improvement in calcification by irradiation was less than when the diet was adequate. Her results were confirmed in 1932 by Blackberg and Berke.

The effects of a diet low only in calcium and phosphorus on the teeth of puppies was studied in 1923 by Marshall. This study revealed at least two abnormalities in the teeth: a marked delay in dentition and a relative absence of dentine although there was a normal amount of enamel. The deciduous teeth were apparently normal in size and shape but were not shed in the usual manner. It was also observed that calcification of the permanent teeth proceeded normally at first and then ceased altogether or at least was very much retarded. Calcification was later resumed but the period of subnormal growth remained definitely
inscribed in the tooth structure. Again the evidence points to the profound effect of a faulty diet upon the development of the teeth.

A relationship between rickets and tooth structure was reported in 1921 by Shipley, Park, McCollum, and Simmonds who found that the incisors of rats with experimental rickets were frequently loose, fragile, and sometimes fractured. These conditions were studied more in detail in 1922 by McCollum, Simmonds, Kinney, and Grieves who found the greatest percentage of oral defects in the rats fed diets deficient in protein, calcium, and fat soluble vitamin. The next highest incidence occurred in rats fed a diet low in calcium; and a still lower percentage occurred on diets low in both calcium and fat soluble vitamin (contrary to Mellanby's findings). None of the defective structural conditions occurring in these rats, namely, osteo-dentine and pulp exposure, was observed in the stock rats fed a complete diet. Similar observations were noted in 1931 by Becks and Ryder who demonstrated a progressive decrease in the deposition of calcium salts in the dentine and final atrophy of the odontoblasts with cessation of calcification and formation of cementing substance in rats maintained on a rickets-producing diet for varying periods of time. The changes in the dentine were similar to those produced in the long bones. Poorly calcified dentine was also produced by Arnim, Clarke, Anderson, and Smith in 1955 by feeding rats diets low in calcium but
adequate in phosphorus. Rosebury and Karshan, however, in 1931 were unable to produce defects in the enamel of the incisors and molars of rats by feeding diets low in calcium and vitamin D.

The effect of a diet deficient in calcium or phosphorus or in calcium and phosphorus on the development of the teeth, as measured by the rate of eruption of the incisors, was reported by Downs in 1931. He found that normally the lower incisors of the rat erupted at a rate of 2.78 mm. per week. In rats given a diet low in calcium and phosphorus the rate of eruption was retarded to 2.14 mm. per week whereas on a phosphate free or calcium free diet the weekly rates of eruption were 2.15 mm. and 2.30 mm. respectively.

Therefore, from the evidence presented, it would appear that diets deficient in calcium, phosphorus, and the antirachitic vitamin retard the growth in body weight, produce skeletal deformities, and result in poorly developed teeth.

Variations in the Vitamin A Content of the Diet

One of the first effects of a vitamin A deficiency is the cessation of a gain in body weight in experimental animals. This important fact was first noted simultaneously in 1913 by McCollum and Davis and Osborne and Mendel. Since that time a vast amount of research has appeared in the literature concerning this phenomenon of retarded growth in vitamin A deficient animals. Among the investigators
have been Steenbock, Kent, and Gross in 1918, Drummond and
Coward in 1920, Sherman and Storms in 1925, Sherman and
Boynton in 1925, Sherman and Munsell in 1925, Chick in
1926, Sherman and Burtis in 1928, Hume and Smith in 1928,
Sherman and Batchelder in 1931, and Sherman and Todhunter
in 1934. Because the change in body weight has been so con-
sistent an observation in rats deprived of vitamin A, it
now serves as a criteria in the quantitative determination
of vitamin A in foods. The method was introduced by
Drummond and Coward in 1920 and later developed by Sherman
and Munsell in 1925 and modified by Sherman and Burtis in
1928.

Comparatively few studies have been reported on the
effect of a vitamin A deficiency on the skeleton. Much
of the early work done in this connection was complicated
by a lack of the anti-rachitic vitamin D, hence it is not
included here. In 1925 Sherman and MacLeod determined the
effects on the skeleton of a diet lacking in vitamin A by
determinations of the calcium content of rats who had died
as a result of feeding a diet lacking in this factor.
Their results showed that such animals contained more cal-
cium than would be expected in normal rats of their maxi-
mum body weights and less than half as much as in normal
animals of the age at which they died. These data in-
dicated that some calcification of bone had continued after
growth had ceased due to a lack of fat soluble vitamin A,
or, in other words, the lack of vitamin A did not interfere with the storage of calcium to so great an extent as it interfered with growth measured by body weight. However, their results were complicated by a lack of vitamin D. In 1933 (74) Smith and Lantz analyzed the tibias of animals fed a standard vitamin A diet, for calcium and phosphorus and compared and compared such analyses with controls receiving the same diet supplemented with a liberal amount of vitamin A as cod liver oil. It was found that the vitamin A deficient animals were smaller in size, their bones weighed less and contained a correspondingly smaller quantity of both calcium and phosphorus; but the differences in percentages of ash, calcium, and phosphorus in the A deficient animals and the normal controls were not significant.

Perhaps the best and most conclusive investigation as to the effects of a diet deficient in vitamin A on the skeletal development of the animal was that reported in 1929 (52) by Quinn, King, and Dimit. They kept eight animals on a basal vitamin A free diet from twenty-eight days of age until their death and forty-six animals were continued on the A free diet from weaning until the surplus stored vitamin of their bodies was depleted, at which time the basal diet was supplemented with an amount of whole milk powder which induced a gain of 22-30 gms. during an eight week period. At death measurements of the trunk length, chest girth, hip width, and length and diameter of the tibia, humerus, femur,
and radius were made and ratios of the weight of the animal to their length of body, chest girth, hip width, and length of the long bones of the leg; and also ratios of the length of body to chest girth, hip width, and length of the long bones of the leg were calculated. The same procedure was followed with normal animals fed Sherman's diet B. It was found that the length of the body, girth of chest, width of hip, and length of the long leg bones of the vitamin A deficient animals increased during the time of restricted feeding to a relatively greater extent than did the weight of such animals during this period, ratios of weight to each of these measurements being lower than the corresponding ratios for normal rats of the same age. Those animals receiving the supplements of milk powder showed about the same chest girth in proportion to their body length as normal animals of the same age and weight. In proportion to their body length these animals had slightly longer leg bones than normal animals of the same weight and age. The hip width of these animals was relatively narrow when comparison was made with the corresponding measurements of normal animals of the same age but the difference was negligible when comparison was made with normal animals of the same weight. Therefore, this study reveals that animals deprived of vitamin A show a lesser retardation of increase in the length of the body and of the legs, the chest girth, and the width of hips than in the body weight, or, in other words, a deficiency of vita-
min A affects the skeletal development of the animal to a lesser degree than it affects the body weight.

Relatively little work has been done on the effects of an uncomplicated vitamin A deficiency on the development of the teeth. Howe and Wolbach in 1925 noted that vitamin A deficient diets produced a specific effect upon the secretory epithelium, making the functional cells non-secretory. During the process of tooth development enamel-forming cells ceased to function and were "shrunken and atrophic or replaced by stratified non-keratinizing epithelium." However, it is questioned as to whether or not their results were complicated by a lack of vitamin D as well as of vitamin A in the ration. Bloch in 1931 found no greater incidence of dental caries in children blinded as a result of a severe vitamin A deficiency in infancy than in normal ones and concluded that in man, at least, a dietary deficiency of vitamin A has no effect upon the development of the teeth. He referred, however, to the unpublished work of Gudjonsson who found that the teeth of vitamin A deficient rats were more friable than those of normal animals.

Shibata in 1931 attributed the deposition of calculus to a vitamin A deficiency in rats. He noted abnormal enamel formation and a decrease in the calcium and phosphorus content of the teeth of rats fed a diet deficient in vitamin A. Similar observations were reported by Masaki who noted that the changes in the teeth of rats fed a vitamin A deficient diet were characterized by "osteomalacia,
disturbance of calcific deposits, and dislodgment of the calcium salts of the already calcified tissue. More recently Smith and Lantz in 1933 observed that the incisors of rats deprived of vitamin A ceased to grow at the normal rate, lost their luster and normal orange pigment and became short and blunt as well as white and opaque. Viosterol (vitamin D) failed to prevent such changes but the administration of cod-liver oil (largely vitamin A) resulted in normal appearing incisors. It was also noted that the percentage of ash was lower, the percentage of calcium higher, and the percentage of phosphorus slightly lower in the incisors of the vitamin A deficient animals than in normal controls. As noted previously, these investigators found no significant differences in the composition of the bones of the two groups of rats. They suggest that since enamel originates from epithelial tissue which is affected specifically by a deficiency of vitamin A, the incisors would be affected by such a deficiency whereas bone would not.

It appears, therefore, that a dietary deficiency of vitamin A results not only in slowing up the growth rate but also in interfering with the normal development of the bones and teeth.

the Variations in Vitamin B (Complex) Content of the Diet

As was the case with vitamin A the growth, as
measured by increase in body weight, of animals deprived of vitamin B or G is greatly retarded. This fact has been noted consistently by many investigators, chief among whom have been Osborne and Mendel in 1917, in 1922, and in 1923, McCollum and Davis in 1915, Chick and Rume in 1919, Steenbock Sill, and Nelson in 1923, Sherman and Spohn in 1925, Chick and Roscoe in 1929, Sandels in 1930, Sherman and Chase in 1931, Sherman and Sandels in 1931, and Sherman and Bourquin in 1931. Since the gain in weight in rats is so consistently influenced by the amount of vitamin B or vitamin G in the diet, the growth rate of rats is now an important criteria in the quantitative determination of vitamins B or G in foods. The method generally used for the determination of vitamin B is the devised by Sherman and Spohn as modified by Sherman and Chase, and for vitamin G is that developed by Sherman and Bourquin.

Accompanying the loss in body weight animals deprived of vitamin B show a marked change in body form characterized chiefly by a humping of the back. This is one of the symptoms of polyneuritis and is observed in rats whose rapid loss in body weight has not been the result of starvation caused by a total lack of vitamin B in the diet. In other words, the diet must contain a small amount of vitamin B, otherwise the appetite is impaired so greatly that the animal dies of starv ation before developing any signs of polyneuritis. This fact was noted by Sandels and Sherman and Chase and others.
In an effort to determine the exact nature of the skeletal deformity accompanying heri-heri in rats, Shipley, McCollum, and Simmonds in 1921 found that vitamin B deficient rats showed no gross skeletal deformity but histologically showed lesions identical with those seen in guinea pigs with scurvy. Findlay and MacKenzie in 1922 likewise found that diets deficient in vitamin B produced hemorrhagic congestion in the bone marrow of the femur in the rat. A more extensive investigation as to the effects of a vitamin B deficiency on the skeleton was that reported by Quinn, King, and Dimit in 1929. They fed thirty-four animals a diet deficient in vitamin B (B and G) supplemented with an amount of whole milk powder to allow them just to maintain their weight during an experimental period of eight weeks. Forty-four animals were fed from weaning age the basal B-free diet supplemented with an amount of milk powder to allow them to make a total gain of 50-55 gms. in an eight week experimental period. At eight weeks the animals were killed and measurements of the body length, chest girth, hip width, and length of the long bones of the leg were made. It was found that relative to their body weight both groups of animals showed longer bodies, greater chest girths and hip widths, and longer leg bones than did normal animals of the same age. Relative to their body length they were found to have smaller chests and longer leg bones than normal animals of the same age and weight. Therefore, in animals deprived of vitamin B
there was a lesser retardation of growth of the body and legs, chest girth, and hip widths than in body weight.

Very little work has been done on the effects of a vitamin B and G deficiency on tooth development. Mellanby found that additions of yeast to a diet deficient in vitamins A and D fed to puppies did not prevent the formation of poor teeth. Further investigation showed that diets in that were adequate in vitamins A and D but deficient in B and G resulted in the formation of teeth which were perfect in structure.

Evidence indicates, therefore, that a lack of vitamins B or G in the diet causes a marked retardation in the growth rate, and subnormal development of the skeleton, but it appears that the teeth may be relatively stable to these deficiencies.

Variations in the Vitamin C Content of the Diet

The observation that a loss in body weight is a frequent accompaniment of a vitamin C deficiency (scurvy) in guinea pigs has been noted by many investigators among whom have been Holst and Frohlich in 1907, Cohen and Mendel in 1918, Hess and Unger in 1918, Chick, Rume, and Skelton in 1918, Hess in 1920, Sherman, LaMer and Campbell in 1921, Bessesen in 1923, and others. Since the appearance of scurvy in an experimental animal (guinea pig) is characterized more specifically by hemorrhages, fragility
of the bones, and looseness of the teeth than by gains or losses in body weight, the growth rate is of only secondary interest in the quantitative determination of vitamin C according to the method introduced by Sherman, LaMer, and Campbell. The weight curve shows a pronounced initial fall when the animal is first placed on the basal C-free diet, followed by a rapid gain in weight to a sharp maximum, then followed by a rapid loss of weight after the onset of scurvy.

The skeletal lesions characteristic of infantile scurvy were summarized by Hess in 1920 as including osteoporosis with fragility and thinning of the cortex in the shaft of the long bones, and frequent occurrence of fractures in severe cases. These lesions were noted in scorbutic guinea pigs in 1907 by Holst and Frolich, in 1916 by Jackson and Moore, and in 1918 by Chick, Hume, and Skelton. Further investigation by Wolbach and Howe in 1925 showed that in scorbutic guinea pigs the formation of bone ceased immediately whereas the osteoblasts continued to proliferate. The administration of orange juice was followed by the prompt appearance of bone matrix between the cells. These authors theorized that "in the formation of intercellular substances, there was a change of the material from the liquid to a solid or jell state and the missing factor in the scorbutic condition was one affecting the jelling or setting of the liquid product." Salter and Aub in 1931 fed experimental animals orange juice at various stages of
the disease, scurvy. They injected them with alizarin which stains newly calcified areas red. In healing scurvy the epiphyseal line was especially well marked indicating a deposition of calcium salts. The bones of the control animals (no vitamin C) remained unstained inspite of the fact that the experimental ration contained large amounts of calcium and phosphorus.

The changes produced in the teeth of guinea pigs and monkeys fed a diet deficient in vitamin C have long been recognized as characteristic symptoms of scurvy. Zilva and Wells in 1919 noted a fibroid degeneration in the pulp and an osteoid degeneration in the dentine of scorbutic guinea pigs and monkeys. Toverud in 1923 and Hojer and Weston in 1925 found that in addition to the defects noted by Zilva and Wells, the odontoblastic layer had disappeared, and the pre-dentine and dentine were amor-

phously calcified. Howe in 1922 noted that when orange juice was fed to guinea pigs whose teeth had become exten-
sively decalcified by a lack of vitamin C for fourteen weeks, calcification occurred immediately. Investigating this point further, Hanke in 1933 found that the teeth of guinea pigs fed less than 3.0 cc. of orange juice daily were extremely decalcified. With the administration of 5 cc. of orange juice daily calcification occurred im-
mediately. He proposed a theory (similar to that of Wolbach and Howe) that "in the scorbutic animal the calcium and
phosphorus salts are in solution and when vitamin C is added, these salts are released and calcium phosphate is crystallized in the decalcified areas."

The fact that the rat is not susceptible to scurvy was first pointed out by Holst and Froligh in 1907. Similarly, Mellanby showed that dogs, and Hess that birds, pigs, and cattle were immune to the disease.

It appears therefore, that when vitamin C is withheld from the diet, there occurs a loss of body weight and decalcification of the teeth and bones.

Variations in the Fluorine Content of the Diet

The effect of graded doses of fluorine, contained in a variety of fluorine compounds, upon the growth rate of young rats was studied in detail by Smith and Leverton in 1934. It was found that the growth rate of animals receiving additions of .05% NaF (.0226% of fluorine) during a six-week test period was about 15% below that of litter-mate controls who received the same basal diet without the addition of sodium fluoride. When this amount was doubled (.1% NaF or .0452% of fluorine) the rate of gain in body weight was less than half the rate of gain recorded for the normal controls, whereas less than .05% NaF did not slow up the rate of growth during this most active period of development. A similar retardation of the growth rate occurred when these percentages of fluorine were fed as potassium or ammonium.
fluoride, or barium or sodium fluosilicate. On the other hand, cryolite and calcium fluoride were found to be less toxic sources of fluorine from the standpoint of their effect upon the growth rate. These findings agree in general with those reported by Sollmann, Schettler, and Wetzel in 1921 and with those of Lamb, Phillips, Hart, and Bohstedt in 1933. An interesting study of the limitation of fluorine toxicosis in the rat with aluminum chloride was reported by Sharpless in 1936. He found that addition of .025% sodium fluoride had no effect on the growth rate but that rats fed 0.1% sodium fluoride grew at approximately two-thirds the normal rate. However, by adding 0.5% aluminum chloride (.056% aluminum) to the diet containing 0.1% sodium fluoride, a normal growth rate was obtained.

The effect of graded doses of fluorine in the diet on the calcium and phosphorus metabolism of rats was reported in 1934 by Lantz and Smith. It was found that growing rats fed a diet containing 0.1% added sodium fluoride retained much less calcium and phosphorus than normal controls reared on the same diet without additions of sodium fluoride. When the ration contained only .05% sodium fluoride, the same differences in calcium and phosphorus metabolism were noted but of a much smaller degree. It was suggested that fluorine affects the metabolism of calcium and phosphorus by interfering with the absorption of calcium. In addition it was observed that animals on a ration containing 0.1% sodium
fluoride were short and stocky and as they became older marked bowing of the legs such as occurs in rickets resulted.

The toxic effect of fluorine on the teeth is now well known. That the dental defect of human teeth known as mottled enamel is caused by the toxic action of fluorides in the drinking water was definitely established in 1931 (113) by Smith, Lantz, and Smith. The defect was produced experimentally in the teeth of dogs, rats, and guinea pigs in 1933 by Smith and Lantz, the results showing that normal calcification of the teeth was interfered with to an extent varying with the concentration of fluorine in the ration. The first indication of a "fluorine effect" occurring at a .0014% level of fluorine was a decrease in the amount of pigment in the lower incisor. When the fluorine intake was greater the whole surface of the tooth appeared white and dull and finally with still larger doses the enamel became so defective that small pits appeared and sections of the enamel chipped off. In another study, Lantz and Smith found that the teeth of animals fed 0.1% sodium fluoride in the diet were lower in ash content but had a greater percentage of calcium and a lower percentage of phosphorus resulting in a higher calcium to phosphorus ratio. Fluorine, as sodium fluoride was also found to have an effect upon the rate of eruption of the incisor teeth of albino rats (116) by Smith in 1934. In this study a total average eruption of 31.7 mm. during a ten week test period was recorded for
normal rats; for rats on rations containing .05% and 0.1% of sodium fluoride during the same length of time, the total eruptions were 27.7 mm. and 16.8 mm. respectively. Sodium fluoride was also found to retard the rate of body growth and retention of calcium and phosphorus. A probable relation between these three effects of sodium fluoride, was suggested by similarity in the degrees of effects produced when the ration contained 0.05% of sodium fluoride, rate of body growth, amounts of calcium and phosphorus retained, and rate of eruption of incisors were 84.1%, 85.9%, 84.3%, and 87.3% respectively, of that of control rats, whereas when the ration contained 0.1% of sodium fluoride these percentages were reduced to 39.8%, 44.2%, 47.8%, and 52.9% respectively. Sharpless in 1936 attempted to limit the toxic effect of fluorine containing rations with aluminum chloride. He found that when the diet contained .025% of sodium fluoride, the incisors appeared dull and white, but if 0.5% of aluminum chloride were included in the same diet, the incisors appeared normal for five to six weeks at which time they became bleached and banded. With 2.0% of aluminum chloride the incisors appeared normal but examining with a small hand lens revealed the presence of fine alternate dark and light pigment lines. When the diet contained 0.1% of sodium fluoride, the incisors were rough, pitted, and chipped but with the addition of 0.5% of aluminum chloride they had a smooth surface but were
bleached and worn down. A 2.0% level of aluminum chloride produced incisors with a pale yellow surface which sometimes showed white and yellow bands. This author assumes that this "protective action must take place in the intestinal tract probably by the formation of an aluminum and fluorine compound which is only very slightly dissociated". He also states that through a private communication, Dr. E. R. Nelson of Iowa obtained similar results with aluminum sulfate.

It seems evident, therefore, that small amounts of fluorine affect the skeletal development and tooth development of the animal body presumably by interfering either with the absorption of calcium from the intestinal wall or with the deposition of calcium in the bones and teeth. The growth rate is not as sensitive to small doses of fluorine as the teeth but larger amounts will produce a definite retardation of the growth rate.

The evidence gathered together on the relationship of diet to the body weight, skeletal development, and tooth development of the animal body indicates that in general diets deficient in certain of the dietary essentials have a marked slowing up effect upon each of these factors, the amount of retardation depending upon the nature and degree of the deficiency in the ration. It was found generally that deficiencies in protein, calories, and vitamins A, B, or C arrested the gain in body weight of animals to a greater extent than they retarded skeletal development. On the other hand, mineral deficiencies, that is calcium, phosphorus, and
the vitamin governing calcium and phosphorus metabolism, had a more pronounced effect upon the skeletal development than on the body weight of experimental animals. The development of the teeth was influenced to about the same extent by mineral deficiencies and deficiencies in vitamins A and C and appeared relatively stable to a lack of the vitamin B complex. It was evident that the relationship between tooth development, skeletal development, and body weight had not been investigated thoroughly enough to be conclusive.

The foregoing review is conspicuous in its lack of information regarding the effect of diet upon the rate of eruption of the teeth. The purpose of this paper is, therefore, to investigate more thoroughly the effect of diet upon the eruption of the incisor, growth in body weight, and skeletal development of rats.
CHAPTER II

EXPERIMENTAL PROCEDURE

The procedure adopted during the course of this investigation of the effect of diet upon the body weight, skeletal development, and eruption rate of the incisors of rats was as follows:

Experimental Animals, Their Housing and Care

White albino rats from our stock colony which is raised on Sherman's diet B (herein referred to as diet 13) consisting of two-thirds ground whole wheat, one third whole milk powder, and sodium chloride equal to two percent of the weight of the wheat, were used throughout these experiments. The rats were taken from their mothers when they were twenty-eight days old (unless otherwise specified) and placed in individual galvanized iron cages with raised screen bottoms. At this time they were given one of the experimental diets under investigation (see below), ten males and/or ten females being used on each diet so as to avoid possible variations in the results due to sex differences. In order to prevent scattering of the food, the food cups were provided with flared metal tops. Tap water (unless otherwise noted) and the experimental diet were given ad libitum. Food records were kept and measurements of the body weight, body length, and tooth eruption were made weekly, the details of which are to be considered later.
At the end of the experimental period the animals were killed and further measurements of the chest girth, lengths of the femur, tibia, humerus, and radius, and weight of the incisors made according to the methods outlined below.

Experimental Diets

The compositions of the experimental diets are given in condensed form in Table I. They can be generally classed as being adequate, deficient in certain minerals, protein-poor, vitamin-free, or containing fluorine, and fluorine with supplements of calcium or aluminum chloride.

The adequate rations include diets 13, 13 m.s., and 16. Diet 13 is composed of two-thirds whole wheat, one-third milk powder and sodium chloride equal to two percent of the weight of the wheat and has been found capable of supporting normal growth and reproduction for many successive generations. Diet 13 m.s. is a modification of diet 13 in which meat scraps have been added, thereby increasing the calcium and iron contents of the ration appreciably. Due to the increased mineral content it has been shown to provide for more optimum growth and reproduction in this laboratory for many successive generations. Diet 16 is the same as Sherman's diet A (117) which is also a modification of diet 13 in that the percentage of wheat is higher and the percentage of milk lower, thus resulting in a decreased calcium content. It is a sub-optimal stock diet since the growth rate of animals maintained on it has been observed to be slightly below the
normal average rate of growth noted with animals on the better diet 13.

The diets containing fluorine (diets 214, 214+ Ca, 214 + Al Cl) were made up as modifications of diet 13. Diet 214 has the same composition as diet 13 with the addition of 0.1 percent of sodium fluoride. The fluorine containing salt was weighed quantitatively, and in the preparation of this diet great care was exercised to ensure uniformity of mixture of the salt with the basal diet since a higher percentage of fluorine in part of the ration resulting from poor mixing would have caused the death of the animal. Diet 214 + Ca is the same diet 214 except that two percent of the wheat has been replaced by calcium carbonate. Unpublished data from this laboratory has shown that the addition of calcium to a fluorine containing ration reduces the toxic effect of the fluorine on the growth rate but does not change the mottled appearance of the teeth of the animal. Hence, this diet was included to throw more light on this point, if possible. Diet 214 + Al Cl is another modification of diet 214 in which 1.47 percent of the wheat has been substituted with aluminum chloride. It will be recalled that Sharpless found it possible to lessen the toxic effects of fluorine on the growth rate of the rat body and on the appearance of the incisors by incorporating in the fluorine-containing ration from 0.5% to 2.0% of aluminum chloride.
Since it was thought desirable to study these points further, diet 214 + Al Cl was included in this investigation.

The rations that are deficient in certain of the minerals are diets 2965 (118), 400, and 600. Diet 2965 is Steenbock's high calcium, low phosphorus, low vitamin D rickets producing diet which consists of yellow corn 76, wheat gluten 20, calcium carbonate 3, and sodium chloride 1. On the other hand, diet 400, composed of yellow corn 75, casein 9, yeast 15, and sodium chloride 1, is low in calcium and vitamin D. Diet 600 is an anemia producing diet since it contains only whole milk powder which is low in iron and copper. Distilled water is given with this diet so that the intake of iron and copper will be at a minimum.

The low protein diet 110 contains only eight percent of the adequate protein, casein, yet it has all of the other dietary essentials, namely, carbohydrate, minerals, and vitamins A, B, G, and D in the correct proportions.

The vitamin A, B, or G deficient rations are, respectively, diets 379, 513, and 555. From an inspection of their compositions it will be noted that each is adequate in every respect other than the vitamin in question. Sufficient protein has been supplied by the casein which has been freed from interfering vitamins by extraction with alcohol. (119) Cornstarch and Osborne and Mendel's salt mixture, both of which are relatively free from all vitamins, provide for
normal amounts of carbohydrate and minerals. Vitamins B (B and B ) and D are supplied in the A-free ration, diet 379, by brewers' yeast and 100 D viosterol, respectively. Diet 513 receives its source of vitamins A and D from cod liver oil and butter fat, and its vitamin C (B ) from autoclaved yeast. Similarly, vitamins A and D are supplied in the G-free ration, diet 555, by cod liver oil and butter fat, and vitamin B (B ) is included by the addition of an alcoholic extract of whole wheat.

Experimental Period

The animals were maintained on the experimental diets for a test period of six weeks from the time of weaning except in the cases of the vitamin A-free and iron and copper (anemia) deficient animals. Due to the nature of these deficiencies the following special methods had to be adopted in these cases.

In our laboratory it has been found by Smith that if rats to be used for vitamin A experiments are weaned at twenty-eight days, they have so large a storage of vitamin A in their bodies that it is difficult to deplete them of such in the interval allowed for this depletion. This large storage of vitamin A is prevented by weaning the rats at twenty-one instead of twenty-eight days. In this study, therefore, the rats were taken from their mothers at twenty-one days, placed in individual galvanized iron cages with raised screen bottoms, and given the basal A-free ration (diet 379) with tap water ad libitum for a test period of seven weeks, the specified
measurements being made during this time from the fourth week of age (28 days) on.

It has been found impossible to produce anemia in animals raised according to the normal procedure. Therefore, in this study, animals were made anemic according to the method of Elvehjem and Kemmerer which is briefly as follows:

When the rats were two weeks old, the mothers were given whole milk powder only in place of the high iron and copper containing stock diet 13. In this way production of anemia in the young was hastened since they had access only to the milk powder which is low in iron and copper. They were weaned at twenty-one days and placed in individual galvanized iron cages with raised screen bottoms and given diet 600 (whole milk powder) and distilled water ad libitum provided in glass containers to which no copper wires were attached for a test period of seven weeks. When the animals were four weeks of age the weekly measurements specified previously were begun. In addition to the measurements taken at the end of the experimental period, hemoglobin determinations were made according to the Newcomer method.

Experimental Methods of Measurement

It was found that measurements of the body length and tooth eruption could be made with less difficulty when the animals were under mild anaesthesia since in this condition they were easier to handle and the measurements could be made more accurately.
The body or trunk length was determined by flattening the anaesthetized animal face downward, with nose and tail extended but not under tension, upon a sheet of paper and marking carefully the points at the tip of the nose and the end of the hair line, the distance between these points then being measured with a metric rule.

The weekly tooth eruption was measured according to the method suggested by Dr. Isaac Schour who has studied at length the effect of hypophysectomy upon the eruption rate of rat incisors. This method is as follows: At the beginning or when the animals were four weeks of age, a horizontal mark was made with a very fine jeweller's file at the gum line of the lower right incisor of each animal. A week later the distance between this mark and the gingival line was measured carefully with an adjustable caliper. The distance between these points represented the weekly rate of eruption. Each week, throughout the test period, the animals were marked again in the same manner. The mark could be made more visible by rubbing ink into the scratch. As the enamel chipped off the incisors of the fluorine-fed animals it was necessary to cut through enamel into dentine to avoid loss of the mark.

Chest girth measurements were taken by wrapping a cotton string around the chest at the smallest girth and pulling taut but avoiding wrinkling of the skin. This string length was then carefully measured with a metric rule.
The leg bone measurements were determined with calipers adjusted to fixed points on the ends of each bone which had been freed from adhering flesh as nearly as possible. According to Quinn, King, and Dimit there is no difference in the measurements between the right and left legs; hence only the right legs were measured in this investigation.

The removal of the incisors from their sockets was facilitated by decapitating the animals and boiling the heads for about five minutes in water. The incisors were weighed quantitatively to the third decimal place immediately after they were removed from the animal, thus avoiding error due to dehydration.

Similar measurements were made on animals who had died before the end of the experimental period.
Chapter III
EXPERIMENTAL DATA

The results of the investigation of the relative effects of diet upon the body weight, skeletal and tooth development of rats are given in Tables II to XIII, inclusive, and the correlations are shown graphically in Figure 1.

Tables II and III were compiled to avoid repetition of general information in other tables concerning the number and final age of rats used on each diet and the total of average values for the body weight, body length, and food consumption of male and female rats respectively.

Tables IV and V give the average total gains in body weight, average weekly gains in body weight, and the average gain in body weight per 100 grams of food consumed for males and females, respectively, on the different diets studied. The weekly gain in body weight made by rats on each diet is compared with that made by those maintained on the normal stock ration, diet 13.

The effect of different diets on the average total gain in body length, average weekly gain in body length, and the average chest girth and length of the long leg bones at death for males and females is given in Tables VI and VII, respectively. The weekly gain in body length and the chest girth at death
as shown by animals on each diet is also compared with those shown by rats subsisting on diet 13.

Tables VIII and IX give the weekly rate of eruption of the incisors and the weight of the incisors at death of male and female rats, respectively, on each diet, comparisons with the normal eruption rate and incisor weight also being recorded.

Ratios between the average total gain in body weight and the average total gain in body length, chest girth, chest girth, and lengths of the femur, tibia, humerus, and radius are given in Tables X and XI for males and females, respectively. Similarly, the relationship between incisor growth (as measured by total incisor eruption and fresh weight of the incisors) and the average total gains in body weight and body length of males and females on each diet has been computed and recorded in Tables XII and XIII.

In order to facilitate discussion of the various groups and to enable comparisons to be more clearly presented, the results are discussed for each diet separately.

Discussion of Experimental Results

Animals on the Normal Ration, Diet 13.

The average gains in body weight and body length per week for animals on diet 13 during the six weeks test period and the average weekly incisor eruption were found to be 22.45 gms., 1.25 cms., and 3.98 mm., respectively, for males and 14.73 gms.,
The average chest girth was 11.38 cms., and 10.86 cms., and the weight of the incisors 130 mgs. and 121 mgs. for males and females, respectively. It was found that males gained an average of 30.17 gms. and females 22.5 gms. in body weight for every 100-gms. of food consumed. The lengths of the femur, tibia, humerus, and radius at death were 3.02, 3.43, 2.78, and 2.50 cms. respectively, for males and 2.72, 3.03, 2.53, and 2.33 cms., respectively, for females.

In the discussions which follow these measurements were considered "normal" or equal to 100 per cent and were used as such in making the comparisons between the experimental diets studied.

Animals on Diet 13 m.s.

When the calcium and iron contents of diet 13 were increased by the addition of meat scraps to the diet, male rats showed slightly greater gains in body weight and body length during the six weeks experimental period and slightly larger chest girths than those recorded for animals on diet 13 alone. The incisors also erupted at a slightly faster rate than those of animals on the unsupplemented diet 13, but a statistical analysis of the actual differences in these eruption rates shows them to be only slightly greater than their probable errors. The ratios of the gain in body weight to the gain in body length, girth of chest, and length of the leg bones and
When the calcium content of diet 13 was lowered by decreasing the amount of milk and increasing the amount of wheat (i.e., diet 16), it was found that the average gains in body weight and body length during the six-week test period were slightly below the normal average gains recorded for animals on the unaltered diet 13. Male rats gained a total of 121.4 grams in body weight and 7.10 cms. in body length in comparison with gains of 134.7 gms. and 7.51 cms. for weight and length, respectively, recorded for normal males subsisting on the better diet 13. The average chest girth of male rats was slightly greater and the leg bones slightly shorter than those of animals on the normal diet 13 but these differences were found to be statistically insignificant. There appears to be little or no difference in the length of the body, girth of chest, or length of the leg bones relative to the gain in body weight between animals of the same age on diet 16 and diet 13.

Again, in some cases, the incisors of male rats were noted to erupt at a slightly faster rate than those of normal controls on diet 13, but a statistical analysis of the differences shows them to be insignificant. The average weekly incisor
eruption rate for males was 4.12 mm. and that recorded for normal males on diet 13 was 3.98 mm. The incisors of the two groups of animals weighed exactly the same. Relative to the gains in body weight and body length the incisor eruption and incisor weight showed no significant differences from those of normal animals of the same age.

Animals on Diet 214.

In accordance with the observations noted by many investigators concerning the effects of a fluorine containing diet on the growth rate, skeletal development, and appearance of the incisors of rats, it was found that when 0.1 percent of sodium fluoride was added to diet 13, badly stunted animals and severe mottling of the enamel resulted.

The body weight of male rats was markedly affected, the average weekly gain being only 8.3 gms. or 36.97 percent of the normal gain. Food consumption was less than normal but not proportionately so, for the decrease in food intake did not run parallel to the inhibition in growth. Males consumed about 76 percent as much food as the normal animals but grew at only approximately 37 percent of the normal rate. Therefore the efficiency of the utilization of the food was lower on the fluorine containing ration than on the normal diet 13.

Skeletal development as measured by the body length, chest girth, and length of the leg bones was also interfered with greatly by the inclusion of fluorine in the basal ration. The
total gain in body length for male rats during the six weeks test period was 3.71 cms. in comparison with a total gain of 7.51 cms. recorded for normal males of the same age. An inspection of Table VI reveals that the femur, tibia, humerus, and radius were shorter than those of normal controls, though the length of these bones was not affected to so great an extent as was the length of the body. Such a phenomenon resulted in animals that were about 51% shorter than normal with leg bones that were only about 15% shorter than normal. It was also found that the chest girth was not retarded to so great an extent as was the body length, the average chest girth of male rats at death being 10.00 cms. which is approximately 88% of that recorded for normal animals of the same age. From a study of the ratios given in Table X it is found that the length of the body, chest girth, and length of the leg bones increased during the time of the consumption of the fluorine containing ration to a relatively greater extent than did the body weight of such animals during this period, since the ratios of the gain in body weight to each of these measurements are considerably lower than the corresponding ratios for normal rats of the same age.

The change in the appearance of the incisors was striking, the first "fluorine effect", that is loss of pigment and dullness, appearing about the second week. The erupted portion of the incisor was not altered, but as the incisor renewed itself, abnormal areas were evident at the gum line. By the end of the
six weeks experimental period the incisors of all animals had become severely mottled being short, blunt, chipped, and chalky white in appearance. The rate of eruption of the incisors was also markedly retarded. Male rats showed a total incisor eruption of 19.86 mm. in six weeks or 3.31 mm. per week which was 83.16% of the normal eruption shown by animals on diet 13. The actual difference between the rates of eruption of the incisors of animals on the fluorine containing ration and on the normal diet 13 is significant since a statistical analysis shows the difference to be over five times the probable error of this difference. Also, the incisors of the fluorine fed males weighed only 66.1% as much as those of normal controls. An inspection of the ratios given in Table XII shows that in comparison with normal animals of the same age the incisors of the fluorine fed group erupted at a faster rate than the body gained in weight or length during the feeding of the fluorine containing ration, since ratios of incisor eruption to the gain in body weight and length are higher than those computed for normal animals of the same age. Likewise, the incisors of males weighed somewhat more relative to the gains in body weight and body length than those of normal animals subsisting on diet 13.

Animals on Diet 214 + Calcium.

It was found that when calcium was added to the fluorine containing diet 214, the rate of growth was greater than it was when the same ration did not contain the added calcium,
yet it was by no means normal. Male rats consuming diet 214 alone gained an average total of 49.8 gms. in six weeks whereas when this diet was supplemented with calcium the average total gain during this period was 73.5 gms., the average gain recorded for normal animals being 134.7 gms. There was a greater economy in the use of the food than there was on the unsupplemented fluorine ration as shown by the fact that males on diet 214 + calcium gained 19.9 gms. for every 100 gms. of food consumed in comparison with a gain of only 14.5 gms. recorded for males subsisting on the unsupplemented fluorine diet 214.

The average body length gain was greater on the calcium supplemented fluorine ration than it was on diet 214 alone, male rats showing a weekly body length gain of .796 cms. (63.68% of the normal gain) in contrast to a weekly gain of .618 cms. (49.44% of the normal gain) recorded for animals on the fluorine diet without the added calcium. The chest girth and lengths of the leg bones were slightly below the average normal but appear to be within the normal range.

The retardation in the gain in body length was proportional to the retardation in the gain in body weight since the ratio of the gain in body weight to the gain in body length was about the same as that calculated for normal animals of the same age.

The incisors were severely mottled, being short, blunt, chalky, and chipped in appearance. Likewise, the incisor eruption rate was retarded yet it was greater than when the
fluorine containing diet did not contain the added calcium. The lower incisors of male rats erupted 3.51 mm. per week whereas the average weekly eruption rates for normal males and for males consuming the unsupplemented fluorine ration were, respectively, 3.98 mm. and 3.31 mm. A statistical analysis of these differences shows them to be significant. The incisors of male rats fed the fluorine diet plus calcium weighed considerably more than those of rats fed the fluorine diet alone and only slightly less than those of rats fed the normal diet 13, the respective weights being 120 mgs., 86 mgs., and 130 mgs.

The ratios given in Table XII show that in comparison with normally growing controls the eruption rate of the incisors of male rats was not retarded as greatly as was the gain in body weight and gain in body length. The inclusion of calcium in the fluorine containing ration slightly improved the rate of gain in body weight and body length and slightly accelerated the rate of eruption of the incisors. The greatest benefit resulting from the addition of calcium to the fluorine diet was, however, the marked increase in the weight of the incisors over that recorded for fluorine animals not receiving the calcium supplement. Evidently fluorine did not prevent the assimilation of the additional calcium in the body yet this additional supply of calcium was not capable of counteracting the "mottling" effects of the fluorine contained in the basal diet.
Animals on Diet 214 + Al Cl₃

In accordance with the results reported by Sharpless, it was found that aluminum chloride added to a fluorine containing ration produced a greater gain in body weight during the six weeks test period than that resulting from the feeding of diet 214 alone but that such a gain was not as great as that recorded for animals of similar ages on the fluorine-free diet 13. Male rats gained 16.26 gms. per week or 72.74% of the normal gain in contrast to gains of 8.3 gms. on the same diet without the addition of the aluminum salt and 22.45 gms. for animals on diet 13. The aluminum chloride also increased the efficiency of the utilization of the food since males gained 24.5 gms. for every 100 gms. of food consumed whereas this gain was only 14.5 gms. when the fluorine ration did not contain the aluminum salt.

The gain in body length was less than it was for animals on the normal diet 13 but considerably more than it was for animals on the unsupplemented fluorine diet, the total gains in length for male rats on the fluorine diet alone, on the fluorine diet plus aluminum chloride and on the fluorine-free diet 13 being respectively, 3.71 cms., 5.98 cms., and 7.51 cms. The girth of the chest and the length of the leg bones appear to be within the normal range.

The ratios given in Table X show almost a normal relationship between the rate of gain in body length and the
rate of gain in body weight. However, the chest was larger and the leg bones longer relative to the gain in body weight than those of normal animals of the same age.

It was interesting to note that the incisors were less severely mottled than those of animals on the fluorine diet without the additional aluminum chloride, but they were far from normal in appearance. They showed the characteristic "fluorine effects" at the beginning of the second week of the experimental period and at the end of the test period were white and dull yet none showed chipping or the excessive wearing down characteristic of severely mottled enamel. The rate of eruption of the incisors was only slightly retarded which is in marked contrast to the findings reported for animals on diet 214 alone. The weekly eruption rate for male rats was 3.71 mm. in comparison with an eruption rate of 3.98 mm. for normal animals and 3.31 mm. for males on the unsupplemented fluorine diet. The incisors weighed about one and one-third times as much as those of animals on diet 214 alone and about nine-tenths as much as those of normal animals consuming diet 13.

The ratios given in Table XII show that the rate of eruption of the incisors closely paralleled the rate of gain in body weight and rate of gain in body length since ratios of the total incisor eruption to each of these measurements are approximately the same as those recorded for male rats on
diet 13. The same table shows almost a normal relationship between incisor weight and gains in body weight and body length.

Animals on Diet 2965.

At the end of the six weeks test period all of the animals on the high calcium, low phosphorus, low vitamin D diet 2965 showed the characteristic symptoms of rickets such as a retarded gain in body weight accompanied by skeletal deformities which included marked bowing of the legs and stunted bone development. Male rats gained an average of only 6.55 gms. in body weight per week or 29.17% of the gain shown by normal controls of the same age. It was also found that the lowered food consumption did not run parallel to the retarded gain in body weight since males consumed 68% as much food as normal controls yet they made only 29.17% of the normal gain in body weight. Therefore, the efficiency of the utilization of the food was lower than it was on the normal diet 13.

The skeletal development was decidedly different from that of normal controls of the same age. The average gain in body length for males was only 39.6% of the gain shown by normal animals. The leg bones did not grow as long as those of animals on diet 13 yet their growth was not as greatly retarded as was the growth in body length. Similarly, the chest girth at death was more nearly normal than was the
length of the body. Thus, the greatly retarded body trunk and the only partly retarded leg bones and chest girth resulted in a striking distortion of bodily contour that was distinctly different from normal.

The ratios given in Table X indicate that the body length, chest girth, and length of the leg bones increased to a relatively greater extent during the experimental period than did the weight of such animals since the ratios of the gain in body weight to each of these measurements are all found to be lower than the ratios calculated for diet 13 animals of the same age.

The rickets producing diet not only retarded the body weight and skeletal development of rats but also had a decided slowing up effect upon the rate of eruption of the incisors. The average weekly rate of incisor eruption for males was 3.14 mm. per week whereas the average weekly eruption rate for normal males was 3.98 mm. This difference in the eruption rates is significant since it is over eight times its probable error. It is also interesting to note that the incisors weighed less than normal, those of male rats weighing approximately 78% as much as those of control rats consuming diet 13.

The ratios given in Table XII show that the incisor eruption was not retarded to the same extent as was the body weight and body length of experimental animals since
ratios of incisor eruption to the gain in body weight and length are higher than the same ratios calculated for animals subsisting on diet 13. The incisors also weighed more in proportion to the gains in body weight and body length than those of normal animals of the same age.

Animals on Diet 400.

The general appearance of the animals on diet 400 at the end of the six weeks test period was different from normal, the animals being markedly underdeveloped in both weight and length. The body weight was retarded greatly, males making only $33.85\%$ of the normal gain in weight during the six weeks test period. There was a lower efficiency in the utilization of the food as shown by the fact that males gained only 16.1 gms. per 100 gms. of food consumed in comparison with 30.17 gms. for males consuming the same amount of the normal diet 13. Males consumed approximately $64\%$ as much food as their controls, but they grew at only $33.85\%$ of the normal rate.

The development of the skeleton was also interfered with as shown by the fact that males gained an average of only 3.32 cm. in six weeks, the normal gain in length during this time for male rats being 7.51 cms. The leg bones were also shorter than those of normal controls, but it can be seen that the retardations in the length of the leg bones was not as great as was the retardation in the length of the body. The chest girth and length of the leg bones of male rats were retarded to about the same degree.
From a study of the ratios given in Table X it is found that the gain in body length of male rats was greater in proportion to the gain in body weight than that recorded for normal animals of the same age for the same length of time. The gain in body length for female rats, however, was proportional to the gain in body weight since the ratio of these two measurements is the same as that calculated for normal female rats of the same age. In other words, the gain in body length was retarded to the same degree as was the gain in body weight. The chest was larger than normal and the leg bones longer than normal relative to the gain in body weight since the ratios of the gain in body weight to each of these measurements are considerably lower than those calculated for normal animals of the same age.

The growth of the incisors as measured by the rate of eruption and weight of the incisors was also distinctly retarded. The incisors of male rats erupted at a rate that was 76.13% of the normal rate and weighed about 68% as much as those of normal controls. The actual weekly rate of eruption and the actual weight of the incisors of male rats were respectively, 3.03 mm. and 89 mgs.

An analysis of the ratios given in Table XII shows that on this low calcium, low vitamin D diet the eruption of the incisors of male rats proceeded at a faster rate than the body increased in weight or length. The incisors of female rats erupted at a slightly faster rate than the body increased in weight but at
a much faster rate than the body increased in length. The incisors weighed more relative to the gains in body weight and body length than those of normal animals of the same age.

Animals on Diet 110.

It is seen from a study of Table IV that animals on a diet low only in protein had lost in body weight by the end of the six weeks experimental period, males having lost an average of 1.21 gms. per week. When the loss in weight for male rats was expressed on the basis of the amount of food consumed, it was found to be 2.7 gms. per 100 gms. of food consumed. The gain in body weight per 100 grams of food consumed was much lower (loss) than that recorded for normal animals consuming the same amount of a normal diet, thus the efficiency of the use of the food was very low on this diet. Even though males consumed 59% as much food as their controls, they lost weight markedly by the end of the test period.

The rate of gain in body length was considerably retarded though not to the same degree as was the rate of gain in body weight. The length of the leg bones was also retarded somewhat.

The ratios of the gain in body weight to the skeletal measurements given in Table X are very much lower than the corresponding ratios for normal animals of the same age. Therefore, the body length, length of the leg bones, and chest girth increased to a much greater extent during the
time of the restricted feeding than did the body weight of these animals. Such a phenomenon resulted in stunted animals whose body form was very different from that of normal controls.

It is interesting to note that whereas the gains in body weight and body length were far from normal, the incisors erupted at a rate that was only slightly below the normal rate. The weekly incisor eruption rate for males was 3.38 mm. or 84.92% of the normal rate. Similarly, the incisors weighed 82.3% as much as those of normal male controls. The relationship between incisor growth and the gains in body weight and length is striking. The ratios of the incisor eruption to the gain in body weight and gain in body length and of the incisor weight to the gain in body weight and gain in body length are much higher than those recorded for normal controls. Thus, the incisors erupted at a faster rate than the body increased in weight and length during the six weeks experimental period. Also, the incisors weighed more in proportion to gains in the body weight and body length than those of normal animals of the same age.

Animals on Diet 600.

By the end of the six weeks test period animals (females only) on the milk diet appeared distinctly anemic, the ears and eyes having lost a great deal of the normal red color. It was also noted that the teeth had lost their orange pigment, appearing white and lustrous. Hemoglobin determinations
confirmed the anemic state of the animals, the values lying between 3.55 and 6.03 gms. of hemoglobin per 100 cc. of blood with an average of 4.78 gms.

The gain in body weight was retarded as shown by the fact that the animals gained only 6.1 gms. per week in body weight which is 41.41% of the gain shown by normal animals in the same length of time. However, there was a great economy in the use of the food since the animals gained 20 gms. in body weight for every 100 gms. of food consumed which is approximately the normal gain that results from the consumption of the same amount of the adequate ration, diet 13. In other words, the retarded gain in body weight was parallel to the lowered food consumption, the rats consuming about 46% as much food as their controls and growing at 41.41% of the normal rate. However, recovery of the food was difficult in some cases and may have caused an error in the food consumption record.

The skeletal development of the anemic animals was also found to be retarded as evidenced by the facts that the gain in body length was only 57.22% of the normal gain and the chest girth was only 69.40% of the normal. The leg bones appeared to be only slightly shorter than those of the normal controls of the same age.

According to the ratios recorded in Table XI the body did not increase as fast in weight as it did in length during
the experimental period. Also the girth of the chest and
length of the leg bones were greater relative to the gain
in body weight than those of normal animals of the same age.

The eruption rate of the incisors and the weight of the
incisors were 3.47 mm. and 92 mgs., respectively, or 86.10%
and 76.0% of the normal rate and weight, respectively. The
incisor eruption proceeded at a faster rate than the body
increased in weight or length during the six weeks test period
since ratios of incisor eruption to each of these measurements
are higher than corresponding ratios computed for normal
animals of the same age. Also the incisors weighed more in
proportion to the gains in body weight and body length than
those of normal controls.

Animals on Diet 379.

When the animals (males only) had been on the vitamin A-
free diet for a period of four weeks from the time of weaning
they began to show the symptoms of a vitamin A deficiency.
There was a marked slowing up of the growth rate and the first
sign of opthalmia, namely, sensitiveness of the eyes to light,
appeared. From this time until death the animals lost in
weight consistently and the opthalmia progressed until total
blindness in many cases was evident. The animal died as a
result of a deficiency of vitamin A in the ration. Autopsy
findings at death revealed the presence of pus, principally
in the bladder and at the base of the tongue, thus the
existence of a true vitamin A deficiency was confirmed.

The marked retardation in body weight, a characteristic symptom of a vitamin A deficiency, was observed, the animals making a gain in weight in six weeks that was only 2.67% of the gain shown by normal animals in the same length of time. The amount of food consumed did not run parallel to the marked retardation in body weight, food consumption being about 56% of the normal and the gain in body weight only 2.67% of the normal. Therefore, efficiency in the utilization of the food was much lower on this diet than it was on diet 13.

Skeletal development was retarded though not to the same degree as was the gain in body weight. The average gain in body length was .346 cms. per week or 27.68% of that recorded for normal controls. The chest girth at death was 79.80% as large as the chest girth of normal animals of the same age. Table VI shows that the leg bones were not as long as those of normal controls but the length of these bones did not appear to be as retarded as the length of the body.

The ratios of the gain in body weight to the different measurements given in Table X show that growth in the length of the body, leg bones, and chest had continued, though not at the normal rate, during the progress of the vitamin A deficiency in spite of an almost stationary body weight level.

The effect of a vitamin A deficiency on the rate of
eruption of the incisors was striking. It was noted that
the eruption rate proceeded normally at first, and as the
effects of the deficiency became more evident there was a
marked slowing up of the eruption rate until, in many cases,
the incisors stopped growing altogether. The average weekly
incisor eruption was only 2.27 mm. or 57.03% of the normal.
Also, the incisors weighed only 70.8% as much as those of
normal controls.

Table XII shows that the incisor eruption proceeded at
a faster rate than the body increased in weight or length
since ratios of the eruption of the incisors to the gains in
body weight and length are higher than those recorded for
normal animals of approximately the same age. Also, the
incisors weighed more relative to the gains in body weight
and length than those of normal animals.

Animals on Diet 513.

Symptoms of a vitamin B deficiency were evident when
the animals (females only) had been on the experimental B-
deficient diet for two weeks from the time of weaning.
From this time until death the rats lost weight and showed
the marked humping of the back which is a characteristic
symptom of polyneuritis. Therefore, the rats had died as
a result of a lack of vitamin B in the basal ration.

The loss in body weight which has been observed con-
sistently in connection with a vitamin B deficiency was
noted here. The animals lost on the average of 4.08 gms. per week during an average survival period of sixty-one days on the B-free diet. When the loss in body weight was expressed in terms of the amount of food consumed, it was found that the rats lost on an average of 12.10 gms. in body weight for every 100 gms. of food consumed. Thus, the efficiency of the utilization of the food was extremely low.

The gain in body length was also retarded, though not to the same extent, as was the gain in body weight, it being 41% of the normal gain. Compared to the chest girth of normal animals (70 days of age), the average chest girth of the stunted animals (61 days of age) was found to be 70.4% as large as that of normal animals. The leg bones were nearly normal in length even though the B-deficient animals were slightly younger than the normal controls.

The ratios given in Table XI show clearly that the body length, chest girth, and length of the leg bones had increased during the time of the feeding of the vitamin B-deficient diet whereas the body weight had decreased considerably.

In spite of the fact that the animals were so greatly distorted at death, the incisors of such animals grew at a rate very nearly equal to the rate of eruption shown by normal animals on the adequate diet 13. The eruption rate of the lower incisors was 3.67 mm. per week or 90.57% of the normal rate. The incisors of the stunted animals at
death (61 days) weighed 79.3% as much as those of normal animals at death (to days).

The ratios of incisor eruption to the gains in body weight and length given in Table XIII are much higher than the corresponding ratios calculated for normal animals of approximately the same age. Therefore, the incisors grew faster than the body increased in weight and length during the time of the restricted feeding. The incisors weighed more relative to the gains in body weight and body length than those of normal animals.

Animals on Diet 555.

Animals (males only) on the vitamin G-free diet gained slightly in body weight up until about the end of the second week on the experimental diet, the weight then remaining fairly constant until the animals were killed at the end of a ten weeks test period, or when they were ninety-eight days old. Characteristic symptoms of a G-deficiency were not evident until the animals had been maintained on the experimental diet for a period of six weeks or more. From this time until the animals were killed, progressive symptoms of a G-deficiency such as redness and soreness of the feet and nose, loss of hair, and roughness of the coat were consistently found. It was interesting to note that in three cases cataracts were plainly visible to the naked eye, an observation which has been recorded for G-deficient animals, heretofore, in other laboratories.
It was found that during the ten week test period the animals gained only 10.1 gms. or 4.49% of the normal gain in weight. The amount of food consumed did not run parallel to the inferior growth curve, the rats consuming about 43% as much food as normal controls but growing at only 4.49% of the normal rate. The gain in weight per 100 gms. of food consumed was also considerably less than that recorded for normal animals, hence, the efficiency of utilization of the food was very low. The skeletal development was also greatly retarded though not to the same extent as was the body weight.

The rats gained an average of .298 mm. in length per week which is only 23.84% of the normal gain in length. The chest girth at death (33 days) was 75.4% as large as the chest girth of normal animals at death (70 days). Also, the leg bones of the stunted animals were considerably shorter than those of the younger animals.

The relationship between the body weight and the skeletal development is brought out in Table X. It is seen that the body length, chest girth, and length of the leg bones had increased to a greater extent than had body weight during the time of the restricted feeding.

The incisors erupted at approximately the normal rate as shown by Table VIII, the eruption rate being 3.56 mm. per week or 89.44% of the normal rate. The incisors weighed 109 mgs. in comparison with 130 mgs. recorded for the younger
normal males on diet 13.

The calculated ratios given in Table XII show that the incisors erupted at a faster rate than the body increased in weight or length and that they weighed more in proportion to the body weight and length than those of normal animals who were twenty-eight days younger.
CHAPTER IV

SUMMARY AND CONCLUSIONS

Male and female rats were weened at twenty-eight days and placed on an experimental diet that was either adequate in all respects, deficient in certain minerals, deficient in vitamins A, B, or C, deficient in an adequate protein, or supplemented with fluorine, fluorine plus calcium, or fluorine plus aluminum chloride for an experimental period of six weeks. Weekly measurements of the body weight, body length, and incisor eruption were made, and at the end of the test period the animals were killed and additional measurements of the chest girth, length of the femur, tibia, humerus and radius, and weight of the incisors were recorded. Food consumption records were kept. The average weekly gains in weight and length and the average weekly incisor eruptions were calculated for animals on each diet and these figures compared with the corresponding measurements computed for normal animals (diet 13) of the same age. Ratios of the total gain in body weight to the total gain in body length, chest girth, and length of the femur, tibia, humerus, and radius, and ratios of the total incisor eruption and weight of the incisors to the total gains in body weight and body length were calculated for animals on each diet.
Conclusions from this study of the relative effects of diet upon the body weight, skeletal development, and rate of eruption of the incisors of albino rats are as follows:

1. The rate of gain in body weight and the rate of gain in body length of male and female rats during a six weeks experimental period were slightly accelerated when the calcium and iron contents of the normal diet 13 were increased, as in diet 13 m.s. The retardation of the rate of gain in body weight and body length was considerable when the calcium or phosphorus content of the diet was lowered, the amount of retardation being greatest when the diet was extremely low in calcium (diet 400) or phosphorus (diet 2965) and least when the diet was only partially deficient in calcium (diet 16). A deficiency of iron and copper in the ration resulted in a retardation of the gain in body weight and body length similar to that resulting from an extreme deficiency of calcium or phosphorus. The inclusion of fluorine in the diet (diet 214) severely retarded the rate of gain in body weight and body length, the amount of retardation being less severe when calcium was added and still less severe when aluminum chloride was added to this diet. A deficiency of the principal dietary essentials, namely, protein and vitamins A, B, or G produced the most severe retardation of the rate of gain in body weight and body length.

2. The chest girth of male and female rats at the end of the six weeks experimental period was slightly greater than
normal when the calcium and iron contents of diet 13 were increased, as in diet 13 m.s. A partial decrease in the calcium content of the diet, as in diet 16, did not affect the chest girth of the animal, but when the calcium or phosphorus content of the diet was greatly reduced as in diets 400 and 2965, respectively, the chest girth was markedly smaller than normal. The addition of sodium fluoride to diet 13, (diet 214) produced animals with smaller chest girths than normal, but when this diet was supplemented with calcium or with aluminum chloride, the chest girth approximated that recorded for normal animals of the same age. The girth of the chest of animals maintained on a diet that was either low in an adequate protein, low in iron and copper, or lacking in vitamins A, B, or G was considerably smaller than normal.

3. The leg bones of male and female rats at the end of the six-weeks experimental period were considerably shorter than normal when the calcium or phosphorus content of the diet was extremely low as in diets 400 and 2965, respectively, but when the calcium content of the diet was only slightly decreased, as in diet 16, these bones were normal in length. The inclusion of fluorine in the diet also caused the leg bones to be shorter than normal, yet when such a diet was supplied with calcium or with aluminum chloride, the leg bones were normal in length. The length of the leg bones was not seriously affected by deficiencies in iron and copper, vitamins A, B, or G, or an adequate protein, the bones being approximately normal in
length at the end of the experimental period.

4. In comparison with normally growing controls the rate of gain in body length was proportional to the rate of gain in body weight when the calcium and iron contents of diet 13 were increased, as in diet 13 m.s., and also when the calcium content of the diet was partially decreased as in diet 16. On the other hand when the calcium content of the diet was considerably decreased, as in diet 400, male rats increased in length at a slightly faster rate than they increased in weight, yet female rats on this diet increased in length at the same rate that they increased in weight. Reducing the phosphorus and reducing the iron and copper content of the diet, as in diets 2365 and 600, respectively, resulted in a slightly greater increase in body length relative to the increase in body weight than that resulting from the consumption of the normal diet 13. The inclusion of fluorine in the diet also retarded the body weight to a slightly greater extent than the body length, but when calcium or when aluminum chloride were added to this diet, the rate of gain in body length was proportional to the rate of gain in body weight. When the diet was lacking in certain of the important dietary essentials, namely, protein and vitamins A, B, or G, the body increased in length at a much faster rate than it increased in weight, though the increase in length was far from normal.

5. In general the girth of the chest and length of the leg bones were not retarded to the same extent as the body weight.
on any of the diets studied.

6. The rate of eruption of the incisors of male and female rats was considerably retarded when the level of calcium or phosphorus in the diet was extremely low as in diets 400 and 2965, respectively, but when the amount of calcium was only partially reduced as in diet 16, the rate of eruption of the incisors was normal. Sodium fluoride added to diet 13 (diet 214) retarded the rate of eruption of the incisors, but this effect could be partially counteracted by the addition of calcium, and could be still further counteracted by the addition of aluminum chloride to the fluorine diet. The rate of eruption of the incisors was only slightly retarded by diets that were deficient in an adequate protein, low in iron and copper, and deficient in vitamins B and G. On the other hand a deficiency of vitamin A caused a severe retardation of the rate of eruption of the incisors.

7. In comparison with normally growing controls the rate of eruption of the incisors was proportional to the rate of gain in body weight and to the rate of gain in body length when the calcium and iron contents of diet 13 were increased, as in diet 13 m.s., and when the calcium content of diet 13 was slightly decreased as in diet 16. However, decreasing the calcium content further, as in diet 400, caused the incisors of male rats to erupt at a faster rate than the body increased in either weight or length, and it caused the incisors of female rats to erupt at a slightly faster rate than the body increased
in weight but at a much faster rate than the body increased in length. A reduction of the phosphorus content (diet 2965) or of the iron and copper content (diet 600) also caused the eruption of the incisors to proceed at a faster rate than the body increased in weight or length. When calcium was added to the fluorine containing diet 214, the incisors erupted at a rate that was more in proportion to the rate of gain in body weight and body length than when the fluorine diet did not contain this supplement. Furthermore, the addition of aluminum chloride to the fluorine diet resulted in almost a normal relationship between incisor eruption and the gains in body weight and body length. Diets lacking in protein or in vitamins B or G caused the eruption of the incisors to proceed at almost the normal rate, whereas the rate of gain in body weight and the rate of gain in body length were extremely subnormal. The incisors of animals fed a diet deficient in vitamin A erupted at a somewhat faster rate than the body increased in length and at a much faster rate than the body increased in weight.

8. In all cases the weight of the incisors was proportional to the rate of eruption of the incisors.
Table I
Composition of Diets

<table>
<thead>
<tr>
<th>Food</th>
<th>Diet Number</th>
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</tr>
</tbody>
</table>

* One drop each of cod liver oil and vitamin B-G extract given daily.
** One drop 100 D viosterol given daily.
*** Alcohol extract of 50 gms. of whole wheat per 100 gms. of food mixture included.
Table II

Age, Body Weight, Body Length,
and Food Consumption Records of Male Rats

<table>
<thead>
<tr>
<th>Diet No.</th>
<th>No. of Rats</th>
<th>Average age of rat at end of test period days</th>
<th>Average weaning body weight gms.</th>
<th>Average final body weight gms.</th>
<th>Average weaning body length cms.</th>
<th>Average final body length cms.</th>
<th>Average total food consumed gms.</th>
</tr>
</thead>
<tbody>
<tr>
<td>13</td>
<td>10</td>
<td>70</td>
<td>63.0</td>
<td>197.7</td>
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<td>21.19</td>
<td>446.9</td>
</tr>
<tr>
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<td>70</td>
<td>69.0</td>
<td>214.3</td>
<td>13.66</td>
<td>21.40</td>
<td>506.0</td>
</tr>
<tr>
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<td>10</td>
<td>70</td>
<td>60.6</td>
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<td>66.7</td>
<td>116.5</td>
<td>14.08</td>
<td>17.79</td>
<td>343.0</td>
</tr>
<tr>
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<td>79.6</td>
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<td>14.93</td>
<td>19.71</td>
<td>369.3</td>
</tr>
<tr>
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<td>70</td>
<td>69.2</td>
<td>166.8</td>
<td>14.33</td>
<td>20.31</td>
<td>397.9</td>
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<td>60.5</td>
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<td>96.8</td>
<td>13.79</td>
<td>17.11</td>
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<td>67.9</td>
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<td>10</td>
<td>73</td>
<td>69.3</td>
<td>73.5</td>
<td>14.91</td>
<td>17.12</td>
<td>275.4</td>
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<tr>
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<td>61.5</td>
<td>71.6</td>
<td>13.49</td>
<td>16.47</td>
<td>323.6</td>
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</table>

* Weight at 28 days.
** Length at 28 days.
Table III

Age, Body Weight, Body Length, and Food Consumption Records of Female Rats

<table>
<thead>
<tr>
<th>Diet No.</th>
<th>No. of Rats</th>
<th>Average age of rat at end of test period (days)</th>
<th>Average weaning body weight (gms.)</th>
<th>Average final body weight (gms.)</th>
<th>Average weaning body length (cms.)</th>
<th>Average final body length (cms.)</th>
<th>Average total food consumed (gms.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>13</td>
<td>10</td>
<td>70</td>
<td>57.3</td>
<td>146.7</td>
<td>12.92</td>
<td>18.90</td>
<td>393.2</td>
</tr>
<tr>
<td>13 m.s.</td>
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<td>70</td>
<td>65.4</td>
<td>169.3</td>
<td>13.61</td>
<td>19.80</td>
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<tr>
<td>16</td>
<td>10</td>
<td>70</td>
<td>60.1</td>
<td>140.9</td>
<td>13.22</td>
<td>18.49</td>
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<td>70</td>
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<td>86.1</td>
<td>12.91</td>
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<td>70</td>
<td>77.0</td>
<td>134.5</td>
<td>14.73</td>
<td>18.56</td>
<td>343.3</td>
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<td>214 + Al Cl₃</td>
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<td>70</td>
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<td>13.90</td>
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<td>70</td>
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<td>77.9</td>
<td>68.0</td>
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<td>76.3</td>
<td>12.48</td>
<td>15.90</td>
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<td>61</td>
<td>60.4</td>
<td>40.9</td>
<td>13.56</td>
<td>15.51</td>
<td>148.7</td>
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</table>

* Weight at 28 days.
** Length at 28 days.
Table IV
The Effect of Different Diets
Upon the Body Weight of Male Rats

<table>
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<tr>
<th>Diet No.</th>
<th>Average total body weight gain</th>
<th>Average body weight gain per wk.</th>
<th>Percentage of normal gain in body weight</th>
<th>Average food consumed per wk.</th>
<th>Average gain in body weight per 100 gms. food consumed</th>
</tr>
</thead>
<tbody>
<tr>
<td>13</td>
<td>134.7</td>
<td>22.45</td>
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<td>84.33</td>
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<tr>
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<td>121.4</td>
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<td>90.42</td>
<td>69.86</td>
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<td>8.30</td>
<td>36.97</td>
<td>57.16</td>
<td>14.50</td>
</tr>
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<td>12.25</td>
<td>54.56</td>
<td>61.55</td>
<td>19.90</td>
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<td>214 + Al Cl₃</td>
<td>97.6</td>
<td>16.26</td>
<td>72.42</td>
<td>66.31</td>
<td>24.50</td>
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<td>- 5.38</td>
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* Diet 13 used as a base or 100%.
Table V
The Effect of Different Diets
Upon the Body Weight of Female Rats

<table>
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<tr>
<th>Diet No.</th>
<th>Average total body weight gain</th>
<th>Average body weight gain per wk.</th>
<th>Percentage of normal gain in body weight</th>
<th>Average food consumed per wk.</th>
<th>Average gain in body weight per 100 gms. food consumed</th>
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<td>100.00</td>
<td>65.53</td>
<td>22.50</td>
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<tr>
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<td>91.38</td>
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<td>71.69</td>
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<td>5.05</td>
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<td>-1.65</td>
<td>-11.20</td>
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<td>30.50</td>
<td>20.0</td>
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<td>33.71</td>
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* Diet 13 used as a base or 100%.
Table VI

The Effect of Different Diets Upon the Skeletal Development of Male Rats

<table>
<thead>
<tr>
<th>Diet No.</th>
<th>Average total body length gain</th>
<th>Average body length gain per wk.</th>
<th>Percentage of normal gain in body length</th>
<th>Average chest girth at death</th>
<th>Percentage of normal chest girth</th>
<th>Average length of the leg bones at death</th>
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<td>13</td>
<td>7.51</td>
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<td>11.38</td>
<td>100.00</td>
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</tr>
<tr>
<td>13 m.s.</td>
<td>7.74</td>
<td>1.29</td>
<td>103.20</td>
<td>12.25</td>
<td>107.70</td>
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<tr>
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<td>7.10</td>
<td>1.18</td>
<td>94.40</td>
<td>12.00</td>
<td>105.50</td>
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<td>.618</td>
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<td>88.00</td>
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<td>.796</td>
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<td>96.3</td>
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<td>83.6</td>
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<td>.553</td>
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<td>85.4</td>
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<td>72.2</td>
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<td>79.8</td>
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<td>2.63  3.24  2.33  2.49</td>
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</table>

* Diet 13 used as a base or 100%.
### Table VII

The Effect of Different Diets Upon the Skeletal Development of Female Rats

<table>
<thead>
<tr>
<th>Diet No.</th>
<th>Average total body length gain</th>
<th>Average body length gain per wk.</th>
<th>Percentage of normal gain in body length</th>
<th>Average chest girth at death</th>
<th>Percentage of normal chest girth</th>
<th>Average length of the leg bones at death</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>cms.</td>
<td>cms.</td>
<td>%</td>
<td>cms.</td>
<td>%</td>
<td>Femur</td>
</tr>
<tr>
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<td>5.98</td>
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<td>100.00</td>
<td>10.96</td>
<td>100.00</td>
<td>2.72</td>
</tr>
<tr>
<td>13 m.s.</td>
<td>6.19</td>
<td>1.03</td>
<td>103.41</td>
<td>11.23</td>
<td>103.5</td>
<td>2.87</td>
</tr>
<tr>
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<td>98.6</td>
<td>2.73</td>
</tr>
<tr>
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<td>.505</td>
<td>50.70</td>
<td>8.85</td>
<td>81.5</td>
<td>2.26</td>
</tr>
<tr>
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<td>64.05</td>
<td>10.32</td>
<td>95.1</td>
<td>2.72</td>
</tr>
<tr>
<td>214 Al Cl</td>
<td>4.54</td>
<td>.756</td>
<td>75.90</td>
<td>10.20</td>
<td>94.0</td>
<td>2.73</td>
</tr>
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<td>85.8</td>
<td>2.26</td>
</tr>
<tr>
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<td>2.09</td>
<td>.348</td>
<td>34.94</td>
<td>9.12</td>
<td>84.0</td>
<td>2.38</td>
</tr>
<tr>
<td>110</td>
<td>1.88</td>
<td>.313</td>
<td>31.42</td>
<td>8.44</td>
<td>77.8</td>
<td>2.53</td>
</tr>
<tr>
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<td>.570</td>
<td>57.22</td>
<td>7.53</td>
<td>69.4</td>
<td>2.41</td>
</tr>
<tr>
<td>513</td>
<td>1.95</td>
<td>.409</td>
<td>41.00</td>
<td>7.64</td>
<td>70.4</td>
<td>2.49</td>
</tr>
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</table>

* Diet 13 used as a base or 100%.
Table VIII

The Effect of Different Diets Upon the Incisor Growth of Male Rats

<table>
<thead>
<tr>
<th>Diet No.</th>
<th>Average incisor eruption per week ( + )</th>
<th>(-) F.E.</th>
<th>( \text{Average fresh weight of incisors} )</th>
<th>( \text{Percentage of normal weight of incisors} )</th>
</tr>
</thead>
<tbody>
<tr>
<td>13</td>
<td>3.98 ( + ) ( + )</td>
<td>- .0607 &amp; 100.00 &amp; 130 &amp; 100.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13 m.s.</td>
<td>4.12 ( + ) ( + )</td>
<td>- .0552 &amp; 103.51 &amp; 128 &amp; 98.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>4.12 ( - ) ( + )</td>
<td>.0454 &amp; 103.51 &amp; 130 &amp; 100.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>214</td>
<td>3.31 ( - ) ( + )</td>
<td>-.1155 &amp; 83.16 &amp; 86 &amp; 66.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>214 Ca</td>
<td>3.51 ( + ) ( + )</td>
<td>-.0890 &amp; 88.19 &amp; 120 &amp; 92.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>214 Al Cl(_2)</td>
<td>3.71 ( + ) ( + )</td>
<td>-.0379 &amp; 93.21 &amp; 118 &amp; 90.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2965</td>
<td>3.14 ( + ) ( + )</td>
<td>-.0710 &amp; 78.89 &amp; 102 &amp; 78.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>400</td>
<td>3.03 ( + ) ( + )</td>
<td>-.0458 &amp; 76.13 &amp; 89 &amp; 66.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>110</td>
<td>3.38 ( + ) ( + )</td>
<td>-.0469 &amp; 84.92 &amp; 107 &amp; 82.3</td>
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<td></td>
</tr>
<tr>
<td>379</td>
<td>2.27 ( + ) ( + )</td>
<td>-.1236 &amp; 57.03 &amp; 92 &amp; 70.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>555</td>
<td>3.56 ( + ) ( + )</td>
<td>-.0429 &amp; 89.44 &amp; 109 &amp; 83.9</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Diet 13 used as a base or 100%.
Table IX

The Effect of Different Diets Upon the Incisor Growth of Female Rats

<table>
<thead>
<tr>
<th>Diet No.</th>
<th>Average incisor eruption per week + P.E. mm.</th>
<th>Percentage of normal rate of incisor eruption</th>
<th>Average fresh weight of incisors</th>
<th>Percentage of normal weight of incisors</th>
</tr>
</thead>
<tbody>
<tr>
<td>13</td>
<td>4.03 ± .0559</td>
<td>100.00</td>
<td>121</td>
<td>100.00</td>
</tr>
<tr>
<td>13 m.s.</td>
<td>3.98 ± .0539</td>
<td>93.75</td>
<td>136</td>
<td>112.80</td>
</tr>
<tr>
<td>16</td>
<td>4.09 ± .0521</td>
<td>101.48</td>
<td>120</td>
<td>99.2</td>
</tr>
<tr>
<td>214</td>
<td>2.83 ± .0822</td>
<td>70.22</td>
<td>92</td>
<td>76.0</td>
</tr>
<tr>
<td>214 Ca</td>
<td>3.47 ± .0691</td>
<td>86.10</td>
<td>106</td>
<td>87.6</td>
</tr>
<tr>
<td>214 AlCl</td>
<td>3.49 ± .0023</td>
<td>86.60</td>
<td>114</td>
<td>94.2</td>
</tr>
<tr>
<td>2965</td>
<td>2.98 ± .0710</td>
<td>73.94</td>
<td>98</td>
<td>80.9</td>
</tr>
<tr>
<td>400</td>
<td>3.12 ± .0435</td>
<td>77.41</td>
<td>89</td>
<td>73.6</td>
</tr>
<tr>
<td>110</td>
<td>3.41 ± .0431</td>
<td>84.61</td>
<td>103</td>
<td>85.1</td>
</tr>
<tr>
<td>600</td>
<td>3.47 ± .0924</td>
<td>86.10</td>
<td>92</td>
<td>76.0</td>
</tr>
<tr>
<td>513</td>
<td>3.67 ± .0329</td>
<td>90.57</td>
<td>96</td>
<td>79.3</td>
</tr>
</tbody>
</table>

* Diet 13 used as a base or 100%.
Table X

Relationship of the Total Gain in Body Weight to the Total Gain in Body Length, Chest Girth, and Lengths of the Femur, Tibia, Humerus, and Radius of Male Rats on Different Diets.

<table>
<thead>
<tr>
<th>Diet No.</th>
<th>Average total gain in body weight</th>
<th>Average total gain in body length</th>
<th>Average chest girth</th>
<th>Body weight gain to body length gain</th>
<th>Body weight gain to chest girth</th>
<th>Body weight gain to femur length</th>
<th>Body weight gain to tibia length</th>
<th>Body weight gain to humerus length</th>
<th>Body weight gain to radius length</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>gms.</td>
<td>cms.</td>
<td>cms.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>134.7</td>
<td>7.51</td>
<td>11.28</td>
<td>17.93</td>
<td>11.89</td>
<td>44.60</td>
<td>39.27</td>
<td>48.45</td>
<td>53.88</td>
</tr>
<tr>
<td>13 m.s.</td>
<td>145.3</td>
<td>7.74</td>
<td>12.25</td>
<td>18.77</td>
<td>11.85</td>
<td>48.92</td>
<td>42.61</td>
<td>50.81</td>
<td>56.98</td>
</tr>
<tr>
<td>16</td>
<td>121.4</td>
<td>7.10</td>
<td>12.00</td>
<td>17.09</td>
<td>10.11</td>
<td>41.86</td>
<td>37.01</td>
<td>46.69</td>
<td>51.44</td>
</tr>
<tr>
<td>214</td>
<td>49.8</td>
<td>3.71</td>
<td>10.00</td>
<td>13.42</td>
<td>4.98</td>
<td>19.37</td>
<td>16.76</td>
<td>20.75</td>
<td>22.84</td>
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<tr>
<td>+ Ca</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>214 + Al Cl</td>
<td>73.5</td>
<td>4.78</td>
<td>10.94</td>
<td>15.37</td>
<td>6.67</td>
<td>26.43</td>
<td>22.68</td>
<td>27.84</td>
<td>29.65</td>
</tr>
<tr>
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<td>5.98</td>
<td>11.20</td>
<td>16.32</td>
<td>8.71</td>
<td>34.00</td>
<td>28.47</td>
<td>36.96</td>
<td>38.42</td>
</tr>
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<td>9.52</td>
<td>9.19</td>
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<td>10.87</td>
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<td>3.32</td>
<td>9.72</td>
<td>13.73</td>
<td>4.69</td>
<td>18.68</td>
<td>15.09</td>
<td>17.74</td>
<td>19.57</td>
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<tr>
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</tr>
<tr>
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<td>- 2.24</td>
<td>- 3.16</td>
<td>- 3.20</td>
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<td>9.09</td>
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<td>1.62</td>
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<td>1.75</td>
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<td>3.38</td>
<td>1.17</td>
<td>3.84</td>
<td>3.11</td>
<td>4.33</td>
<td>4.05</td>
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</table>
Table XI

Relationship of the Total Gain in Body Weight
to the Total Gain in Body Length, Chest Girth, and Lengths
of the Femur, Tibia, Humerus, and Radius of
Female Rats on Different Diets

<table>
<thead>
<tr>
<th>Diet No.</th>
<th>Average total gain in body weight</th>
<th>Average total gain in body length</th>
<th>Average chest girth</th>
<th>Body weight gain to body length</th>
<th>Body weight gain to chest girth</th>
<th>Body weight gain to femur length</th>
<th>Body weight gain to tibia length</th>
<th>Body weight gain to humerus length</th>
<th>Body weight gain to radius length</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>gms.</td>
<td>cms.</td>
<td>cms.</td>
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<td></td>
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</tr>
<tr>
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<td>10.86</td>
<td>14.78</td>
<td>8.14</td>
<td>32.50</td>
<td>29.17</td>
<td>34.94</td>
<td>37.94</td>
</tr>
<tr>
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<td>6.19</td>
<td>11.23</td>
<td>16.78</td>
<td>9.25</td>
<td>36.20</td>
<td>31.58</td>
<td>38.34</td>
<td>41.39</td>
</tr>
<tr>
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<td>80.8</td>
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<td>7.54</td>
<td>29.59</td>
<td>26.66</td>
<td>31.68</td>
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</tr>
<tr>
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<td>3.03</td>
<td>8.85</td>
<td>9.57</td>
<td>3.27</td>
<td>12.74</td>
<td>10.82</td>
<td>13.00</td>
<td>12.39</td>
</tr>
<tr>
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<td>57.5</td>
<td>3.83</td>
<td>10.32</td>
<td>15.01</td>
<td>5.57</td>
<td>21.14</td>
<td>18.19</td>
<td>22.91</td>
<td>23.41</td>
</tr>
<tr>
<td>214 + Al Cl&lt;sub&gt;3&lt;/sub&gt;</td>
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<td>4.54</td>
<td>10.20</td>
<td>13.96</td>
<td>6.21</td>
<td>23.22</td>
<td>19.27</td>
<td>24.19</td>
<td>26.31</td>
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<td>8.89</td>
<td>2.42</td>
<td>10.00</td>
<td>9.22</td>
<td>10.81</td>
<td>11.53</td>
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<td>2.09</td>
<td>9.12</td>
<td>14.49</td>
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<td>10.27</td>
<td>13.17</td>
<td>13.77</td>
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<td>1.88</td>
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<td>-5.26</td>
<td>-1.17</td>
<td>-5.91</td>
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<td>-4.23</td>
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<td>-7.83</td>
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<td>-8.26</td>
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</table>
Table XII

Relationship of Incisor Growth to
The Total Gains in Body Weight and Body Length
of Male Rats on Different Diets

<table>
<thead>
<tr>
<th>Diet No.</th>
<th>Average total gain in body weight</th>
<th>Average total gain in body length</th>
<th>Average incisor eruption</th>
<th>Fresh weight of incisors</th>
<th>Incisor eruption to total gain in body weight</th>
<th>Incisor eruption to total gain in body length</th>
<th>Incisor weight to total gain in body weight</th>
<th>Incisor weight to total gain in body length</th>
</tr>
</thead>
<tbody>
<tr>
<td>13</td>
<td>134.7</td>
<td>7.51</td>
<td>23.87</td>
<td>130</td>
<td>.177</td>
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<td>.1856</td>
<td>.965</td>
</tr>
<tr>
<td>13 m.s.</td>
<td>145.3</td>
<td>7.74</td>
<td>24.71</td>
<td>128</td>
<td>.170</td>
<td>3.19</td>
<td>.1931</td>
<td>.361</td>
</tr>
<tr>
<td>16</td>
<td>121.4</td>
<td>7.10</td>
<td>24.71</td>
<td>130</td>
<td>.203</td>
<td>3.48</td>
<td>.1900</td>
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</tr>
<tr>
<td>214</td>
<td>49.8</td>
<td>3.71</td>
<td>19.86</td>
<td>86</td>
<td>.399</td>
<td>5.35</td>
<td>.231</td>
<td>1.72</td>
</tr>
<tr>
<td>214 + Ca</td>
<td>73.5</td>
<td>4.78</td>
<td>23.11</td>
<td>120</td>
<td>.314</td>
<td>4.83</td>
<td>.1925</td>
<td>1.63</td>
</tr>
<tr>
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<td>5.98</td>
<td>22.26</td>
<td>118</td>
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<td>.1888</td>
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<td>2.97</td>
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<td>102</td>
<td>.679</td>
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<td>.1855</td>
<td>3.73</td>
</tr>
<tr>
<td>400</td>
<td>45.6</td>
<td>3.32</td>
<td>18.21</td>
<td>89</td>
<td>.399</td>
<td>5.48</td>
<td>.2047</td>
<td>1.95</td>
</tr>
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<td>- 7.3</td>
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<td>20.28</td>
<td>107</td>
<td>2.76</td>
<td>8.97</td>
<td>.1895</td>
<td>14.65</td>
</tr>
<tr>
<td>379</td>
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<td>2.21</td>
<td>14.65</td>
<td>92</td>
<td>3.48</td>
<td>6.63</td>
<td>.1593</td>
<td>21.90</td>
</tr>
<tr>
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<td>10.1</td>
<td>2.98</td>
<td>35.59</td>
<td>109</td>
<td>3.52</td>
<td>11.94</td>
<td>.3265</td>
<td>10.79</td>
</tr>
</tbody>
</table>
Table XIII

Relationship of Incisor Growth to the Total Gains in Body Weight and Body Length of Female Rats on Different Diets

<table>
<thead>
<tr>
<th>Diet No.</th>
<th>Average total gain in body weight (gms)</th>
<th>Average total gain in body length (cms)</th>
<th>Average total incisor eruption (mm)</th>
<th>Fresh weight of incisors (mgs)</th>
<th>Incisor eruption to total gain in body weight</th>
<th>Incisor eruption to total gain in body length</th>
</tr>
</thead>
<tbody>
<tr>
<td>13</td>
<td>98.4</td>
<td>5.98</td>
<td>24.19</td>
<td>131</td>
<td>0.273</td>
<td>4.04</td>
</tr>
<tr>
<td>13 m.s.</td>
<td>103.9</td>
<td>6.19</td>
<td>23.91</td>
<td>156</td>
<td>0.230</td>
<td>3.86</td>
</tr>
<tr>
<td>16</td>
<td>90.3</td>
<td>5.27</td>
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Fig. 1. Correlations Between Body Weight, Skeletal Development, and Incisor Growth of Male Rats on Different Diets
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