CHANGES IN THE NUTRITIONAL STATUS SINCE 1929
AS SHOWN BY SEVERAL REPRESENTATIVE GROUPS IN THE TEMPERATE ZONE

by

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In the following pages an attempt is made to correlate some material presented from several localities in the temperate zone in regard to changes in the nutritional status of the population during the period 1929 to 1933.

I

Groups from which the Studies were Made.

The groups from which these studies were made include all ages and subjects living under varied economic and environmental situations.

The reports of Dr. H. T. Price[1] are based on studies made of the nutritional status of 988 children of Erie Pennsylvania, 812 children of a Pittsburgh section, reports of the public schools of Pittsburgh over a five year period (1929-33) and reports from the files of the Public Health Nursing Association of Pittsburgh regarding infants from one to two years of age.

Dr. Allen M. Kerr[2] reports the changes in the nutritional rating of Pittsburgh school children as determined by comparing the results of 500,000 individual weighings done before 1928 with 300,000 weighings done since that time.

Dr. Dr. P. C. Jeans[3] reports the incidence of night blindness in children admitted to Iowa City Children's Hospital.

The changes in the mortality rates and factors influencing these changes, (over the periods 1922-27 and 1927-32) of 710 members of a slum population who were moved

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1. Henry T. Price, M.D., Nutrition in Children.
2. A. M. Kerr, M.D., Effect of Economic Crisis on Nutrition of School Children.
3. P. C. Jeans, M.D., Vitamin Deficiency in Childhood.

into modern dwellings in 1927, have been reported by Dr. G. C. M. M'Gonigle.[4]

Esther Jacobs[5] has reported the increase in the number of diagnoses of malnutrition made on all patients who came to the Community Health Center Diagnostic Clinics over the five year period 1928 through 1933. The age range of this group was from one month to fifty years.

Dr. J. G. Slevin[6] investigated the relationship of malnutrition in school children to physical defects and the social status. For these studies 1,140 children of five to fifteen years from three public schools in Detroit, Michigan were examined during the school year of 1931-32.

The results of feeding 350 children on diets varying in amounts of phosphorus, calcium, and vitamin D over a year period (1932-33) have been published by Dr. Brown and Dr. Tisdall of Toronto, Canada.[7]

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4. G. C. M. M'Gonigle, M.D., Poverty, Nutrition and Public Health.
5. Esther Jacobs, Is Malnutrition Increasing?
6. John G. Slevin, M.D., Some Physical and Social Aspects of Malnutrition in School Children.
7. A. Brown, M.D., and F. Tisdall, M.D., Effect of Vitamins and Inorganic Elements on Growth and Resistance to Disease in Children.

II

A Brief Review of the Vitamins and Inorganic Elements Essential to Life.

Although the advanced clinical entities of the vitamin deficiency diseases will not be considered, a brief review of the pathologic changes and symptoms attributed to deficiency of the various vitamins and inorganic elements, and of the sources of these substances is made in an effort to establish the relationship between a vitaminosis and the criteria used to classify the nutritional status of the subjects.

Vitamin A is considered necessary for the "health" of epithelial tissues and tissues of epithelial origin.[1] It is fat soluble and heat stable.[2] The known food sources for this vitamin are butter fat, egg yolk, carrots, spinach and fish oil.[3]

The absence of vitamin A leads to a real metaplasia (and not an inanation) of the cuboidal, cylindrical and transitional cells into squamous, keratinizing type of cells. The process first affects the deepest cells of a layer and gradually extends to the surface. When this surface is exposed to the outside (as, for example, tissues in the respiratory tract and in the conjunctiva) infection occurs.[4]

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1. D. L. Tilderanut, Eye Manifestations of Avitaminosis.
2. George D. Burr, Role of Vitamins in Nutrition.
3. A. Brown and F. Tisdall, Effect of Vitamins and Inorganic Elements on Growth and Resistance to Disease in Children.
4. D. L. Tilderanut, Op. cit.

The changes in the conjunctiva are said to begin with disappearance of the goblet cells followed by metaplasia of the entire cellular structure making it similar to epidermis.[5] The symptoms which accompany these changes are:

1. Stickiness of the lids.
2. The conjunctiva becomes dry and white. Glistening (Bitot's) spots appear and are often covered by a whitish foam.
3. Tears do not seem to moisten the spots and the surface has a "greased" appearance. There may be a mild brownish-grey pigmentation of the conjunctiva at this time.
4. The conjunctiva becomes thick and lies on the eyeball in folds.
5. Later the process advances in the cornea which becomes cloudy.
6. Softening and breaking down of the corneal tissue follows.
7. The ulceration progresses until there is rupture of the cornea, infection and its usual sequellae.

There is no demonstrable retinal change accompanying vitamin A deficiency. However, the retina has been shown (by Holm) to store large quantities of vitamin A. This is thought to be stored in the lipiodal cell content at the periphery of the retina. With vitamin A deficiency,

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5. D.L. Tilderanut, Op. cit.

an early diminution of visual acuity in dim light occurs.[6]

Skin changes occurring with, and usually preceded by, xerophthalmia have been described by Dr. S. E. Sweitzer.[7] There is hyperplasia and hyperkeratinization of the epithelium of the hair follicles and epidermis with associated metaplasia of the epithelium of the sweat glands to that of the keratinizing type, degeneration of the glandular structures of the skin leading to infection.

Clinically, the skin of the upper and lower extremities, shoulders, lower abdomen, and to a less extent that of the chest, back and buttocks becomes rough and dry, usually several weeks preceding the conjunctival symptoms. Later, spinous papules appear at the sites of the hair follicles. Occasionally, associated with the above changes, there was absence of visible sweating, the articular folds were covered with dry, delicate scales, and the skin was darker than normal. The follicular papules varied in size and each had a hard, keratotic plug at the apex projecting above the surface of the lesions as a spinous process. There are usually large numbers of somedomes on the face associated with a very dry skin. The eruption is abundant and symmetrical.[8]

The other systems in which there are demonstrable epithelial changes ascribed to vitamin A deficiency are the

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6. Ibid.
7. S. E. Sweitzer, M.D., Eye Manifestations of Avitaminosis.
8. Ibid.

respiratory system, gastro-intestinal system and the genito-urinary tract. Symptoms believed to result from vitamin A deficiency changes in structures of the above systems are anorexia, hematuria, respiratory disorders, such as coryza, sinus infections and bronchitis, slowness of movement and mental sluggishness[9], urolithiasis, emaciation, and possibly neoplasms.[10]

Vitamin B is known as the anti-beri-beri or anti-neuritic vitamin. It is water soluble and heat labile especially in alkalies.[11] The sources of this vitamin are yeast, wheat germ, milk, liver, egg yolk, whole grain cereals, fresh fruit, leafy vegetables and glandular organs.[12]

To the deficiency of vitamin B the following changes have been ascribed: atony in the intestinal tract leading to constipation and digestive disturbances, polyneuritis, paralysis, cardiac weakness, emaciation, growth failure, anemia, edema and impaired lactation.

> "A moderate deficiency of vitamin B cannot be adequately detected because any well-balanced diet of common and natural foods supplies adequate vitamin B, and there is no need for special vitamin B preparations for every day use."[13]

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9. Ibid.
10. George D. Burr, Op. cit.
11. A. Brown and F. Tisdall, Op. cit.
12. George D. Burr, Op. cit.
13. P. C. Jeans, Vitamin Deficiency in Childhood.

Vitamin C is the anto-scorbutic vitamin (hexauronic acid) and it is water soluble and heat labile especially in the presence of oxygen.[14] Its sources are citrus fruits such as oranges and lemons, turnips,[15] tomatoes, cabbages and other leafy vegetables.[16]

The deficiency of vitamin C is manifested by an increased capillary fragility (ascribed to a capillary degeneration[17]) leading to ulcerations and hemorrhages in the mouth, subcutaneous hemorrhages and anemia, impairment of the teeth, lowered vitality, growth failure, skeletal, visceral and muscular degeneration and secondary infections.[18,19] Mild forms of vitamin C deficiency are best detected by Gothlin's test for capillary fragility. Frank scurvy is considered to be rare in this country.[20]

Vitamin D, the anti-rachitic vitamin is fat soluble and heat stable. Its edible sources are cod liver oil, irradiated ergosterol, irradiated foods, summer milk, butter and egg yolks. Exposure to ultra violet light is the most valuable method of preventing vitamin D deficiency.

Vitamin D deficiency is very common in the temperate zone.[21] It produces disturbances of phosphorous-calcium

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14. George D. Burr, Op. cit.
15. E. W. McHenry, An Economic Anticorbutic Settlement.
16. A. Brown, and F. Tisdall, Op. cit.
17. George D. Burr, Op. cit.
18, 19. Ibid., S. E. Sweitzer, Op. cit.
20. P. C. Jeans, Op. cit.
21. Ibid.

metabolism. It is known that a decreased retention of calcium and phosphorous accompanies vitamin D deficiency. The ash content of bones taken from rachitic animals is decreased to 30% as compared with a normal of 55 to 60%. This is due to a deficient calcification of all osseous structures.

The clinical defects produced by vitamin D deficiency are dental caries, weakness, ricketts, osteo-malacia and predisposition to infection.

Vitamin E is known as the anti-sterility vitamin. It is fat soluble and heat stable. It is present in wheat germ oil, whole grain cereals, green vegetables, muscle and glandular organs. To its deficiencies have been attributed sterility caused by gonadal degeneration in the male and imperfect placentation in the female, embryonic death and resorption, muscular dystrophy and paralysis in the young.[22]

Vitamin G (B_2 or PP), the anti-pellagra vitamin, is water soluble and heat stable. It is present in yeast, lean meat, milk, eggs, wheat germ and green vegetables.[23] Its deficiency results in dermal, neural and digestive lesions.

The skin lesions are bilateral and symmetrical, appearing on the backs of the hands, wrists, some part of the face and neck and on the feet and ankles in persons who do not customarily wear shoes. The location of the eruption is determined by the areas exposed to sunlight.

The eruption begins as a macular erythema which gradually fuses. There may be a moderate swelling at this

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22. George D. Burr, Op. cit.
23. Ibid.

time. After a few days the lesions deepen in color to a reddish-brown hue. Seven to ten days later desquamation occurs, producing a rough scaly surface. The more severe cases may be complicated by bullous lesions with erythematous bases. Ecchymoses may occur. Secondary infection is apt to follow the formation of bullae. This may progress to severe ulceration with edema.

After the eruption has disappeared the skin is thickened, crusted and may, in some cases, be more deeply pigmented, while in other cases the pigmentation of the involved areas is lessened. The extreme terminal stage, after many exacerbations have ensued, is atrophy of the skin leaving a thin, parchment-like scar.

The tongue is usually red, smooth and swollen over the tip and margins. Atrophic patches in the mucous membrane throughout the gastro-intestinal tract and a papillomatous condition at the cardiac end of the stomach have been described.

Degeneration (of a varying degree) may occur in the ganglion cells of the brain and spinal cord and in the posterior and poster-lateral columns of the spinal cord in the cervical and lumbar regions.

The clinical manifestations of vitamin G deficiency include the dermatitis described above, anorexia, diarrhea (in about 25% of the cases) and mental apathy which may progress to euphoric dementia. [24]

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24. S. E. Sweitzer, Op. cit.

The inorganic elements essential to life are sodium, potassium, calcium, magnesium, phosphorous, chlorine, sulphur, iodine, iron and copper. Of these, three must be given special attention because deficiency of them in a careless dietary is common.

Calcium is present in milk and leafy vegetables. The child's diet must have an adequate amount of calcium which is one grain a day for a child of ten years, and necessitates 24 ounces of milk a day. If all milk is eliminated from the child's diet the other foods supply but 0.17 grams of calcium a day. 25

Iron is present in egg yolk, liver, spinach and other green vegetables.

The iodine content of food and water is low in central North America and iodized salt should be used to secure an added source of this element.

These substances above mentioned are all essential to life and an animal which is deprived of any of them will lose weight and die.[26]

The caloric value and fat-carbohydrate-protein ratio of an adequate diet has been given by Dr. H. T. Price as follows:

1. Adult ration..... per day

Minimum of	2500 calories
" "	20% fats
Maximum of	65% carbohydrates
Minimum of	12% protein
" "	2 tablespoons of canned tomatoes
" "	1 teaspoonful of cod liver oil

25. A. Brown and F. Tisdall, Op. cit.
26. Ibid.

2. Adolescent rations (8 to 15 years) per day

Minimum of	2100 calories
" "	33% fats
Maximum of	52% carbohydrates
Minimum of	15% proteins
" of	two teaspoons of tomato juice or equivalent
" of	one teaspoonful of cod liver oil

Whole grain cereal, fresh vegetable including potatoes.

3. Children's ratio (2 to 8 years) per day

Minimum of	1400 calories
" "	30% fats
Maximum of	52% carbohydrates
Minimum of	18% protein
" of	three teaspoons of cod liver oil
" of	two tablespoons of canned tomato juice or equivalent

50% of all cereals to be whole grain, one fresh vegetable a day, and eggs should be included in the diet.

III

Criteria Used by Various Examiners.

Dr. Allen M. Kerr[1] of Pittsburgh used the following height-age-weight index for classifying the group of school children he studied. The study was based on changes in group placement rather than on changes in the individual weight of the child.

Group 1. 5% or more above average ... very good

2. Average to 5% above average weight ... good

3. Average to 81/3% below average weight ... fair

4. 8½% below average to 142/7% below average ... poor

5. More than 142/7% below average weight ... very poor

The averages were taken from the figures given in the Baldwin-Woods tables. The total change from one weight group to another was used as an index of the changes in nutritional status of the entire school population.

Dr. J. G. Slevin[2] used the following clinical criteria for classification of his subjects as to their

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1. A. M. Kerr, M.D., Effect of Economic Crisis on Nutrition of School Children.
2. J. G. Slevin, M.D., Some Physical and Social Aspects of Malnutrition in School Children.

nutritional status:

Lack of muscular turgor

Pallor

Lack of adipose tissue

Signs of circulatory stagnation

Underweight

Those children 2 to 15% below standard weight but without the above clinical criteria were classified as underweight. The children 10% or more underweight having two or more of the above clinical signs were considered to be malnourished. All other children examined were referred to as the average group.

The infants reported by Dr. H. T. Price of Pittsburgh[3] were judged by failure to gain adequately in weight, or by loss of weight.

The slum population reported by G.C.M. M'Gonigle[4] was studied in regard to changes in mean crude death rate and the dietary factors operating to influence an increase in the death rate.

The clients coming into the Community Health Center Diagnostic Clinics of Philadelphia[5] were graded on nutritional status by:

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3. H. T. Price, M.D., Nutrition in Children.
4. G. C. M. M'Gonigle, Poverty, Nutrition and Public Health.
5. Esther Jacobs, Is Malnutrition Increasing?

Height and weight

Condition of skin and tissues

Mucous membranes and musculature

General physical condition [6]

The conditions of the teeth, general tone of the gingivae and buccal mucous membranes were used as criteria for showing effects of diets varied in phosphorus, calcium and vitamin D in the studies made by Dr. Brown and Dr. Tisdall of Toronto Canada.[7]

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6. Ibid.
7. A. Brown and F. Tisdall, Effect of Vitamins and Inorganic Elements on Growth and Resistance to Disease in Children.

IV

Prevalence of Malnutrition Found in Various Localities

In January 1933 988 children of Erie, Pennsylvania were examined over a twelve day period. The chief findings were;

Dental Caries	532
Diseased Tonsils	469
Enlarged Cervical Glands	265
Mouth Breathing	205
Cardiac Abnormalities	85
Enlarged Thyroid	147
Underweight	132
Markedly Malnourished	75

Re-examination of this group the following school year showed 619 gained an average height of 14/5 inches

521 gained an average weight of 14/5 pounds

65 failed to gain weight

35 lost weight

Changes in the nutritional status of the school children of Pittsburgh noted for the year 1932-33 as compared with 1927-28 were;

	Poor and very poor nutrition	Fair nutrition	Good nutrition
1927-28	20%	28%	52%
1932-33	39%	24%	37%

The report from the Public Nursing Association of Pittsburgh shows that the average weight gained by children from one to two years over periods of six months each was 3 pounds 9 ounces in 1931, 3 pounds 2½ ounces in 1932, and 2 pounds 12½ ounces in 1933.[1]

According to the report given by Dr. A. M. Kerr, 7% of all of the school children examined during the 5 year period before 1927 had shown a very poor nutritional status. During the 5 years following 1927 the percentage of school children having very poor nutritional status (more than 142/7 % below average weight) was,

1927-28	8%
1928-29	9.1%
1929-30	10%
1930-31	10.5%
1931-32	12.6%

In 1928 492% of the school children were at or above normal weight. The school year 1931 to 1932, 37% remained in this classification. Correspondingly, 20% of the school children were in the poor and very poor classifications in the school year 1927-28, and in the school year 1931-32 this figure had increased to 34.2%.

Statistics of the nutritional status of the pre-school children, as given by Dr. Kerr, showed that in June

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1. Henry T. Price, M.D., Nutrition in Children.

1932 44% were of normal nutrition and 80.7% had fair nutrition or better.

Dr. Slevin of Detroit, Michigan[2] states that there were five million children having some degree of malnutrition in 1930. He reports the findings from the examination of 1140 children during the year of 1932 and gives the following data;

122 children (10.7%) were malnourished (See criteria on page 12)

85 children (7%) were underweight

Demonstrable physical defects were found in

70% of the average group

89% of the undernourished

75% of the underweight

The physical defects, in order of frequency, were;

Anemia - 60% more frequent in malnourished group.

Diseased Tonsils - 15% more frequent in malnourished group.

Dental Caries -

Adenoids - 10% more frequent in malnourished group.

Heart Disease - 3% more frequent in malnourished grap.

Thyroid Enlargement.

Dr. Slevin reports that of the 207 under average children, 92 had anorexia and that 72 of those with anorexia

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2. J. G. Slevin,M.D., Some Physical and Social Aspects of Malnutrition in School Children.

were malbourished, 17% of these latter had no other demonstrable physical defects.

The previous school records of 77 of the 122 malnourished children showed that 64 (83%) had demonstrable physical defects during their school careers, 20 (26%) were not malnourished before 1931, 24(32%) were malnourished in 1930-31, 15 (19%) were malnourished in 1929 and 18 (23%) gave history of nutritional deficiency antedating 1929.

Dr. P. C. Jeans in his article published in 1933[3] reports that 20% of the children admitted to the Iowa City Hospital have some degree of hemeralopia, and that 80 to 90% of the children have dental caries.

The slum populations studied by Dr. G.C.M. M'Gonigle[4] showed an increase in the mean crude death rate of the population that was moved into modern dwellings as compared with its mean crude death rate before removal and as compared with the death rate of the population in the unaltered slum areas and as compared with the entire community.

Changes in the mean crude death rate from 1927-32 (as compared with 1922-27)

England and Wales	6.6% higher
Stockton-on-Tees	1.9% lower
Mt. Pleasant (alter slum pop.)	44.6% higher
Riverside area(unaltered slum pop.)	2.9% lower

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3. P. C. Jeans, M. D., Vitamin Deficiency in Childhood.
4. G.C.M. M'Gonigle, M.D., Poverty, Nutrition and Public Health.

The net increase of deaths in the altered slum population over the five year period of 1927-32 was;

	Net increase
0-10 years	9.2%
10-55 years	18.4%
55-75 years	16.9%

Over this same five year period the death rate of the unaltered slum population decreased from 26.1% (1923-27) to 22.78% (1927-32).

Of the certified causes of death there were increases in measles, cancer, heart disease, bronchitis, pneumonia, diarrhea, nephritis, puerperal sepsis.

During the five year period of 1928-32 there was a marked increase in malnutrition among the clients coming into the Community Health Center Diagnostic Clinics of Philadelphia.[5]

1928 - 2130 clients - 23% (490) showed malnutrition

1932 - 1688 clients - 36.5%(616) showed malnutrition

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5. Esther Jacobs, Is Malnutrition Increasing?

V

Factors Contributing to Malnutrition.

Dr. A. H. Parmelee[1] cites the following physical factors which contribute to malnutrition. Defects mechanically interfering with food intake include carious teeth, infected teeth and gums, improper occlusion, enlarged tonsils, nasal obstructions such as hypertrophied adenoids, nasal polyps and deviated septum. The physical defects which contribute to impaired appetite are purulent or muco-purulent discharges in the naso-pharynx and factors causing fatigue such as obstructions to nasal breathing, defective eyesight and defective locomotion.

Dr. Slevin[2] quotes the statistics offered by Dr. Kaiser that in observations done on 4,400 children over a four year period, half of whom had had tonsillectomies, more than 10% of the non-tonsillectomized were malnourished. Dr. Slevin also quotes the statement of Dr. Perlman that in a group of 400 malnourished children focal infection was the commonest contributing agent.

According to Dr. Max Einhorn[3] psychogenic anorexia and organic lesions interfering with food intake are important factors in malnutrition of adults. Persons suffer-

1. A.H. Parmelee, M.D., Physical Defects Interfering with Nutrition.
2. J. G. Slevin, M.D., Some Physical and Social Aspects of Ma lnutrition in School Children.
3. M. Einhorn, M.D., Diet and Subnutrition.

ing from this psychogenic anorexia can be classed, usually in one of three main groups.[4 & 5] There are those who have had temporary functional disturbances necessitating a diminution in food intake which has been continued by the individual and developed into a habit. Often these individuals exclude essential foods from their diets over long periods of time.

In another group are those who have always been below normal weight and who have unconsciously balanced their food intake and energy output so that the body weight remains below normal.[6] In the third group are those whose loss of appetite has been preceded by a definite cycle of events: worry, overwork, fatigue, loss of weight and anorexia.

Dr. C. A. Aldrich[7] states that the error of advocating large amounts of carbohydrate to increase the caloric intake has been responsible for an increased incidence and severity of ricketts. He also states that the inconsiderate forcing of large amounts of food upon a child in order to increase the caloric intake has often produced a resistance to food and consequent anorexia.

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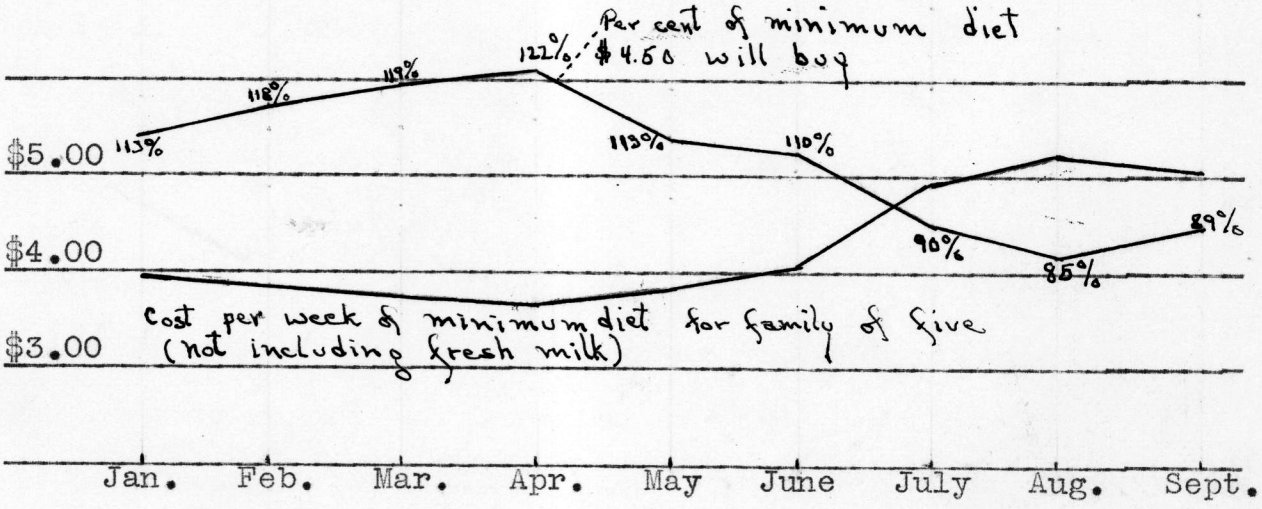
4 & 5. E.P. Rolli and M.S. Brown, Dietary Treatment of Undernutrition.
6. M. Einhorn, M.D., Op. cit.
6. E.P. Rolli and M. S. Brown, Op. cit.
7. C. A. Aldrich, M.D., Causes for Poor Nutrition other than those Purely Physical.

Economic and environmental factors contributing to malnutrition have been of grave importance during the five year period following 1929. In 1933 more than one tenth of the population of Pennsylvania was on relief and more than one half of this population was comprised of children.[8] During this same year the purchasing power of the dollar decreased as indicated on the following graph:



The industrial communities were affected most severely by this unemployment. The importance of this situation is stressed by the statement made by Dr. Aldrich[9] that 30% more food must be purchased by promiscuous buying to secure the same nutritional value as can be obtained by scientific selection.

Dr. Kerr[10] mentions the fact that unemployment effects were beginning to be felt in 1928 and that such

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8. H. T. Price, M.D., Nutrition in Children.
9. C. A. Aldrich, M.D., Op. cit.
10. A. H. Kerr, M.D., Effect of Economic Crisis on Nutrition of School Children.

effects occur first in that group which is "always on the edge of poverty, is first to be dismissed from the payroll and is first to feel the effects of underfeeding." He substantiates this statement with the observation that the shift to lower nutritive levels occurred first in schools in the poorer sections of Pittsburgh. He also mentions that high school children suffered more than elementary children of the same district because the parents could not provide money for the noonday lunch and there was not only a decreased number of children patronizing the school cafeterias but also a decreased outlay for each child.

In the slum population, studied by M'Gonigle, the hygiene and sanitation of the dwellings had been vastly improved and, after a careful survey, the only adverse factor which could be discovered to be at least partially responsible for the increased death rates following removal to modern dwellings was a slight increase in weekly rent.

Owing to this increase in rent the food expenditures were cut down to 36.1% of the income of the unemployed members of this group. The deficiency in protien allotment (100 grams per person per day being used as the normal average) amounted to slightly more than 40% for each family. In the years 1927-32, 40% of the total male population (14 to 65 years) of Stockton-on-Tees was unemployed. Only once, since 1922, has the unemployment of this community fallen below 20%. [11]

11. G.C.M. M'Gonigle, Poverty, Nutrition and Public Health.

Dr. Slevin[12] reports that 74 of the 207 malnourished children in his series did not get a sufficient amount of food and that these 74 children came from families where the wage-earner was unemployed. He also states that of 1,420 families included in the three schools, 253 heads of families were unemployed. 27% of these unemployed parents had malnourished children. He also states that one third of the malnutrition in the group which he studied was closely related to economic depression.

Dr. Alldrich[13] states that poverty directly contributes to malnutrition because of its consequent evils of overcrowding, lack of sanitation, poor heating, lack of sunshine, fatigue, worry and overwork.

12.J. G.Slevin, M.D., Op. cit.,
13.C. A. Alldrich, M.D., Op. cit.

VI

Suggested Therapeutic Measures to Combat Malnutrition

The first consideration in the treatment of malnutrition is prophylaxis. Esther Jacobs has suggested periodic health examinations done with special attention to early signs of subnutrition. The eradication of physical defects and foci of infection which contribute to fatigue and anorexia has been urged by Dr. Parmelee, Dr. Price, Dr. Kerr, Dr. Slevin and Miss Jacobs.

Dr. Price recommends the use of low cost, scientifically selected, balanced rations in community relief markets supplemented by dietetic education programs.

Dr. Kerr advocates the slight addition of 600 calories a day to the diets of school children by giving them milk on each school day.

Dr. Brown and Dr. Tisdall[1] state that the average person suffers from insufficient amounts of minerals, vitamins and protiens due to the ease with which purified carbohydrates are obtained. They suggest that diets be built around milk to supply calcium and protein, meat to supply proteins, eggs to supply proteins, vitamins and iron, vegetables and fruits to supply minerals and vitamins.

Dr. McHenry[2] recommends the use of turnip juice as a source of vitamin C to replace orange or tomato juice

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1. A. Brown and F. Tisdall, M.D., Effect of Vitamins and Inorganic Elements on Growth and Resistance to Disease.
2. E. W. McHenry, M.D., An Economic Anticarbutic Settlement.

in relief diets. He states that an ounce of turnip juice is as high in vitamin C as an ounce of lemon juice (280 vitamin C units in one ounce). The much lower cost of turnips makes them more easily available to people in straitened economic circumstances.

Insulin treatment of cases of marked subnutrition has been favorably reported by Dr. Barron.[3] This requires hospitalization of the patient. The patient is given twenty to thirty units of insulin before each meal (an average of seventy units a day). With this a high caloric diet of 3500 to 4000 calories a day is given in three meals and three lunches.

Many beneficial effects have been claimed for the use of insulin. Among them are the acceleration of the secretory center of gastric, pancreatic and biliary juices by hypoglycemia, increased utilization of fats and carbohydrates through a heightened potency of pancreatic enzymes, and a greater secretion of bile. This leads to a more complete digestion, absorption and assimilation of food. It is claimed to produce increased gastric motility and tonicity with a prolonged hunger period. Of the thirty two cases treated by Dr. Barron, 75% showed an immediate weight gain and those who did not gain weight felt a definite tonic effect. However, most of the patients lost their weight three to six months after treatment although Dr. Barron states that many patients have kept the added weight for years.

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3. M. Barron, M.D., Treatment of Malnutrition in Adults.

Dietary treatment of subnutrition can be accomplished without insulin by gradually increasing the food intake. [4 &5] This obviates the danger of hypoglycemia, dispenses with the bother of taking insulin and does not necessitate hospitalization. The patient must be convinced of the necessity of taking more food. A mild stomachic may be used to stimulate the appetite and a gastric sedative often aids in overcoming the early satiety experienced by the patient when the diet is first instituted. Once the patient has managed to eat the entire diet, he becomes accustomed to the increased bulk and the appetite returns. Of six cases thus treated by Dr. Ralli and Dr. Brown[6], the weight gain was from eleven to twenty-three pounds in six week's time, with an average gain of seventeen pounds. In this series, insulin was not found to increase the weight gain above the above-mentioned figure. This seems the more satisfactory method of treatment for the average case of subnutrition.

The factor of organic disease must be ruled out in all cases of subnutrition. The irradication of focal infections with special attention to tonsillectomies and adinectomies is urged. [7,8,9].

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4 and 5. E.P. Ralli and M. S. Brown, M.D., Dietary Treatment of Undernutrition.
M. Einhorn, M.D., Diet and Subnutrition.
6. E.P. Ralli and M.S. Brown, M.D., Op. cit.
7,8, and 9. A.H. Parmelee, Physical Defects Interfering with Nutrition.
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