

# DIET PREVENTS POLIO

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REFERENCES

## INTRODUCTION

Medical science has been able to prevent diphtheria, smallpox, and typhoid fever by vaccination. We have been able to reduce the mortality from tuberculosis by pasteurization of milk and improved general hygiene. Typhoid fever and cholera have been prevented by chlorination of water and proper sewage disposal. Humans have been protected against these diseases because they have been artificially immunized and because their environment has been controlled to some extent, and *not because the body's inherent ability to prevent infection has been built up.*

It is far more important to enable the human to maintain his natural powers against infection than it is to keep bacteria and viruses away from his door, especially since we do not have vaccines available for many common infections. I do not believe that Nature over intended that man should suffer sickness, crippling, and death through infections. From my observations and studies in human nutrition I have become convinced that Nature originally did endow us with natural powers of protection and that we have lost these powers through errors in diet. A polio vaccine may some day be a reality. Until then, it is necessary that we fortify ourselves with good food.

The purpose of this book is to show how one may fortify the body through diet and thus prevent infection. The method, which I offer as a protection against polio, is based on the concept that the maintenance of normal blood sugar levels will prevent the invasion of the body's tissues by the poliovirus and thereby prevent the infection. I shall offer evidence to show that a lowering of the blood sugar to abnormally low levels is the most important factor of susceptibility to infection. Since the maintenance of normal blood sugar levels is fundamentally dependent on the food we eat, the method of prevention becomes a matter of diet. Thus the human has the power of preventing this crippling disease because he has control over what he eats. Knowledge is power. I state without reserve that we have this knowledge.

The concept that low blood sugar is a factor of susceptibility to polio also helps us to understand why polio is more prevalent during the summer, why polio often follows physical exertion, fatigue, and swimming in cold water. It also helps us to understand why rest helps prevent infection. I shall present evidence to show that these predisposing factors may cause low blood sugar and thus favor polio.

I shall not discuss the geographic distribution of polio, nor the fact that polio is worse during certain years, nor the effects of rainfall on the incidence of polio because we do not have the answers to these problems. I regard them as unimportant when it comes to preventing polio. Even if we knew the answers to these questions, it is doubtful that we could do anything about them. The human is always at the mercy of the weather. Weather is part of his natural environment. The human organism has, however, been endowed physiologically with the means to adjust to changes in his environment and to withstand its rigors and hazards.

Intensive research during the past twelve years on the relationship between diet and susceptibility to infection, not only in polio but also in common respiratory infections and tuberculosis, has convinced me that the human organism can protect itself against infection virtually completely by proper nutrition.

That there is a direct relationship between nutritional standards and susceptibility to infection is shown by the great increase in the incidence of tuberculosis in countries that experience a reduction in nutritional standards during wartime. Knud Faber, a Danish physician, in an analysis of factors responsible for the increase in tuberculosis mortality in Denmark, Sweden, and England during World War I, concluded that the reduced consumption of meat and fish was the

most important nutritional factor. Dr. Faber began his study without any preconceived ideas as to what were the responsible factors for this increased tuberculosis mortality. He writes:

“In Denmark the increase in tuberculosis mortality took place in 1916-1917 simultaneously with a greatly reduced consumption of meat and fish, and the decrease of the mortality coincided with a greatly increased consumption of these foods. If we take beef, veal, and fish alone, the fall in consumption was extraordinary during 1915-1916. The total calories consumed were not diminished because there was sufficient bread and flour.” He continues:

“We see the same thing in Sweden, though to a less marked degree. The rise in tuberculosis mortality was recorded in 1914-1916, and in those years the consumption of bread and flour rose, whereas that of meat decreased. After 1916 we see a steady and continuous fall in tuberculosis mortality, and at the same time flour foods fell off while the consumption of meat and fish rose rapidly. It may be added too, that in England, a rise in tuberculosis mortality coincided with a lower consumption of meat and butter and an increased consumption of flour foods.”

There has been a similar rise in tuberculosis mortality in practically all belligerent countries in Europe during and since World War II and for exactly similar reasons, namely, a great reduction in the consumption of protein foods, such as, meat, fish, and eggs, along with an increased consumption of the more available and cheaper starchy foodstuffs.

## **THE BLOOD SUGAR AND ITS REGULATION**

Sugar is an essential constituent of the blood. It is also called “blood glucose.” In the fasting state, as in the morning before breakfast, the blood sugar concentration is between 80 and 90 mg. per 100 cc. of blood. Even after several days of fasting the blood sugar will be maintained around this level in a well-nourished individual. It is essential to normal health that the blood sugar be maintained at this level, and that it should not fall below this level for periods longer than an hour. After a meal containing sugar the blood sugar rises at once, usually reaches a concentration of 120 to 140 mg. after 1 1/2 to 2 hours, and then gradually falls during the third and fourth hours to the previous fasting level. (Fig. 1.) In some individuals the blood sugar may reach 180 mg. and higher after a meal containing sugar, and, accompanying this high level, sugar may be found in the urine.

This abnormally high concentration of blood sugar is called in medicine Hyperglycemia (Hyper—excessive; Glycemia—blood sugar). The opposite state, an abnormally low blood sugar concentration, is called Hypoglycemia (Hypo—less; Glycemia— blood sugar). Fig. 2 shows the hypoglycemic type of curve, and Fig. 3 shows the hyperglycemic type of curve.

The maintenance of the blood sugar at normal levels is brought about by an efficient regulatory mechanism. The main organs in this mechanism are the liver, the autonomic nervous system, and certain glands of internal secretion called endocrine glands. The liver is at the center of this mechanism and serves as a storehouse of the blood sugar supply. The foods we eat are digested and broken down into simpler chemicals which are absorbed from the gastrointestinal tract, carried to the liver, and there built up (synthesized) by the liver cells into a complex compound called glycogen.

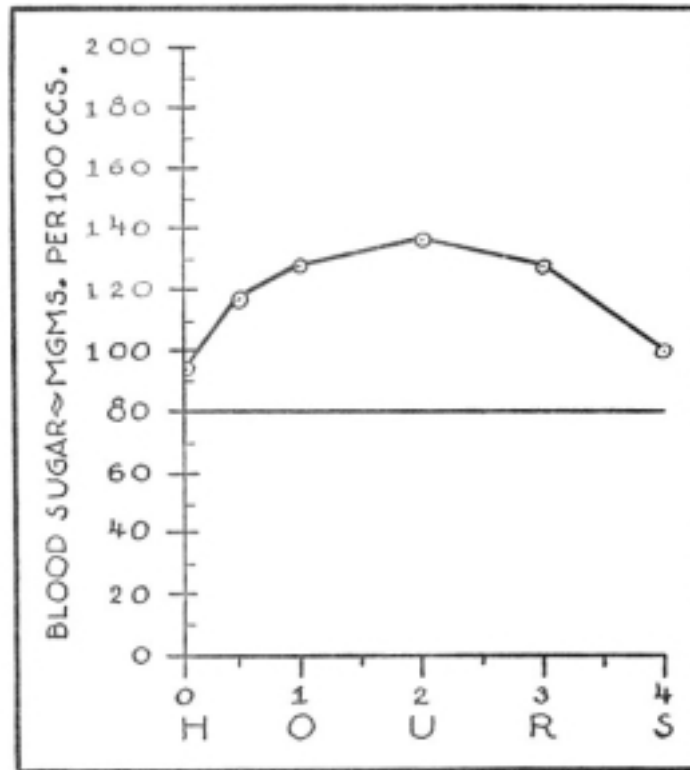
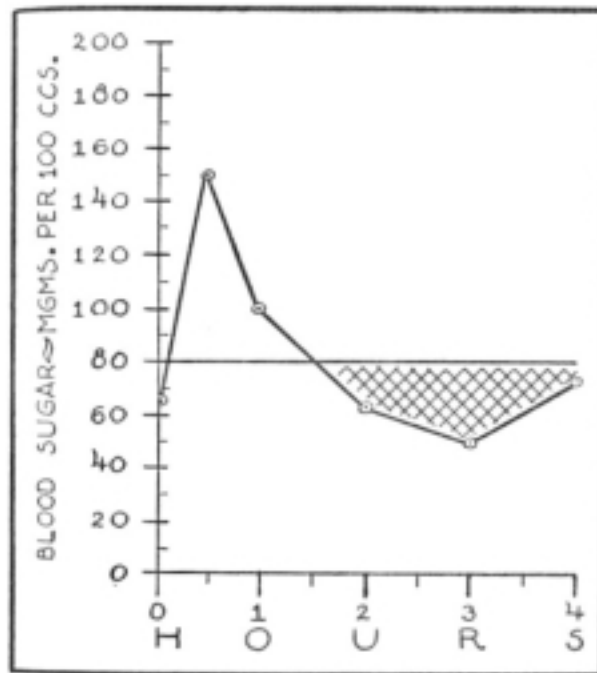


FIG. 1

*Normal glucose tolerance curve. Subject drank a solution containing 100 grams of glucose. The blood sugar level rises gradually to a peak during the first two hours and then falls during the third and fourth hours to the previous fasting level. All blood sugar values lie well above the 80 mg. baseline.*

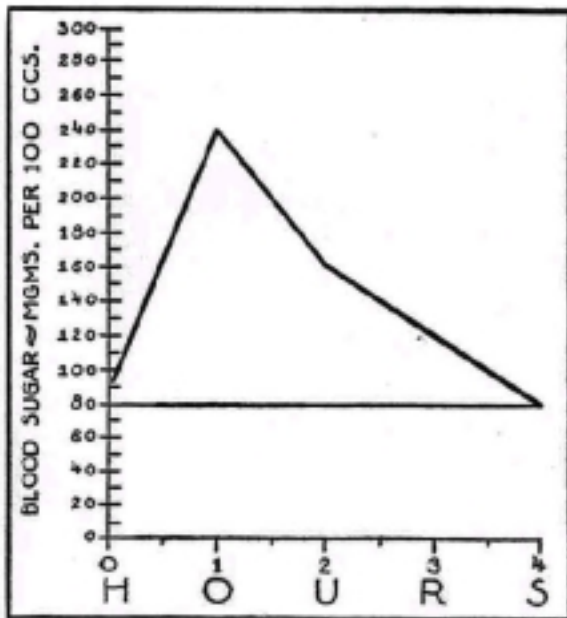
In the fasting state glycogen is constantly being broken down in the liver cells into a simpler chemical called glucose or sugar which is then liberated into the blood stream at such a rate as to maintain the blood glucose level at around 80 mg. per 100 cc. This breakdown of liver glycogen is controlled chiefly by nerves of the autonomic system, which consists of two divisions, the sympathetic and the parasympathetic.



**FIG. 2**

*Low blood sugar (hypoglycemic) type of curve obtained after 100 grams of glucose. Note the sharp rise to 150 mg. at ½ hour followed by a sharp fall to abnormally low levels after 1½ hours. Note that the blood sugar remained at abnormally low levels for about 2½ hours. Hatched area indicates the extent and duration of the low blood sugar period.*

The autonomic nervous system is so called because it is not under direct voluntary control; it regulates involuntary functions such as digestion, intestinal movements, the heart rate, the blood pressure, breathing, and body temperature.



**FIG. 3**

*High blood sugar (hyperglycemic) type of curve obtained after 100 grams of glucose. Note sharp rise to around 250 mg. after 1 hour with rapid fall during the second and third hours. Individuals with this type of curve have symptoms resembling those seen in hypoglycemia and may also be susceptible to polio.*

In general, the action of the sympathetic division is to facilitate and hasten the breakdown of liver glycogen with subsequent elevation of the blood sugar level, and the action of the parasympathetic division is to favor the synthesis and storage of liver glycogen. The two divisions are thus essentially antagonistic, and, at any moment, the blood sugar level will be the resultant of these two opposing forces.

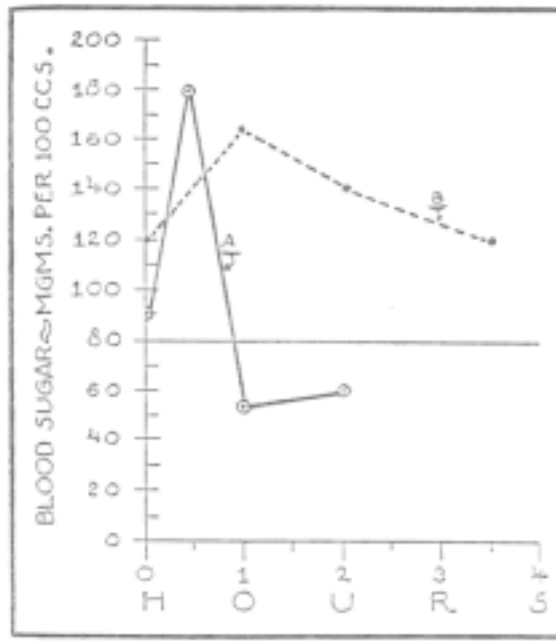


FIG. 4

*Glucose tolerance curves obtained in the monkey (curve A, after Jungeblut and Resnick) and in the rabbit (curve B, after du Vigneaud and Karr). Curve A, monkey: note wide fluctuation in blood sugar range with fall to low value of 50 mg. after one hour. Curve B, rabbit: note that all blood sugar values lie well above the 80 mg. baseline.*

As for the endocrine glands, the important ones in blood sugar regulation are the pancreas and the adrenals, both of which are under the control of the pituitary gland. The pancreas secretes the hormone insulin, which brings about the storage of glycogen and thus prevents the development of hyperglycemia. (Diabetes, a state of chronic hyperglycemia, is characterized by an insufficiency of insulin.) The adrenal glands produce a hormone called adrenalin whose action on the liver is to facilitate the breakdown of liver glycogen with subsequent elevation of the blood sugar. Adrenalin works in conjunction with the sympathetic nerves, and, since both produce the same effect, the two together are known as the adrenal-sympathetic system.

Since the heart of the blood sugar regulatory mechanism is the liver with its glycogen storage, and since glycogen is built up from the products of digestion, it becomes apparent that the amount of glycogen will depend on the nutrition of the individual. This is most important because it is the one factor that the individual can control.

In the fasting state glycogen is constantly being broken down into glucose because glucose is continually being removed from the blood stream since practically every organ in the body utilizes glucose as a source of energy. Glucose combines with oxygen, a process called oxidation, in order to provide the energy needed by the cells of the body to perform their particular functions. An important function of every cell is its ability to resist infection.

The body's cells are working every moment of life and a steady supply of glucose must be supplied by the blood. The brain and spinal cord (which make up the central nervous system), the heart, the muscles, utilize glucose practically exclusively for energy purposes in performing their functions. A constant delivery of glucose is needed at every moment of life in order that such vital organs be adequately provided with their source of energy. These organs are never in a resting state.

The constant demand of the tissues of the body for glucose necessitates a constant source of supply. Failure of this supply mechanism may result in death. If the supply of glucose fails the body will draw on its own tissues (muscle and fat) to get glucose. Under such circumstances these tissues break down yielding split-products, which are synthesized to glycogen in the liver and then paid out as glucose into the blood. If this state is prolonged, weight loss results with eventual emaciation. This mechanism explains the weight loss and emaciation that often accompany worry and anxiety states. In these states the individual loses his appetite, becomes malnourished, and draws on his own tissues to maintain blood sugar levels.

I wish to emphasize the fact that the blood sugar supply to the central nervous system is particularly important. The blood sugar must not only be supplied continuously, but must also be maintained at optimum level, around 80 mg. per 100cc. 'When the blood sugar falls below 80 mg. certain organs, especially the nervous system, will be embarrassed and signs and symptoms of disturbance in function make their appearance. The severity of the signs and symptoms will depend on how low the blood sugar falls.

When the blood sugar falls to 60 to 70 mg., symptoms are usually mild and may consist of slight headache, faintness, muscular weakness, hunger, irritability, and perhaps a feeling of nervousness or tension.

When the blood sugar falls to 50 to 60 mg., the symptoms are more marked and will consist of headache, dizziness, unsteady gait, faintness, weakness, fatigue, marked irritability, pallor, sweating, tremors, palpitation, and general nervousness.

If the blood sugar falls to 40 mg. or lower, unconsciousness usually occurs. This unconsciousness is exactly like that seen in the ordinary fainting spell and is usually accompanied by pallor, sweating, and feeble, rapid pulse. Recovery from such faints is usually spontaneous and is brought about by a rise in blood sugar, which occurs as a protection against further fall in blood sugar level. The abnormally low blood sugar level stimulates certain centers in the central nervous system, and from these centers impulses go out over the sympathetic nerves to the liver with a resultant increase in breakdown of liver glycogen and subsequent rise in blood sugar.

At the same time the adrenal glands respond to the fall in blood sugar by an increased output of adrenaline into the blood. The adrenaline acts upon the liver to step-up the breakdown of glycogen so that more glucose will be liberated into the blood. The effects of this adrenal-sympathetic stimulation with rise in blood sugar level brings about spontaneous recovery from the unconscious state. The cells of the brain now receive normal amounts of sugar and resume their normal function.

The hypodermic injection of adrenalin will also hasten recovery because the injected adrenalin has the same effect upon the liver as the body's adrenalin. The common practices of applying cold water to the head, the inhalation of vapors of spirits of ammonia, and vigorous rubbing of the skin, hasten recovery from simple faints because these measures can likewise cause stimulation of the adrenal-sympathetic system.

If the adrenal-sympathetic response is weak, recovery may be slow; if it is absent, recovery will not occur and death ensues. If the adrenal-sympathetic response is adequate, but the liver glycogen storage deficient, the blood sugar may not be restored to normal levels simply because there is not enough glycogen available. Such deficient storage will result from poor nutrition.



Summarizing, it is evident that the blood sugar must be maintained at 80 mg. for normal bodily function, and that symptoms appear if the blood sugar falls below this level. These symptoms will be mild, moderate, or severe, depending on how low the blood sugar falls. Spontaneous recovery from low blood sugar results from adrenal-sympathetic stimulation of the liver with subsequent rise in blood sugar. Recovery from low blood sugar will thus depend chiefly on (1) an adequate protective response by the adrenal-sympathetic mechanism and (2) the amount of glycogen stored in the liver, which, in turn, will depend on the nutritive habits of the individual.

## **LOW BLOOD SUGAR AND SUSCEPTIBILITY TO POLIO**

During my research I observed a large number of patients who had symptoms that were caused by low blood sugar. They complained of the symptoms previously described, namely, headache, dizziness, weakness, fatigue, abdominal pain, nervousness, palpitation, frequent sweats, and occasional fainting spells. Most of these patients were malnourished, which, physiologically, meant subnormal liver glycogen storage. Their diet was deficient in protein and consisted largely of the cheaper starchy foods.

I noted that these patients also had poor resistance to infections such as colds, sore throat, grippe, influenza, bronchitis, and pneumonia. By increasing the protein content of their diet and by reducing the sugar and starch content, they improved considerably. They became stronger, more vigorous and buoyant, and had fewer infections.

A few of these, patients had had polio in childhood. Observations of these patients over a long period of time led me to suspect that their susceptibility to infection was possibly due to their poor diet with its high sugar and starch content. Their increased resistance to infection with a better diet confirmed this suspicion. It then occurred to me that their susceptibility to polio could be explained on a similar dietary basis.

Specifically, I suspected that children and adults contracted polio because of low blood sugar brought on by a diet containing sugar and starch. I reasoned that the poliovirus was able to cross tissue barriers, reach the brain and spinal cord, invade the nerve cells, damage or destroy them and cause paralysis. And I further reasoned that if the blood sugar never fell below 80 mg. polio could never result. I suspected that during a polio epidemic only those children and adults who experienced periods of low blood sugar would contract the disease and that those individuals who were in actual contact with the virus but who maintained normal blood sugar levels would not contract the disease.

Thus, it remained to prove that low blood sugar could be a factor in susceptibility to polio. And, after this had been proved, the following questions had to be answered:

What causes low blood' sugar in humans? How can low blood sugar be prevented? The prevention of low blood sugar would thus mean the prevention of polio.

Before describing the experiments performed, I should like to make a preliminary summary and state without reserve that: (1) low blood sugar is a factor of susceptibility to polio; (2) low blood sugar occurs frequently in children and adults and is caused chiefly by a dietary error, namely, the consumption of sugar and starch; and (3) correction of this dietary error will prevent low blood sugar and thus prevent polio.

An experimental method to prove that low blood sugar was a factor of susceptibility to polio was readily available. In 1938, the only laboratory animal that could contract polio by experimental inoculation was the monkey. All other laboratory animals were completely resistant to the poliovirus. The rabbit is one of these resistant animals.

Without knowing the blood sugar range in the monkey and rabbit, it was suspected that the blood sugar in the monkey reached lower levels than in the rabbit. These suspicions were found to have a basis in fact through the investigations of Drs. Jungeblut and Resnick of Columbia University who studied blood sugar levels in monkeys, and through the investigations of Drs. du Vigneaud and Karr of Cornell University who studied blood sugar levels in rabbits. (Fig. 4).

In monkeys, blood sugar values as low as 50 mg. were observed, whereas in the rabbit, values below 100 mg. were never observed. In numerous determinations made on rabbits I have never obtained values below 100 mg. It was therefore concluded that the susceptibility of the monkey to the poliovirus was due to the fact that its blood sugar fell to subnormal values, and that the resistance of the rabbit might be associated with the fact that its blood sugar never fell below 100 mg, and that at this concentration cellular oxidation of glucose in the nervous system and other organs would be maintained at such a level as to enable the cells to protect themselves against invasion by the virus.

Physiologists have stated that the normal blood sugar level of 80 mg. holds true for all mammals.

The next step was to lower the blood sugar of the rabbit to subnormal values with insulin injections, and then inoculate the rabbit with poliovirus. This was done and *it was found that the rabbits became infected and developed the disease.*

The details of these experiments were published in the *American Journal of Pathology*, January, 1941. Some rabbits showed signs of infection 8 to 10 hours after inoculation. I wish to stress this short period of incubation in the rabbit because it demonstrates that polio can develop in a short period of time. This is important, as we shall learn later, when we discuss the onset of polio in humans within 24 hours after severe physical exertion.

The rabbit is also resistant to the dog distemper virus. One of the largest research laboratories has conducted much research with this virus and when I informed the members of the staff about my success in inoculating rabbits with poliovirus after lowering the blood sugar, they inoculated rabbits with the dog distemper virus after insulin and reported to me that they observed signs of infection in the rabbit for the first time. This corroborating experiment indicates that low blood sugar may cause susceptibility to many infections.

I was thus satisfied that low blood sugar was a factor of susceptibility to the poliovirus in monkeys, and that rabbits could be rendered susceptible after their blood sugar was lowered with insulin (Insulin, as you probably know, is the hormone which diabetics inject into themselves in order to keep their blood sugar within normal range. It is a quick-acting drug and can lower the blood sugar within an hour or so after injection). I concluded that the concept that low blood sugar created susceptibility to polio in both monkeys and rabbits could be applied to humans as well.

The next step in the solution of the polio problem was to find out the causes of low blood sugar in humans. Fortunately the answer to this problem was already at hand. It has been found that the consumption of sugar and starch and foods containing these substances were the chief cause of low blood sugar. The accompanying charts show clearly how the blood sugar is controlled by

what we eat. These graphs were obtained from patients whom I was studying and treating for low blood sugar (Figs. 5 & 6).

You will note that when these patients drank a solution of pure glucose they had a period of low blood sugar which began one to two hours after the glucose was taken and which lasted for one to two hours, and longer. This study of the blood sugar is called the “glucose tolerance test” and is employed for the detection of hypoglycemia or hyperglycemia. You will note that when they ate a meal containing sugar and starch they also had periods of low blood sugar which came on an hour or so later and which lasted for from one to two hours. The low blood sugar was more marked and lasted for a longer time after the glucose solution than after a meal containing starch.

It is an established fact that this paradoxical depressant effect on the blood sugar level is more readily exerted by sugar than it is by starches. I have observed these results in hundreds of cases and similar results have been obtained by other investigators.

It is a surprising paradox: the more sugar (and starch) you eat, the more likely you will develop low blood sugar. Drs. E. P. McCullagh and C. R. K. Johnston have shown how the glucose tolerance test is readily influenced by diet. Thus the second problem: What can cause low blood sugar in the human was solved.

The third problem, how can low blood sugar could be prevented, was the only one left and this, too, was readily solved. It had been found by other investigators that a meal consisting of protein, fat, and carbohydrates, but with no sugar or starch, never caused low blood sugar. The addition of sugar and starch to such a meal could readily produce low blood sugar. Figs. 5 and 6 show clearly how the blood sugar is maintained at 80 mg. and higher after a meal containing no sugar and little or no starch, whereas, in the same individual, a meal containing sugar and starch will cause low blood sugar.

Thus I arrived at a simple formula for preventing polio: eliminate from the diet sugar and foods containing sugar, and reduce the consumption of foods containing starch.

Since eating sugar and starch during a meal may cause low blood sugar after one to three hours, and since elimination of sugar and starch prevents low blood sugar, the invasion of the body by the poliovirus will be prevented by a diet containing no sugar and no starch. *Protection against polio would thus begin on the very day such a diet was started and protection would last just as long as such a diet was adhered to.* I have found that a diet completely free of sugar and starch and consisting of proteins, fats, and non-starchy vegetables, may be adhered to for years with beneficial effect and absolutely without harmful effect. There is no supporting evidence to indicate that sugar and starch are necessary for health or for energy purposes. The human is a carnivore and can thrive on protein and fat alone, if necessary.

The Eskimos thrive well on meat and fish, which yield only protein and fat, and polio is unheard of among them. American and European explorers in the Arctic regions have lived on meat and fish for as long as 18 months and have maintained perfect health all the time on such a diet. Vilhjalmur Stefansson, the Arctic explorer, has described his existence on such a diet in great detail. He states that he was in perfect health on such a diet, which consisted of protein and fat alone.

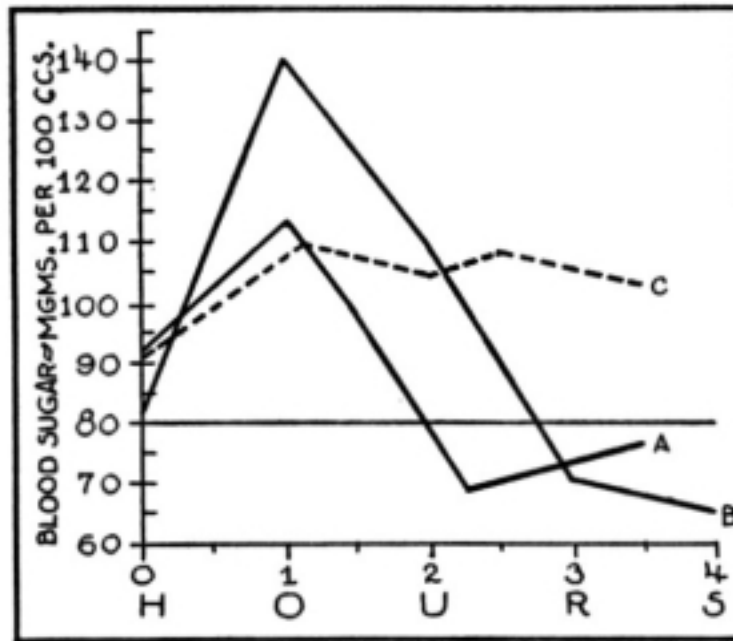
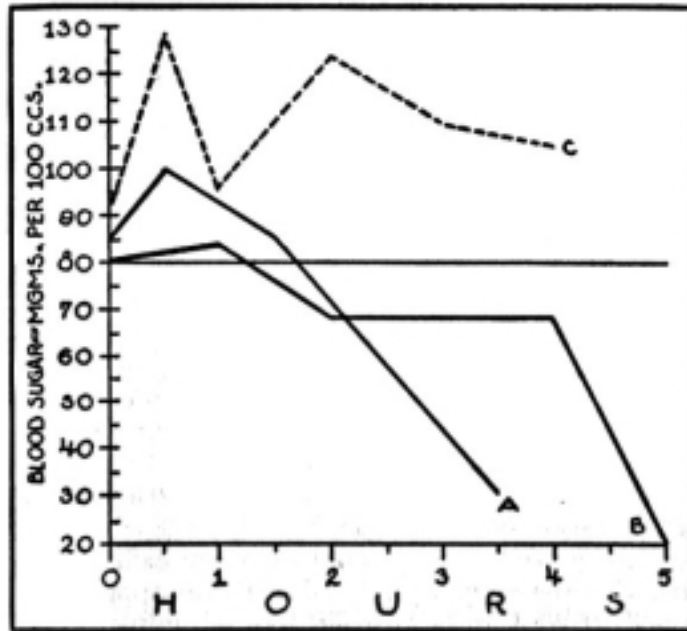


FIG. 5

The *effect* of various meals on the blood sugar in an individual with severe chronic low blood sugar. Curve A shows the course after 100 grams of glucose. Curve B shows the course after a high carbohydrate meal consisting of 1 orange, a bowl of oatmeal with cream and sugar, 2 rolls and butter, and a cup of sweetened coffee. Curve C shows the course after a low carbohydrate meal consisting of 1 orange, 2 eggs, 1 slice of bread and butter, a glass of milk to which 1 ounce of cream was added. About 2 hours after this meal the subject took a glass of milk.

Note that the greatest hypoglycemic effect is produced by the pure glucose solution (curve A), the blood sugar reaching subnormal levels sooner than after the high carbohydrate meal. Note that after a low carbohydrate meal the blood sugar levels are elevated and stabilized at normal levels. All curves obtained on same subject.

Eskimos who live on meat and fish are not susceptible to infectious diseases. They do become susceptible when they live amongst white men and eat the white man's diet with its sugar and starch. It is true that the Eskimo's fresh contact



**FIG. 6**

The effect of various meals on an individual with chronic low blood sugar of more severe degree than shown in Fig. 5. Curve A shows the course after 100 grams of glucose. Curve B shows the course after a high carbohydrate breakfast consisting of 1 orange, a bowl of oatmeal with cream and sugar, a piece of cake, and a cup of sweetened coffee. Curve C shows the course after a low carbohydrate meal consisting of 1 orange, 2 eggs, 1 slice of bread and butter, a glass of milk-cream mixture (4 oz. milk and 3 oz. cream).

A low carbohydrate meal elevates and stabilizes the blood sugar levels. This stabilizing effect is important because some of the symptoms of low blood sugar are due to rapid fall in blood sugar level which accompany wide fluctuations in blood sugar levels following the ingestion of sugar and starch.

with the white man exposes him to infectious diseases to which he (the Eskimo) has not had the opportunity to become immune. The presence of sugar and starch in the Eskimo's new diet is of greater significance. A U. S. public health officer stationed in Alaska has blamed this dietary factor for the great susceptibility of the Eskimo to tuberculosis.

## THE SIGNIFICANCE OF THE HYPERGLYCEMIC CURVE

There is a group of individuals who show high blood sugar with the glucose tolerance test. These individuals may have symptoms exactly like those found in individuals who show hypoglycemia with the glucose tolerance test and it is impossible to differentiate between the two except with the glucose tolerance test. Individuals with hyperglycemia are mild diabetic cases and sugar may be found in the urine after a meal containing sugar and starch, but not consistently, and the urine may be free of sugar after an all night fast. Fully developed cases of diabetes usually show urinary sugar consistently. Mild hyperglycemic patients are readily controlled by a low carbohydrate diet and usually have normal blood sugar levels on such a diet without the use of insulin, as shown in Fig. 7.

Mild hyperglycemia can cause symptoms similar to those found in hypoglycemic individuals and these symptoms are readily relieved by the same diet used in treating hypoglycemia, namely, a low-carbohydrate high-protein diet. Figs. 3, 5 and 6 readily show how the blood sugar is elevated to normal in hypoglycemic individuals; and Fig. 7 shows how the blood sugar is lowered to normal in hyperglycemic individuals. Severe hyperglycemic patients (frank diabetics) usually require insulin in addition to a diet. It is important to recognize mild hyperglycemic individuals because, if neglected, they may become frank diabetics.

The mild hyperglycemic individual has symptoms because of an inherent inability to utilize sugar and starch in the diet and because of the wide fluctuations in blood sugar levels. Fig. 7 shows how the blood sugar rises rapidly from a normal fasting level to an abnormally high level after one hour and then falls rapidly to around previous

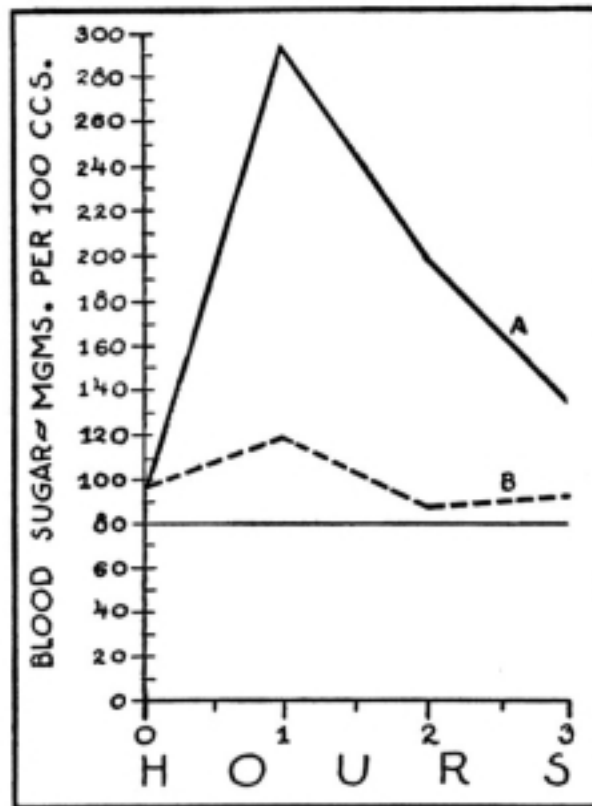


FIG. 7

This figure shows the stabilizing effect of a low carbohydrate meal on the blood sugar in a hyperglycemic individual. Curve A was obtained after 100 grams of glucose; note sharp rise to abnormally high level followed by a

rapid fall to previous fasting level which was within normal range. Symptoms exactly like those seen in hypoglycemia may occur during the period of rapid fall. Curve B was obtained after a low carbohydrate meal; note that blood sugar levels are within normal range and do not show wide fluctuations.

fasting level. Symptoms usually occur during the period of rapid fall. It has been found that symptoms depend not only on *how low* the blood sugar falls but also on the *rate of fall* in blood sugar level. Thus an individual with mild hyperglycemia may have symptoms while the blood sugar falls from 150 mg. to 100 mg. if the fall is rapid, despite the fact that the fall has occurred through normal range. This has been called "relative hypoglycemia." Symptoms of hyperglycemia are readily relieved by a low carbohydrate diet because the wide fluctuations are prevented and the blood sugar becomes stabilized.

Individuals with mild hyperglycemia, like untreated diabetics, are thus more susceptible to infection with polio because two factors are operating to produce susceptibility:

(1) The wide fluctuations in blood sugar levels with periods of rapid fall to relative hypoglycemic levels; and (2) the mild diabetic state which signifies an inherent inability to utilize sugar and starch in normal amounts. In individuals with hypoglycemia only one factor is operating to produce susceptibility, namely, the reduced utilization of sugar during the period of low blood sugar.

### **PHYSICAL EXERTION AND POLIO**

It has frequently been reported that attacks of polio have occurred after moderate to severe exertion. Football players, marathon runners, and other athletes have contracted polio so soon after drills, contests, and the like, as to suggest a causal relationship between the exertion and the onset of the disease. Medical journals contain such reports. Drs. Voss, Bremer, de Rudder, Petersen, Nase have made such reports. (5) Voss' case was that of a 17-year-old girl who contracted polio after a strenuous tennis match. Bremer's case was that of a student who contracted polio after a difficult mountain climb. Petersen and de Rudder observed two small epidemics following athletic contests. The athletes, who were between 13 and 19 years of age, were reported as being in good health and trained in athletics at the time of the contests. Eleven boys became ill with polio within a week after the contest. Another group of six boys became ill a few days after a second contest. Nase reports the case of a 26-year-old soldier who participated in a cross-country run. At the end of the run he was completely exhausted, began to vomit, and had a high temperature. He was admitted to the hospital the next morning. At this time he was unable to sit up in bed, was unable to move his legs. He died nine hours after admission with paralysis of the respiratory muscles. An autopsy revealed polio.

Football players in this country have contracted polio after games and after arduous drills. One southern team in 1948 had three players who developed polio during the same week. One of them died. The college authorities canceled the games scheduled for the rest of the season. I have questioned young polio patients during epidemics and have found that many of them took sick the day after an all day outing or picnic during which they played games, swam long and hard, and ate a good deal of sweets, ice cream, and soda pop

How does exertion bring on or predispose one to polio? I attribute this increased susceptibility to polio after exertion to low blood sugar. Physical exertion, when immoderate, is accompanied by strong and prolonged muscular contractions which are performed at the expense of the blood sugar and result in a severe drain on liver glycogen stores. The depletion of liver glycogen results

in a reduced output of glucose into the blood and the blood sugar concentration falls. Drs Levine, Gordon, and Derick of Boston found low blood sugar in six of nine marathon runners at the end of a long race. The blood sugar values of the six were 45, 47, 49, 50, 65, and 65 mg per 100 cc. All six finished the race in poor general condition and one of them was on the verge of collapse. Three who finished in good condition had normal blood sugar values, viz, 82, 82, and 89 mg (6)

Physical exertion, especially in summer, is accompanied by much sweating, thirst, and fatigue. The desire for cool sweet drinks is great and thirst is most pleasantly quenched by sweet drinks and ice cream. This ingestion of sugar may cause a fall in blood sugar. Thus the combined effect of physical exertion and the ingestion of sweets may greatly depress the blood sugar and thereby favor the onset of polio. Under such circumstances polio may have its onset within 24 hours.

This rapid onset may be explained by stating that the individual was in apparent good health at the beginning of the exertion and was a carrier of the virus, i.e., harbored the virus in the nose, throat, or gastrointestinal tract, like many other healthy individuals during an epidemic (It has been stated that for every frank case of polio during an epidemic there are about 200 healthy carriers of the virus). During the period of low blood sugar following the exertion, the virus crossed tissue barriers that harbored it, invaded the nervous system and attacked the nerve cells, with eventual paralysis. That polio may develop in such a short time after exertion is further suggested by the fact that I was able to infect rabbits after 8 to 10 hours when the rabbits' blood sugar was experimentally lowered with insulin.

Dr. W. J. McCormick of Toronto has also implicated sugar and starch as dietary factors responsible for polio (7). He believes that the ingestion of refined sugar and starch eventually cause a metabolic disturbance in the tissues of the nervous system with development of the disease. Dr. McCormick took careful dietary histories on many patients during polio epidemics. The following case report is an example:

Case 1). I. C., a boy eleven years of age, onset of illness at a beach resort on July 29. Prior to illness the patient had been in the habit of bicycling 18 miles daily to and from the beach, where, in addition to swimming, he played eight holes of golf daily. On the evening before the onset he went on a raft which he propelled by "poling" for half a mile, after which he was "very tired."

Usual diet prior to illness: *Breakfast*: cornflakes, or other toasted cereal, bacon, eggs, white bread toast, two or three slices with jam or marmalade, milk or cocoa, canned fruit juice. *Lunch*: white bread sandwiches, milk, cake, white buns. *Dinner*: boiled potatoes and meat, white bread and butter, cabbage salad, watermelon. *Bedtime*: white bread, jam, chocolate milk. *On the day before the onset the weather was very hot, and he had five drinks of pop, several chocolate bars, and a popsicle.*

My interpretation of the sequence of events in this case is as follows: After a long day of excessive physical exertion, i.e., bicycling, swimming, golfing, and poling a raft, the boy felt very tired at night. This tired feeling no doubt reflected a state of low blood sugar. Since the day was very hot he consumed much sugar in the form of soda-pop, ice cream, and candy — all this in addition to the sugar and starch contained in his three regular meals. The combined effects of the physical exertion and the heavy consumption of sugar could have produced a state of very low blood sugar lasting for several hours. During this state any poliovirus present on the surface of the membranes of the nose, or gastrointestinal tract could readily cross tissue barriers, invade the nervous system and cause the disease within 24 hours.

Thus, during epidemics of polio and even during non-epidemic periods, severe physical exertion should be avoided. This means complete avoidance of, or reduced participation in, such sports as



long distance running, distance running, tennis, and the like. Short periods of exercise are permissible. Swimming in very cold water should be avoided. Cold water chills the body, and brings about an increased oxidation of sugar to provide heat in order to maintain normal body temperature. This extra combustion of sugar occurs at the expense of the blood sugar and liver glycogen. Marked shivering after cold-water bathing is due to involuntary muscular contractions, the purpose of which is to produce extra heat.

Dr. Levinson found that monkeys forced to swim to the point of exhaustion in cold water developed more severe paralysis than did either those that remained in cages or those that were immersed in water at body temperature and protected from exercise and chilling (8).

The incidence of polio in any town or city during an epidemic is usually spotty. Cases occur here and there and it is usually impossible to establish that a polio victim had been in previous contact with another polio victim. In this respect, polio does not behave like measles and scarlet fever, which are highly contagious. Some researchers state that during an epidemic the virus is universally distributed in the epidemic area and that many healthy individuals harbor the virus on surface membranes of the nose, throat, and gastrointestinal tract. Such individuals are called carriers.

Most researchers also believe that there must be some inherent factor of susceptibility present in the bodies of those who fall victims of the disease, a factor which lowers the resistance of the body for a period of time and permits the virus to penetrate the surface membranes and invade the central nervous tissues. I maintain that the presence of low blood sugar is this factor of susceptibility. The lowering of the blood sugar lowers the gates and permits the virus to enter.

When I injected the poliovirus into rabbits whose blood sugar had been lowered by insulin, some of the rabbits showed signs of infection after only eight hours. This is an unusually short incubation period (The length of time between injection of the virus and the appearance of symptoms is called the incubation period). In monkeys, the incubation period is from 5 to 10 days, sometimes longer. Its exact duration in humans is not known and has been variously given as from 5 to 20 days. From my rabbit experiments I have concluded that, in the presence of low blood sugar, the virus is able to travel from surface membranes to the central nervous tissues in a matter of hours. The speed with which the virus travels will depend on the degree to which the blood sugar is lowered and on the duration of the low blood sugar. The lower the blood sugar and the longer it persists the greater the speed of invasion, the greater the multiplication of the virus, and the more severe the infection.

Those cases of polio that progress rapidly to extensive paralysis and death are cases that had prolonged low blood sugar at the time of invasion. Abortive polio cases are those that exhibit signs and symptoms of infection without developing gross signs of paralysis and which recover without evidence of paralysis. I would say that such cases had short periods of moderately low blood sugar and so only a small amount of virus reached the central nervous system, caused some inflammation but no paralysis.

In line with what has been previously stated about protective adrenal-sympathetic system, I would say abortive cases were able to recover from low blood quickly enough to prevent extensive invasion of the virus and subsequent injury to the cells of the nervous system. Between the two extremes of extensive paralytic and abortive cases, there are many degrees of involvement depending on: (1) how low the blood sugar falls, (2) how long the period of low sugar lasts, (3) the amount of glycogen stored in the liver, and (4) the effectiveness of the adrenal sympathetic response.

There are other physiological and immunological factors concerned with resistance to infection but I believe that they play a minor role at the onset of the infection. Potent immunological factors do not come into play until the infection is well established I regard the above listed factors as the most important, by far, since they play a leading role in prevention.

## THE DIET

The various foods we eat supply the body with three kinds of foodstuffs — proteins, fats, and carbohydrates. In general, animal foods (beef, pork, fish, poultry, dairy products, eggs) supply proteins and fats, with a small amount of carbohydrate in the form of glycogen. Grains, vegetables, and fruits supply mainly carbohydrates with varying amounts of protein and fat.

Protein constitutes by far the greater part of animal tissues and it is essential that human diet contain adequate amounts of good protein. Proteins are essential for growth, repair, replacement, the production of immune bodies needed to combat infection, the production of glandular substances, enzymes, etc. Life without protein is impossible. The importance of protein is suggested by the fact that the term is derived from the Greek and means “of first importance.”

All proteins are made up of carbon, hydrogen, oxygen, and nitrogen. Some contain, in addition, iron, phosphorus, or sulphur. Proteins are usually classified as high-grade proteins, which are those of high biological value; and low-grade proteins, which are those of low biological value. Proteins of high biological value are found in animal foods; those of low biological value are found in plant foods. High biological value proteins more nearly resemble the proteins of human tissues in chemical make-up than do the proteins of lower biological value. Obviously, it is more advantageous to eat animal protein than plant protein.

Carbohydrates consist of carbon, hydrogen, and oxygen, and are produced by all plant life from carbon dioxide and water under the influence of sunlight, in a process called photosynthesis. Under the general heading of carbohydrates we distinguish sugars, starches, and forms which are neither sugar nor starch since they do not react specifically to chemical tests for sugar and starch. This distinction is extremely important because there is evidence that only sugar starch can cause low blood sugar. *Carbohydrate such as, carrots, lettuce, tomatoes, cabbage, which contain no sugar and no starch do not cause low blood sugar.* This difference in behavior is due, no doubt, to a difference in chemical structure.

Fats consist of carbon, hydrogen, and oxygen, and chemically are made up of glycerol in combination with palmitic, and oleic acids. Fats are found in all animal foods, dairy products, eggs, some grains, vegetables, and fruits. Fats do not have any depressant effect on the blood and so there are no restrictions in their consumption. Fats may be eaten, therefore, in any quantity according to individual taste and tolerance. In humans, fats are less responsible for obesity than sugar and starch. In those persons with a tendency to obesity all excess sugar and starch is readily converted to fat and stored as such throughout body. I have observed individuals for years on a diet of protein, fat, and little or no sugar and starch, and maintained normal weights in spite of increased fat. Livestock are fattened for market chiefly by feeding them corn, grain, feeds, all of which contain large of starch.

### GENERAL DIET INSTRUCTIONS DURING AN EPIDEMIC

I advise that, during an epidemic, sugar and all foods containing sugar, be avoided, and that foods containing starch be consumed in reduced amounts. Those foods containing no sugar and

no starch may be eaten in unlimited quantity. Below are listed common foods containing either sugar or starch or both, and those containing no sugar and no starch. Since sugars are to be eliminated and starch consumption reduced, the calories needed for proper nutrition will be derived from an increased consumption of proteins, fats, and permitted carbohydrate foods.

*The following foods should be avoided:* sugar, soft drinks, ice cream, ices, sherbets, cakes, candies, cookies, wafers, pastries, pies, fruit juices, canned and preserved fruits, jams, jellies, marmalades, puddings, honey, syrups.

Coffee, tea, cocoa, lemonade, etc., may be sweetened with saccharin. Ice cream, ices, and custards may be prepared with saccharin. So-called “diabetic desserts” and food preparations may be used.

Nuts may be eaten in unlimited quantity except the starchy ones such as peanuts, cashews, chestnuts, and cocoanuts. These should be eaten sparingly.

The following foods should be eaten in reduced quantity because they contain starch:

beans, dried	tapioca	crackers
beans, lima	macaroni	cereals
corn	spaghetti	oat preparations
peas, dried split	vermicelli	rice preparations
potatoes, white or sweet	bread	rye preparations
yams	buns	corn preparations
lentils	biscuits	wheat
rice	rolls	preparations

The following fresh fruits should be eaten only in limited quantity because of their sugar content:

oranges	cantaloupe	pineapples	raspberries
grapefruit	watermelon	strawberries	grapes
lemons limes	apples	blueberries	cherries
honey dew	pears	blackberries	plums
melons	peaches		

Fresh fruits are permitted, but only one portion should be taken with a meal, i.e., one apple or one orange. The sugar in fruits may cause low blood sugar if they are eaten in excess. Fruit juices, canned fruits, dried fruits, preserved fruits, should be avoided. Fruits may be stewed without sugar. Apples may be baked without sugar. Tomato juice is allowed since it contains no natural sugar.

The following carbohydrate foods contain little or starch and may be eaten in unlimited quantity:

artichokes	Celery	mushrooms	spinach
asparagus	chard, swiss	okra	tomatoes
avocados	collards	onions	turnips
bamboo	cucumbers	parsley	watercress
shoots beans,	eggplant endive,	parsnips	horseradish
wax beans,	leaves greens,	peas, fresh	olives
string	beet	peppers	mustard
beans, soy	greens,	pumpkins	vinegar
beets, red	dandelion	radishes	capers
broccoli	greens,	rhubarb	mayonnaise
brussels-	turnip	rutabagas	
sprouts	leeks	sorrel	
cabbage	kale		
carrots	kohlrabi		
sprouts	lettuce		
cauliflower			

All animal foods may be eaten in unlimited quantity Such foods are: beef, pork, lamb, mutton, veal, poultry, fish These may be purchased fresh, canned, smoked, dried, etc. Eggs can be used freely whether fresh or dried. All dairy products may be eaten in unlimited quantities milk, buttermilk, fermented milk, butter, sweet and sour cream and all cheeses. Milk may be fresh, evaporated, or powdered.

#### SUGGESTED MEALS

##### *Breakfast*

fresh fruit or tomato juice

eggs, any style; two or more eggs, if desired bacon, ham, fish, cheese, or other meat

1 slice bread or 4 soda crackers

butter or oleomargarine

beverage; milk, coffee, tea, cocoa, all without sugar; saccharin may be substituted for sugar

This breakfast may be varied according to taste, capacity, and appetite. Some individuals are satisfied with fruit, eggs, bread and butter, coffee. Others wish to eat meat, fish, or cheese. There is no limitation on the amounts of permitted foods. If cereals are eaten at all, the quantity should be very small, and only saccharin should be used for sweetening.

##### *Lunch*

tomato juice, broth, or soup containing permitted vegetables, but no rice, noodles or other starch

meat, fish, or poultry (as much as desired) permitted vegetables, raw or cooked salad, with or without dressing

1 slice bread or 4 crackers

butter or oleomargarine

beverage; milk, coffee, tea, or cocoa, with saccharin, if desired

permitted fresh fruit

*or*

tomato juice, or broth, or soup

combination salad: egg, sardines, salmon, cold cuts, etc. cheese, sour cream

1 slice bread or 4 crackers butter or oleomargarine beverage, as above permitted fresh fruit

##### *Dinner*

oysters, shrimp, tomato juice, broth, soup meat, fish, poultry, omelet permitted vegetables salad

1 slice bread or 4 crackers butter or oleomargarine beverage, as above nuts, permitted fresh fruit, cheese

*or*

oysters, shrimp, tomato juice, broth, soup combination salad, cold cuts, cheese, eggs, sour cream  
permitted vegetables salad

1 slice bread or 4 crackers butter or oleomargarine beverage, as above nuts, permitted fresh fruit,  
cheese

Soybean muffins made from pure soy flour may be used in unlimited quantity as a substitute for  
bread and crackers. Natural gravies are preferred to gravies thickened with wheat flour.

#### Box LUNCH SUGGESTIONS

1. Cold cuts of meat or poultry; parsnips fried in butter; pickles; lettuce; tomato juice.
2. Hard-boiled eggs; sliced cold boiled carrots; cheese; walnuts; milk.
3. Cold roast lamb or fried eggs; fried in egg batter and soybean flour; season; slices of eggplant;  
piddes; milk or or beef ion, hot or cold.
4. Slices of tripe fried in batter of egg and soybean flour; or hard boiled egg; lettuce; celery stuffed  
with cheese; milk or tomato juice.
5. Vegetable salad (no potatoes) with dressing, carried in a jar; soybean muffins or soda crackers;  
salted nuts; clear milk or tomato juice.
6. Hot soup or broth carried in a thermos bottle; soda crackers soybean crackers or muffins; cheese; milk or  
tomato juice.
7. Summer squash dipped in beaten egg and fried in oil or butter and sprinkled with grated  
Romano or other cheese, well seasoned; cold roast chicken or turkey; milk or tomato juice  
consommé.
8. Left-over meat or poultry chopped fine with grated cheese, seasoned, and stuffed into green  
peppers; olives, radishes, milk or tomato juice.
9. Finely chopped cabbage fried slowly in bacon fat; add green peas and some tomato paste, a  
dash of paprika, strong cheese, to make an omelet; may be carried in a covered with waxed paper;  
olives; milk or tomato juice.
10. Chopped nuts mixed with a paste of hard boiled eggs to which a little oil is added, a pinch of chili  
powder; this may be rolled in tender cabbage leaves steamed for 30 minutes; may be eaten cold with  
pickles and cheese; milk or tomato juice or bouillon.
11. Cocktail frankfurters; pickled beets; olives; lettuce; chopped mixed nut salad; hot consomme.
12. Pickled peppers with thin slices of roast or boiled chicken or turkey, dusted with chili powder  
and rolled into lettuce leaves; milk or tomato juice.
13. Pickled eggs; sauerkraut; cheese; soybean crackers or soda crackers; milk, buttermilk, or  
tomato juice.
14. Left-over chicken, veal, roast pork, chopped fine and mixed together, well seasoned and  
pressed into patties, fried in oil or butter to make a sandwich surface for pimento cheese as a filler;  
milk or tomato juice.
15. Thin codfish cakes with a slice of ripe tomato placed between the cakes; tomato juice or milk.

16. Italian or other sausage fried in oil; celery; cheese; crackers; tomato juice.

17. Flank steak, laid out flat, covered with tomato paste, add finely chopped green peppers and celery, mashed green peas, season with chili powder, salt, pepper (garlic if desired), sprinkle with a few mint leaves and grated cheese, roll entire mass tightly, tie every two inches with string, steam on rack for 30 minutes and then bake until done in hot oven; cool. Can be carried as a meat roll for lunch.

The above suggestions may be altered to taste. Ripe tomatoes, green peppers, cucumbers or celery may be added to any lunch; pickled pigs' feet, cold boiled lobster meat, canned salmon or tuna may be added to lunch box.

### **THE BRIEF 1948 DIET CAMPAIGN**

I shall now relate an unusual experience for a physician who was personally responsible for a short, intensive, country-wide diet campaign against polio of one day's duration conducted through the co-operation of the press and radio. Before I embarked on this campaign, I realized that I would be adversely criticized by many, regardless of the soundness and truth of my ideas about preventing polio. Many regard me as a seeker of publicity. I was not in a position to gain financially from the publicity because I was engaged in private practice but was a full-time federal employee. Organized medicine frowns upon such steps as mine and I endorse this attitude on general principles.

Then why did I go ahead with the campaign? The simple truth is that I felt I had something concrete to offer in the prevention of polio. For several weeks before the diet story was publicized on August 4, 1948, I felt profoundly frustrated. I felt I had a means of preventing polio but could not reach the public to tell them about it. I hoped that publication of my experiments in the medical journal in 1941 would stir public health authorities to explore the possibilities implicit in my experimental results. However, nothing ever came of the publication. The data lay buried on the bookshelf. And so I decided to assume personal responsibility by informing the public. It was a bold step and required some courage because a professional career could have been jeopardized. Looking back, I am happy I took the step.

For several years after 1941 I was faced with the problem of testing the efficacy of the diet in the prevention of polio. Experimenting with the diet on humans presented obvious difficulties. An ideal test would be to put a particular area of a city on the diet during an epidemic and compare the results with those observed in the rest of the city that was not put on the diet. Such a test can be conducted only by a public health agency. My experimental work with rabbits had been published in January, 1941, in the *American Journal of Pathology*. Polio has been prevalent every year since then and it reached epidemic proportions in 1944 and 1946. In the summer of 1944 I wrote to a public health agency and suggested that the people in epidemic areas be advised to adhere to a sugarless and starchless diet for the duration of the epidemic. However, no action was taken.

The summer of 1948 presented an opportunity to test the diet. I was living in the city of Asheville, N. C., which had a population of 55,000. In May and June it was evident that the state of North Carolina was headed for a major polio epidemic. Asheville was having many cases for a city its size. The number of cases increased during July. State and local health officers, after meeting with the Buncombe County Medical Society, finally recommended strong restrictive

measures. Churches, theaters, swimming pools, parks, and recreation areas were closed. Public gatherings were discouraged. Children were not permitted to ride in buses. They were kept at home all day long, their activities confined to the home and front yard. Families that could do so, quit the state.

Asheville, a city that does a large tourist business, became a “ghost town.” There was no panic among the citizens. There was a subdued fear and an air of helplessness in spite of restrictive measures. Quarantine has never proved of value in the control of polio and some health authorities state that it is valueless. Besides being valueless, it depresses morale.

The epidemic showed no signs of abatement. August 1 arrived, with the worst weeks ahead. I decided to approach the Asheville newspapers. I told the editors of my animal experiments and researches in diet and outlined a method of control. The editors were impressed and convinced of my claim to authority in matters of nutrition. A feature writer, Mr. James K. Hutsell, was assigned to write the news story. This was intended only for the city of Asheville and Buncombe County. On August 4, 1948, the Asheville *Times*, an afternoon paper, carried a detailed article telling about my experience in nutrition research and experiments with rabbits and monkeys. The following diet suggestions were printed: (See Appendix for news story.)

1. Eliminate from the diet sugar and foods containing sugar, such as soft drinks, fruit juices (except tomato juice), ice cream, cakes, pastries, pies, candies, canned and preserved fruits. Saccharin may be substituted for sugar.
2. Cut down the consumption of starchy foods, such as bread, rolls, pancakes, potatoes, rice, corn, cereals, grits.
3. Substitute for starchy foods the following: tomatoes, string beans, cucumbers, greens, lettuce, turnips, carrots, beets, cabbage, onions, soybeans, cauliflower.
4. Do not eat fruits or melons more than once daily, and then only in small quantities.
5. Eat more protective foods, such as pork, eggs, beef, fish, poultry, milk, cream, cheese.
6. Eat three substantial meals a day. Avoid exertion and fatigue because they are known to be associated with low blood sugar. Avoid swimming in cold water. Rest as much as possible.
7. The diet should be followed until the polio danger is officially declared over by local health authorities.

The story printed the following two direct statements made by me:

“I am willing to state without reserve that such a diet, strictly observed, can build up in 24 hours time a resistance in the human body sufficiently strong to combat the disease. Of course, the diet must be followed throughout the period of the epidemic.”

One of the puzzling characteristics of polio has been its prevalence in warm weather. Many people cut down on protective foods such as meats, fish, and poultry because of a mistaken idea that a “light” diet is better for them in warm weather. And they increase the consumption of cooling foods and beverages, most of them heavily sweetened. It is this increase in consumption of sugar that produces a lowering of blood sugar and thereby a lowering of the body’s resistance to the poliovirus.

The Asheville *Times* meanwhile had released the story on the morning of August 4 to the Associated Press and United Press wire services. In the afternoon and evening of August 4 local

radio stations broadcast the diet suggestions at frequent intervals. Many afternoon and evening newspapers in distant cities printed AP or UP dispatches. Coast-to-coast newscasts carried the story all during the evening of August 4. On August 5, the Asheville *Citizen*, the morning paper, printed the story as it had appeared in the *Times* the day before. During August 5 newspapers throughout the country had either AP or UP stories. Some papers had the story on the front page. During August 5 radio stations throughout the country were still broadcasting the diet. Some newspapers did not carry the news until August 6. Thereafter several weekly publications had articles about the diet. The Asheville papers carried follow-up stories about the diet and polio for several days in order to impress the public because of the severity of the epidemic in this area. Thus a virtual alarm was carried across the country by press and radio.

The people of Asheville co-operated to an unexpected degree. They welcomed the opportunity to help themselves. The restrictive measures had been depressing. The confinement of children to home all summer was trying to all concerned. The statements about the diet were made in such strong, positive, and optimistic tone that they were readily taken up and adhered to. Since adults as well as children were being attacked by the virus, many grown-ups followed the diet.

One of the striking effects was the immediate improvement in morale. Parents felt that they were doing something constructive instead of just standing by and hoping the disease would not strike their homes. Store sales of sugar, candy, ice cream, cakes, soft drinks, and the like, dropped sharply and remained at low level for the rest of the summer. One southern producer of ice cream shipped one million fewer gallons of ice cream than usual, during the first week following the release of the diet story. Saccharin sales mounted sharply.

*The Results in Asheville.* Up until August 4, 1948, the city of Asheville had 55 cases of polio. If one assumes arbitrarily that the peak had been reached on that date, one could have expected about 55 cases during the decline until the end of the year, since in general during polio epidemics the number of cases following the peak is about equal to the number of cases preceding the peak. However, instead of 55 cases there were only 21 new cases in Asheville from August 4 to December 31.

Actually, however, in the southeastern United States, polio epidemic peaks are usually reached during early September. If the epidemic had been allowed to run its course without the diet story, there might have been around 75 cases in Asheville by the first week in September (a conservative estimate), with a similar number following the peak. Thus there could have been a total of 150 cases in Asheville for the entire season. Actually, there were 76 cases for the entire season, or about half the expected number.

The city of Asheville is located in Buncombe County. Both the city and the county have approximately the same population, about 55,000. The total number of cases for the county, excluding Asheville, was 102 cases for the entire year. If isolation had any effect, it should have been manifest in the county, which is largely rural with widely separated homes. Many of the county cases came from farms. Lack of contact was thus no protection against the disease. The city of Asheville was subjected to a heavier, effective, and more sustained propaganda regarding the protective possibilities of the diet than the county. Further, the epidemic and the diet were major topics of discussion in the city during August and September. The people of the city were more alert to the dangers of the epidemic and hence more receptive to any measure, which offered some degree of protection. The lack of close contact in the county rural areas could have created a false sense of security. These factors could readily account for the lower incidence in the city proper.

*The Results in the Country as a Whole.* The most striking effect on the course of the polio epidemic is noted in the number of cases reported from the entire country. This is clearly shown by the graph, Fig. 8, which shows the course of the 1946 and 1948 epidemics.

The graph shows that 1948 was running well ahead of 1946. The 1948 curve lies well above the 1946 curve up until the week ending July 31. For the next six weeks the 1948 curve falls below



the 1946 curve, a phenomenon which has never been observed to occur in previous polio epidemics. After the week ending July 31, the 1948 curve runs a highly irregular course. The break in the 1948 curve occurred during the week ending August 7 and coincided with the release of the diet story on August 4 and 5. This immediate effect need not be surprising since it was stated "without reserve" that strict adherence to the diet would afford protection within 24 hours, because the change in diet has an immediate effect on blood sugar levels.

The graph in Fig. 9 is also very striking. It shows an immediate effect on the number of cases per week for the entire country. From the week ending May 8 through the week ending July 31, the number of cases by which 1948 was leading 1946 was climbing, so that by the week ending July 17, there were 420 more cases in 1948 than for the corresponding week in 1946. For the week ending July 31, there were 304 more cases in 1948 than in the corresponding week in 1946. Then a sudden change occurred. For the next six weeks 1948 fell behind 1946 by 1581 cases. For the week ending

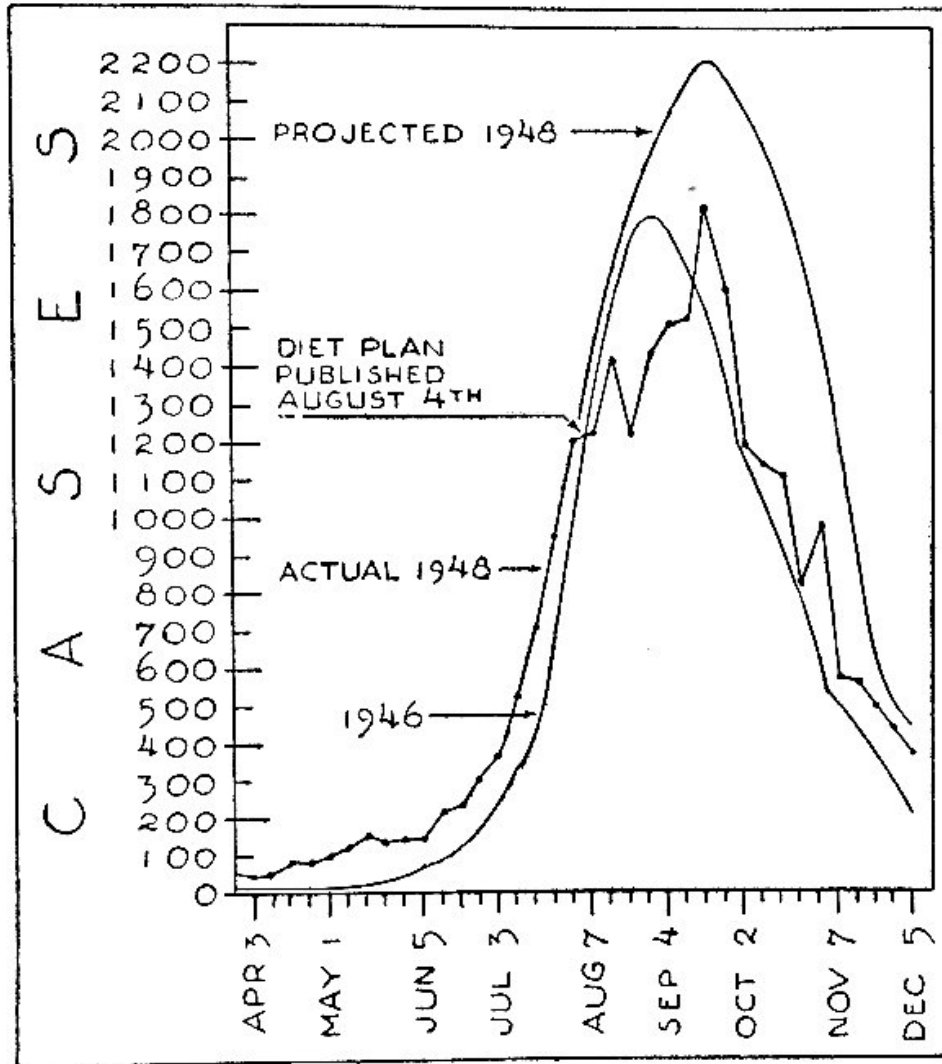


FIG. 8

This graph compares the 1946 and 1948 polio curves for the entire Note the smooth contour of the 1946 curve. The 1948 curve abruptly irregular coincident with the week ending August 7 when campaign began, and pursues an irregular course thereafter. The 1948 curve shows what the 1948 curve would have looked like if the epidemic had run its natural course without the diet campaign. The actual 1946 and 1948 curves were constructed from figures compiled by the U.S. Public Health Service.

August 7, there were 45 fewer cases in 1948 than in 1946. For the week ending August 14, there were 166 cases, and for the week ending August 21, there were 504 fewer cases. Since the diet campaign began on August 4 the diet was able to produce an effect during the

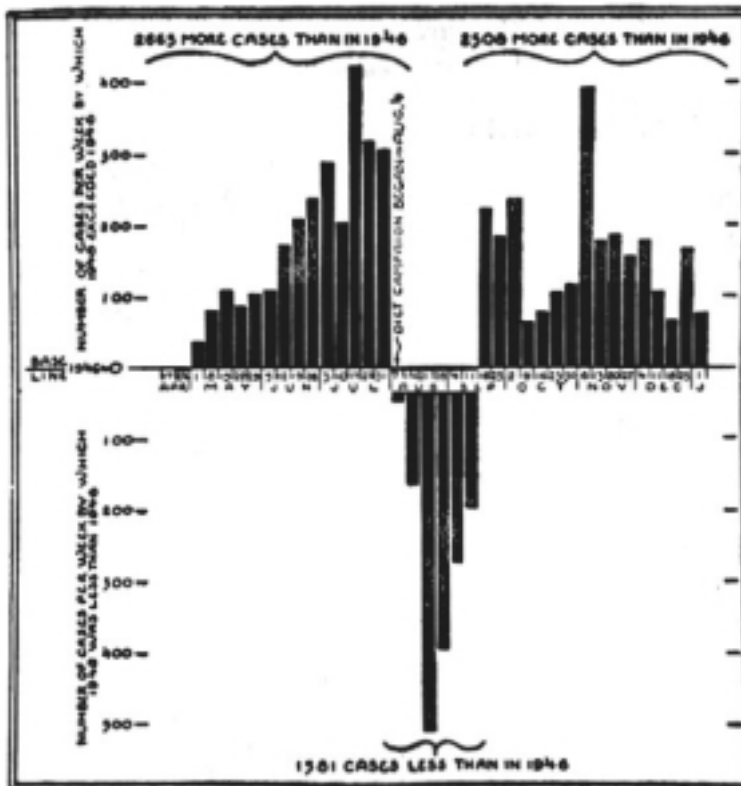


FIG.9

This graph shows the number of cases per week by which 1948 exceeded or fell behind the corresponding week in 1946. Up till the week ending August 7, 1948, the weekly number of cases in 1948 exceeded the weekly number of cases in 1946, as shown by the upright position of the solid bars on the graph.

One would have expected 1948 to maintain its lead over 1946 right through August and September when the peak is usually reached. However, 1948 suddenly falls behind 1946 for a period of six weeks, a period when polio epidemics are most severe and when one would have expected 1948 to exceed 1946 rather than fall behind 1946. Note that 1948 falls behind 1946 during the week of the diet campaign.

week ending August 7 on only three days: August 5, 6, and 7. The greater reduction in incidence during the weeks following can be explained by the fact that the diet was in effect every day of each of those weeks. Study of all previous epidemic years reveals that when one year has more cases than another year, the greater year always runs ahead of the lesser year. Thus, according to previous experience, 1948 should never have fallen behind 1946, especially during the peak weeks in August and September. From the week ending September 18 until the end of the 1948 is once more ahead of 1946, but not by as much as one would expect. If the diet campaign had been kept up all during the epidemic season, 1948 could have been kept below 1946 and thousands of cases could have prevented.

If we consider that 1948 is running ahead of 1946 or the average by 250 cases each week for the six weeks from June 26 to July 31, then the total for the six weeks August 7 to September 11, 1948, would have exceeded the total

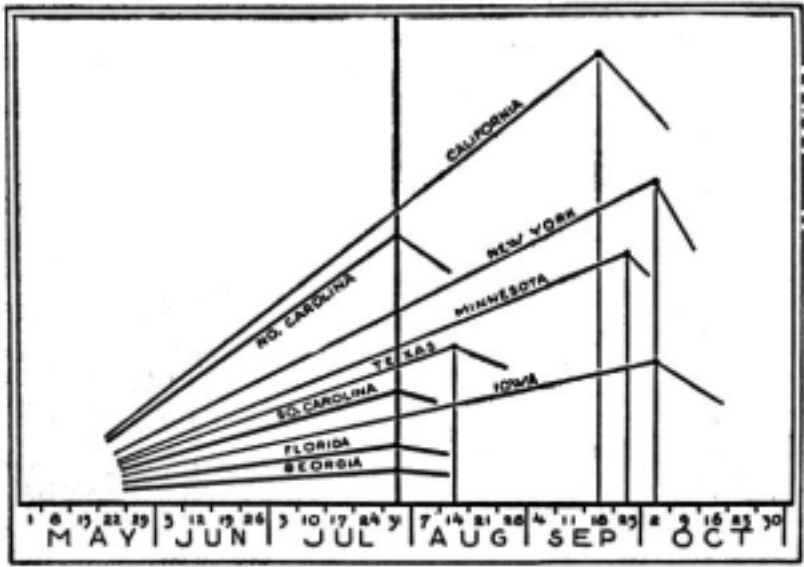


FIG. 10

This graph shows the dates when the epidemic peaks were reached for several states. Note that four southeastern states, North & South Carolina, Georgia, and Florida, reached their peaks during the week ending July 31, an unusually early date. The other states reached their peaks later. The North Carolina curve was closely paralleling the California curve and most likely headed for a peak date much later than July 31.

for the corresponding six weeks by 1,500 cases. Actually, the total for the six weeks August 7 to September 11, 1948, is 1,581 cases *fewer* than for the corresponding six weeks in 1946. Thus, one can estimate that the diet campaign prevented around 3,000 cases during the six week period August 7 to September 11, 1948. This is a conservative estimate.

TABLE NO.1

ACTUAL FIGURES FOR CORRESPONDING WEEKS IN 1948 AND 1946 SHOWING ABRUPT CHANGE IN NUMBER OF CASES

Weekending	1948	1946	Difference	(more in 1948)
May 22				
May 29	127	38	89	
June 5 June				
12 June 19	138	34	104	
June 26 July				
5 July 10	149	42	107	
July 17 July				
24 July 31	219	48	171	
Aug. 7 Aug.				
14 Aug. 21	253	45	206	
Aug. 28				
Sept. 4	309	74	235	
Sept. 11				
	362	78	284	
	513	311	202	
	717	297	420	
	982	668	314	
	1,215	911	304	(fewer in 1948)
	1,239	1,284	45	
	1,409	1,575	166	
	1,307	1,816	509	
	1,412	1,806	394	
	1,512	1,780	268	
	1,527	1,726	199	

These figures were obtained from Public Health Reports of the U. S. Public Health Service.

*The peak dates in various states.*

In past polio epidemics, peaks usually have been reached in late August or early September. The 1946 peak for the entire country was reached on September 25. Fig. 10 shows that in four southeastern states, the peak date was the same and occurred during the week ending July 31. These four states were: North and South Carolina, Georgia, and Florida. In five widely separated states the peaks occurred as follows: Texas, August 18; California, September 18; Minnesota, September 25; New York and Iowa, October 2.

The earlier peak date for the four southern states may be attributed to the fact that the press and radio publicity given the diet control plan was widespread and readily taken up by the citizens in this area because they were much concerned with the severe epidemic in Carolina. The fact that the diet plan originated in this area also served to create great interest in it. The later peak dates in the other states suggests that the publicity was not as concentrated as it was in the southeast. Observations by me indicate that practically every newspaper in the east published the story. In

New York City, for example, only one newspaper, *The New York Times*, carried the story. Southern editors were eager to present their readers a story of prime importance and of great interest.

In summary, I would say that the diet campaign 1948 prevented 3,000 cases during the six week August 7 to September 11. Although 1948 ran ahead of 1946 from the week ending September 18 until the end of the year, there were not as many cases in 1948 as one would have expected. I have estimated, conservatively that 1,600 cases were prevented during the period September 18 to December 31, 1948. Thus, I estimate that the campaign of 1948 prevented about 5,000 cases August 7 and December 31, 1948.

The country as a whole had more cases of polio in 1949 than in any previous year. There were 38,153 cases in 1949 against 23,418 in 1948. There was no diet campaign during 1949.

However, a study of the 1949 statistics yields some interesting data. I wish to call attention to a great decrease in polio cases in Asheville and in the state of North Carolina during 1949, in spite of the fact that 1949 for the country as a whole was far worse than 1948. In 1949, 39 states showed an increased number of cases over 1948. Ten states and the District of Columbia show a fall in the number of cases, and of these, North Carolina shows the most striking reduction. The following figures released by the National Office of Vital Statistics of the U. S. Public Health Service are submitted:

**Examples of states showing increases in 1949:**

Massachusetts	1,705	+181
New York	5,072	+1,321
New Jersey	1,350	+715
Michigan	2,568	+662
Texas	2,123	+1,611
Illinois	2,705	+1,013
Oklahoma	1,216	+339

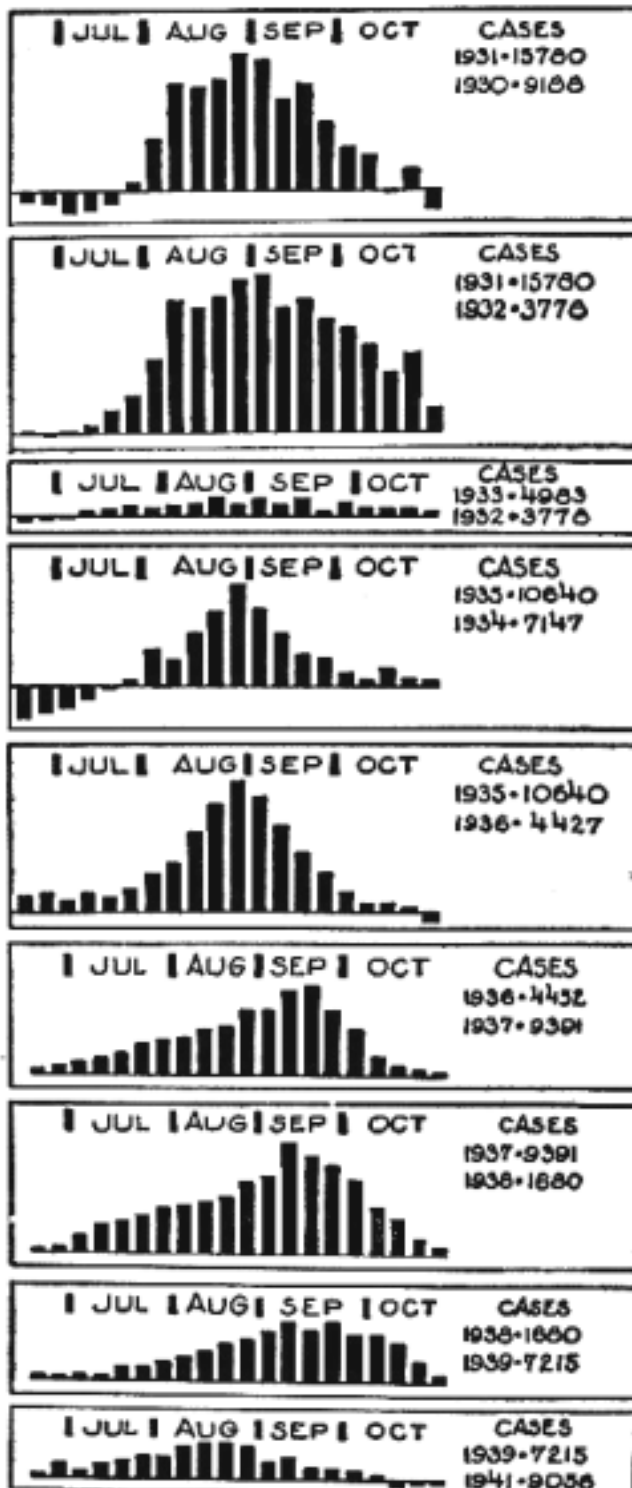
**Examples of states showing decreases in 1949:**

North Carolina	214	-2,402
South Carolina	98	-355
Georgia	201	-215
Florida	228	-245
California	2,156	-4,150

Total Cases for the United States: 38,153\* -23,418

\*In 1952, the worst year, 57,600 Americans contracted polio.

The most striking figure is the great reduction in number of cases in North Carolina. In 1949, there was about one-twelfth the number of cases in 1948. The city of Asheville had only 5 cases in 1949 as against 76 in 1948. A study of these figures also indicates that a heavy epidemic (as in 1948) is not followed necessarily by a reduced epidemic (as in 1949). There is no evidence to support contention that a city or state develops an immunity during a severe epidemic year. If this were true, then 1949 should never have been so much more severe than 1948. Those who believe that the reduced number of cases in Carolina and Asheville in 1949 was due to immunity acquired in 1948, will find difficulty in explaining the rise in number of cases in states that had many cases in 1948.



The fact that four of the ten states showing a number of cases in 1949 were located in the southeast part of the country may be attributed to the widespread publicity given the diet suggestions in that area. Because of the severity of the North Carolina epidemic, adjoining southern states had wide newspaper and radio publicity made available by the AP and UP wire services and practically every newspaper carried the story. Countrywide newspaper and radio publicity was also obtained but to a lesser extent. For example, in New York City, only one newspaper, *The New York Times*, carried the story.

I am convinced that the unique and striking reduction in the number of cases in 1949 for Asheville and Carolina is not accidental. Practically all citizens in Asheville with whom I have discussed the matter are of the same opinion. I have ample and reliable evidence obtained by direct questioning that the parents of Asheville remembered the diet suggestions of 1948 and followed them in 1949. They had cut the diet story from their newspapers to save for future reference.

The National Foundation for Infantile Paralysis has informed me that there was a sharp and significant drop in the sales of soft drinks and ice cream in North and adjoining states. The National Foundation had made their own investigation to ascertain to what extent the public had followed my suggestions. They have also stated that the consumption of soft drinks was less in 1949.

FIG 11

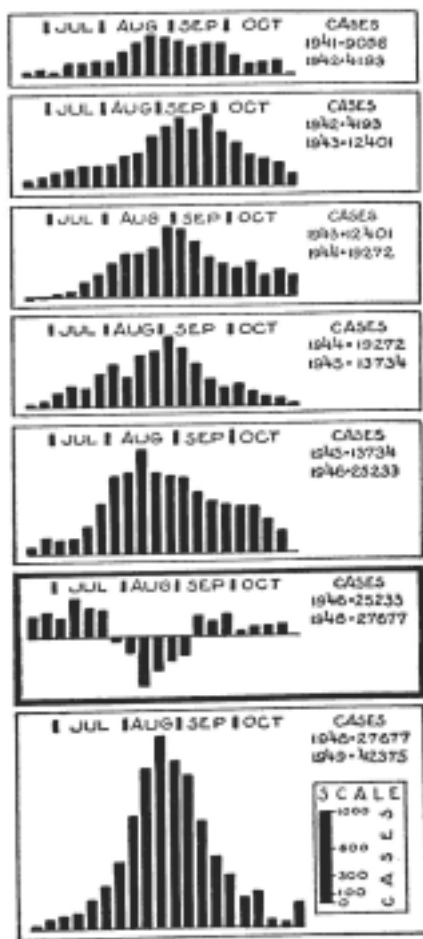
Ten states and the District of Columbia had fewer polio cases in 1949 than in 1948, as

against 38 states which showed marked increases in 1949 over 1948. Of the ten states showing

declines in 1949, the greatest decline in the state of North Carolina, with South Carolina running second. In 1948 the state of North Carolina had a case rate of 66.3, the second highest rate in the country for that year. In 1949, the state of North Carolina had a case rate of only 6.3, the second lowest in the country for that year. In 1948 the state of South Carolina had a case rate of 19.2, and in 1949 had a case rate of 5.4, the lowest in the country that year.

Figs. 11 and 12 show a very significant event in the history of polio in this country since 1931. Each graph represents a comparison between two years, one of the years having had more cases than the year with which it is being compared. Each solid bar on a graph represents the number of cases by which the greater year exceeded the lesser year during a corresponding week. You will notice that once it is apparent that a particular year is running ahead of the year with which it is being compared, the bars on the graph lie above the baseline for duration of the epidemic.

This pattern is true for all the graphs except the graph which compares 1946 and 1948. You will notice that for the first time the bars on the graph become inverted during August and September due to the fact that 1948 fell behind 1946 in number of cases for six consecutive weeks during those months, although 1948 had been running well ahead of 1946 during June and July. This difference in for 1948-1946 is striking and unique. It is unique because it had never happened before, and it is striking because the change in pattern occurred exactly during the week when the diet story for preventing polio was released-throughout the country.



Certainly such a radical change in epidemic pattern cannot be regarded as accidental. The burden of proof falls on those who would regard it as accidental. The usual pattern is present in the graph comparing 1949 and 1948. Nineteen forty-nine greatly exceeded 1948 in the number of cases and the bars on the graph are all in the upright position because 1949 was always ahead of

1948. There was no diet campaign during 1949 and so the epidemic ran its usual course in most of the country.

## **THE PREVALENCE OF LOW BLOOD SUGAR**

Personal observations since 1937 have convinced me of the frequency of low blood sugar in children, adolescents, and adults. I have performed hundreds of glucose tolerance tests routinely on patients attending a medical clinic for disorders and diseases common in everyday practice. I found evidence of low blood sugar in more than half the cases. I found that a diet aimed to correct low blood sugar did away with or greatly alleviated many symptoms not only in those who showed low blood sugar with the tolerance test but also in those who did not reveal low blood sugar at the time. I also found that an individual may show normal blood sugar levels on one occasion and low blood sugar on another occasion. I have concluded that any human can experience low blood sugar as long as he or she consumes sugar and starch. Other researchers have shown how the blood sugar level is controlled by what one eats.

Many healthy people have symptoms of low blood sugar without realizing that the symptoms are due to low blood sugar. For example, many individuals experience a physical let-down in their daily activities around 11 A.M. and 4 P.M. At those hours they get a little tired, may have a slight headache or a sensation of lightheadedness, become a little moody or depressed or irritable, and usually are hungry, especially for something sweet to serve as a "pick-up." And so they will usually partake of the following: a cup of coffee or tea or chocolate, pie, cake, pastry, cookies, candy bars, ice cream, soft drinks, or the like. These sweet foods and beverages afford a rapid relief from their symptoms because they cause a rapid rise in blood sugar level.

I regard as artificial the rapid rise in blood sugar produced by eating sugar. The sugar is an artificial stimulant; and in some people the desire for sweets amounts to a craving, and the demand for something sweet during this craving amounts to an addiction. I regard this craving for sweets as abnormal.

In the first place the low blood sugar is abnormal and could have been prevented. However slight, it is abnormal and is caused by a dietary error, namely, the ingestion of sugar and starch. The low blood sugar that comes on around 11 A.M. is due to eating sugar and starch at breakfast, and the low blood sugar at 4 P.M. is due to eating sugar and starch at the noon meal. On a high protein low carbohydrate diet the fall in blood sugar at 11 A.M. and 4 P.M. does not occur and so there is no physical let-down and no need for a pick-up. Cigarette smoking can also serve as a pick-up because nicotine can cause an immediate rise in blood sugar level by stimulating the adrenal-sympathetic system, the rise occurring at the expense of liver glycogen.

The physical and mental pick-up which follows eating something sweet is accompanied by a rise in blood sugar which lasts for about 30 to 60 minutes and which is soon followed by another fall in blood sugar. A vicious cycle is thus set up.

This rise and fall in blood sugar may occur several times during the waking hours and, as a result, many individuals acquire the habit of drinking 4 to 10 cups of coffee or tea or cocoa daily, or they eat candy at frequent intervals, or drink 4 to 10 bottles of soda pop, or find it necessary to smoke at frequent intervals. The desire for a cigarette actually coincides with a fall in blood sugar and the feeling of satisfaction that comes with a smoke is due to a rise in blood sugar. Denicotinized cigarettes do not satisfy because they do not cause a rise in blood sugar.



Coffee, tea, cocoa, not only cause a rise in blood sugar by reason of the added sugar, but also because they contain caffeine or related chemical compounds that stimulate the adrenal-sympathetic system and thus cause a rise in blood sugar at the expense of liver glycogen. People who smoke a great deal and drink much coffee and who have poor appetites are in a chronic state of malnutrition because they are steadily depleting the liver glycogen stores and are failing to replenish the stores with proper food.

Many people are irritable in the morning before breakfast because of low blood sugar. Some people are so irritable, moody, morose, or depressed, before breakfast that they dare not say anything or do anything until they have had something to eat, if only a cup of coffee. They have learned that they are more tolerable after having eaten something. The blood sugar usually reaches its lowest level in the morning before breakfast, especially after the all-night fast. I have found that eating less sugar and starch at dinner the night before helps prevent low blood sugar in the morning. (See Fig. 13.)

The effects of low blood sugar on morning behaviour and mood is profound. It is a matter of common knowledge that individuals who are irritable and cranky and prone to start arguments before breakfast experience a remarkable change after they have had something to eat. Mothers know that an irritable, cranky, and crying infant is usually a hungry infant. And most wives know that a grumpy husband will feel better after he has had something to eat. It is of great advantage to the wife and mother to know that the blood sugar level is fundamentally related to the behavior of husband or child. Having this knowledge she will readily excuse misbehavior in hungry members of the family. Thus much domestic friction can be avoided.

Statesmen are learning that properly fed citizens are happier, more satisfied, and easier to handle; and that poor nutrition and starvation go hand in hand with unrest, violence, and rebellion. The stability, behavior, and morale of a nation is the sum total of the stability, behavior, and morale of its individual citizens. A properly fed people is a healthy people.

Errors in judgment in all life's activities are often due to the fact that the individual made a decision during a period of low blood sugar at which time he may have been moody, irritable, depressed, or unstable. I have schooled myself never to make an important decision in the morning before breakfast because my frame of mind may be such as to lead to a decision that I would later regret having made. I therefore always delay making a decision until after I have had a good breakfast.

It is surprising how one's attitude toward a problem or a person can change after a good meal, and this only because a rise in blood sugar brings about better brain function by increasing glucose-oxygen consumption. Thus decisions are more likely to be correct when made after a good meal. Experienced negotiators in business and government have

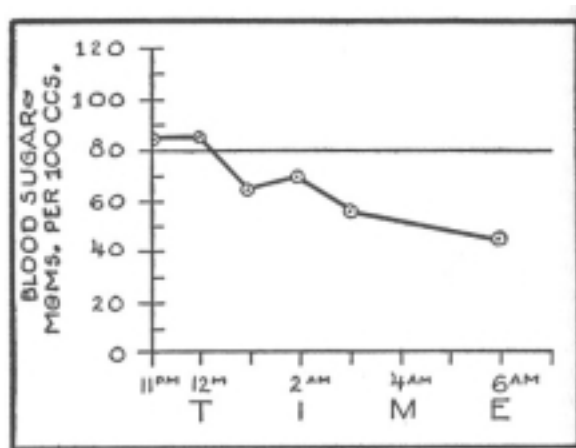


FIG. 13

Course of the blood sugar between the hours of 11 p.M. and 6 A.M. in a patient who had hypoglycemic symptoms around 5 to 6 A.M. Note gradual fall during sleep to the low level of 50 mg. at 6 A.M. Symptoms were readily controlled with a low carbohydrate diet.

learned that the dinner table is a good place to accomplish things.

I have digressed somewhat merely to point out that low blood sugar can happen to everybody, at any age, and often because we all eat sugar and starch.

I have found that low blood sugar is found just as frequently in children as it is in adults. Since both children and adults may have low blood sugar, how is it that polio attacks children so much more often than adults? There is an answer to this question.

Dr. S. B. Wortis of New York found that, weight for weight, the nervous tissue of the young in any species consumes more oxygen, i.e., needs more oxygen, than that of the adult. For this reason, the nervous tissue of the young is more vulnerable to low blood sugar than that of the adult. As has been previously mentioned, nervous tissues consume oxygen in proportion as they utilize glucose. Therefore, in the presence of low blood sugar, the tissues will consume less oxygen and so suffer an oxygen lack. Periods of low blood sugar mean periods of reduced oxygen consumption with resultant increased susceptibility to infection.

Polio may strike children and adolescents who are in apparent good health, i.e., individuals who have no complaints or symptoms, and who eat and sleep well. They may have a history of no more illness than an occasional head cold, or sore throat, or stomach upset. Yet they contract polio. Why? Because low blood sugar can occur at any time if the individual is consuming sugar and starch. Excessive consumption of sweets during a single day, with or without physical over-exertion, may cause a period of prolonged low blood sugar which will lower protective barriers and permit the poliovirus to invade the central nervous system.

Good health is not something one is born with and which persists from year to year as a permanent physical characteristic. Good health is largely the result of bodily chemical conditions which fluctuate normally at every moment within a narrow range. The range of fluctuation is controlled by several factors, especially diet and physical activity. And one of the most important chemicals is the blood sugar. To a great extent, then, health will depend on the fluctuation range of the blood sugar. Good health thus becomes a condition which is at any moment dependent on labile (unstable) chemical states in the blood and tissue fluids, and which may be lost or impaired temporarily when the chemical equilibrium is upset. If an individual is able to eat regularly a diet

adequate in quantity and quality, indulge in normal physical and mental activity, and get adequate rest and sleep, he may be reasonably assured of continuing good health. Some individuals seem constitutionally healthier and sturdier than others, but they are only relatively so. Healthy and sturdy individuals can succumb to disease very readily when they are under stress, when they become careless in dietary habits, and when they over-exert physically or mentally.

Several observers have remarked on the fact that polio frequently attacks children and adolescents who are larger and heavier than the average for their age. Some of them are actually obese. I would say that such individuals eat excess amounts of sugar and starch and the excess is laid down in the body's tissues as fat. Such individuals often have an inordinate craving for sweets and starches; and it is their excessive consumption which causes low blood sugar and subsequent susceptibility to polio.

I have had frequent occasion to observe and treat many adolescents who came to me complaining of symptoms which were found to be due to chronic low blood sugar as determined by the sugar tolerance test. That their complaints and symptoms were due to low blood sugar was confirmed readily by the fact that they were relieved by the elimination or drastic reduction in the consumption of sugar and starch. Individuals with chronic low blood sugar are particularly vulnerable to invasion by the poliovirus. As I mentioned previously, the appearance in the clinic of individuals who had had polio several years before and who were now complaining of symptoms that proved to be due to low blood sugar, led me to suspect that there might be some connection between low blood sugar and susceptibility to polio.

I shall now cite several case histories of adolescents with chronic low blood sugar whom I have observed and treated. The reader will thus get an idea of the many and varied complaints that such individuals have. I want to emphasize that such cases are by no means rare; they are seen frequently in clinics. I shall give the results of the sugar tolerance test in each case. In this test the individual, in the fasting state in the morning, drinks a solution containing 50 grams of glucose. A blood sugar specimen is taken immediately before the solution is drunk. Blood sugar specimens are then taken one half hour, 1 hour, 2 hours, 3 hours, and sometimes 4 hours after the solution is drunk. You will notice that in each case the blood sugar was well below the lower limit of normal of 80 mg. at some time during the test. The duration of low blood sugar varied in each individual but in each case it lasted for a significant period of time.

*Case No. 1.* J. D., male, 13 years old, was brought in by his mother who stated that he was listless, had no desire to play with other boys, had no desire to do his school homework, and preferred to rest most of the time. She had much difficulty waking him from sleep in the morning. A year before he had a fainting spell while in church. Physical examination of the boy revealed moderate underweight but no physical abnormalities. He ate fairly well and consumed average amounts of sugar and starch. A sugar tolerance test revealed: fasting blood sugar, 78 mg.; ½ hour, 115 mg.; 1 hour, 55 mg.; 2 hours, 46 mg.; 3 hours, 58 mg. On a high protein diet with reduced consumption of sugar and starch he improved within a few weeks.

*Comment.* This case may be regarded as a severe example of chronic low blood sugar. His fasting blood sugar was practically normal and he showed a rise ½ hour after taking 50 grams of glucose. However, for the next three hours the blood sugar remained at low levels. Apparently at the time of his fainting spell a year before, the blood sugar had fallen to a level much lower than 46 mg. which he had at the end of 2 hours.

*Case No. 2.* A. Z., male, 12 years old, came in complaining of daily generalized abdominal rumbling of several months' duration. The rumbling was felt in the morning before breakfast and

disappeared after food was taken. It recurred about 15 minutes after breakfast was eaten and would last practically all morning at school until lunch was taken when it disappeared again. Soon after lunch, on his way to school, it would recur and keep up till 3 P.M. when he took a glass of milk which afforded relief. It soon recurred and kept up till supper when it was relieved by eating. It reappeared shortly after supper and lasted till he fell asleep. Sleep was sound. Physical examination revealed no physical abnormality. He was not underweight. His diet contained considerable amounts of sugar and starch with each meal.

A sugar tolerance test revealed: fasting, 60 mg.; ½hour, 100 mg.; 1 hour, 55 mg.; 2 hours, 50 mg.; 3 hours, 60 mg. He was readily relieved by a high protein low carbohydrate diet after a few days.

*Comment.* This case illustrates one of the common symptoms caused by low blood sugar, namely, abnormal contractions of the stomach and intestinal musculature. You will note that he had rumbling before meals when his blood sugar was low and that the rumbling disappeared immediately after eating because the blood sugar rose, though temporarily. Rumbling reappeared 15 to 30 minutes after eating because low blood sugar came on again, and the rumbling lasted as long as the low blood sugar persisted. In cases of sharp abdominal pain the blood sugar level is usually even lower and causes stronger contractions of the intestinal musculature.

At this point it would be well to digress and discuss the mechanism of the normal hunger sensation because the symptoms, rumbling and abdominal pain, are actually abnormally exaggerated hunger phenomena. In an individual with normal blood sugar levels and with a normal sugar tolerance curve, the blood sugar rises to around 100 mg. after ½ hour; 120 mg. after 1 hour; 140 mg. after 2 hours, with a gradual fall during the third and fourth hours to the previous fasting level of 80 to 90 mg. (Fig. 1).

The only sensation occurring during the fall is that of ordinary hunger. The fall in blood sugar stimulates in the brainstem certain nerve centers which control stomach and intestinal muscular contractions. The fall in blood sugar serves as an automatic mechanism to inform the organism that it is time to eat. As a result, the stomach starts to contract rhythmically and vigorously and the sensation of hunger is felt. Normal hunger thus depends on a gradual fall in blood sugar within the normal range. Abnormal hunger and hunger pangs are due to abnormally strong gastric (stomach) contractions brought on by a too rapid fall, especially when the fall is to abnormally low levels. Not only is the stomach stimulated, to strong contractions by the low blood sugar, but also the intestines; and the strong contractions of the latter give rise to generalized rumbling and pain and cramps.

Thus, in case No. 2, the patient's generalized rumbling was actually an abnormal exaggeration of a normal physiologic function, namely, the hunger mechanism. This patient's blood sugar would rise briefly and then start to fall to low levels, causing strong contractions of the stomach and intestines. Instead of rising steadily during the first two hours to around 140 mg., the blood sugar rose to 100 mg. at the end of ½ hour, and then fell to 50 mg. at the end of one hour; and it was during this fall that his rumbling would begin and keep up till he ate some food, when the rumbling disappeared coincident with a rise in blood sugar. If the blood sugar had fallen to 40 mg. the stomach and intestinal contractions would have been more violent and would have given rise to sharp abdominal pain or cramps.

*Case No. 3.* F. M., male, 13 years old, was referred to the clinic by a school physician for failure to gain weight, frequent headaches, restlessness, nervousness, and "fidgeting" in class. He also had attacks of sharp abdominal pain at irregular intervals. These attacks would come on about 15 minutes before a meal, causing him to double up. He had no nausea, vomiting, or fever with the attacks. He also tired easily on ordinary exertion. His mother stated that the boy had had a

“nervous breakdown” at the age of 9 years. Physical examination revealed a thin, pale boy, with no evidence of physical abnormality. His diet contained much sugar and starch.

A sugar tolerance test revealed: fasting, 65 mg.; ½ hour, 90 mg.; 1 hour, 75 mg.; 2 hours, 55 mg.; 3 hours, 50 mg. He responded readily to a high protein low carbohydrate diet. His nervousness and abdominal pain were relieved after a week on the diet and after a few months he showed a gain in weight.

*Comment.* This patient’s symptoms were chiefly nervous in origin. This is to be expected, since the nervous system is very vulnerable to low blood sugar, especially in the young. The fact that his attacks of abdominal pain came on before meals is a clue that they were most likely due to periods of low blood sugar. His response to the change in diet confirmed this suspicion.

Case No. 4. B. P., male, 14 years old, was brought in by his mother because of nervousness, dizzy spells, frequent headaches, night sweats, and abdominal pain after hard running (a “stitch in the side,” as boys commonly call it). He also had frequent attacks of coldness and blueness of the hands and forearms in cold weather and even in moderate weather. The mother also stated that he was cranky, easily angered, and “always wants to eat,” especially something sweet. Because the family was poor, the diet consisted chiefly of the cheaper starchy foods. He also ate excessive amounts of sugar. Physical examination revealed a nervous, restless, alert, thin boy. There were no physical abnormalities.

The sugar tolerance test revealed: fasting, 75 mg.; ½hour, 100 mg.; 1 hour, 90 mg.; 2 hours, 60 mg.; 3 hours, 60 mg. Financial difficulties prevented giving him the prescribed high protein diet but he did improve considerably by reducing the consumption of sugar and starch.

*Comment.* This case illustrates the abnormal craving for food, especially sweets, in the presence of chronic low blood sugar. The low blood sugar causes frequent and strong stomach contractions which give rise to frequent hunger sensations. In this case, although the consumption of sweets was excessive, the boy was not overweight. (In other cases, where there may also be some disturbed function of the endocrine glands (thyroid, pituitary, pancreas) the excessive consumption of sugar and starch may lead to obesity). As has been stated, irritability is a common symptom of low blood sugar. This boy was very cranky, easily provoked, and subject to temper tantrums. Low blood sugar readily explains the irritability that accompanies marked hunger.

Case No. 5. J. S., male, 11 years old, was brought in by his mother because he was thin, overactive, and a poor eater. He also had attacks of numbness and coldness of the hands. Occasionally he had chills soon after eating. He had frequent attacks of upper and lower abdominal pain on exertion, such as running. He often felt dizzy on getting up from the lying position. Physical examination revealed an alert, thin, intelligent boy with no physical abnormalities. A sugar tolerance test revealed: fasting, 80 mg.; ½ hour, 80 mg.; 1 hour, 95 mg.; 2 hours, 75 mg.; 3 hours, 55 mg. With a change to a high protein low carbohydrate diet he had prompt relief from the abdominal pain and showed improvement with respect to his nervous complaints.

Case No. 6. R. S., male, 15 years old, came in because of failure to gain, frequent head colds, headaches, restlessness at school and at the movies. Apparently he was unable to remain seated for any length of time. He was a chronic fingernail biter. Physical examination revealed a thin, nervous boy with no evidence of physical abnormality. Because of financial difficulties his diet consisted chiefly of the cheaper starchy foods. The sugar tolerance test revealed: fasting, 75 mg.; ½ hour, 85 mg.; 1 hour, 68 mg.; 2 hours, 68 mg.; 3 hours, 68 mg.

*Comment.* This boy showed a very slight rise to 85 mg. at the end of ½ hour, and for the next 2½ hours the blood sugar was maintained at the low level of 68 mg.

## **THE CONCEPT OF GAMMA-GLUCOSE**

I believe the reader will benefit by a discussion of the concept of gamma-glucose. Ordinary glucose is a mixture of two chemical forms called alpha-glucose and beta-glucose. Alpha-beta-glucose mixture is derived from the cane sugar we eat, and from the sugar present in fruit; and it is an end product of the digestion of starch. Gamma-glucose is so labile (unstable) that it has never been isolated in the test tube and identified as such. The evidence for its existence however, while indirect, is strong. Many authorities in chemistry and physiology believe it exists since it helps explain certain phenomena which otherwise would be obscure. (Just as in nuclear physics where faith in the existence of protons, electrons, and mesons, helps to explain many phenomena).

Gamma-glucose is considered to be labile, highly reactive, and readily oxidized. It is very likely that the cells of the body prefer to oxidize gamma-glucose to the exclusion of alpha-beta-glucose. Dr. Shaffer, an authority on the subject, in a review of the literature on gamma-glucose, has stated that the hypothesis appears attractive that alpha-beta-glucose is converted under the influence of insulin into gamma-glucose. In this discussion, and in the present state of our knowledge, it is sufficient to believe that alpha-beta-glucose is synthesized to glycogen in the liver under the influence of insulin and that the glycogen is subsequently broken down into gamma-glucose which is then liberated into the blood.

When a human lives on meat and fish exclusively, as does the Eskimo or Arctic explorer, he is living on protein and fat. Yet glycogen is stored in the liver in normal amounts and there is a constant normal blood sugar level. Where do the glycogen and glucose come from on such a diet? They are derived from protein and fat. Fifty-eight percent of protein and 10% of fat can be converted by the body to glycogen and ultimately to glucose. I want to point out that the blood sugar on such a diet may be said to exist exclusively in the gamma form. On such a diet the blood sugar is produced entirely within the body and so may be called endogenous glucose as opposed to the exogenous alpha-beta-glucose which is introduced from the outside in the foods we eat. Certainly it would appear plausible to regard the sugar derived from meat and fish (protein and fat) as being different chemically from the sugar derived from cane sugar, fruits, and starch. From my studies I have been forced to conclude that alpha-beta-glucose is so foreign to the cells of the body as to be harmful to the body's economy.

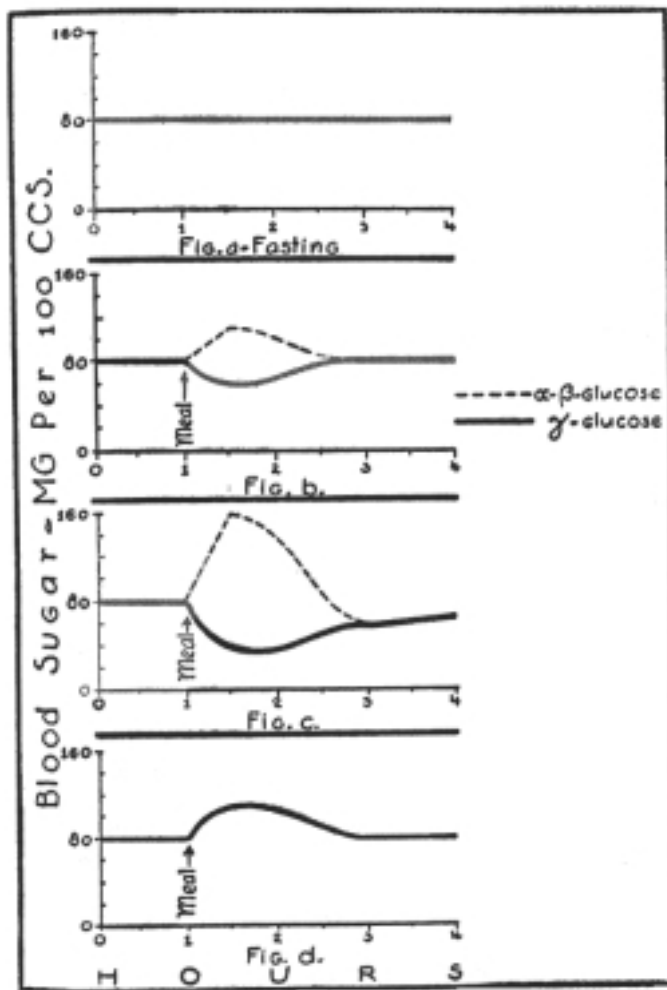


FIG. 14

Fig. a. In the fasting state the blood glucose (or blood sugar) is derived from the breakdown of liver glycogen and may be said to exist in the endogenous gamma-glucose form.

Fig. b. Following the ingestion of a moderate amount of exogenous alpha-beta-glucose the blood sugar level will rise, and the blood glucose value will now be the sum of the gamma-glucose plus the alpha-beta-glucose. However, the exogenous alpha-beta-glucose inhibits the output of endogenous gammaglucoase from the liver; and the concentration of the gamma-glucose will fall as shown by the heavy line in the figure. Thus there may be a fall in total body oxygen absorption because of the fall in gamma-glucose concentration and in spite of the rise in blood glucose level.

Fig. c. Following the ingestion of a larger amount of exogenous alpha-beta-glucose the blood glucose level rises to a high value and the liver output of endogenous gamm2.glucoase is proportionately depressed. Under such circumstances an individual may experience hypoglycemic symptoms even though the blood sugar actually rises. The gamma-glucose output *may* be subnormal for 1 to 3 hours. After such a meal there may be a fall in total body oxygen absorption.

Fig. d. Following the ingestion of proteins and fat the blood glucose level rises moderately. Since no sugar or starch is ingested no exogenous alpha-beta-glucose is available to inhibit the output of endogenous gamma-glucose from the liver. The blood sugar will thus consist of the gamma-glucose form exclusively. The rise in blood sugar after protein and fat is due to the rapid conversion of the digested protein and fat to gamma-glucose. A meal consisting of protein, fat, and carbohydrate foods containing no sugar or starch will also cause a moderate rise in blood sugar and with no inhibition of liver output of gamma-glucose. After such a meal there will be a rise in total body oxygen absorption.

There is ample evidence for this statement from observations made by two of this country's leading researchers in metabolism, Benedict and Carpenter. These workers determined the minimum (basal) oxygen requirements of normal human subjects and then measured the oxygen consumption of these subjects after various test meals of sugar, starch, protein, and fats, alone and in combination.

After a protein meal they always observed a marked rise in oxygen absorption (consumption), a phenomenon called "the specific dynamic action" of protein and which is regarded as highly beneficial to the body. After a fat meal there was a slight rise in oxygen absorption or none at all. After sugar and starch they found a slight initial rise followed by a fall in oxygen absorption in some experiments, and in other experiments they observed a consistent fall in oxygen absorption, with no initial rise. Benedict and Carpenter were unable to explain this fall in oxygen absorption after sugar and starch. They were much surprised to discover that the ingestion of sugar and starch actually could cause a depression of total bodily oxygen absorption below basal requirements.

Here are some of the actual experimental results reported by Benedict and Carpenter. They fed one subject 400 grams of bananas (413 calories) and observed an increase in oxygen absorption of 5.5 grams during the first hour, and a fall in oxygen absorption below basal requirement during the second, third, and fourth hours. Bananas contain chiefly sugar and starch. In the same subject, after 217 grams of beefsteak, (451 calories) which consists of protein and fat, there was an increase in oxygen absorption during each of four hours, with a total increase of 17.5 grams of oxygen against a net increase of only 1.5 grams of oxygen after the bananas.

With larger meals the results were even more striking. For example, after 1382 calories of a sugar mixture, there was a fall in oxygen absorption of 11 grams over an eight hour period with no initial increase; in other words the subject would have consumed 11 grams more oxygen if he had fasted for the eight hours. On the other hand, after 1305 calories of beefsteak there was an increase in oxygen absorption of 40 grams during an eight-hour period.

Now, how does the ingestion of sugar and starch depress oxygen absorption? There are two ways by which this can be brought about: first, by causing low blood sugar, and second, by depressing the liver output of gamma-glucose. As has been previously explained, sugar and starch may cause low blood sugar about an hour after eating, and this period of low blood sugar may last from one to three hours. During the period of low blood sugar there will be a reduced delivery of glucose to all the cells of the body with a resultant reduced oxidation of glucose; as a result, less oxygen will be absorbed by the body since cells utilize oxygen in proportion as they utilize glucose. Also, during the period of reduced glucose-oxygen consumption, less heat will be generated and so the body temperature may fall. This reduced heat production during low blood sugar readily explains the presence of subnormal body temperature in certain individuals in the morning before breakfast, and even after meals. Such individuals tolerate cold weather very poorly.

The second mechanism whereby sugar and starch may cause a depressed oxygen consumption involves the concept of gamma-glucose. During fasting, the blood sugar is derived entirely from the breakdown of liver glycogen and may be regarded as existing in the readily oxidizable gamma form. Now Dr. Soskin and his co-workers have found that when ordinary alpha-beta-glucose is injected into the blood the greater the amount of alpha-beta-glucose injected the greater the depressant effect on the liver output. This reduced output by the liver occurs because there is no apparent need for the liver to pour out endogenous glucose as long as exogenous alpha-beta-glucose is being introduced from the outside. The injection of exogenous glucose raises the blood sugar level and, to prevent too great a rise, the liver responds by lowering its



output. The oxygen absorption of the body may fall, however, in spite of the rise in blood sugar level, which followed the injection of glucose, because of the reduced output from the liver of the more readily oxidizable form, which I believe to be the gamma form.

The influx of alpha-beta-glucose into the blood drives the gamma-glucose out of the circulation, just as bad money drives good money out of circulation. The oxygen absorption of the body falls in proportion as the output of gamma-glucose falls. This concept will explain why Benedict and Carpenter observed a moderate fall after small sugar meals and a greater fall after larger sugar meals. After a pure protein and fat meal (beefsteak) there is no introduction of alpha-beta-glucose and hence there will be no depressant effect on the liver output of gamma-glucose and there will be no fall in oxygen absorption. The marked increase in oxygen absorption after beefsteak may be readily explained by the oxidation of the products of digestion resulting from the digestion of the protein and fat.

Summarizing, the ingestion of sugar and starch may depress oxygen absorption by causing low blood sugar and/or depressing the output of gamma-glucose from the liver. In some instances only one of these mechanisms may be operating, and in other instances both mechanisms may be operating. In the latter, the fall in oxygen absorption will be greater than in the former, and the fall in oxygen absorption may be so great and so prolonged that susceptibility to infection will occur.

I wish to impress the reader with this concept of gamma-glucose. Gamma-glucose is to be regarded as the "natural sugar," preferred by the cells of the body for energy purposes because it is more readily oxidized with greater release of energy. Alpha-beta-glucose is to be regarded as a "foreign sugar," and, in a sense, artificial. Alpha-beta-glucose may be oxidized directly but not as efficiently or as rapidly as gamma-glucose. The reader should remember that the ingestion of sugar and starch with resultant liberation of alpha-beta-glucose after digestion exerts a depressant effect on the liver output of the preferred gamma-glucose.

Knowledge in medicine and physiology is in a constant state of flux. New discoveries open new doors and confirm or alter previous concepts. In the light of our present knowledge, the concept of gamma-glucose presented here is fundamentally sound and warranted by supporting data, and it explains certain phenomena that would otherwise be baffling. Regardless of whether or not the above explanation is the correct one, the fact remains that the ingestion of sugar and starch may readily cause a fall in total bodily oxygen absorption through adverse effects on the blood sugar regulatory mechanism, and it is this state of oxygen lack that is responsible for susceptibility to infection.

## THE PUZZLER IN POLIO EPIDEMICS

In an article in the June 28, 1947 issue of the *Journal of the American Medical Association*, Dr. A. B. Sabin, a leading investigator in polio, discusses certain problems, which have baffled students of the disease. I shall state some of the problems he mentions and shall attempt to throw some light on their solution.

1. Dr. Sabin states: “*No circumstance in the history of poliomyelitis is so baffling as its change during the past 50 years from a sporadic to an epidemic disease.*”

An increasing consumption of sugar, as shown in the following table, helps explain this change in the incidence of the disease.

<u>Years</u>	<u>Yearly Average</u>
1880-1890	44
1890-1900	56
1900-1910	65
1910-1920	82
1920-1930	100
1930-1937	95
1939-	103.2

2. Dr. Sabin writes: “*Another peculiar circumstance which may contain an important clue is that epidemics have occurred with greatest frequency and severity in the very countries in which sanitation and hygiene have undoubtedly made the greatest advances.*”

I offer the following explanation: Advanced sanitation and hygiene are, as a rule, to be found in the wealthier countries. Advanced sanitation and hygiene have helped prevent such diseases as typhoid fever, cholera, malaria, and tuberculosis, because the environment has been controlled by purification of water and milk, by drainage of swamps, by proper sewage disposal, and the organisms responsible for these diseases have been “kept away from our doors.” The fact that polio has not been prevented by advanced sanitation and hygiene indicates that its incidence is controlled and influenced by factors quite different from the factors that bring about the spread of typhoid and the other diseases. As previously stated, advanced sanitation and hygiene are to be found in the richer countries, and one of the unfortunate evils that accompany wealth is the consumption of sugar in the form of luxury foods such as ice cream, candies, soft drinks, cakes, pies, pastries, and the like. Poor countries cannot afford luxury foods, sanitation and hygiene. That is how I would explain the greater incidence of polio in countries with advanced sanitation and hygiene. The following table shows the extreme differences in sugar consumption in various parts of the world and it will be readily noted that the countries with the lowest sugar consumption are most backward in sanitation and hygiene.

Thus we see that sugar consumption is by far the greatest in the richer countries where one would also expect to find advanced sanitation and hygiene. Epidemics have occurred with the

greatest frequency and severity in the high sugar consuming countries. In fact, epidemics have never been reported in the natives of the low sugar consuming countries, such as China.

3. Dr. Sabin states: *“In my opinion, one of the most important problems in the epidemiology of poliomyelitis is the determination of factors relative to virus, host, and environment, which are different in cities like New York, Chicago, Minneapolis, Los Angeles, and Denver, and many others in the United States with histories of large outbreaks of the disease, and cities like Peiping, Tientsin and Shanghai, occupying the same latitude in China in which only rare sporadic cases have been reported thus far, despite the presence in these cities of western-trained physicians who could not have missed such outbreaks in the native population if they had occurred.”*

**TABLE  
NO. 3**

**SUGAR CONSUMPTION BY GEOGRAPHICAL DIVISIONS**

**(Crop Year Sept. 1, 1938, to Aug. 31, 1939)**

**Per Capita Consumption**

<b>Country</b>	<b>(Pounds — Raw Value)</b>
<u>United States</u>	<u>103.2</u>
<u>Canada</u>	<u>102.9</u>
<u>Mexico</u>	<u>37.1</u>
<u>Other North America</u>	<u>41.2</u>
<u>Argentina</u>	<u>71.0</u>
<u>Brazil</u>	<u>51.7</u>
<u>Other South America</u>	<u>28.8</u>
<u>Sweden</u>	<u>119.3</u>
<u>United Kingdom</u>	<u>112.6</u>
<u>Switzerland</u>	<u>85.1</u>
<u>Holland</u>	<u>89.4</u>
<u>Germany</u>	<u>63.3</u>
<u>Italy</u>	<u>21.3</u>
<u>Poland</u>	<u>29.8</u>
<u>Rumania</u>	<u>14.6</u>
<u>Spain</u>	<u>17.2</u>
<u>China</u>	<u>3.2</u>
<u>India</u>	<u>24.3</u>
<u>Japanese Empire</u>	<u>29.1</u>
<u>Java (Dutch East Indies)</u>	<u>11.6</u>
<u>Other Asia</u>	<u>13.0</u>
<u>Algeria, Morocco, and Tunisia</u>	<u>44.4</u>
<u>British South African Union</u>	<u>58.6</u>
<u>Egypt</u>	<u>20.4</u>
<u>Other Africa</u>	<u>37.0</u>
<u>Australia</u>	<u>114.5</u>
<u>Other Oceania</u>	<u>81.0</u>

In line with the explanation offered to problem 2, I would state that here again the extreme differences in per capita sugar consumption between China (3.2 lbs.) and the United States (103.2 lbs.) afford a ready explanation for the occurrence of epidemics in the cities of the United States and the absence of epidemics in the cities of China.

4. Dr. Sabin tells how polio occurred among American troops in China, Japan, and in the Philippines, in spite of the fact that there were no outbreaks of polio at the time among the native children and adults in those areas in which the troops were located. A report on polio in the Philippines in 1936 stated that 16 of 17 patients with the disease in Manila were Americans. In 1945 there were 246 cases of polio with 52 deaths among American troops in the Philippines according to reports to the Office of the Surgeon General. And, since the end of combat in the Philippines, polio has been among the leading causes of death in American troops; but checks have revealed no outbreaks of polio among the surrounding native population. In fact, epidemics of polio have never been observed among the natives of the Philippines. Why has the disease been confined to the American troops in these countries?

Dr. Sabin also witnessed an outbreak of polio in the summer of 1946 among American marines stationed in the Tientsin area of North China. Four men died, one was severely paralyzed, and at least 25 others had nonparalytic attacks. There was no outbreak of polio among the natives at the time. Dr. Grice, a British physician in Tientsin for 25 years, informed Dr. Sabin that while he infrequently saw paralytic polio in children in the foreign colony he rarely saw the disease among the Chinese. The extraordinarily uncommon occurrence of polio among the yellow races living in North China was also reported by Zia in 1930.

I offer the following explanation for the occurrence of polio among American troops in China and the Philippines: The Americans took their dietary habits with them overseas. All during the war, as soon as local combat conditions permitted, ice cream, candies, soft drinks, cakes, and the like, became available to American troops. Ice cream manufacturing equipment followed soon after combat equipment. I saw American troops consume great quantities of candy bars when they lost their appetite for the monotonous K and C rations. It was felt that our men would feel at home, not get homesick, and have better morale if sweets were available. Thus, I submit, polio occurred among the Americans and not among the natives because the natives did not consume the amount of sugar the Americans did.

5. Dr. Sabin writes: *“Intimate human contact...does not by itself explain the recurrent summer epidemics of paralysis.... With the present high incidence of the disease among children of school age in the United States, it is remarkable that, unlike certain other infections of childhood, the epidemics of paralysis occur during the very months when the children are away from school.”*

I submit that this problem may be answered, in part, by stating that when children are away from school during the summer they have more time for physical activities which may at times be excessive and so they become predisposed to polio as previously discussed in the chapter on Physical Exertion and Polio. Also, during the summer they get hot and thirsty, consume more sweet cooling beverages and foods, and thereby run the risk of low blood sugar.

6. Dr. Sabin also states: *“All this brings one back to another old question in the epidemiology of poliomyelitis, namely: do more people acquire the poliomyelitis virus during the summer and early autumn months, or is there only a difference in the ratio between the number exhibiting the paralytic and inapparent nonparalytic forms of the disease during the different times of the year? If there were only some way of answering this question by direct laboratory tests instead of by speculation, analogy or evaluation of probabilities, one could end the controversy between those who maintain that certain unknown changes in the host rather than increased dissemination of the virus, are responsible for epidemics and for the greater incidence of the disease... Why does paralytic poliomyelitis, with only rare exceptions, remain practically dormant during more than two thirds of each year, appearing in only an occasional person, and then seems to explode during the late summer and early autumn?”*

In line with the ideas heretofore expressed, I would say that polio is more prevalent during the summer because of a change in the host. This change is a chemical one, namely, an increased incidence of low blood sugar brought on by an increased consumption of sugar in the form of cooling foods and beverages and, perhaps, a reduction in protein foods. Excessive physical exertion incidental to summer outings and vacations may further predispose to low blood sugar. Dr. Sabin makes the following significant remarks regarding quarantine measures during polio epidemics: "*Poliovirus is present for a short time in the throat and more frequently and for a longer time in the intestinal tract and stools of certain apparently healthy people as well as of acutely paralyzed patients during an epidemic... There is no evidence that the virus is ordinarily present in the nose or that droplets emitted from the respiratory tract play a significant role, if any, in the dissemination of the virus..... Measures designed to minimize spread by droplet infection such as closing of movies and churches... are not warranted.*"

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### **DIET IS MAJOR FACTOR IN POLIO PREVENTION, DR. SANDLER BELIEVES**

(From *The Asheville Citizen*, August 5, 1948.)

A theory concerning a major cause for human infection with polio has been advanced by Dr. Benjamin P. Sandler, of Asheville, N. C.

Dr. Sandler, a recognized authority in nutrition research, was the first doctor to transmit polio to the rabbit, believed to be immune, a test he completed in 1938.

His theory is two-fold — he believes he has found a major cause for polio in humans, and he believes that preventive measures are simple, easy, and quickly applicable.

Dr. Sandler believes that the major cause is the low blood sugar in the human body, caused, paradoxically as it may sound, by eating too much sweets and starchy food.

The preventive measures? Cut out foods containing sugars and starches.

In 24 hours, according to Dr. Sandler, the body can build up sufficient resistance to the poliovirus to prevent disease. The diet would have to continue, of course.

"The crisis is here and hours have become precious," he said. "I have been impelled to bring this directly to the newspapers because of my profound conviction that, through community cooperation and general acceptance of a diet low in sugars and starches, this epidemic can be got under control in about two weeks time.

"I am willing to state without reserve that such a diet, strictly observed, can build up in 24 hours time a resistance in the human body sufficiently strong to combat the disease successfully. The answer lies simply in maintaining a normal blood sugar."

Here is Dr. Sandler's program:

- (1) Eliminate from the diet sugar and foods containing sugar, such as: soft drinks; fruit juices (except tomato juice); ice cream; cakes, pastries, pies; candies; canned and preserved fruits. (Saccharin may be substituted for sugar.)
- (2) Cut down the consumption of starchy foods, such as: bread, rolls, pancakes; potatoes; rice; corn; cereals and grits.
- (3) Substitute for such starch foods and starchy vegetables, the following: tomatoes, string beans, cucumbers, greens, lettuce, turnips, carrots, red beets, cabbage, onions and soybeans.
- (4) Do not eat fresh fruits or melons more than once a day, and then only in small quantities.
- (5) Eat more protective protein foods, such as: eggs, pork and beef products; fish (fresh or canned); poultry; milk, cream and cheese.

Eat three substantial meals a day, advises Dr. Sandler. And avoid exertion and fatigue because they are known to be associated with low blood sugar content. Avoid swimming in cold water. Rest as much as possible.

Dr. Sandler suggests that the recommended diet be followed until the polio danger season officially is declared over by local health authorities.

“One of the puzzling characteristics of polio,” Dr. Sandler said yesterday, “has been its prevalence in warm weather. Many people cut down on protective protein foods— such as meat, fish and poultry — because of a mistaken idea that a ‘light’ diet is better for them in warm weather. And they increase consumption of cooling foods and beverages — most of them heavily sweetened. It is this increase in consumption of sugar that produces a lowering of blood sugar and thereby a lowering of the body’s resistance to the poliovirus and other diseases.”

Here is the basis for the Sandler theories:

A normal blood sugar content of 100 milligrams in each 100 cubic centimeters of blood is necessary to maintain resistance to bodily infection. Any appreciable lowering of this blood sugar content (say, to from 75 to 55 mg.) can lower the barriers and permit bodily invasion by the virus of polio.

Dr. Sandler offers as the scientific basis for these statements research done with rabbits and monkeys. This research he began at Willard Parker hospital in New York during the metropolitan area’s record polio outbreak of 1931.

Authorities had noted that rabbits normally are resistant to poliovirus. Dr. Sandler, observing that studies showed that in rabbits the blood sugar never dropped below 100 mg., began pondering the far-differing case of the rhesus monkey, a notoriously easy prey to poliomyelitis. In monkeys, blood sugar content frequently was observed to fall to abnormally low levels, around 50 mg.

Furthermore, observations on humans who had recovered from polio revealed low blood sugar — hypoglycemia is the technical term — to be frequently present.

From these — rabbits, monkeys and humans — Dr. Sandler first deduced that low blood sugar could be an important factor in susceptibility to the poliovirus.

The job was to check this deduction through experiments in which the blood sugar content of rabbits would be lowered and their susceptibility to polio again tested.

In the laboratories of the Morrisania hospital in New York 10 years ago, Dr. Sandler began a series of experiments in which insulin was injected in rabbits to lower the blood sugar for periods of four to six hours. Once the blood sugar content had been thus dropped, the doctor attempted again to transmit the poliovirus to the normally highly resistant animals. The rabbits then fell easy victims.

The animals showed evidence of polio infection within eight to 10 hours after intracerebral inoculation with the virus, indicating rapid spread of the disease during the period of hypoglycemia. (Dr. Sandler reported on these studies in the American Journal of Pathology in January, 1941). Some rabbits died within 14 hours after infection. Characteristic nerve-cell destruction with paralysis was in evidence.

Chronic hypoglycemia (low blood sugar) is a common disorder in childhood and adolescence, Dr. Sandler points out, and is readily influenced by diet as well as exertion. This, he believes, serves to explain the high incidence of polio in younger age groups, as well as the frequently reported occurrence of the disease following strenuous physical exertion.

Dr. Sandler received his degree in medicine at New York university in 1931. He interned at Morrisania city hospital in New York city and later was on the staff there as well as Polyclinic and Montefiore hospitals in New York city. From July, 1941, until February, 1947, he was in the U. S. naval medical corps, attaining the rank of commander.

He has done considerable research in polio and the relationship between diet and disease. He has published six papers on the latter subject, as well as papers on other medical subjects. His research includes a period assisting the research staff at Willard Parker hospital in New York city during the epidemic there in 1931, and independent research later, when he "gave" polio to a rhesus monkey, transmitted it to a rabbit, and then to another monkey.

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Summarizing the evidence for my contention that low blood sugar is a factor of susceptibility to polio, and that a diet aimed to prevent low blood sugar can prevent polio, I submit the following:

1. Low blood sugar is not present in the rabbit, a non-susceptible animal.
2. Low blood sugar is present in monkeys, a susceptible animal.
3. Inducing low blood sugar in rabbits with insulin renders the animals susceptible to poliovirus.
4. Physical exertion, swimming in cold water, predispose to polio because they may be associated with low blood sugar.
5. The diet campaign aimed to prevent low blood sugar and thereby prevent polio had a significant effect on the number of cases during the 1948 epidemic both locally in the city of Asheville, the state of North Carolina, and in the nearby southeastern states as shown by the earlier peak dates in those states. The diet campaign also had a significant effect on the number of cases throughout the

country as shown by the change in the trend of the 1948 epidemic when compared with the trend in 1946.

6. The unique change in the graph comparing 1946 with 1948 is exceptional, in that the change occurred immediately after the release of the diet instructions, and because such a change had never before occurred in the history of polio in this country.

7. Although the 1949 polio epidemic for the country as a whole was more severe than the 1948 epidemic, the city of Asheville and the state of North Carolina experienced the greatest reduction in the number of cases in 1949 in spite of the fact that North Carolina had the second highest case rate in the country in 1948. The state of North Carolina had a case rate of 66.3 in 1948 and a case rate of only 6.3 in 1949. South Dakota had a case rate 153.9 in 1948, the highest in the nation, but showed a reduction in 1949 to only 63.0.

8. Polio epidemics have occurred throughout the world in past years *only in those countries with high per capita sugar consumption*. Epidemics are unknown in countries with low sugar consumption. The greater the sugar consumption the more severe the epidemic.

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*“Foods must be in the condition in which they are found in nature, or at least in a condition as close as possible to that found in nature.”*

*Hippocrates*



## REFERENCES

1. Jungeblut, C. W., and Resnick, R.: *Blood Sugar Levels and Dextrose Tolerances in Experimental Poliomyelitis*. American Journal of Diseases of Children, Vol. 51, p. 91, 1936.
2. du Vigneaud, V., and Karr, W. G.: *Carbohydrate Utilization*. Journal of Biological Chemistry, Vol. 66, p. 281, 1925.
3. Sandler, B. P.: *The Production of Neuronal Injury and Necrosis With the Virus of Poliomyelitis in Rabbits During Insulin Hypoglycemia*. American Journal of Pathology, Vol. 17, P. 69, 1941.
4. McCullagh, E. P., and Johnston, C. R. K.: *Manipulation of Glucose Tolerance by Diet*. American Journal of Medical Sciences, Vol. 195, p. 773, 1938.
5. Editorial, Journal of the American Medical Association, Vol. 116, p.2506, May 31, 1941.
6. Levine, S. A., Gordon, B., and Derick, L.: *Some Changes in the Chemical Constituents of the Blood Following a Marathon Race*. Journal of the American Medical Association, Vol. 82, p. 1778, 1924.
7. McCormick, W. J.: *Observations on the 1941 Outbreak of Poliomyelitis and Encephalitis in the Midwest*. Medical Record, Vol. 155, p. 89, 1942.
8. Levinson. Cited in editorial, Journal of the American Medical Association, Vol. 116, P. 2506, May 31, 1941.
9. Wortis, S. B.: *Respiratory Metabolism of Excised Brain Tissue*. American Journal of Psychiatry, Vol. 13, P. 725, 1934.
10. Shaffer, P. A.: *Intermediary Metabolism of Carbohydrates*. Physiological Reviews, Vol. 3, p. 394, 1923.
11. Benedict, F. G., and Carpenter, T. M.: *Food Ingestion and Energy Transformation With Special Reference to the Stimulating Effects of Nutrients*. Carnegie Institute of Washington Publication No. 261, 1918.
12. Soskin, S., Essex, H. E., Herrick, J. F., and Mann, F. C.: *The Mechanism of Regulation of the Blood Sugar by the Liver*. American Journal of Physiology, Vol. 124, p. 558, 1938.