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FOOD POISONING *

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The changes that have passed over the modern world in the last hundred years and that are still in progress have profoundly modified many conditions affecting human nutrition, as they have all other aspects of individual and community life. For the majority of the human race, especially for those living in the vast aggregations known as the modern city, almost every vestige of personal and individual control of food supply has disappeared. Foods are prepared in enormous quantities by machine methods, are transported hundreds and even thousands of miles, are handled by scores of persons before reaching our tables. Cheapness and abundance of food have been attained only at a price. We are dependent as never before on the skill of others in choosing, preparing, conserving, transporting and serving our food; and we are incidentally exposed to the hazards engendered by cupidity, ignorance and carelessness. Small wonder that the problem of controlling the character of city food supply is one of the most fundamental problems of public health.

A bewildering variety of factors has been found to influence the character, palatability and wholesomeness of foods.

Food Idiosyncrasy.—In certain remarkable instances a peculiar condition of the individual consuming the food is found to give rise to physiological disturbance. The human body has become sensitized in some way to a particular protein substance and the ingestion of even small quantities of this protein may produce rash, vomiting and other disagreeable consequences. Such well-known examples of food sensitization as seen in the idiosyncrasy of certain persons to strawberries, shellfish and even to eggs and milk, are obviously an individual physiological quality not to be attributed to any abnormality or inferiority inherent in the food.

Injurious effects due to the composition, contents or contamination of food itself are, however, sufficiently common.

Poisons Inherent in Food.—A relatively serious but fortunately

* Portions of the Gordon Bell Memorial Lecture delivered at Winnipeg, Manitoba presented with some modifications by Dr. Jordan at the School of Tropical Medicine.

rare cause of illness is found in the natural presence in certain plants and animals of highly poisonous substances. Perhaps the most familiar example of the danger from this source is the poisoning due to toxic varieties of mushrooms eaten by mistake for wholesome varieties which they resemble. Numerous other plants, however, contain poisonous substances and are occasionally responsible for illness and death. The spindle-shaped roots of the deadly water hemlock (Cicuta maculata) are not infrequently mistaken by children for horseradish, Jerusalem artichokes, parsnips, or some other edible root.(1) Numerous deaths of stock animals on the range may sometimes be caused by the larkspur, by certain lupines, laurels and other plants. Poisonous honey may be derived from the blossoms of the azalea, the rhododendron and certain other nectar-bearing plants. Professor McCollum has recently recalled to me the description in Xenephon's Anabasis:

> After accomplishing the ascent the Greeks took up quarters in numerous villages, which contained provisions in abundance. Now for the most part there was nothing here which they really found strange; but the swarms of bees in the neighborhood were numerous, and the soldiers who ate of the honey all went off their heads, and suffered from vomiting and diarrhoea, and not one of them could stand up, but those who had eaten a little were like people exceedingly drunk, while those who had eaten a great deal seemed like crazy, or even, in some cases, dying men. So they lay there in great numbers as though the army had suffered a defeat, and great despondency prevailed. On the next day, however no one had died, and at approximately the same hour as they had eaten the honey they began to come to their senses; and on the third or fourth day they got up, as if from a drugging.

The mysterious disease, milksickness, long the scourge of American pioneer settlements in the Middle West, is due to a poison derived from the white snakeroot (Eupatorium ageratoides). In times of drought when pasturage becomes scanty, grazing cattle feed on this plant and the poison passes over into the milk of affected animals in such quantity that it produces a fatal illness in man.(2) The mother of Abraham Lincoln died from this cause in 1818 in southern Indiana.

Rarely the presence of natural poisons in widely purveyed food articles raises this danger to the importance of a public-health problem. One instance is on record where an oil derived from a tropical plant which was used in a commercial butter substitute gave rise to a widespread outbreak of poisoning.(3)

According to recent dietary experiments by Edward Mellanby,(4) certain common food stuffs are thought to contain specific harmful substances as, for example, a substance in cereals interfering with

calcification. Another type of harmful substance is thought to be present in wheat germ. This substance produces severe nervous symptoms. From this point of view vitamins may be supposed to exercise an antidotal or counteracting effect upon the natural poisons present in widely used food stuffs. These hypothetical toxic substances in natural food, like vitamins, are thus far konwn only by their physiological effects.

Poisoning from eating normal animal tissues does not seem to be so common as from plants, and less is known about the specific poisonous constituents. Several varieties of fish, notably some tropical species, are quite poisonous particularly at certain seasons. The famous Japanese Fugu, one of the balloon fish, has been the cause of numerous deaths and has been often utilized for suicidal purposes.

Besides the foregoing instances of poisoning due either to toxic substances in healthy undecomposed plant and animal tissue or to a peculiar sensitive condition in a few individuals, other and more generally significant factors affecting the safety and wholesomeness of food are today matters of much concern to public-health workers. These are: (1) accidental introduction of poisonous substances into food during growth, manufacture or preservation; (2) intentional addition of preservatives to food; (3) contamination with pathogenic microbes through the agency of convalescents or germ carriers engaged in the preparation, transportation or serving of food; (4) infection with microbes or intoxication with the products of microbes present in the bodies of food animals; (5) the deleterious action of poisonous substances formed in partly spoiled or decomposed food.

(1) *Accidental Introduction of Poisonous Substances in Food.*—Even so powerful a poison as arsenic may find its way into widely used food substances. A recent instance is the arsenical poisoning due to cocoa prepared by a well known English firm. On investigation the potassium carbonate used to make the cocoa more soluble was discovered to contain considerable quantities of arsenic, so that the marketed product showed one-tenth of a grain of arsenic to a pound of cocoa. In Germany, potash that had been used in making Pfefferkuchen was recently found to contain arsenic. In this case the arsenic was attributed to potash manufactured from the wool fat of sheep that had been treated with arsenic containing mixture to prevent scab. In 1900 an extensive epidemic of peripheral neuritis in the English Midlands was traced to the presence of dangerous quantities of arsenic in beer; investigation showed that the brewing sugars contained arsenic derived from the sulphuric acid used in their preparation. Food exposed to the gases arising from the

combustion of certain coals may likewise be impregnated with arsenic. In these instances the sources of the arsenic is the same,—the iron pyrites, practically always arsenical, contained in the coal or used in the manufacture of the sulphuric acid. Since this danger became known, great care has been exercised to insure that the sulphuric acid used in making glucose shall be arsenic-free, and there is probably little cause to apprehend serious trouble from this source, although constant watchfulness is of course necessary. Still more recently considerable uneasiness has been expressed over the possibility of arsenic poisoning from fruit that has been sprayed with insecticides. The almost universal use of arsenicals in sprays for apples and other fruit trees and the fact that the presence of arsenic has been demonstrated on the skin and in the calyx of ripened apples shipped to foreign coutries have been used as arguments for placing a ban on sprayed fruits. Caseinates are used extensively in sprays as a means of inducing adhesion, so that it is difficult to free apples from the arsenical deposit. We know little or nothing about the effect on children or on weakened adults of even such small quantities of arsenic as may be present on sprayed apples, and the question should be investigated with an open mind. As yet the reported cases of poisoning from this cause do not seem to be either numerous or severe. It may well be that the lead which is also a usual ingredient of the insecticidal sprays (lead arsenate) is more of a menace than the arsenic.

Lead is, of course, a familiar and dangerous poison, but apart from lead hazards in industry, there is much less likelihood of lead getting into food and drink than there was some years ago. One of the earliest (1767) epidemiological investigations—that upon the famous Devonshire colic due to the action of cider on lead containers —was not without its lesson, and today lead poisoning except in certain trades is a comparative rarity. The use of lead service pipes for water supplies has been largely abandoned, and the lead glazes once commonly used in enameled cooking dishes are now rarely employed. Soluble lead glazes if used are, however, undoubtedly dangerous. The recent study by Monier-Williams for the British Ministry of Health showed that prolonged action of citric acid on lead-glazed cooking ware, mostly imported from France, resulted under certain conditions in a marked degree of contamination. The particular enamel employed in the cooking ware so widely used at present in the United States is lead-free, and is probably quite safe. Monier-Williams considers that "the probability that undesirable constituents in significant amounts may be dissolved from enamel

hollow ware during the ordinary processes of cooking may be regarded as remote."(5)

The great modern development in the consumption of canned foods has caused alarm to be expressed lest tin poisoning might result from corrosive action upon the tin container. It is well established that tin may be dissolved by acid fruits and berries and by certain vegetables. Definitely traced cases of tin posoning due to this cause are, however, very few in number, and most sanitarians who have investigated the matter agree with Lehmann(6) that the amounts of tin ordinarily present in canned foods are not of sanitary significance. The increasing employment of lacquered or enamel-lined cans for those fruits and vegetables especially liable to attack tin has probably reduced this danger to a minimum.

On the other hand, prolonged and frequent contact of copper with food and drink must be looked upon with concern. Although recent observations(7) contradict the reports of Mallory(8) and others that feeding copper salts causes pigment deposits in the liver of the rabbit, there can be little doubt that copper is a protoplasmic poison and as such should not be indiscriminately ingested. The employment of copper sulphate to color canned peas, string beans or other green vegetables should be prohibited generally, as it is now in Denmark, the United States and recently Great Britain.

On the whole, is is probably true that the accidental or incidental contamination of food with poisonous metallic substances is not increasing at the present time. The ordinary sources of danger are today fairly well known, and adequate precautions are being taken voluntarily or are prescribed by official regulation. New difficulties may arise, as exemplified in the case of sprayed fruit, and wherever the human element is involved we shall always have to reckon with stupidity and carelessness. As a public health problem, however, this aspect of food poisoning is better in hand than some of those to be presently considered.

(2) *Food Preservatives.*—From time immemorial the human race has been confronted with the need of carrying over food from a period of superabundance to a period of scarcity. Primitive methods of drying, smoking and salting have been largely supplemented by modern methods of refrigeration and canning. The deliberate addition of chemical substances to food for the purpose of impeding or preventing spoilage is liable to abuse. Since food spoils because of microbic action, the preservative substances must be of a kind and amount to restrict bacterial development; hence they partake of the nature of general protoplasmic poisons. Unfortunately, it is not

easy—witness the laborious experiments of chemotherapy—to find utilizable substances which are injurious to bacteria without being injurious to man. Even so, we cannot be unmindful of the force of the argument that enormous quantities of wholesome foods are now wasted by spoilage and that a really harmless food preservative is much to be desired on economic grounds.

The whole matter of chemical preservatives in food is highly complicated and the diverse opinions held demonstrate the incompleteness of our knowledge. The preservative powers of a briny solution of common salt are well know; small amounts of salt are absolutely necessary to health; large doses may be poisonous. Smoked meats and fish owe their keeping qualities to the presence of creosote and similar substances; creosote is more poisonous than some other chemical preservatives over which much controversy has raged, yet little if any hygienic objection has been offered to the use of smoked foods. It does not follow that the frequent partaking of food impregnated with creosote may not be fraught with injurious consequences.

With respect to more vehemently discussed preservatives, sanitary opinion has gradually crystallized to the conviction that the deliberate addition of formed poisons to food should be reduced to a minimum. The present conditions of commercial competition make it a great temptation for purveyors of food to avoid loss from spoilage. The easiest and least expensive way to do this is to add what seems to the average food-handler an extremely small quantity of some preservative. Some of these substances have been sold broadcast under such alluring names as "freezaline", "preservaline", etc. The use for this purpose of certain chemicals such as formaldehyde, salicylic and sulphurous acid is now prohibited by law in most cililized countries. These compounds are definitely poisonous in relatively small amounts and their injurious action in minute successive doses in animal experiments is quite marked. More debate has occurred about the use of such preservatives as boric and benzoic acids. Benzoic acid, in particular, is present naturally in many fruits and berries, notably cranberries, and while itself poisonous is converted into harmless hippuric acid when taken into the body. Nevertheless, while evidence of harm is lacking, human experiments extending over a sufficiently prolonged period and under suitably varied conditions are necessary for a competent judgment even in this most favorable instance. One unfortunate aspect of the unrestricted use of chemical preservatives is the practical difficulty of controlling the amount added. Savage(9) relates that he found as much as 96 grains of

boric acid per pound of brawn in one sample examined, and the brawn-maker, when invited to explain, stated that since the weather was rather hot he had mixed in a small handful of the boric acid as if it were so much common salt.

While the whole topic of food preservation is far too complicated to be dealt with adequately here, it seems justifiable to conclude that pending further experimental evidence of the effect on man of long-continued ingestion of even the preservatives apparently most harmless, the following procedure is warranted: (1) the use of chemical preservatives in food should be restricted as far as possible and limited at most to a few specified substances such as benzoic acid and its compounds; (2) the use of certain substances such as formaldehyde, sulphurous acid and sodium fluoride should be prohibited; (3) the permissible proportion of any authorized preservative should be prescribed by law, and all food containing preservatives (apart from salt, sugar and other listed substances) should be labeled with the kind and amount of preservative it contains.

(3) *Food Borne Infections.*—In the modern world fewer and fewer families use food that they themselves have produced. Food today is ordinarly brought to the consumer from great distances and on its way is handled by many people unacquainted with the most elementary bacteriological technique. Fortunately the life of most pathogenic microbes outside the human body is brief, otherwise infection due to food handling would be much more common than it actually is. Even so the list of food borne infections is a formidable one. Typhoid fever, the paratyphoid fevers A and B, Asiatic cholera, diphtheria, scarlet fever, septic sore throat and even, recently, acute poliomyelitis have all been traced to the contamination of food by human agency. As might be expected, milk—highly nutritious for microbes as well as man—is the vehicle in many cases, but numerous other foods have been implicated, particularly those commonly consumed uncooked. A remarkable instance of a food-borne scarlet-fever outbreak in three Massachusetts towns has lately been reported.[10] The epidemic was traced to the lobster meat in a lobster salad served by the same caterer at banquets in Weymouth, Lynn and Salem.

Oysters and other shellfish, celery, lettuce, watercress and similar foods which may come in contact with sewage-polluted water or other source of contamination and then are eaten without adequate purification have been shown by more or less convincing evidence to give rise to typhoid infection. In large cities the danger of milk-borne infection has been almost entirely done away with by the

practice of pasteurization; the relatively higher incidence of typhoid at the present time in smaller communities is probably due in large part to the proportionally greater use of raw milk. The oyster industry has recently been subjected to special overhauling following serious outbreaks of typhoid attributed to oysters in New York, Chicago and other large cities in the United States. Control measures have been instituted by State and Federal agencies with the complete cooperation of the oyster dealers, and it is believed that effective safeguards can be, in a considerable degree have been, established. The attempt to form a judgment as to the sanitary quality of shellfish merely by the shellfish score—that is—the relative number of B. coli, seems to be unwise and likely to lead to erroneous decisions. The interpretation of the shellfish score can only be made properly with a full knowledge of the sources and history of the shellfish. Direct inspection and supervision of the oyster beds themselves must, I believe, be our main reliance in preventing oyster contamination.

The likelihood of occasional food contamination conveyed from the hands of typhoid or other germ carriers engaged in preparing or serving food is too familiar to call for extended comment. Difficult administratively as is the control of typhoid carriers, the outlook on the whole is distinctly hopeful. The effect of our preventive measures is cumulative. As typhoid decreases, the number of carriers automatically diminishes. In the United States, we are manufacturing only one-tenth the number of typhoid carriers that we were two or three decades ago. The carrier problem is bound to become less serious.

Great strides too have been made in the matter of general cleanliness in food handling. The traditional peck of dirt which as children we were told appallingly that sooner or later we should have to eat has shrunk to manageable dimensions. The civilized races have—perhaps rather suddenly—grown fastidious. We no longer view with equanimity the tossing of loaves of bread from street wagons by one dirty hand to another. We are not indifferent to flies in the restaurant milk pitcher or to the saliva-moistened finger of handlers of bakery goods. Street dust, now that our imagination correctly pictures its composition, is no longer viewed as a necessary garnishment of food stalls. It is well sometimes to remember that part of the increased cost of our food at the present day is the price we are paying for increased cleanliness and for increased safety from food-borne infections. Pasteurized milk and wrapped bread are worth more than the same foods laden with intestinal microbes.

(4) *The Relation of Animal Infections to Food Poisoning in*

Man.—A particularly interesting and significant phase of the food-poisoning problem concerns the connection between various animal infections and certain human food-borne infections or intoxications. Well known diseases of the domestic animals, such as tuberculosis and foot-and-mouth disease of cattle are sometimes transmitted to man, notably by the medium of milk. Here also the pasteurization of milk constitutes an invaluable safeguard. One class of animal diseases is especially conspicuous in its relation to human food poisoning. This is the group of paratyphoid-enteritidis infections. Many animal species are liable to attack. Cattle, sheep, horses, swine, rabbits, guinea pigs, rats, mice—all suffer at times from infection with members of this group and are sometimes subject to great epidemics. Barnyard fowls such as chickens and ducks also have their paratyphoid diseases. The bacilli causing these infections are closely related to the typhoid bacillus, but may be readily distinguished by appropriate tests.(11) Most of the meat-poisoning outbreaks in Great Britain and Continental Europe have been traced to animal infections of this class. The symptoms are characteristically gastro-intestinal so that the diagnosis of "food poisoning" is naturally and commonly made. The discovery that meat-poisoning might be due to paratyphoid bacilli was made by Gäftner in 1888 in the investigation of an outbreak in the small German village of Frankenhausen. The outbreak was traced to the use of meat from a cow slaughtered because she was ill with a severe enteritis. Fifty-eight persons were affected; one died. Gärtner isolated from the spleen of the fatal case and also from the flesh and intestine of the cow a bacillus to which he gave the name B. enteritidis. Since that time numerous meat-poisoning outbreaks have besen associated with the presence of organisms of this group.

The typical outbreaks that I have cited elsewhere(12) may be quoted in illustration:

Kaensche[13] describes an outbreak at Breslau involving over eighty persons in which chopped beef was apparently the bearer of infection. The animal from which the meat came had been ill with severe diarrhoea and high fever and was slaughtered as an emergency measure *(notgeschlachtet)*. On examination a pathological condition of the liver and other organs was noted by a veterinarian who declared the meat unfit for use and ordered it destroyed. It was, however, stolen, carried secretly to Breslau, and portions of it were distributed to different sausage-makers, who sold it for the most part as hamburger steak *(Hackfleisch)*. The meat itself presented nothing abnormal in color, odor or consistency. Nevertheless, illness followed in some cases after the use of very small portions. With some of those affected the symptoms were very severe, but there were no deaths. Bacilli of the *Bacillus enteritidis* type were isolated from the meat.

A large and unusually severe outbreak reported by McWeeney[14] occurred in November, 1908, and among the inmates of an industrial school for girls at Limerick, Ireland. There were 73 cases with 9 deaths out of the total number of 197 pupils. The brunt of the attack fell on the first or Senior class, comprising 67 girls between the ages of thirteen and seventeen. Out of the 55 girls belonging to this class who partook of beef stew for dinner 53 sickened, and 8 of these died. One of the two who were not affected ate the gravy and potatoes but not the beef. Some of the implicated beef was also eaten as cold meat by girls in some of the other classes, and also caused illness. Part of the meat had been eaten previously without producing any ill effects. "The escape of those who partook of portions of the same carcass on October 27 and 29 (five days earlier) may be accounted for either by unequal distribution of the virus, or by thorough cooking which destroyed it. Some of the infective material must, however, have escaped the roasting of the 29th, and, multiplying rapidly, have rendered the whole piece intensely toxic and infective during the five days that elapsed before the fatal Tuesday when it was finally consumed." The animals from which the fore quarter of the beef was taken had been privately slaughtered by a local butcher. No reliable information could be obtained about the condition of the calf at, or slightly prior to, slaughter. The meat, however, was sold at so low a price that it was evidently not regarded as of prime quality. In this outbreak the agglutination reactions of the patients and the characteristics of the bacilli isolated showed the infection to be due to a typical strain of *Bacillus enteritidis*.

It is highly significant that most outbreaks of this type have been due to the use of meat from an animal known or suspected to be ailing at the time of slaughter. The thrifty European peasant, noticing that one of his farm animals is out of condition, kills it "to save its life" and avoid loss. The recognition that the bodies of animals dying a natural death are not altogether fit for food is very ancient. The 21st verse of the 14th chapter of Deuteronomy reads: "Ye shall not eat of anything that dieth of itself: thou mayest give it unto the sojourner that is within thy gates that he may eat it; or thou mayest sell it unto a foreigner." It is also noteworthy that raw or partly cooked portions of the animal have proved more likely to cause illness than thoroughly cooked portions and that there is often no appearance of abnormality in the meat itself. This latter circumstance greatly increases the difficulty of preventing food poisoning by any system of meat inspection. In the Ghent outbreak of 1895 a veterinary surgeon who was a slaughterhouse inspector was firmly convinced that the meat which he had passed could have had no connection with the outbreak and ate several pieces to demonstrate its wholesomeness, an experiment that had a tragic ending, as the inspector was shortly afterwards attacked with severe choleraic symptoms and died five days later, paratyphoid bacilli being found at necropsy.

For a time great confusion existed about the bacilli responsible for the various outbreaks of meat poisoning, many writers, particularly certain German investigators, confounding the meat poisoning bacilli with the bacilli that cause human paratyphoid fever of the B type. In an attempt to clear up some of the uncertainties, I have had under investigation for a number of years a collection of several hundred strains of exactly known origin. It appears to be true that the majority of meat-poisoning outbreaks that have been adequately investigated bacteriologically are due to one of two organism—either to B. enteritidis (Gärtner's bacillus) or to B. aertrycke, an organism that is closely related to B. paratyphosus B. (B. schottmülleri) which causes paratyphoid fever, but is cleary separable from it by cultural and immunological tests. English bacteriologists for a time mistook B. aertrycke for B. cholerae suis, a common member of this group in swine. A certain specialization with respect to animal hosts seems to be in progress in this large and unstable group of microbes. B. paratyphosus A infection, so far as known, is confined to man; B. abortivo equinus to the horse; B. cholerae suis to swine. B. aertrycke is less restricted; it seems to be commonly present in infections of calves and adult cattle, but has been also found in sheep epidemics and in some rodents. B. enteritidis has been found in cattle, in swine and in rodents. B. paratyphosus B (B. schottmülleri), like B. paratyphosus A, has been isolated chiefly in connection with human paratyphoid fever, but—or an organism hardly separable from it has been occasionally isolated from swine although it is not known to cause natural infections in those animals.

In their relation to food poisoning, as has been said, the two most important types appear to be B. enteritidis and B. aertrycke; B. cholerae suis has been found in a few cases; the equine strain has never been definitely reported in connection with food poisoning, although at last two outbreaks due to horse meat have been recorded in Germany.(15) The two human types (A and B) sustain the same relation to human infection that the typhoid bacillus does—that is, they are of human rather than animal origin and when they cause disease in man seem to be always derived from human sources. So far as epidemiological significance is concerned, they thus occupy a position somewhat apart from the other members of the group.

To the two types of infection with members of the paratyphoid group already mentioned—(1) human infections transmitted by direct or indirect contact; (2) food derived from animals suffering from paratyphoid infection must be added a third, (3) food contaminated by animal carriers of paratyphoid bacilli. Examples of this type

occur in the contamination of food by rats and mice. These rodents are liable to individual infection and also to extensive epidemics, sometimes from B. enteritidis, sometimes from B. aertrycke. Among these animals in a state of nature, carriers are found not infrequently. Elizabeth Verder in the University of Chicago, who has examined the bodies of 114 freshly caught wild rats in localities where rat virus had not been used isolated B. enteritidis in five animals, B. aertrycke in one.

Direct evidence of rodent contamination in a given case of food poisoning may be difficult to secure. The relatively large number of outbreaks of food poisoning with which B. enteritidis or B. aertrycke has been found and in which careful investigation has failed to secure evidence either of human-contact infection or of the use of food from ailing animals leads, however, to the suspicion that the contamination of food by rats and mice may be of fairly frequent occurrence. The suspicion is strengthened by the existence of a number of recorded instances in which the careless use of bacterial rat viruses has led to outbreaks of food poisoning. It may be added parenthetically that the use of these viruses has not proved of material value in the destruction of rodents and is open to the serious sanitary objection that the animals after apparent recovery may continue to carry the bacilli of the virus and so contaminate food. On the whole, the agency of rats and mice in causing food poisoning outbreaks has hardly been given due weight. It will be found the most plausible hypothesis in numerous instances where other sources can be definitely excluded.

Perhaps the best way of preventing those types of food poisoning that are due to an infected condition of food animals is, first of all, inspection of the living animals giving milk or destined for slaughter. If this is done, ailing animals will in most instances—although not invariably—be detected. In any event, examination of the live animals is likely to be much more efficacious in discovering infection than examination of the milk or meat. An additional safeguard—and one which is desirable on other grounds—is the thorough cooking of all foods of animal origin. The protection of food, cooked or uncooked, from occasional contamination with vermin is not an easy matter either for country or city dwellers. It is, however, a question that will undoubtedly receive more attention in the near future from health authorities, particularly as regards conditions in restaurants, hotels and public institutions. The use of bacterial rat viruses should be prohibited.

(5) *Food Spoilage.*—It has long been a seductive hypothesis that

during the course of the decomposition of food substances, poisons are generated and that much so-called food poisoning is due to the poisonous products of bacteria. This opinion has found expression in many civilized countries in official enactments prohibiting traffic in decomposed meats; vegetables and fruits. The interpretation of the conventional legal phraseology has, however, given full play for expert ingenuity. Just what constitutes a foodstuff "filthy, decomposed or putrid" is a question on which there may be totally different conceptions in the minds of witnesses, experts, lawyers, judge and jury. In point of fact, as every one knows, partly decomposed foods are consumed every day, often in large quantities, without evidence of injury. Many kinds of cheese are valued chiefly for the particular decomposition products that they contain. Well-ripened Brie, or Camembert cheeses are evidently far advanced in decomposition. The Chinese delicacy known as *pidan* consists of preserved ducks eggs that have been stored for months in a paste of lime, salt, wood ashes and tea. Much decomposition of protein has taken place in these eggs as indicated by the amount of ammoniacal nitrogen that they contain, which is considerably higher than in the eggs known to egg chandlers under the unsavory name of black rots.

It is plain, therefore, that bacterial growth in substances used for food is not necessarily injurious, and that in some cases it may increase the palatability of food without demonstrably impairing its wholesomeness. In recent years natural and artificially soured milk has been expertly recommended and widely used as a food or beverage for persons in delicate health in spite of the fact that it contains may millions of bacteria and their decomposition products.

At one time a particular class of decomposition products, the alkaloidal substances known as ptomaines were supposed to be responsible for many cases of food poisoning. These ptomaines are split products arising from the decomposition of the protein molecule; some of them are undoubtedly poisonous. It seems quite unlikely, however, that they are concerned in any great degree, if at all, in the causation of food poisoning. They do not usually begin to appear until putrefaction has been in progress a week or more. As Savage([17]) expresses it: "Under ordinary commercial conditions no one would be stupid enough to vend such food, no one would be rash enough to eat it."

It is true also that belief in the toxicity of ptomaines rests upon inoculation experiments and that, so far as I know, feeding experi-

ments with ptomaines prepared from decomposed meat have never been carried out. While the diagnosis of ptomaine poisoning has its uses as a convenient refuge from etiological uncertainty, it can hardly serve as a precise designation of an actual pathological event. It is highly significant that with the advance in our knowledge of food poisoning and its causation there are fewer and fewer cases in which the diagnosis of ptomaine poisoning is warranted. The majority of the carefully investigated outbreaks of acute gastro-intestinal disturbance of the type that at one time were denominated ptomaine poisoning are now found to be associated with the presence of bacilli of the paratyphoid group. Indeed, that ptomaines possess any practical significance has yet to be demonstrated.

In contrast to ptomaines, which are non-specific, are derived from the splitting of proteins and are not the direct product of bacterial metabolism are those poisons which, like tetanus and diphtheria toxins, are specific to the microbes that produce them. The poison ergot is generated by a fungus attacking rye, as has long been known; the use in times of famine of the rye in which this fungus growth has taken place gave rise in the Middle Ages to extensive outbreaks of a disease designated as "saint's fire" or "the fire of Saint Anthony"; over 40,000 persons were said to have perished from this cause in an outbreak in Limoges in the Tenth Century.

The question of a specific formed poison in food has come up especially in connection with the paratyphoid group. There seems little doubt that in many—perhaps most—of the food-poisoning cases associated with these bacilli, the presence or probable presence of living paratyphoid organisms can be demonstrated. There remains a residue of cases, however, in which living organisms have not been found either in the suspected food or in the tissues of the patient. There are some instances too where violent gastro-intestinal symptoms have appeared within so short a time after the incriminated food had been eaten as to suggest the direct action of poisonous products. Experiments in feeding animals with dead paratyphoid bacilli, with culture filtrates or with incriminated food have not been conclusive, although there is abundant evidence that the bacterial products when injected parenterally are toxic. Since these latter substances are heat resistant, it has been surmised, especially by Savage and White,(18) that they may persist for a long time even after food has been subjected to thorough cooking. In this way the authors cited would explain the occurrence of certain food-poisoning outbreaks in Great Britain attributed to the use of canned South American meat. The matter is extraordinarily difficult for experimental verification

since not only is the epidemiological evidence often incomplete so that there is doubt about the incriminated food articles, but also, as already stated, the feeding of animals is quite negative. The method of testing the patient's serum for its agglutinative action against suspected paratyphoid strains seems inapplicable here, since it is well established immunologically that the introduction of heat-killed (100°C.) bacilli into the alimentary tract fails to give rise to agglutinins in demonstrable quantity. There seems indeed at the present no satisfactory evidence that heat-resistant paratyphoid toxins using this term broadly are the cause of food poisoning.

At the present time the most important, as well as the best studied example of food poisoning due to a bacterial product is botulism. In this disease the active poisonous substance is a true toxin which may be produced in food substances, is destroyed by heat, is potent in extremely minute doses and gives rise to a specific antitoxin. The toxin of Clostridium botulinum is in fact strictly comparable immunologically with the toxins produced by the tetanus and diphtheria bacilli. Unlike the latter, however, it is dangerous when swallowed. Fortunately the conditions under which the botulism toxin is produced are somewhat unusual. Strictly anaerobic conditions are necessary, the reaction of the medium must be suitable, temperature and composition of the medium are important determining factors; the presence of certain saprophytic microbic species such as Clostridium sporogenes may interfere with toxin production.(19) It is not surprising, therefore, that botulism poisoning is relatively very rare despite the fact that the germ itself is quite widely distributed in nature. The name botulism (Lat. botulus-sausage) must now be recognized as somewhat inappropriate since, although some of the earliest outbreaks were traced to sausage, the majority of recent cases, at least in the United States, have been due to canned vegetables, such as corn, string beans and spinach.

Botulism has always been one of the rarest known diseases and affects only exceptionally an individual or a small group. The dramatic suddenness of its onset and its high fatality have, however, made it a conspicuous and much dreaded malady. From 1912 to 1917 records of 28 outbreaks of botulism were obtained in the United States and Canada. The greater attention that began to be paid to this disease about 1917 perhaps caused an increase in the number of recognized cases put on record. Other factors, however, may have been concerned. At all events the years 1918–1923 were marked by a considerably larger number of reported outbreaks than the preceding six years.

Number of Reported Outbreaks of Botulism

1912–1917	28
1918–1923	83
1924	8
1925	8
1926	3
1927	5

Since 1923 there has been a definite reduction in the number of reported outbreaks, there being but 16 outbreaks in 1924–1925 as contrasted with 33 for 1922–1923. In 1926 there were only 3 outbreaks reported, one due to home-canned asparagus, one to home-canned fish, one to home-canned string beans. Since 1917 our knowledge of botulism has been greatly extended by the researches of Dickson, Meyer, Geiger, Bengston, Bronfenbrenner, Esty, Dack, Starin and many others.

One of the remarkable features of botulism is its singular distribution. Of 56 outbreaks of botulism in the United States recently tabulated,(20) 49 of which occurred during 1922–1926 it is said that 46 occurred in the Western States; in 4 of the 7 outbreaks reported in the Middle West, the food was canned in the Western States. Corresponding with this distribution of cases, the specific microbe has been found with particular abundance in the soil of certain regions, notably the Rocky Mountains and Pacific Coast States; it is less frequent in the Atlantic States and relatively rare in the region of the Great Lakes and Mississippi Valley.(21) In Canada soil samples in significant numbers have been examined from only three localities: the neihborhood of Vancouver, the Canadian Rockies (Glacier, Lake Louise) and the Province of Quebec. Clostridium botulinum was found in a large proportion of the samples collected in the two former localities (31 per cent of the Glacier samples), but was absent in the 16 samples from the Province of Quebec.[22] There seems to have been but one outbreak of botulism reported in Canada(23)—near Dawson City, Yukon—and nothing is certainly known about the causative food. If as thought possible, commercially canned beets were responsible, they were doubtless prepared at some distant point.

Botulism outbreaks have been reported in Germany, Belgium, Switzerland and other European countries. The first recognized outbreak in Great Britain occurred at Lock Maree Scotland, in 1922. No systematic soil surveys have been made in Europe, but scattered observations show botulism spores to be widely distributed in the soil in Belgium, Denmark, Holland, Switzerland and England. The

question naturally arises why botulism has not occurred more frequently in England and some other countries if the spores are actually present in the soil. While the answer to this question is not wholly clear, some of the perplexing factors of botulism may be explained by the relative numbers of botulinum in different localities.

As a rule the spores are decidedly less numerous in European than in American soil specimens.(5) They are apparently more abundant in the Rocky Mountain soils than in other parts of the United States. Another significant fact is that there are two immunologically distinct varieties of Cl. botulinum designated provisionally as Type A and Type B. Type A has been much more frequently isolated than Type B in outbreaks of botulism in the United States; it also was isolated from the wild duck paste that caused the Lock Maree outbreak in Scotland. Type A is more commonly found in the soils of the Rocky Mountain region, Type B in the Mississippi Valley and the Atlantic States. Only Type B was found by Meyer in European soils. A third Type—Type C—has been especially studied by Dr. Ida Bengston of the United States Health Service.(25) It is much less heat resistant than the other strains and has not been casually associated with any of the reported outbreaks of botulism.

The interrelationship of these three immunologically distinct types of Clostridium botulinum is not clearly understood. It has been conjectured that B type is a degenerate descendant of the more highly toxic and resistant A type, but proof of this connection is still lacking. Why Type A should be so abundant in the virgin uncultivated soil of certain regions is a mystery as yet unsolved.

The association of the several botulinum organisms with various animal diseases presents an interesting but complicated problem. Varying degrees of susceptibility to the toxin of the different types undoubtedly exist, chickens, for example, being much more susceptible to Type A toxin than to either B or C. Both B and C toxins are, nevertheless, highly toxic. The disease known as "limberneck" in chickens may be caused by the consumption of the larvae of the green fly (Lucilia caesar) which contain botulinum organisms or botulinum toxin. Ida Bengtson has shown that Type C is frequently implicated in these natural outbreaks in chickens. Limberneck may also result when chickens feed upon the remnants of spoiled canned foods thrown out on the ground in the barnyard. A number of human outbreaks of botulism have been traced to their source through the evidence afforded by outbreaks of limberneck. It is still uncertain how far botulinum toxin is responsible for cases of "forage poison-

ing'' in horses and other stock animals. Observations reported by Seddon from Australia and by others in various parts of the world have not led to a unified scientific judgment. Since Clostridium botulinum is a common soil organism in many localities, its presence in the animal intestines or even in the internal organs after death is not conclusive evidence that death was due to botulinum intoxication.

The queston has been frequently asked whether Cl. botulinum is, strictly speaking, a pathogenic organism, whether it can grow and generate toxin within the animal body. The conclusion of some of the earlier investigators that this organism ''is absolutely incapable of reproducing itself in the animal body'' seems to have been overthrown by more recent and more extensive studies. Especially the experiments of Coleman and Meyer([26]) and of Starin and Dack([27]) show that carefully detoxified spores are capable of germination in the bodies of laboratory animals, that spores and vegetative forms become widely disseminated through the tissues and that toxin is formed in sufficient quantity to cause death. It seems most unlikely, however, that under natural conditions tissue invasion by living botulinum organisms plays any part in the causation of botulism in man. Massive doses must be used to produce this effect upon experimental animals and there is no authentic instance on record of the development of botulism in human beings except when formed botulinum toxin has been taken into the alimentary tract.

The symptoms of botulism are strikingly different from those of paratyphoid infection: onset is less sudden, gastro-intestinal symptoms are relatively rare, temperature is subnormal and there is seldom any abdominal pain; especially characteristic of botulism are the disturbances of vision and the difficulty in swallowing; the mortality in botulism averages over 60 per cent as contrasted with about one per cent in demonstrated paratyphoid infection. Preserved foods are to be suspected, particularly when signs of spoilage are present. If samples of the incriminated foods are available for examination, toxin tests controlled with specific antitoxin should be made.

The treatment of botulism is not on a satisfactory basis. In the first place, while botulinum toxin is unique in being the only known exotoxin that is absorbed from the digestive tract, there are many essential things about its absorption and its action that are still quite obscure. Experimental animals, although susceptible to feeding, are much more susceptible to subcutaneous or intraperitoneal injections. Different animal species evince marked differences in susceptibility, these differences being especially marked with respect

to poisoning by mouth. Mice are very susceptible to subcutaneous inoculation, but show no effects when much larger doses of toxin are given by mouth.([28]) Swine are resistant to enormous doses of toxin given by mouth; as much as 10,000,000 MLD (for mice) have been given without ill effect.([29]) Rabbits and guinea pigs are quite susceptible to oral administration. The conditions affecting the permeability of the intestinal wall to botulinum toxin are not understood. Dack and Gibbard([29]) have shown that only a very small amount of toxin could be demonstrated in the venous blood from a perfused loop of intestine containing toxin, and that there was no noteworthy difference between the highly resistant hog and the relatively susceptible rabbit in this respect.

In the second place the obstacles to successful antitoxin treatment are very great. While true, specific and—in animal experiments—therapeutically efficacious antitoxin can be produced just as in tetanus and diphtheria, the time necessary to produce an antitoxin of even moderate potency is very great, and may be as long as six months. This adds to the expense of producing antitoxin on a commercial scale and is a barrier to its general distribution. The therapeutic need of prompt administration is even greater in botulism than it is in tetanus and diphtheria since a large amount of the toxin is usually introduced into the body at one time instead of being as in the latter diseases, absorbed slowly from the site of infection. Prompt administration is however peculiarly difficult to achieve. Many of the outbreaks have occurred in thinly settled regions where botulism antitoxin is quite unavailable. Sometimes the course of the disease is very rapid; in the Lock Maree outbreak two of the victims died within 18 hours after the first appearance of symptoms. A further difficulty in providing for antitoxin treatment is that the types of organism are immunologically specific. Type A antitoxin does not protect against Type B toxin and vice versa. When botulism symptoms appear there is no time for type differentiation; to be on the safe side a polyvalent serum should be given. All these difficulties render the successful application of antitoxin treatment to botulism highly problematical. Indeed, the administration of botulism antitoxin in human cases cannot yet be said to have had a fair trial in a single instance. Other modes of treatment have been suggested. Dickson and Sheyvsky([30]) have observed that the toxins of both Types A and B exert an influence upon the endings of the motor fibers of the voluntary nervous system which leads to a marked susceptibility to fatigue. Since death from botulism is practically always due to failure of the respiratory muscles or the heart, more

often the former, any means for conserving muscular strength may be serviceable. Bronfenbrenner and Weiss ([31]) have advised that patients be given morphine for this purpose, a procedure that seems to be justifiable if dosages that tend to depress the respiratory functions are avoided. For the present, measures of prevention seem more hopeful than those of cure.

Fairly effective measures to prevent botulism may be taken. These fall into two groups, those concerned with the initial methods of preservation and those having to do with the inspection and treatment of the food before serving. Since the spores of Clostridium botulinum apparently get into foods chiefly from the soil, measures of cleanliness are strongly indicated. It may be safely predicted that painstaking attempts to free vegetables from soil particles before canning will be rewarded by a greater freedom from botulism. In those regions where botulism spores are particularly numerous in the soil such precautions are especially necessary. The most serious difficulty is due to the high heat resistance of the botulinum spore. While the majority of spores are killed at the ordinary temperature at which foods are canned or processed, certain strains apparently possess an abnormally high resistance and may survive the usual heating procedure. Esty([32]) found that the heat resistance of 81 Type A strains at 105°C. varied from 3 to 75 minutes. It is evident that at high altitudes where water boils at temperatures considerably under 100°C. especial care is necessary in attempting to process foods by heat. Experiments upon the degree of heat and time of exposure necessary to kill even the most resistant forms and upon the heat penetration in different foods have shown ways of reducing the likelihood that any botulinum spores will survive large-scale, carefully controlled processing.

The second group of safeguards is likewise quite efficacious. Nearly all—some investigators would say all—canned foods implicated in botulism afford to the senses unmistakable evidences of being spoiled. There is no doubt that the instant rejection without tasting of any preserved food that gives the slighest indication of spoilage would practically do away with the menace of botulism. Finally it is worth noting that the botulism toxin is readily destroyed by boiling and that in consequence the custom—already followed quite generally—of heating canned foods before serving interposes still another barrier to botulism poisoning. Rare as botulism is at the present day, there is the best of reasons for believing that it can be entirely eliminated by application of the measures just described. The progress already made is most encouraging.

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