FOOD POISONING IN PUERTO RICO

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In this paper, the term "food poisoning", will be used to indicate gastro-intestinal or other specific symptoms produced as a result of deleterious substances of varied nature and origin which are either naturally present in the food or which may have gained access to it through different channels before or after its preparation. In our tropical island, food poisoning is common; all the ideal elements necessary for the occurrence of these conditions are present throughout the whole year. The public sale of popular food products—fritters of all kinds, fried meats, sausages, and the like—by street vendors at open stands, which viands are handled by the buyer and the seller and exposed continually to flies, affords ample opportunities for contamination and transmission of disease to other individuals (1, 2).

Food poisoning has been known in this island for a long time, but it was not until recent years (3) that the actual cause of many of these pathological processes were found to be more or less the same as in temperate climates where the conditions have been thoroughly studied (4, 5, 6, 7, 8, 9, 10, 11, 12).

In the early days of the Spanish colonization in Puerto Rico (15th century) diarrhea was considered as one of the gravest problems with which the settlers were confronted (13). Its actual cause was unknown but the disease invalidated and killed many individuals. Excess in food, in alcoholic beverages, and poor attention to mild intestinal disorders, were then considered as predisposing causes. Dysentery was also very prevalent among the Indians and early settlers of Puerto Rico, especially so after hurricanes, when great famine occurred and conditions were so altered as to make the transmission of pathogenic organisms from man to man easier.

* Presented at the School of Tropical Medicine in the Seminar of April 6, 1933.
Received for publication April, 1933.

During the epidemic of bacillary dysentery which followed the big hurricane of 1928 (San Felipe), the opinion was advanced by certain people that this was an epidemic outbreak of food poisoning and not dysentery. A mere epidemiological study of such an outbreak rules out this erroneous statement, which was based on personal appreciation and not on scientific evidence; the clinical and bacteriological studies carried out prove conclusively that the epidemic was one of bacillary dysentery produced by an organism of the paradysentery group, Flexner type (14) epidemic, which reappeared after the hurricane of 1932 in practically the same region and with similar epidemiological and bacteriological findings.

The first historically authentic incidence of food poisoning in Puerto Rico is recorded in the year 1824 when several cases produced by eating crab meat happened in different localities of the island (15). Several deaths occurred as a result of the poisoning, and the prevalent idea at the time was that the crabs had eaten the fruits of the common manchineel—(manzanillo), and thus became poisonous. In Peñuelas four deaths were recorded at intervals of 8, 12, 14 and 15 hours after ingesting crab meat. In Guayanilla there also occurred one death 8 hours after eating crabs. The symptoms in all these cases began from 4 to 8 hours after partaking of the crabs and were characterized chiefly by abdominal pains, rapid and weak pulse, cold and clammy skin, thirst, occasional convulsions, pain in the extremities and joints of the body, and as a rule, nausea and vomiting.

The cause and mechanism of food-borne infections and intoxications has been the subject of many investigations which can be found in the medical treatises of recent years (16, 17, 18).

The following brief outline, by no means complete, gives an idea of the numerous and varied agents which may give rise to food poisoning.

I. FOOD INFECTIONS

1. BACTERIA

a. Organisms of the *Salmonella* group (paratyphoid or food poisoning group) universally accepted as incitants of food infections. Other bacteria whose status as incitants of true food poisoning is as yet under scientific discussion.

b. Bacterial products (toxins) such as botulinus toxin and products of the staphylococcus.

c. Other bacteria which produce specific pathologic conditions—not true food poisoning—which may gain access to the human body through foods.

2. FUNGI, which by products of metabolism or through their action on certain foodstuffs may cause food poisoning.

II. FOOD INTOXICATIONS

1. ACCIDENTAL OR CRIMINAL. Poisoning produced by the presence of deleterious products in food.
2. NATURAL. Produced by poisonous substances contained in food products, whether animal or vegetable in origin.

III. FOOD ALLERGY

IV. UNDETERMINED ETIOLOGICAL AGENTS

At present, gastro-intestinal conditions, acute and chronic, accompanied by diarrhea, constitute one of the main public health problems in the Island of Puerto Rico. In our mortality statistics, under the term "Diarrhea and enteritis" are included all sorts of intestinal infections, as well as the true food infections and intoxications.

How frequently food poisoning actually occurs in Puerto Rico is unknown as it is not included at present in the list of reportable diseases, but in the new regulations on Transmissible Diseases pending approval by the Insular Board of Health, it is included as compulsory information by physicians. In America, cases of food poisoning are required to be reported in eight states: Georgia, Illinois, Kansas, Maryland, Missouri, Montana, New Mexico and Ohio (19). Botulism is of compulsory notification in eleven states and also in Puerto Rico.

The term "Ptomaine poisoning", as in other places, continues to be popular with many physicians who use it vicariously in relation to food poisoning. The term is in disuse in most countries, and is being replaced by the more specific terms of food infection or intoxication. Ptomaines are basic alcaloidal substances derived from the disintegration of proteins; they appear in food products only in the latter stages of putrefaction, thus it is easy to understand why true ptomaine poisoning is rare or almost unknown. This is one reason why it is recommended to discontinue the use of the term "Ptomaine poisoning". It has also been definitely demonstrated that in most instances of the so-called ptomaine poisoning, the responsibility can be placed on other definite

etiological factors(19, 20). In a special article of the Journal of the American Medical Association (4) there appears a careful analysis of a number of deaths diagnosed as ptomaine poisoning; 43 autopsies were made, and it was found that the original diagnosis had to be changed in every case to that of peritonitis, appendicitis, malaria, tuberculosis or some other condition. In spite of all this, the term continues to be popular and is often erroneously used.

The committee of the American Public Health Association in dealing with the question "When are meats or fish spoiled" declares frankly that "it is extremely difficult to tell with accuracy when food is spoiled or decomposed" (19). Many attempts have been made to find some relation between the numbers of bacteria present and the quality of certain foods, particularly meat, but so far without much success. Chemical criteria of the presence of injurious decomposition are likewise lacking. Most observers agree that, in order to interpret correctly the results of either bacterial or chemical examination, it is necessary to know the sanitary history of the food in question(19).

BACTERIAL INFECTIONS

Under this heading which includes the commoner and most important cases of food poisoning may be included the following:

1. Specific Infections: In this group are often included a certain number of specific diseases—not true food poisoning —whose causative organism may be transmitted to the human body through the agency of food. Among these, the most important are typhoid fever, asiatic cholera, bacillary dysentery, tuberculosis; a group of diseases which may be transmitted through milk but which apparently do not constitute serious problems in Puerto Rico; also other diseases caused by specific microorganism such as scarlet fever, septic sore throat and diphtheria, all of which may attack man through food agency.

2. True Food Infections: In this group are included gastrointestinal conditions which can be traced to organisms of the paratyphoid (*Salmonella*) group which reach the body of man through foods. Some of the organisms of this group are primarily animal pathogens and reach man by means

of contact through carriers, or material infected with the etiological agent. The actual mechanism in the production of symptoms is still a matter of discussion; some investigators have evidence which suggests that they may be actual infections brought about by the colonization and growth of these bacteria in the intestinal tract(21).

In the true sense of the word all the organisms classified under the *Salmonella* group do not cause true food poisoning. The well known *Bacillus paratyphosus A* (*Salmonella paratyphi*) and *B. paratyphosus B* (*Salmonella schottmülleri*) produce paratyphoid fevers which can be acquired through food, but which are infections in many respects similar to typhoid fever, and cannot be grouped as true food poisoning. *B. enteritidis* (5, 22, 23, 24) and *B. aertrycke* (25), similar in many respects to the schottmülleri type, and the *Cholera suis* (*suipestifer*) *bacillus* (26, 27), are the chief producers of true food poisoning. Other bacilli (28, 29, 30) have again and again been implicated as incitants of food poisoning, among which *B. proteus* (31, 32, 33) tops the list. A critical examination of the evidence submitted in favor of *B. proteus* is not convincing; such is the opinion of two authorities on food poisoning, Jordan (19) in the United States and Savage in England (34), who consider that no toxic products of these organisms capable of producing symptoms have as yet been definitely demonstrated. Filtrates of the organisms, heated or unheated, do not seem to produce gastro-intestinal symptoms when given to human volunteers or monkeys (35). The chief organisms of the *Salmonella* group which are capable of producing food poisoning are not generally parasitic to man, but when so localized, are mere accidental invaders. They are, however, parasitic in other animals; *B. aertrycke,* found in parrots, as in psittacosis (19); *B. enteritidis* occasionally found in rats (36, 37), and *B. cholera suis* found at times in hogs (19). These organisms are sometimes found in ducks' eggs (38), and at times in infected meat.

B. dysenteriae Sonne (39) has been recently accused of causing an outbreak of true food poisoning due to the eating of fish cakes contaminated with such organisms.

Among the cases of food poisoning in Puerto Rico which we have found to be caused by organisms of this *Salmonella* group, might be briefly mentioned the most important ones:

The first case (3) was that of a white man, a laborer, 28 years old, healthy and well up to November 27, when he ate supper at a friend's home. The meal consisted of lobster and coffee, the lobster having been prepared the previous day and kept at room temperature until it was consumed. Twelve hours after partaking of this meal the patient was seized with violent abdominal cramps, so intense that he could not touch the abdomen or even stand a hot water bag over it. Soon diarrhea appeared, the stool being liquid, accompanied by tenesmus and pain. There was very slight fever, 37.6°C. during the attack. In two days the patient had entirely recovered. The stool was liquid and very foul with considerable mucus and specks of blood. Large amounts of thread-like material resembling bits of intestinal mucous membrane were noticed. Microscopic examination revealed large numbers of neutrophilic polynuclear leucocytes, many intact red blood corpuscles, and enormous numbers of epithelial cells of the columnar and stratified squamous types. No ova or parasites of any kind were found. Bacteriological examination revealed a non-lactose fermenting, Gram negative bacillus which we classified as *Salmonella suipestifer* from its morphological characteristics, cultural reactions, and agglutination tests. The blood of the patient agglutinated *Salmonella suipestifer* * in dilutions 1:150 and *Salmonella enteritidis* only in dilutions as high as 1:50.

It was impossible to get a sample of the lobster for examination, and all data available was obtained through the patient, since the other people affected refused to be examined or to answer questions. There were three persons besides the patient present at the meal and all showed the following symptoms:

One developed abdominal pains and diarrhea twenty hours after the meal. There was no blood or mucus in the stool.

Another developed slight pain, nausea and diarrhea thirty hours later.

The third had mild pains in the abdomen, slight diarrhea, and weakness. Symptoms appeared twelve hours later but lasted only a few hours.

A second group of cases (3) came under our observation

* Culture supplied to us by Dr. John Reichel of the then Mulford Laboratories of Philadelphia.

during April 1928. A dinner party was held at one of the clubs of San Juan at which there were eight persons present, but other people at the Club were served the same food with the exception of one or two items. Next day, i.e. about 18–20 hours after the dinner, four of the eight present at the party became ill. The symptoms were more or less alike though differing in severity. They experienced malaise, slight headache and weakness. Soon there was griping and diarrhea, which were the most pronounced symptoms. The stools were liquid, foul and contained slight bits of mucus, large numbers of polynuclear neutrophiles, abundant epithelial cells but no blood. The stools from all four cases were examined bacteriologically. Three of the stools revealed the presence of an organism of the *Salmonella* group which we classified as *S. enteritidis* from its morphological characteristics, cultural reactions and simple agglutination tests. In the other case no organism of the *Salmonella* group could be detected in spite of repeated trials.

A complete epidemiological study was not made. It was impossible to obtain any of the food served at the dinner for examination. The stools from all the help employed at the Club were examined bacteriologically in an attempt to determine a possible carrier, but the results were negative.

The third group of cases occurred in San Juan (3). These really occurred in two separate outbreaks, the first in March 1928 and the second in May of the same year, but since the source of infection was supposedly the same, the two are considered together. In the earlier group (March) there were altogether 25 cases occurring among five families. Two small dogs belonging to one of the families also ate fish and died with symptoms of food poisoning. The fish eaten by all these five families was traced to the same source. Some of the original lot of fish consumed was obtained for bacteriological examination and a Gram negative bacillus was isolated which was classed as *S. enteritidis* from its cultural characteristics and agglutination reactions.

The second outbreak was seen during the early part of May 1928, in the city of San Juan. All the cases occurred in people who ate regularly at the same place, a small restaurant which catered to 45 customers. Of the 45 who ate fish, 29 became ill, 13 remained well, and 3 could not be traced. This

fish came from the same source as in the March outbreak, but no sample of it could be obtained since all had been consumed; however, samples from a new lot were secured and examined bacteriologically. No organisms of the *Salmonella* group were found.

No feces could be obtained from any of the patients in this last group, but blood was later obtained for agglutination tests from ten of the cases.

In 50 per cent of the cases examined, agglutinins for *B. enteritidis* or some organism of the *Salmonella* group were detected.

The symptoms following the ingestion of fish were alike in both groups. From two to twenty hours after the ingestion of the fish the patient would notice a feeling of distress in the epigastrium, slight malaise and headache, chilliness, or at times a violent abdominal pain followed by griping and diarrhea. Later there was a feeling of thirst and intense weakness which would persist for quite a long time. There was very little fever. It was common to observe pain in the back, knees and other joints of the body. None of the patients noticed blood in the stools. The bowel movements varied from 3 to 30 in 24 hours.

3. Bacterial Toxins (or products). Infection produced by toxins or bacterial products, among which must be specially mentioned botulism and intestinal disorders produced by toxic products of the staphylococcus. Lately toxic products of an organism of the *aerogenes cloacae* (40) group have been implied as causing a large outbreak of food poisoning.

BOTULISM

Botulism is one of the outstanding examples of true food poisoning in which toxic products—a toxin elaborated by the organism outside of the body—reach the human intestines in foods (41, 42, 43, 19, 44, 45, 46, 10, 47, 48, 49, 50, 51). The condition is a readily recognizable one on account of the characteristic symptomatology which accompanies it. In cases of this form of food poisoning the nervous system is affected in a characteristic way; there is dizziness, double vision, difficulty in swallowing, and definite involvement of the nerve elements which persist for some time. There is progressive muscular weakness, subnormal temperature as a rule, absence of pain or other abdominal symptoms, with con-

stipation as the rule. The visual disturbances are characteristic. The mortality is high, generally between 80 and 85 per cent. If the causative food is subjected to examination, *Bacillus botulinus* and its toxin can be demonstrated. The symptoms are produced by a soluble exotoxin elaborated by the organism. This toxin has the property of traversing the gastro-intestinal mucous membrane and reaching the nervous system, thus giving rise to the characteristic symptomatology.

Judging from statistics, botulism does not appear to be very common in Puerto Rico. There is no reason whatsoever why the condition should not occur in this Island where food products are sold in a popular way which offers all the ideal requirements for the development and growth of the causative organism (52), but so far no case of botulism has come to our notice since we have been interested in this matter.

STAPHYLOCOCCI

Poisoning by toxic substances elaborated by staphylococci present in food products, has gained scientific recognition since the studies carried out by Jordan and other investigators of the University of Chicago (53, 19, 54, 55, 56, 57). These studies leave little doubt as to the rôle of this important group of bacteria in the etiology of food poisoning. The toxic substances that give rise to the symptoms are only produced in so far as has been determined by the hemolytic variety, although the food poisoning substance (enterotoxin) is different from the hemolytic substance (58, 59). The enterotoxin is not however found in all strains of hemolytic staphylococci. When filtrates of the organisms are given to human volunteers they give rise to typical symptoms. The toxin as a rule is present in the food which is ingested, and is directly responsible for the production of symptoms. Woolpert and Dack (60) of the University of Chicago have been able, by growing staphylococci under special conditions, to obtain a toxin which will cause typical reactions when given to monkeys. Due to the distribution in nature of the hemolytic staphylococcus these organisms are of the utmost importance in regards to food poisoning. Barber (61) was the first to call attention as to the possibility of the staphylococcus as an incitant of food poisoning.

POISONING BY CHEESE

Outbreaks of food poisoning ascribed to cheese are very common in Puerto Rico and samples of cheese are frequently being sent to the public health laboratories for bacteriological examination.

In February 1930 there occurred an outbreak of food poisoning in Yauco produced by eating cheese. Four persons were intoxicated, but no death was registered and the patients recovered promptly without any untoward effects. Symptoms consisting chiefly of dizziness, vomiting and marked diarrhea without blood or mucus appeared two to four hours after ingestion of the food. Examination of this cheese revealed the presence of a hemolytic staphylococcus. The organism was also isolated from the cheese by Professor Jordan of the University of Chicago who found out that filtrates of this organism produced symptoms similar to those of the intoxication when given to human volunteers (54, 55).

During August 1930 there occurred another case of poisoning produced by cheese. This happened in the town of Aguadilla in six people of the same family who ate cheese in a restaurant in Arecibo. All six persons ate the cheese about 7:00 P. M., and between 10:00 and 11:00 P. M. the symptoms of intoxication started. These were vomiting, pain, a feeling of collapse, and diarrhea, which lasted practically all the night and subsided next day. In one case there was no vomiting. This was a woman sixty years old who had severe symptoms, intense pain in the abdomen, violent diarrhea and was very toxic. All the patients recovered. Bacteriological examination of samples of the cheese revealed the presence of a hemolytic staphylococcus whose filtrate was toxic when given to human volunteers.

During July 1930 there was an outbreak of food poisoning in the city of Humacao. In this outbreak there were eight persons affected. From three to four hours after eating cheese all the persons suffered with vomiting, abdominal pain, tachycardia, headache, a feeling of weakness with cramps in the muscles, articular pain and profuse diarrhea. The symptoms subsided after six hours. Bacteriological examination of this cheese revealed the presence of a hemolytic staphylococcus. Unfortunately the toxicity of this organism was not tried upon human volunteers.

OTHER OUTBREAKS

During May 1932 we studied a case of food poisoning produced by eating sweet potato (batata) candy. In this instance only one case occurred, an adult male who suffered with nausea, abdominal cramps and slight diarrhea. These symptoms occurred two hours after the candy was ingested and subsided after 6 hours. Bacteriological examination of the candy revealed the presence of a hemolytic staphylococcus.

POISONING WITH HAM

During the month of April there occurred an outbreak of food poisoning among 3 people, the apparent cause being a ham (62) which had been boiled and used for two or three days previously. It was served at breakfast to four people on the day before the attack without their noticing any untoward symptoms. Next day, however, six people were at breakfast, three of whom ate the ham, and soon (two hours) afterwards developed symptoms of severe intoxication. The three people who did not eat of the ham were not ill. The symptoms were vomiting, nausea, severe abdominal pains, a sensation of oppression of the chest, clammy perspiration and diarrhea. Chemical examination of the ham revealed the presence of ammonia, according to the report of the Director of the Chemical Laboratory of the Health Department, who thought this was indicative of a certain degree of putrefaction in the ham. Bacteriological examination of several portions of the ham revealed the presence of a hemolytic staphylococcus and a *bacillus coli communior*. Several laboratory animals (guinea pigs and white rats) were fed with the ham and with extracts of the ham without visible effect. Filtrates of 48-hour old growths of the staphylococcus and *B. coli* isolated were administered to guinea pigs and injected intraperitoneally without any undue effects whatsoever. The filtrate was not tested on human volunteers.

FUNGI

There is a definite type of poisoning produced by rye known as ergotism or "Saint's fire". This is due to the action of a fungus, *Claviceps purpurea* (63, 19) on the rye. Fungi have occasionally been implied as causing food poisoning (64) but this question has not yet been definitely established.

FOOD INTOXICATION

1. **Accidental** or **criminal poisoning** produced by the presence of deleterious products in food is just as common in Puerto Rico as in other parts of the world and criminal attempts to produce death by using the different poisonous salts of the heavy metals are not unusual occurrences. An interesting case of such poisoning in Puerto Rico has been reported by Costa Mandry and Garrido (65). It consisted of the poisoning of several members of a family by arsenic contained in fritters. Out of 14 persons in a family, 12 ate the fritters and developed symptoms of poisoning. Chemical examination revealed the presence of arsenic in the fritters, but its presence could not be detected in the different components which were used in the preparation of the dish. How the arsenic reached the fritters could not be determined, although the epidemiological study pointed to it as the cause of the poisoning.

2. **Natural Poisoning**, or that produced by the presence of poisonous substances contained in food products, either animal or vegetable in origin.

Animal Products:

This type of poisoning common in other parts of the world and produced by the consumption of poisonous flesh of certain animals, is not common in Puerto Rico. Among the more important of the alleged poisonous animals are certain species of fish which are common in Oriental waters (63, 19). In a study of the fish fauna of Puerto Rico, Poey (66) recorded 301 species. None of these fish have been definitely proved to be poisonous up to the present time. In 1899 a federal commission made a very thorough study of the fish fauna of Puerto Rico (67), pointing out the fish that were suitable as food. There is a tradition among the fishermen that certain fish, like the picúa (*Sphyraena*), are poisonous during the period of sprawing. The idea also exists that at certain times of the year there is a species of marine plant called "cobriza" which is poisonous and is ingested by the smaller fish who when eaten by the larger varieties, poison these. These facts have not been confirmed scientifically. Among the fish which have been indicated at times as the cause of food poisoning in Puerto Rico are found the following:

1. Barracudas (*Sphyraena barracuda*, picuda).

2. King fish (*Scomberomorus cavalla,* pintado, sierra).

Walker (68) and Gregory (69) have made studies of fish poisoning in the Virgin Islands. Gregory, in his study, mentions the King fish as poisonous and cites Gatewood, who claims that in Puertorrican waters it is not poisonous. This fish is one of the most commonly used by the people of Puerto Rico as a food, and we know of no instance in which the flesh of the fish itself has been found poisonous.

3. Caranx, (yellow jack, arrayado). This fish was also classed as poisonous in the Virgin Islands study, but the same authors claim again that in Puertorrican waters it is not poisonous.

4. Swellfish (tamboril, *Spheroides spengleri*).

There is a notion among the fisherman that this type of fish is poisonous. However, it is hardly ever used as food.

Jarvis (70), in the report of the studies on the fisheries of Puerto Rico remarks in regard to fish poisoning "that the fish decompose in a very short time and are exposed to sun and flies before reaching the market; therefore, the so-called 'fresh fish' is often stale, sometimes tainted, and will impart gastro-intestinal poisoning. It is believed that the great majority of cases of fish poisoning are caused by decomposed fish. A few of this region (Puerto Rico) are poisonous, but these fish are seldom if ever used as food".

This view held by Jarvis in regard to poisoning by fish in Puerto Rico does not seem justifiable to us. We rather believe that it is not the fish itself which gives rise to gastro-intestinal symptoms, but the pathogenic microorganisms with which it is contaminated through unsanitary handling from the time it is caught to its consumption. Most of the fish poisoning outbreaks which have been reported in Puerto Rico could have been traced epidemiologically or bacteriologically to infected fish after it was caught, if proper studies had been conducted on time. We had the opportunity of studying a serious outbreak of fish poisoning in San Juan, Puerto Rico, which was traced to contaminated fish caught in the waters of Fajardo.

VEGETABLE PRODUCTS

De-Grosourdy (71) in his Treatise on Botany in the Antilles, makes a detailed classification of all the plants found

in these islands which in one way or another produce alarming symptoms, and divides them into three groups:

1. Poisonous group, in which are included 35 different types.

2. Dangerous plants, in which are included 14 different varieties.

3. Suspicious group, in which he classifies 31 species of which we should beware.

Besides these three groups he also classifies forty-five other plants under the group of cathartics or purgatives, all of which when ingested give rise to gastro-intestinal disturbances and in many instances to poisoning.

BURMA OR RANGOON BEAN *(Phaseolus lunatus)*

This bean is abundant in certain localities of the Island of Puerto Rico where it is grown and cultivated. It was introduced into the Island in 1915, and before its poisonous effects were detected it was cultivated by many farmers. It has been the cause of numerous outbreaks of poisoning in the Island of Puerto Rico. The poisonous principle is a cyanogenetic glucoside which when acted upon by the intestinal ferments, liberates hydrocyanic acid. In the year 1917 following the detection of this bean in the local markets, the Chemical Laboratory of the Health Department of Puerto Rico carried on special studies to detect the amount of hydrocyanic acid contained in the different types of commercial beans sold in Puerto Rico (72). The consensus of opinion is that if Burma bean produces 100 mgs. or more of hydrocyanic acid per kilogram it is poisonous and its use must not be permitted. In the year 1917, 59,754 pounds of beans resembling the Burma type were confiscated and chemical examination (72) revealed the hydrocyanic acid content per kilogram to be as follows:

White	460 mgs.
Gray Burma (baya)	160 mgs.
Red Burma	190 mgs.
Black Burma	290 mgs.
Pink Burma	170 mgs.
Light red Burma	170 mgs.
Spotted Burma	560 mgs.
Portorrican Burma	260 mgs.
American red bean	negative

Santo Domingo bean	negative
Black Venezuela bean	negative
Japanese bean	negative
American Chocolate bean	negative
Chile bean	negative
American spotted bean	negative
Lima bean	20 mgs.
Spotted Lima bean	negative
American chicory bean	negative
White native bean	negative
Red native bean	negative
California bean (marietas)	negative
California baby bean	negative

Ingestion of Burma beans produces marked gastro-intestinal symptoms with a fatal outcome in many cases. The symptoms produced by the intoxication are as a rule a sensation of soreness and pain in the abdomen, vomiting and a feeling of collapse. Diarrhea with blood and mucus is a rare manifestation.

In April 1929, there were two cases of poisoning (with one death) in Camuy, produced by the white Burma. Besides the Camuy poisoning there are several other cases on record which were caused by Burma beans. In 1930, in the town of San Sebastián, an outbreak of poisoning in four individuals (with two deaths) was investigated and traced to the consumption of Burma beans.

In Maricao, where the bean is grown and raised locally, there occurred several deaths of pigs, produced by feeding them with beans, which were later identified as Burma beans. In 1932 Dr. E. Garrido Morales, Epidemiologist of the Department of Health of Puerto Rico (62), investigated an outbreak of food poisoning in Utuado. Eight persons were involved (with two deaths) and the investigation pointed to some dried "gandules" as the cause. Upon examination it was detected that the gandules had been mixed with Burma beans.

The real danger in Puerto Rico as regard to Burma beans is that these are often mixed with beans of other types, making it difficult for the people to recognize the poisonous variety until it is too late.

YUCA (yuca amarga, brava o agria, cassava, *Manihot Manihot* (L.) bitter cassava).

Yuca is a tuberous vegetable which grows abundantly in the West Indies and especially in Puerto Rico. There are two types of yuca (71), the sweet or inoffensive product which is used as a vegetable and constitutes a delicious food, and the poisonous variety, which causes symptoms of poisoning and death when eaten in sufficient amounts. The two types are similar in many respects, and it takes considerable experience on the part of the farmers to detect one type from the other. The non-edible type contains a very violent poison which gives rise to symptoms soon after it is eaten. The active principle is supposed to be hydrocyanic acid in the form of a water soluble compound which is readily extracted with water; thus, when the root is cooked the acid is liberated. Poisoning by yuca used to be very common in Puerto Rico and those physicians who have practiced in the smaller towns of the island have had the experience of seeing poisoning in an individual or family produced by yuca. The symptoms of poisoning begin with a violent pain in the pit of the abdomen followed by generalized pain throughout the whole abdomen, with vomiting, repeated liquid depositions, as a rule accompanied by blood and mucus, and marked headache with visual disturbances. There is a cold feeling of the extremities and of the whole body, a clammy perspiration and collapse, followed by death in the very severe cases.

If we take the boiled pulp of the yuca brava, extract the water, and dry it, we have what is called cassava, a harmless substance used in the manufacture of bread.

CASTOR BEAN (Palma Christi, *Ricinus communis* L., higuereta)

This bush is very abundant in Puerto Rico. The seeds are small beans which contain a powerful toxin (ricin) which when taken in sufficient amount gives rise to definite symptoms of poisoning, often with a fatal outcome. The bush grows wild everywhere, particularly in certain localities of the island and the seeds are frequently eaten by children or people ignorant of their poisonous qualities. When ingested, the bean gives rise to vomiting, abdominal cramps, severe diarrhea generally without blood or mucus, and often collapse.

BARBADOS NUT (Piñón de botica, tártago, *Curcas Curcas* (L.) English physic nut tree)

The seeds of this tree, commonly found in Puerto Rico, have drastic purgative effects if taken, and in a sufficient quantity may produce death. The active principle of these seeds is an oil which resembles in many respects croton and castor oils. The seeds have a very agreeable taste similar to that of hazel nuts and are often eaten by children or adults. The symptoms produced by this fruit are, as a rule, vomiting, frequent liquid depositions, profuse sweating, weakness, fainting and collapse. In severe cases there is delirium and collapse, and the pulse rate becomes accelerated.

COMMON MANCHINEEL (Manzanillo, arbre de mort, *Hippomane mancinella* L.)

The fruit of the common manchineel resembles small apples of an agreeable odor but with an acrid taste. Eating these fruits gives rise to definite symptoms of gastro-intestinal disturbances manifested by a burning sensation in the lips and bucal mucosa, larynx and stomach. There is marked abdominal distention and pain, clammy perspiration, and feelings of dizziness and collapse.

The tree is not abundant in Puerto Rico at present, but in 1827, before a general order of the Spanish Government to destroy all the trees on account of their poisonous qualities, they were abundant. It was a general belief in the first quarter of the 19th century that crabs and fish in the dry seasons fed on these fruits common in swampy areas and became poisoned, so that when they were consumed as food, they too gave rise to poisoning (15). We have no knowledge of recent cases of poisoning by eating this fruit.

MUSHROOM POISONING Food poisoning by mushrooms is very common in the United States and results from confounding poisonous with edible mushrooms (19). The symptoms produced by poisoning with mushrooms (*amanita*) vary from a mild type accompanied by diarrhea, nausea, pain and ultimate recovery, to violent types with very high mortality accompanied by gastro-intestinal symptoms, abdominal pain, delirium, convulsions and death. In Puerto Rico, mushrooms are not commonly found. They are rarely used as a food except in the large hotels which import the product directly from the

United States and serve it in their menus. We have no knowledge of poisoning occurring from mushrooms in the city of San Juan during the past ten years. The mushrooms which may be present in the Island of Puerto Rico have not as yet been studied or classified and our knowledge concerning them is therefore very limited.

FOOD ALLERGY

Gastro-intestinal manifestations and urticaria are of frequent occurrence in some individuals as a result of eating foods non-injurious to others. It is supposed that the symptoms produced in these individuals are the result of special manifestations of hypersensitiveness to foreign protein substances. Food allergy is primarily due to a special condition of the individual himself, and not to the food. The symptoms are varied, ranging from pain, distension of the abdomen, nausea, vomiting and diarrhea accompanied or not by a rash, to violent gastro-intestinal symptoms, migrain and at times asthma and angioneurotic edema.

This idiosyncrasy, though occasionally met with, is not very frequent in Puerto Rico. According to Dr. Ramón M. Suárez (73) who probably sees the largest number of cases of allergy and asthma in Puerto Rico, the commoner manifestations of allergy produced by food are as mentioned above. In his experience the food articles which most commonly give rise to these conditions are in the order of their frequency, as follow: fish, particularly shell fish, eggs, wheat, milk (especially in children), egg plant (berenjena), pork and salami.

UNDETERMINED ETIOLOGICAL AGENTS

Investigators of experience on food poisoning epidemics know that there are numerous outbreaks in which the epidemiological evidence points to certain food products, yet bacteriological examination does not support the view.

In Puerto Rico, as in other parts of the world, this same experience is met with.

In the latter part of November 1927, Costa Mandry and Marín (74) studied an epidemic outbreak of diarrhea in a convict camp near San Juan. Thirty-two out of 40 persons living under similar conditions in this convict camp were attacked with a mild dysentery, characterized by nausea, vomit-

ing, generalized pain in the abdomen, and mild diarrhea with blood and mucus. The attack as a rule started from 2 to 8 hours after eating the food. A careful bacteriological and parasitological study did not reveal the causative agent of the outbreak. There is the possibility that the outbreak could have been caused by food poisoning.

In the Insular Asylum of Puerto Rico there occurred a small outbreak of abdominal pain accompanied by nausea and diarrhea among the domestic staff of the Institution. All the people that were affected ate at the same table and became ill after a certain meal. Bacteriological examination of the stools of those affected (food was not available) did not reveal any bacterium that could be ascribed as the causative agent.

SUMMARY

We have attempted in this paper to present our personal observations together with a review of the regional treatises as regards to food poisoning, with special reference to the prevalence of the condition, the common etiological agents, and the characteristic modalities of the disease in this tropical island.

In the brief outline which we have given of the causes of food poisoning we have only included those conditions which do occur in the Island of Puerto Rico or which are likely to occur because conditions prevalent facilitate their occurrence at any time. However, a good many substances, animal and vegetable in origin, which are common causes of food poisoning in other countries, are not present in Puerto Rico as far as our knowledge goes, and for that reason we have purposely omitted them.

Food infections and intoxications are of common occurrence in Puerto Rico.

The number of deaths from food poisoning which was negligible in Puerto Rico before the year 1927, apparently has increased since that year, seven deaths having been recorded from this cause in 1927 and fifteen in 1928, as against six deaths in the five-year period from 1922 to 1926. The death rate from this cause has risen from less than one half point per 100,000 population in 1927 to 1.2 per 100,000 population in 1931.

The probable reason for the increase of 1928 which has been maintained during the years following, is that since that year scientific attention has been directed towards this group of conditions, bacteriological and epidemiological studies of outbreaks have been made and reported, public and medical conferences have been given, and facilities placed at the disposal of practicing physicians to recognize and diagnose these conditions. The interest of the many members of the medical profession rose with the corresponding increase in the number of deaths certified from this cause.

The death rate from food poisoning in Puerto Rico is rather low if compared with the rates recorded in the southern States, and is comparable with the rates recorded in the United States Registration Area and in the northern States as shown in Tables I and II.

I

DEATH RATES BY 100,000 POPULATION FROM POISONING BY FOOD IN PUERTO RICO AND CERTAIN STATES OF THE UNITED STATES DURING THE YEARS 1924, 1925, 1927, 1928, 1929

	1924	1925	1927	1928	1929
New York	0.26	0.34	0.28	0.15	0.13
Pennsylvania	0.57	0.67	0.39	0.66	0.32
Maryland	1.38	0.64	0.75	0.62	0.74
North Carolina	1.65	1.45	1.13	1.07	1.50
South Carolina	1.19	1.38	0.70	1.04	0.46
Georgia	1.68	.....	.....	1.30	2.02
Tennessee	1.82	1.18	0.96	1.01	0.84
Virginia	1.19	1.11	0.94	0.79	0.78
California	1.32	0.95	1.10	0.66	0.67
Colorado	1.39	0.96	1.11	1.37	1.84
Vermont	0.28	0.85	1.13	0.83	0.55
Massachusetts	0.39	0.48	0.18	0.16	0.26
Alabama	.....	2.48	1.80	1.84	1.63
Mississippi	0.89	1.56	1.61	1.57	0.80
Louisiana	1.82	1.47	1.44	1.41	1.10
U. S. Reg. Area	0.9	0.9	0.7	0.7	0.7
Puerto Rico	0.2	.....	0.5	1.0	0.6

II

DEATH RATES PER 100,000 POPULATION FROM FOOD POISONING IN PUERTO RICO DURING THE YEARS FROM 1922 TO 1931

Year	Rate Per 100,000 Population
1922	.....
1923	0.14
1924	0.21
1925	.....
1926	0.06
TOTAL	0.41
1927	0.47
1928	0.99
1929	0.58
1930	0.90
1931	1.20
TOTAL	4.14

Food poisoning is produced in Puerto Rico more or less by the same etiological agents (bacterial or otherwise) as in temperate climates, and the condition follows the same characteristic trends as regards to epidemiology and symptomatology. The condition is very important in this Island where gastro-intestinal disturbances are one of the main problems with which the Department of Health has to deal. Foodborne infections occur throughout the year but are more common during the spring and summer months. The implicated food as a rule looks normal in every respect. Complete bacteriological and epidemiological studies of all cases of food poisoning occurring in Puerto Rico are necessary. In a good many outbreaks which have been investigated no definite etiological agent could be found.

As Jordan says, cases of food poisoning of undetermined etiology, "are sufficient to show that in a considerable proportion of cases of alleged food poisoning there is a large measure of uncertainty about the real source of trouble. Although the trend of opinion has been in the direction of an increased recognition of the share of certain bacteria, especially those of the *Salmonella* group, there is an important residue of un-

explained food poisoning that needs further skilled investigation".

Let us all together cooperate in the study of these conditions in Puerto Rico by soliciting, when cases come to our hands, the help of those investigators who are experienced and interested in the study of this important group of conditions. It is our part now to try to elucidate in every way possible this problem which is linked with a still greater one —gastro-intestinal disturbances—which has baffled our physicians since the early days of the Spanish colonization of this Island, and still baffles our competent health authorities.

REFERENCES

1. Costa Mandry, O. Infecciones intestinales por ingestión de alimentos proteicos contaminados. Bol. Asoc. Méd. de P. R. **23**: 414. 1931.
2. Costa Mandry, O. Envenenamientos por substancias alimenticias. P. R. y su enfermera. 8: Nos. 26 y 27. 1932.
3. Costa Mandry, O. Food infections in Puerto Rico. A bacteriological and epidemiological study of three outbreaks. P. R. Rev. of Pub. Health & Trop. Med. **4**: 128. 1928.
4. Geiger, J. C. Special Article—Food Poisoning. J. A. M. A. **90**: 459. 1928.
5. Geiger, J. C.; Nelson, M.; Gray, J. P.; Firestone, F. and Wynns, H. L. Three outbreaks of food poisoning. Public Health Reports. **46**: 1565. 1931.
6. Jordan, Edwin O. The epidemiology of paratyphoid infections. J. Prev. Med. **3**: 279. 1929.
7. Jordan, Edwin O. Food Poisoning. P. R. Jour. of Pub. Health & Trop. Med. **4**: 517. 1929.
8. Jordan, E. O. and Geiger, J. C. Two "Food Poisoning" outbreaks apparently due to bacilli of the paratyphoid enteritidis group. J. Infect. Dis. **32**: 471. 1923.
9. Koser, S. A. Development of paratyphoid enteritidis group in various foodstuffs. J. Infect. Dis. **31**: 79. 1922.
10. Salthe, O. and Krumwiede, C. Studies on the paratyphoid enteritidis group. VIII. An epidemic of food infection due to a paratyphoid bacillus of rodent origin. A. J. Hyg. **4**: 23. 1924.
11. Savage, W. G. and White, P. B. An investigation of the Salmonella group, with special reference to food poisoning. Med. Research Council, Special Report Series. No. 91. His Majesty's Stationery Office. London. 1925.
12. Savage. W. G. and White, P. B. Food Poisoning. A study of 100 recent outbreaks. Med. Research Council. Special Report Series No. 92. His Majesty's Stationery Office. London. 1925.
13. Acosta, José Julián. Historia Geográfica, Civil y Natural de la Isla de San Juan Bautista de Puerto Rico de Fray Iñigo Abbad. Librería Acosta, P. R. 1866.
14. Costa Mandry, O. and Garrido Morales, E. La disentería bacilar en Puerto Rico. Bol. Asoc. Méd. de P. R. **23**: 22. 1931.

15. RAMOS, F. Prontuario de disposiciones oficiales del Gobierno de la Isla de Puerto Rico desde 1824 a 1865. Imp. González. P. R. 1866.
16. BRANHAM, S. E. HUMPHREYS, E. M. . Soluble antigens of *Bacterium enteritidis*. J. Infect. Dis. **40**: 516. 1927.
17. DACK, G. M.; CARY, W. E. and HARMON, P. H. Unsuccessful attempt to produce Salmonella intoxication in man. J. Prev. Med. **2**:479. 1928.
18. ECKER, E. E. and MEGRAIL, E. Production of toxic substances in young cultures of single cell strains of *B. paratyphosus* B. J. Infect. Dis. **37**:546. 1925.
19. JORDAN, E. O. Food Poisoning and food-borne infection. University of Chicago Press. 1931.
20. GEIGER, J. C. Poisoning by food probably due to contamination with certain bacteria. J. A. M. A. **81**: 1275. 1923.
21. DACK, G. M.; JORDAN, E. O. and WOOD, W. L. "Food Poisoning" produced in monkeys by feeding living Salmonella cultures. J. Prev. Med. **3**: 153. 1929.
22. KINLOCH, J. P.; SMITH, J. and TAYLOR, J. S. The Aberdeen outbreak of milk-borne Gaertner enteritis, July 1925. J. Hyg. **25**: 434. 1926.
23. O'KELLY, W. D. Report of an outbreak of bacterial food poisoning. J. Hyg. **21**: 113. 1922.
24. PECKHAM, C. F. An outbreak of pork-pie poisoning at Derby. J. Hyg. **22**: 69. 1923.
25. ROWLAND, F. M.; MARSHALL, F. W. and MENTON, J. An unusual case of food poisoning. Brit. Med. J. **1**: 439. 1928.
26. KRUMWIEDE, C.; PROVOST, D. J. and COOPER, G. M. Studies on the paratyphoid enteritidis group. VII. Enteric infection ("Food poisoning") due to tapioca pudding contaminated with *B. cholerae suis* (*B. suipestifer*). J. Med. Research. **43**: 53. 1922.
27. SCOTT, W. M. Food poisoning due to *B. suipestifer*. J. Hyg. **25**: 406. 1926.
28. CARY, W. E.; DACK, G. M. and MYERS, E. An institutional outbreak of food poisoning possibly due to a streptococcus. Proc. Soc. Exp. Bio. & Med. **29**: 214. 1931.
29. LEVIN, W. Cheese poisoning. A toxicogenic bacillus isolated from cheese. J. Lab. & Clin. Med. **2**: 761. 1917.
30. LINDEN, B. A.; TURNER, W. R. and THOM, C. Food poisoning from a streptococcus in cheese. Pub. Health Reports. **41**: 1647. 1926.
31. HENRY, S. D. Bacterial food infection. Kansas Med. Soc. J. **28**: 159. 1928. (Abst. J. A. M. A. **90**: 1979. 1928.)
32. ILLSLEY, M. L. Food poisoning probably caused by *B. proteus*. J. A. M. A. **90**: 292. 1928.
33. WARREN, S. and LAMB, M. E. A fatal infection with an organism of the proteus group. J. Med. Research. **44**: 375. 1923–1924.
34. SAVAGE, W. G. Some problems of Salmonella food poisoning. J. Prev. Med. **6**: 425. 1932.
35. DACK, G. M.; HARMON, P. H. and JARRA, I. E. Negative results obtained on introducing heat-killed cultures of *Salmonella aertrycke* and *Salmonella enteritidis* into the intestinal tract of monkeys and other animals. J. Prev. Med. **2**: 461. 1928.
36. MEYER, K. F. and MATSUMURA, K. The incidence of carriers of *B. aertrycke*

(*B. pestis caviae*) and *B. enteritidis* in wild rats in San Francisco. J. Infect. Dis. **41**: 395. 1927.

37. VERDER, E. Wild rat as a carrier of organisms of the paratyphoid enteritidis group. Am. J. P. H. **17**: 1007. 1927.
38. Foreign Letters. Food poisoning from duck eggs. J. A. M. A. **99**: 1274. 1932.
39. KELLERT, ELLIS. Food poisoning following the eating of codfish cakes. Am. J. P. H. **21**: 1352. 1931.
40. GILBERT, R.; COLEMAN, M. B. and LAVIANO, A. B. Food poisoning due to toxic substances formed by strains of cloacae-aerogenes group. Am. J. P. H. **22**: 721. 1932.
41. DEBORD, G. G.; EDMONDSON, R. B. and THOM, C. Summary of Bureau of Chemistry investigations of poisoning due to ripe olives. J. A. M. A. **74**: 1220. 1920.
42. EMERSON, H. W. and COLLINS, G. W. Botulism from canned ripe olives. J. Lab. & Clin. Med. **5**: 559. 1920.
43. JENNINGS C. G.; HAASS, E. W. and JENNINGS, A. F. An outbreak of botulism. J. A. M. A. **74**: 77. 1920.
44. KOSER, S. A., EDMONDSON, R. B. and GILTNER, L. T. Observations on *B. botulinus* infection of canned spinach. J. A. M. A. **77**: 1250. 1921.
45. KOSER, S. A. and REITER, D. O. Botulism resulting from consumption of canned onions. J. Prev. Med. **3**: 499. 1929.
46. NEVIN, M. Botulism from cheese. J. Infect. Dis. **28**: 226. 1921.
47. SISCO, D. L. An outbreak of botulism. J. A. M. A. **74**: 517. 1920.
48. STARIN, W. A. The persistence of botulinum toxin in discarded canned foods. J. Infect. Dis. **40**: 579. 1927.
49. STARIN, W. A. and DACK, G. M. Pathogenicity of *Clostridium botulinum*. J. Infect. Dis. **36**: 383. 1925
50. THOM, C.; EDMONDSON, R. B. and GILTNER, L. T. Botulism from canned asparagus. J. A. M. A. **73**: 907. 1919.
51. ZLATOGOROFF, S. I. and SOLOVIEV, M. N. Botulism in the Union of Socialist Soviet Republics. J. A. M. A. **88**: 2024. 1927.
52. COSTA MANDRY, O. Envenenamientos por substancias alimenticias. Bol. Asoc. Méd. de P. R. **21**: 31. 1928. Bol. de la Rev. de Hyg. y T. B. C. de Valencia, España. No. 171: 28, No. 172: 38, No. 173: 43. 1930.
53. DACK, G. M.; CARY, W. E.; WOOLPERT, O. and WIGGERS H. An outbreak of food poisoning proved to be due to a yellow hemolytic staphylococcus. J. Prev. Med. **4**: 167. 1930.
54. JORDAN, E. O. Staphylococcus Food Poisoning. J. A. M. A. **97**: 1704. 1931.
55. JORDAN, E. O. The production by staphylococci of a substance causing food poisoning. J. A. M. A. **94**: 1648. 1930.
56. JORDAN, E. O. and HALL, J. R. A case of food poisoning apparently due to staphylococcus. J. Prev. Med. **5**: 387. 1931.
57. JORDAN, E. O. and MCBROOM, J. Results of feeding staphylococcus filtrates to monkeys. Proc. Soc. Exp. Bio. & Med. **29**: 161. 1931
58. BIGGER, J. W. The production of staphylococcal haemolysin with observations on its mode of action. J. Path. and Bact. **36**: 87. 1933.
59. JORDAN E. O. and BURROWS, W. Nature of the substance causing staphylococcus food poisoning. Proc. Soc. Exp. Biol. & Med. **30**: 448, 1933.

60. WOOLPERT, O. C. and DACK, G. M. Relation of gastro-intestinal poison to other toxic substances produced by staphylococci. J. Infect. Dis. **52**: 6. 1933.
61. BARBER, M. A. Milk poisoning due to type of *Staphylococcus albus* occurring in udder of healthy cow. Philippine J. S. **9**: 515. 1914.
62. Epidemiology Division, Dept. of Health of Puerto Rico. Off. reports to the Commissioner of Health.
63. DAMON, S. R. Food infections and food intoxications. Williams & Wilkins, Baltimore. 1928.
64. ZICKGRAF, G. Poisoning from eating figs. Zent. Blatt f. in. Med. **49**: 237. 1928. (Abst. J. A. M. A. **90**: 1758. 1928.)
65. COSTA MANDRY, O. and GARRIDO MORALES, E. Food poisoning in a Porto Rican Family due to arsenic. Bol. Asoc. Méd. de P. R. **22**: 200. 1930.
66. DUMONT, E. Ensayo de una historia médico quirúrgica de la Isla de Puerto Rico. Im. La Antilla. Habana. 1875.
67. U. S. Commission of Fish and Fisheries. Investigations of the aquatic resources and fisheries of Puerto Rico by the U. S. Fish Commission Steamer "Fish Hawk" in 1899: Govt. Print. Off. Wash. 1900.
68. WALKER, P. D. Fish poisoning in the Virgin Islands. U. S. Naval Med. Bull. **17**: 193. 1922.
69. GREGORY, C. R. Three cases of food poisoning attributed to eating freshly-caught fish. U. S. Naval Med. Bul. **23**: 316. 1925.
70. JARVIS, N. D. The fisheries of Puerto Rico (Investigational report No. 13) Govt. Printing Office, Wash. 1932.
71. DE GROSOURDY, R. El médico botánico criollo. Lib. de Brachet. París, 1864.
72. Chemical Laboratory. Official reports to the Commissioner of Health of Puerto Rico.
73. SUÁREZ, R. M. Personal communication.
74. COSTA MANDRY, O. and MARÍN, R. Study of an outbreak of diarrhea in a convict camp near San Juan, P. R. P. R. Rev. of Public Health & Trop. Med. **3**: 311. 1928.