karyocytes, and nucleated red cells were numerous in the red marrow. Spectroscopic examination of the blood for sulphaemoglobinaemia was negative. Death was considered to be due to agranulocytic angina with septicaemia.

Discussion

Between May 3 and 21 54 grammes of prontosil album were given. Agranulocytosis developed some time between May 20 and 25. A feature of the case was the failure of serial leucocyte counts to give warning of the impending disaster, the changes being merely those of a progressively active infection. An increased output of young polymorphs (Arneth classes I + II, Table II) was well maintained till May 20, after which a rapid failure in their production evidently occurred. The types of myeloid bone marrow found in cases of agranulocytosis include aplastic, normal, and hyperplastic varieties (Beck, 1933). The present case belongs definitely to the aplastic type.

Of the various drugs administered, chloral and the barbiturates do not contain the benzene ring. Barbiturates have been frequently used in association with amidopyrine in recorded cases, and an adjuvant effect has been suggested, but available evidence is against their being independently effective. Salicylates contain the benzene ring with no amino group; there is little evidence that they can cause agranulocytosis, and in any case none had been given within twenty-four days of the onset of the condition. Kracke (1932) produced experimental agranulocytosis in rabbits with benzene, but concluded that oxidation products were probably responsible. Kracke and Parker (1934) later recorded that there was constantly increasing evidence that the condition was caused for the most part by easily oxidizable benzene-ring drugs.

Marshall, Emerson, and Cutting (1937) have investigated the excretion of sulphanilamide, and found that 100 per cent. of the daily dose could frequently be recovered in the urine as free sulphanilamide and a conjugated form—para-acetylaminobenzene sulphonamide. They add, however, that it is possible under certain conditions that a small amount is excreted in some other form or eliminated through some other channel than the kidneys. They showed in a case with impaired renal function that there was delay in excretion of the drug.

Cases of agranulocytosis have been recorded in which no drugs appear to have been taken and no obvious cause could be found, so that a definite conclusion in regard to the causation in the present case is not justified.

Summary

1. A case of fatal agranulocytic angina is recorded.
2. The bone marrow showed complete myeloid aplasia.
3. The only benzene-ring drug administered within twenty-four days of the development of agranulocytosis was sulphanilamide.
4. The agranulocytosis appeared to be either of the idiopathic type of unknown aetiology or to be due to sulphanilamide.

I am indebted to Dr. J. K. Rennie for the clinical material and data, and to Dr. J. A. Price and Mr. G. C. Holliday for assistance with some of the investigations.

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FOOD POISONING—ITS EPIDEMIOLOGY AND BACTERIOLOGY*

BY

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The term "food poisoning" is conventionally restricted to an acute gastro-enteritis caused by the ingestion of food or drink which either contains living bacteria capable of setting up an acute inflammatory condition of the alimentary tract or contains toxic substances produced by various types of bacteria that have an irritant effect upon the gastro-intestinal mucosa. This definition does not include "botulism," a disease which presents no symptoms of gastro-enteritis, but which is nevertheless a form of food poisoning caused by the absorption from the alimentary canal of a specific toxin elaborated under suitable conditions by the *Clostridium botulinum*.

During recent years the number of organisms proved to be capable of giving rise to food poisoning in one or other of its forms has greatly increased. Since the isolation of *B. enteritidis* by Gaertner in 1888—followed ten years later by the isolation of *B. aertrycke*, or *Bact. typhi murium* as this organism is now called, by Durham in England and de Nobele in Belgium, both organisms being undoubtedly responsible for cases of food poisoning—at least twenty-seven members of the Salmonella group have been identified with actual outbreaks of food poisoning in human beings. It is of importance to note, too, that some pathogenic intestinal organisms normally giving rise to one particular type of disease may on occasion cause another kind of illness—for example, the dysentery organisms may produce the typical symptoms of food poisoning.

It is difficult to assess with any degree of accuracy the prevalence of food poisoning. Many solitary cases must undoubtedly go undetected, but with increased laboratory facilities such cases are being detected with greater frequency. In some form or other cases occur fairly often in this country—for example, fifty-eight outbreaks were recorded at the Ministry of Health in 1934, and of 137 outbreaks of suspected food poisoning in 1935 only twelve were excluded as not food poisoning. In America and Germany the incidence seems to be considerably higher.

Food poisoning may be conveniently discussed under three headings: (1) that due to the multiplication within the body of pathogenic bacteria present in food, and referred to as the "infection" type; (2) that due to the ingestion of certain substances formed as the result of bacterial multiplication in the food before ingestion—the "toxin" type; and (3) that due to the toxin of *Clostridium botulinum*—"botulism."

The "Infection" Type

Among the organisms responsible for the "infection" type members of the Salmonella group are the most common, but members of the dysentery group, more especially the Sonne bacillus and *B. dysenteriae* Flexner, must be included. In 1934 Salmonella infections were discovered in twenty-two instances, of which fourteen were, so far as could be ascertained, solitary cases, and during 1935 these were found on fifty-three occasions; twelve outbreaks during the latter year, however, were caused by dysentery bacteria, of which eight were due to the Sonne bacillus, three to *B. dysenteriae* Flexner, and

* Paper read at the Congress of the Royal Institute of Public Health and Institute of Hygiene, Margate, 1937.

one to the Newcastle-Manchester type of dysentery organisms. An outbreak of acute gastro-enteritis affecting about eighteen individuals who had partaken of cockles presumably contaminated with *Proteus vulgaris* is recorded by Dr. Allan Gray (1937).

I have included in the Salmonella group only those organisms which give rise to acute gastro-enteritis, as distinct from those causing the "enteric" type of disease. By some authorities *B. typhosus* as well as the paratyphoids are placed together in the Salmonella group; and from antigenic analysis this is quite justifiable. The "O" or somatic antigen of *B. typhosus* is exactly the same as the somatic antigen of *B. enteritidis* and its numerous varieties: *B. paratyphosus* B has the same somatic antigen as *B. aertrycke*, *stanley*, *derby*, *reading*, and so on. But in other respects the enteric group differ greatly from those I have included in my Salmonella group: the incubation period of the disease they produce is considerably longer, the symptomatology is different, the enteric group are not natural pathogens of animals, and transmission of the disease is confined ultimately to the human carrier. In fact, the whole problem of the prevention of enteric fever consists in stopping the various routes by which the bacteria may pass from the intestine of one human being to the mouth of another, whereas in the food-poisoning organisms there are additional factors to consider.

But seeing that all these bacteria are so closely related antigenically it is not surprising that some of them give rise on occasion to one type of disease and on occasion to another—for example, *Bact. cholerae suis*, var. Kunzendorf, often known as the Western European type of *paratyphosus* C or *Bact. suipestifer*, usually gives rise to a continued fever of the enteric type, but infection may take the form of an acute gastro-enteritis; and, similarly, *Bact. typhi murium* or *B. aertrycke*, though generally causing an acute gastro-enteritis, does occasionally bring about a continued fever. Even varieties of the same type of organism may cause different symptoms—for example, there are eight varieties of *Bact. enteritidis*, most of them antigenically similar and differing from one another only in some of their fermentation reactions (the Stern glycerol reaction, Bitter's rhamnose fermentation test, and *d*-tartrate fermentation reaction). Some of these give rise to the enteric type of disease and others to acute gastro-enteritis. Some time ago I was able to isolate *Bact. senftenberg*, var. Newcastle, from the faeces of a patient who showed the typical symptoms of a paratyphoid infection. An extract from his case sheet on admission to hospital on July 10, 1935, reads: "In bed since the 4th inst., complained of severe frontal headache; patient appeared very ill, feverish; rash fairly well marked over the body, but brighter over the arms, wrists, and legs. Temperature 101.6°, pulse 120, respirations 26. History of constipation; complains of pains and stiffness over the arms, shoulder-joints, and legs." The patient's serum agglutinated Oxford Standards Salmonella group suspension only up to a dilution of 1 in 320, with no agglutination with *paratyphosus* A or *paratyphosus* B suspensions.

In the case of *Bact. thompson*, I have isolated this organism from the faeces of two patients in different parts of this county during the past twelve months; the cases were quite unrelated and both were fatal. Each showed the symptoms of *B. typhosus* infection, but no Widal examination was made. When I rang up the doctor in one case he was rather surprised at the result: the patient had died the previous day, and the doctor had just filled in the death certificate, recording the death as due to

typhoid fever. It might be argued that his patient died of a typhoid infection and that *Bact. thompson* was accidentally found in the faeces. I cannot prove or disprove this, but the only really reliable proof of infection is the isolation of the organism from the patient, and, in the case referred to, *Bact. thompson* was obtained in large numbers on primary culture direct from the faeces by a method which has been tested out carefully and found capable of detecting typhoid organisms when these are relatively very very few.

The dysentery organisms, as I have mentioned, occasionally behave in a similar manner; instead of the usual dysentery symptoms the patient may show the symptoms of acute gastro-enteritis. This, of course, is mainly a problem which concerns the laboratory; it is the duty of the laboratory to identify any pathogenic organism that may be present, and not confine its search to members of one particular group. But it is as well to remember that all cases of continued fever need not essentially be due to a typhoid or paratyphoid infection. And as dysentery organisms may occasionally cause food-poisoning symptoms, these must be kept in mind; they are somewhat delicate organisms, rather apt to die off very quickly outside the human body, so that infected or suspected material should be examined as early as it is available if it is hoped to isolate them. I have found that the method of adding two parts of 30 per cent. glycerol in 0.6 per cent. saline to 1 part of faeces as soon as possible after they are passed helps to keep the dysentery bacteria alive when the services of a laboratory are not immediately available. As far as I know there is no good selective medium invented yet for the isolation of dysentery organisms, but my experience is that the methylene blue-eosin agar used in the examination of acid and gas producers in water is slightly superior to MacConkey's medium.

As has been indicated, the Salmonella group consists of a large number of organisms, and new types are continually being added to the list, but the commonest type in food-poisoning outbreaks is undoubtedly *Bact. typhi murium* (*B. aertrycke*). During the years 1923-33 the Ministry of Health found this organism in 110 out of 186 food-poisoning outbreaks, and in 1934 in twenty-two out of forty-three; of the forty-six outbreaks that occurred in 1935 in which the causative organisms were actually isolated twenty-nine were due to *Bact. typhi murium*. The next most frequently isolated organism is *Bact. enteritidis*. These facts are rather significant, and are of some importance when the cause of outbreaks comes under investigation.

The "Toxin" Type

In spite of exhaustive investigations into many outbreaks it has not been possible to isolate any member of the food-poisoning or dysentery groups. These outbreaks were formerly designated as ptomaine poisoning, caused by the presence in the ingested foods of products of protein decomposition. These so-called ptomaine poisons or toxic amines, however, are not produced until the food reaches an advanced stage of decomposition, and further examination showed them to be relatively non-toxic to most laboratory animals except when given in excessively large doses, far larger than are ever likely to be ingested under natural conditions. Barber in 1914 described a very interesting outbreak where a certain strain of *Staphylococcus albus* was isolated from the milk of a cow. The milk produced no ill effects if consumed immediately after its withdrawal from the udder, but on

being allowed to stand a few hours at room temperature it invariably produced the characteristic symptoms of acute gastro-enteritis. That these results were not due to any other component of the milk was proved by the fact that the same symptoms were produced in human volunteers who drank pure milk into which this particular strain of staphylococcus was introduced. The organism itself obviously did not cause the symptoms: it was necessary to allow it time—two or three hours at 28° to 30° C.—in which to produce something that would give rise to symptoms. Dr. Savage (1920) suggested later that many of the outbreaks from which food-poisoning organisms could not be isolated might be due to the ingestion, not of food-poisoning organisms themselves, but of toxic products elaborated by them, and recent work in America and in this country has furnished evidence strongly supporting this view.

The experimental evidence that staphylococcal toxin does produce gastric irritation has been provided mainly by Professor Jordan of Chicago, and consists in the production of the characteristic sickness and vomiting in human volunteers and in monkeys on the administration of quite small doses by the mouth. Savage and White (1925) paid considerable attention to this aspect of food poisoning in their extensive and valuable investigations, and while the implication of the *Salmonella* group in some of these "toxin" outbreaks was difficult to prove, it was nevertheless highly probable that these were the responsible organisms. Twenty-six of these "toxin" outbreaks are recorded in the Annual Report of the Chief Medical Officer for the year 1934 and forty-seven the following year.

Very little has as yet been discovered as to the nature of these toxic substances. It is not known whether they are specific bodies elaborated by the bacteria or whether they represent the breakdown products of the dead organisms. They appear to be relatively non-toxic when fed to most laboratory animals, though young rhesus monkeys are usually affected after a fairly large dose. On the whole, they are weakly antitoxinogenic. Savage and White were able to show that when toxic canned foods were emulsified and injected into animals, feebly agglutinating sera for *Salmonella* organisms were sometimes obtained, but, in spite of the intense symptoms these toxic foods produced, the sera of sufferers seldom revealed any appreciable developments of agglutinins. In their "Study of 100 Recent Outbreaks of Food Poisoning," they recorded that in nine of the outbreaks, while no specific organisms were isolated, the presence of *Salmonella* toxins in the suspected food was highly probable. Samples of blood from some of the patients showed a slight agglutination with members of the *Salmonella* group, usually up to a dilution of 1 in 20 or 1 in 25. Injections of watery extracts of the food into animals intraperitoneally sometimes resulted in the development of agglutinins; but here again positive agglutination was obtained only up to dilutions of about 1 in 40. The evidence, as may be gathered, is not too conclusive, but taken in conjunction with other factors it lends great support to the "toxin" theory.

Bacteria capable of giving rise to these toxic substances are not by any means confined to members of the *Salmonella* group; many are regarded as non-pathogenic under ordinary conditions, but with certain conditions of growth they are capable of producing these highly irritant toxic substances. *Staphylococcus aureus* is the organism most frequently associated with these "toxin" outbreaks; others are *Staphylococcus albus*, *B. coli*, *Proteus vulgaris*, *Proteus morgani*, *Streptococcus viridans*, and a micro-

coccus found in milk. Not all members of any particular group have the power to produce these substances—for instance, only a very few staphylococcal strains possess this particular property, and the toxin is quite distinct from the haemolytic or necrotic toxins of the staphylococcus. Up to the present we have no means, other than the direct test on human volunteers or susceptible monkeys, of determining which strains are toxinogenic and which are not. It is presumed that the bacteria multiply and elaborate these substances in the food prior to its consumption, the subsequent cooking to which the food is subjected destroying the organisms without materially affecting their toxic products. A consideration of the ubiquity of some of these bacteria, the variation in the potency of the toxins they produce, our inability at present to distinguish the poisonous from the non-poisonous strains except by test on the human subject, and our comparative ignorance of the suitable conditions for the production of toxins, makes this type of food poisoning not only difficult to investigate but also difficult to control.

The seasonal variation of food poisoning of the "infection" type is in all probability only a consequence of rise of atmospheric temperature favouring bacterial growth in infected foods, and thus encouraging the ingestion of large doses of the specific bacteria that seems to be necessary to set up the acute symptoms. The highest incidence occurs between the months of May and October, with the peak incidence in July. In the "toxin" type, too, more outbreaks tend to occur during the summer months, but these are far more evenly distributed throughout the year.

In both types the morbidity rate is high: 75 to 100 per cent. of the persons who eat the affected food are attacked, but the mortality rate is very low. The symptoms produced by the "toxin" type are similar to those of the "infection" type: the incubation period is usually shorter—half an hour to four hours, as compared with six to twenty-four hours in the latter. While they last the symptoms in the "toxin" type are more marked, but recovery is usually more rapid and the patient is well again in two or three days.

Vehicles of Infection

In *Salmonella* infections made-up meat dishes are the commonest vehicles of infection. In twenty-eight cases in which the causative organisms were isolated by Savage and White (1925) ten were caused by made-up meat dishes. Fresh meat, manipulated meat, milk, and milk products, especially ice-cream, are others; fish and fresh eggs, particularly ducks' eggs, have often been found to be responsible. The food associated with the "toxin" type is usually some form of canned meat or potted meat or fish, and milk products, more especially cakes and éclairs filled with cream or custard. "Corned beef" outbreaks are much less frequently met with now than formerly, the more efficient modern methods employed in the canning of this foodstuff being probably responsible for the almost complete disappearance of this one-time common source of "toxin" outbreaks. Obviously any food may act as a vehicle of infection, as, for instance, a dried milk powder from which *Bact. typhi murium* was isolated by Jones and Wright (1936).

Those familiar with the investigation of food-poisoning outbreaks will know that for various reasons it is not always an easy matter to isolate the offending organism or to prove the presence of a specific toxin; much less easy is it to trace the mode of infection of the food by

the organism. The common ways in which food may be infected are: (1) Infected animals may be used for food—for example, meat (beef and pork), ducks or their eggs. (2) Infected animals may excrete specific organisms to contaminate food—for example, rats and mice, bovines. (3) Human carriers may contaminate the food by handling.

There seems to be little doubt that animals, especially cattle and pigs, may suffer from diseases caused by the *Salmonella* organisms. The disease in animals is usually of an acute and often fatal type, but the carcass need not essentially be noticeably unhealthy. A case is reported in the annual report of the Ministry of Health, 1935, in which beef from the carcass of an animal almost certainly suffering from a *Salmonella* septicaemia was responsible for 174 cases, with eight deaths, in Lancashire. The flesh appeared normal, but *Bact. typhi murium* was isolated from all the parts of it examined. In most cases of food poisoning due to the consumption of meat from an infected animal it has been found that the animal has been "emergency slaughtered." Meat from such a source must be eaten in an uncooked or imperfectly cooked condition, as sausages or pressed beef, to cause ill effects. In this country such occurrences must be extremely rare, but in Germany, where more raw meat is eaten, they afford a frequent source of human infection. Within recent years ducks' eggs have on many occasions been responsible for food poisoning: one such outbreak investigated by Dr. Scott of the Ministry occurred in this county in 1933, and an outbreak at Preston in 1934 involved 250 persons who had consumed ice-cream in the preparation of which, it was alleged, the mixture containing ducks' eggs was insufficiently cooked. Infection of ducks by *Bact. typhi murium* and *Bact. enteritidis* is not uncommon; hens are less frequently infected with these organisms.

The contamination of foodstuffs by the excreta of infected animals is probably of more frequent occurrence than is generally supposed. McAllan and Howie (1931) record an outbreak of *Salmonella* infection in which milk was contaminated from the faeces of an infected cow; but rats and mice, in my opinion, are commoner offenders in this respect. Our rodent population is a fairly large one, and both rats and mice suffer naturally from infection with *Bact. typhi murium* and *Bact. enteritidis*, and may excrete the organisms in their urine and faeces for considerable periods. Food exposed in insanitary premises invariably attract these animals, and infection is thereby easily transferred to human beings. In the case of the infected dried milk referred to previously, the food used for a child who subsequently died of an *aertrycke* infection was kept in an uncovered container. Mouse faeces were found in the food, and from these pellets as well as from the powder *Bact. typhi murium* was isolated. From the intestines of mice caught in the house within the next few days the same organism was cultured. In an investigation commenced some time ago about 250 rats and mice were examined, and cultures of *Bact. typhi murium* or *Bact. enteritidis* were obtained in from 10 to 12 per cent. of them, in some cases from the intestine, in others from the intestine and spleen.

The Carrier

With regard to the human carrier as a source of infection, it is generally considered that he plays but an insignificant part. Authenticated cases are on record, but the proved cases are relatively few. The probability of the intermittent carrier state makes it still more difficult

to ascertain the true condition of affairs. The methods employed in the examination for carriers need to be more exacting than in the examination of the frank acute case. Agglutination tests are of little help; they may narrow the field of "suspects," but even in cases of known infection, proved by the actual isolation of the organism, the agglutinin response may be very poor, and agglutinins may be present to some extent in the blood of normal persons. As I have said, the isolation of the specific organism is the only proof of infection, and obviously in the case of a chronic carrier who is excreting but few pathogenic bacteria as compared with non-pathogenic ones, it is only to be expected that such carriers are easily missed unless special methods are employed. The frequency with which I have accidentally found *Salmonella* organisms suggests that carriers may be more common than is generally supposed. Two of these cases were particularly interesting. One was that of a missionary who had been invalided home from India with typhoid fever. He walked into the laboratory one morning with a specimen of faeces; he wished to know whether or not he was a typhoid carrier. After a few formalities the specimen was accepted. No *B. typhosus* was found in the specimen but a large number of *Bact. typhi murium*, and the same results were obtained from another specimen taken a week or so later. The other case concerned a possible typhoid carrier in a family of six persons, one of whom had developed typhoid fever, the organisms having been found in her faeces. No typhoid organisms were recovered from any of the other five persons, but a good growth of *B. aertrycke* was obtained from the faeces of the father, who was quite fit and apparently healthy and could not remember ever having been ill.

And here I must digress a little again to mention the methods employed for the isolation of these pathogenic organisms: the matter has an important bearing on the problem of human carriers and also on the prevalence of these food-poisoning organisms among animals. In 1936 I published a paper in which a comparison is given of results obtained with special selective media and with MacConkey's medium—the medium still used in many laboratories. I shall not go into the details of the different media; it will be sufficient to say that in a very careful investigation of a large number of specimens, both faeces and urines, 34.7 per cent. of the specimens were positive for organisms of the typhoid food-poisoning group on the particular selective media used as compared with 14.5 per cent. positive with MacConkey's medium. At the time when these special media were being tested out many of the cases to which I have referred came into the laboratory for investigation, and all specimens were examined on both media. In the fatal case of *B. aertrycke* infection due to the contamination of the dried milk by mouse faeces, *B. aertrycke* was isolated on both media from the faeces of the patient and from post-mortem material, but only on the selective media was the organism recovered from the infected food and from the mice themselves. Similarly for the two *aertrycke* carriers and the *senftenberg*, var. Newcastle, case: in all these the pathogenic organism could only be recovered on the selective media. I would mention also that convalescents became negative on MacConkey's medium very much earlier than they did when the faeces and urines were examined on these special media. I had occasion some time ago to investigate a small outbreak of dysentery in a children's hospital. Among the specimens examined were eleven mice caught on the premises. No food-poisoning or dysentery organisms were isolated from

these, but Morgan's bacillus No. 1 was recovered from the intestine of two mice. The part these bacteria play in summer diarrhoea of infants is not yet entirely clear, but there is no doubt that they are the organisms most frequently isolated from these cases. It thus appears to me that the dissemination of pathogenic bacteria by human convalescents and carriers and by rodents can very easily occur, and unless special methods for the examination of these are employed many sources of infection are bound to be missed.

The mode of infection of food by organisms giving rise to the "toxin" type of food poisoning is not so difficult to determine. As has been indicated, many organisms of different types have now been identified with outbreaks; most of them are common organisms found almost anywhere, but their presence in various foodstuffs has hitherto been regarded as of no significance. In the light of our present knowledge, however, in cases where these occur in any suspected food and in which no organisms of the *Salmonella* group can be isolated it is as well to bear in mind their possible association with the outbreak.

Botulism

Botulism is a disease which is fortunately rare in this country, and while very few of us have had the opportunity of investigating an outbreak of this disease, it is necessary to keep in mind its existence. My experience is confined to the examination of nut-meat brawn at the time of the occurrence of the few cases in London in 1935; neither *Cl. botulinum* nor its toxin was detected in the specimens examined.

The causative organism is a normal inhabitant of the surface layers of the soil, from which it may easily gain access to various types of food. There is no evidence that it is a normal intestinal inhabitant of man or of animals. *Cl. botulinum* is an anaerobic sporing organism which is incapable of growth in the animal body and giving rise to infection, but under suitable conditions it produces a very potent toxin. We do not know all the factors which determine the growth and toxin production of the organism. The spores are fairly resistant to heat and may withstand boiling for from half an hour to twenty-two hours: the potted-duck paste responsible for the Loch Maree cases in 1922 had been heated on three occasions, at one stage to a temperature of 115° C. for two hours. Media which are acid do not encourage growth, so that canned acid fruits are not likely to give rise to botulism. Vegetables and other products that are not acid and are preserved in air-tight containers which are not or cannot be adequately sterilized by heat—for example, glass containers—are possible sources of danger. The toxin is destroyed by such heat as is ordinarily applied in cooking, so that cooked food eaten within a short time of heating, and also fresh foods, are safe.

In Europe most of the cases of botulism have been caused by preserved meat or potted paste, whilst in America the contaminated foods have been mainly canned fruits and vegetables. Investigations into the different outbreaks have indicated that home-canned food is decidedly more dangerous than commercially prepared food.

Food infected with organisms of the *Salmonella* group usually appears quite normal: there need be no great change in colour and no detectable change in smell or taste. But in the case of botulism the preserved foods are frequently noticeably spoiled, the cans are usually

"blown," the food shows evidence of decomposition in varying degree, and it has a more or less rancid odour.

The methods of investigating outbreaks of food poisoning are not within the scope of this paper, and are fully described in a Memorandum by the Ministry of Health (Memo 188/Med. 1935).

Commentary

From what has been said it is obvious that there is still much to learn about bacterial food poisoning in human beings. The paths of infection of food, the part played by animals in transmitting infection, the conditions under which some normally harmless bacteria as well as natural pathogens are able to elaborate substances highly irritant to the human intestine, and the nature of these toxic substances are matters on which our knowledge is still very deficient and imperfect. The problems met with in investigating even the smaller outbreaks are often numerous and complicated, and entail an enormous amount of work that can only be successfully attempted in a well-equipped laboratory. Fortunately the assistance of the Ministry's laboratory is available for the examination by any local authority of specimens from suspected food poisoning.

Comparatively few outbreaks are investigated completely: this is inevitable in some cases, but an outbreak ended is not a problem solved. Very rarely are negative findings published: this is unfortunate, because a clearer understanding of the factors involved in bacterial food poisoning will more easily be acquired only by a comparison and correlation of all findings, whether these be positive or negative, in the different types of outbreaks.

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A conference of the Tuberculosis Association will be held in Paris from October 14 to 17 under the presidency of the Ministre de la Santé Publique et de l'Éducation Physique. The party will leave Victoria Station, London, by the 9 a.m. train on Thursday, October 14, and will arrive in Paris at 3.50 p.m. At 5 p.m. there will be a reception at the Hotel Lutécia by the Comité National de Défense contre la Tuberculose, followed by a dinner under the presidency of the Minister of Public Health. On October 15 visits will be paid to the Hospital Laennec, the Dispensary Léon Bourgeois, the Pasteur Institute, and to the dispensary, crèche, and open-air school at Suresnes. Luncheon will be given by the Municipality of Suresnes. On October 16 there will be visits to the offices of the Assurances Sociales and to l'École de Puériculture, and luncheon will be arranged by the Office of Public Health of the Seine at the Sanatorium of Saint Martin du Tertre. The party will leave Paris at 7.55 p.m., and will arrive in London on the morning of Sunday, October 17. Members of the Tuberculosis Association who wish to attend the conference are requested to inform the honorary secretary, Dr. Frederick Heaf, at 26, Portland Place, W.1, by July 31. All tickets will be issued by Thos. Cook and Son, Ltd., Berkeley Street, W.1, to whom remittances should be made payable and sent direct. Correspondence with Messrs. Cook should be marked B.H.43. Further information can be obtained from the honorary secretary.