Cheese as the Cause of Epidemics

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SANITATION in cheese has always been taken for granted like a good many other things in this world. Only recently has the importance of certain sanitary procedures and precautions been thrust upon us. As usual it has taken a series of epidemics with several big ones thrown in for good measure to awaken us to the importance of cheese as a carrier of disease germs. In all the fermentation industries such as in the beer, wine, and vinegar industries there have grown up certain practices and procedures based on the trial and error method which have generally come to be known as the "art" of making these fermented products. As science has encroached upon the "art" in these industries, it has frequently been found that there was a scientific basis for many of the procedures. But it has also found that some of the taboos and practices had no foundation in fact.

The making of cheese comes under the heading of a fermentation industry and as such is bound by tradition as regards certain practices. One of the most strongly entrenched traditions in the cheese industry was that pasteurized milk did not make good cheese. Cheese makers fought pasteurization the same as market milk, butter, and ice cream makers had before them. Pasteurization has never been a welcome guest in the dairy industry whose very existence is dependent upon it. Imagine the chagrin of the old time cheese maker since it has been demonstrated that pasteurization is an important step in the production of uniformly high quality cheese. The other two steps are high quality milk and a good starter. Furthermore, if a good starter is added to high quality pasteurized milk, it is possible to reduce the aging time of cheese from 6 to 8 months to 3 to 4 months because it can be ripened at a temperature of 60° F. instead of 40 to 50° F. The quality of cheese made under these conditions develops more and a better flavor.

EARLY HISTORY OF CHEESE POISONING

Cheese has long been considered one of the causes of food poisoning, being about on a par with sausages in this respect. Possibly this was because cheeses were fermented products and underwent digestion by enzymes attendant with odors—especially with certain types of cheese. There were several theories advanced to explain biological food poisoning which was so prevalent in the early days due to the lack of sanitation.

One of the first experimenters in the field was Albrecht von Haller (1708–1777), the distinguished Swiss physiologist, who demonstrated that aqueous extracts of putrid material injected into the veins of animals caused death. After this, much work was done with putrid material from both animals and plants in an attempt to discover the poisonous principle. For example Kerner in 1820 advanced the theory that the effects observed when poisonous sausage was eaten was due to sebacic acid. Later he modified his views, and believed the harmful effects to be due to a compound consisting of sebacic acid and a volatile principle. In 1827 Hunnefeld working with poisonous cheese accepted the ideas of Kerner regarding poisonous sausage but be-

lieved that in the case of cheese, caseic as well as sebacic acid was the active agent. During the early part of the nineteenth century the theory generally prevalent was that fatty acids such as sebacic acid were the active agents responsible for food poisoning.

In 1852, however, Schlossberger demonstrated by experiments that pure fatty acids were free from poisonous properties. In the meantime a great deal of work was being done on the basic constituents of decomposed foods which exhibited toxic symptoms when fed or injected into animals. In 1865 Selmi, an Italian toxicologist, gave the name ptomains (Greek for cadaver) to this class of substances, basic in character, which resulted from the decomposition of foods. Since they resembled the vegetable alkaloids, they were frequently called putrefactive alkaloids. Vaughan and Novy (51) later defined a ptomain as an organic compound basic in character and formed by the action of bacteria on nitrogenous matter.

After Pasteur's work showing the relationship between bacteria and disease, the attitude toward food poisoning changed. The search was for pathogenic or toxigenic bacteria or their products in poisonous foods. This is evident by the nature of the research work which followed in subsequent years.

During the years 1883 and 1884 there were reported to the Michigan Health Department about three hundred cases of cheese poisoning caused by the eating of twelve different cheeses. Vaughan (50) to whom the cheese was submitted for analysis isolated a crystalline substance which he believed caused the illness and to which he gave the name tyrotoxicon (Greek, cheese poisoning). This name he believed at the time to be original but he later found that it had been used as early as 1849 in Germany to designate poisonous cheese. The Michigan State Board of Health in 1884 obtained the aid of Sternberg, one of the outstanding bacteriologists of his time, to aid in solving the problem of poisonous cheese which was so prevalent. In 1885 Sternberg (45) reported that he found "micrococci in the fluid of the cavities of the cheese." The pure cultures which he isolated and injected subcutaneously into rabbits were without effect. So he concluded: "It seems not improbably that the poisonous principle is a ptomain developed in the cheese as a result of the vital activity of the above mentioned micrococcis." For a few years thereafter investigators spent a great deal of time looking for tyrotoxin in cheese. For example Baker (1) in 1884 reported eight outbreaks of gastroenteritis due to cheese and stated that cheese from one factory over a thirteen months period had caused a great many people to become ill. Reed (33) in 1893 reported numerous cases of poisoning in Ohio due to the eating of cheese containing tyrotoxicon. Spoiled milk used in making the cheese was considered the cause; also two sick cows were found among those supplying the milk for the cheese. Spica (46) isolated a chemical substance from cheese which he found toxic for frogs.

TRANSITION PERIOD: PTOMAINS TO BACTERIA

When Pasteur announced the germ theory of disease in 1870, search began anew for the cause of food poisoning. As new discoveries were made in the field of bacteriology, new etiological agents were found to have caused illness from eating the food in question.

Koch invented solid media in 1875 first as a gelatin tube method and later, 1883, as a plate method. About this same time Hesse introduced agar, and in 1887 Petri devised a culture dish. These were the prime requisites necessary to an era of far-reaching discoveries now known as the Golden Age in Bacteriology.

Dr. M. W. Taylor in 1857 reported

the first milk epidemic in Penrith, England, of which there is a record. Later in 1881 Dr. E. Hart of England reported a long list of epidemics due to milk including 50 due to typhoid fever, 15 to scarlet fever, and 4 to diphtheria. Yet, not until after the real cause of the epidemics was discovered was it possible to prevent them. Eberth described the typhoid bacillus in 1880, and Gaffky isolated it in 1884 by use of the new solid medium. Shortly thereafter various species of paratyphoid bacteria were isolated and identified and one of the real causes of outbreaks of disease and illness due to food was discovered for the first time.

In 1888 Gärtner isolated *Bacillus enteritidis* from an outbreak of raw meat which had caused illness in 57 people who had eaten it. This was the first report of food poisoning attributed to the Salmonella group of bacteria. In 1895 van Ermengem isolated *Clostridium botulinum* from ham which had caused illness in 23 persons and which is now recognized as botulism. In 1930 Dack and coworkers "rediscovered" what had been previously "discovered" three times, viz., that certain staphylococci under favorable conditions can cause food poisoning by the production of a toxin in the food before ingestion. It is now known that alpha type streptococci when present in large numbers in food can and do cause food poisoning. As the bacteriological agents which caused food poisoning increased, there was little room left for ptomains. Consequently they have gradually faded out of the picture until today one seldom hears the name ptomains mentioned among scientific men. In fact some doubt that they ever caused food poisoning, certainly not the many cases attributed to them. However, the idea still persists in the mind of the layman and even in the mind of the older physicians.

CHEESE IN ITS MODERN ROLE

Data on cheese as a vehicle for carrying disease germs to humans have been gradually accumulating since the advent of bacteriology and epidemiology. Novy (52) in 1890 isolated an organism from one sample of poisonous cheese which was fatal to cats after incubation at 35° C. for 24 hours in milk of which 100 ml. had been introduced into the stomach. No tyrotoxicon could be found in this or in a number of other samples of cheese which had caused illness in those eating them. Vaughan and Perkins (53) isolated a toxicogenic, micro-aerophilic bacillus from cheese which had caused illness in 12 people. It produced a powerful toxin when grown in broth or milk. Levin (25) studied cheese which had caused illness in six persons who had eaten it. He found no tyrotoxicon but a bacillus which produced a soluble thermostable toxin. Poisoning could be induced in laboratory animals either by feeding the cheese or a broth culture of the isolated organism. In 1923 Rich (35) reported an epidemic of 51 cases and 4 deaths from typhoid fever traced to cheese made from milk produced on a farm employing a typhoid carrier. The cheese was shipped green 3 to 12 days after making, and consumed probably 9 to 30 days thereafter. Another outbreak of typhoid fever, involving 29 cases and 4 deaths traced to cheese, was studied by Wade and Shere (56). The epidemiological data showed that *Eberthella typhosa* probably survived and had lived in the cheese approximately 63 days.

One of the most extensive epidemics of typhoid fever traced to cheese occurred in Canada with 627 cases and 57 deaths (13). After all other sources of infection such as milk, ice cream, butter, water, oysters, and shellfish were eliminated, it was finally traced to a fresh sweet Canadian cheese. The cheese was made by one manufacturer in one municipality of the district from the milk of several producers whose families either had or gave a history of having had typhoid fever. More re-

cently Gauthier and Foley (16) reported an outbreak of typhoid fever due to cheese which caused 6 deaths and 40 cases of disease. The source of the infection was finally traced to a woman who had had typhoid fever 20 years previously and who had milked cows against orders from the health department. This milk was a part of a raw milk supply delivered to the R. cheese factory to which the epidemic had been traced.

An epidemic of gastroenteritis of 22 cases traced to Wisconsin Cheddar cheese in Kansas City, Kansas, and another of 9 cases in Biddeford, Maine, from imported Albanian cheese were reported by Linden, Turner, and Thom (26). They isolated a streptococcus but did not determine its origin or species. They believed that "such outbreaks are exceedingly frequent." Their data indicated that the milk would have to be pasteurized at 145° F. for 30 minutes to destroy the organism.

There are reported several outbreaks of acute gastroenteritis in which the evidence strongly points to cheese but is not absolutely conclusive since no bacteriological investigations of the cases were made. In one of the outbreaks (49) 40 of the crew of one ship who had eaten the cheese in question and 20 of the crew from another ship supplied with the same cheese had acute gastroenteritis but recovered within 24 hours. Another outbreak (34) in which no causative organism was found but in which the cheese was strongly suspected was on the U.S.S. "Ruben James." Forty members of a crew of 105 were poisoned. All messes were affected except the one which had not been served cheese. No evidence of the Salmonella group was found in the cheese upon bacterial examination.

Of 300 group and family outbreaks of food poisoning listed by Jordan (23) in the United States from October 1913 to October 1915, 31 were assigned to cheese. In addition he lists nine individual cases or a total of 40 outbreaks attributed to cheese.

In England Graham-Stewart and associates (17) reported 23 cases of milk infection traced to Italian cream cheese. *Salmonella schottmülleri* was considered the cause since agglutination tests of the blood serum definitely showed it to be the etiological factor. Out of 100 food-poisoning outbreaks in England, Savage and White (38) found 8 due to cheese. They reported (39) one epidemic traced to cheese in which 9 individuals became ill after eating the cheese. No organisms could be found in the cheese but upon the basis of absorption tests on sera from the patients they attributed the illness to *Salmonella suipestifer*. Macaulay (27) reported 126 cases of cheese poisoning which occurred in Dover due to red Canadian Cheddar cheese shipped from Montreal. No tyrotoxicon was found, nor microorganisms which were toxic, but all evidence pointed to a bacterial toxin as the cause. He believed that a toxin produced by a member of the Gaertner group was the cause. He says, "Such delicate organisms as those of the Gaertner group, introduced during the process of manufacture would be unlikely to survive during the maturation of the cheese, although their stable toxin would likely persist."

Dolman (10) stated that in Canada between 1932 and 1939 there had been 6 known cheese-borne epidemics of typhoid involving 760 persons and 71 deaths due to Cheddar cheese manufactured from raw milk. Bowman (5) reported 3 epidemics involving 100 cases of typhoid fever due to eating cheese. Epidemiological data indicated that the cheese in question most likely became infected through a contaminated raw-milk supply from a farm having a typhoid carrier.

Outbreaks of disease due to cheese have been reported in other countries. No attempt has been made to search exhaustively the literature. Levin (25)

has reviewed some of the earlier outbreaks due to cheese. Blix and Tesdal (6) reported two outbreaks in Norway traced to cheese. One of 29 and the other of 26 cases of gastroenteritis. *Bacterium cholerae-suis* was isolated from the stools of two cases and also from the cheese of the one epidemic while *Salmonella aertrycke* from the cheese of the other epidemic.

Schytte and Tesdal (40) reported 65 cases and one death due to gastrointestinal infection caused by *Salmonella aertrycke* in cheese. The onset of symptoms appeared in 21 to 42 hours after ingestion of the cheese.

Swanner (43) according to Yale and Marquardt (57) has summarized 31 cheese-borne epidemics which occurred throughout the world from 1883 to 1939.

Since 1935 A. W. Fuchs, Senior Sanitary Engineer of the U. S. Public Health Service, has been including data on epidemics caused by milk products as well as milk in his yearly compilation of epidemics. To save space a tabulation of the epidemics caused by cheese has been made from these reports. This information is given in Table 1. In this 10-year period, there have been 21 epidemics of 824 cases and 18 deaths that have been attributed to various kinds of cheese or an average of more than two per year. Since these disease outbreaks were reported from only 13 states, one can hardly believe that this represents the actual total. Rather, as Fuchs has frequently pointed out, the logical explanation probably lies in their efficient epidemiological organization for discovering the outbreaks and their willingness to report them.

LONGEVITY OF PATHOGENIC BACTERIA IN CHEESE

The length of time pathogenic bacteria will live in cheese is of interest from a public health standpoint in view of certain State regulations.

Typhoid Fever and Cheese

As early as 1895 Rowland (37) investigated the viability of this group of bacteria in cheese by inoculating cheese with *Eberthella typhosa;* after a few days he could find no organisms. Later Wade and Shere (56) seeded milk with *Eberthella typhosa* and made 18 batches of experimental cheese from it. The organism died out within 8 days in 16 batches but persisted for 34 and 36 days respectively in the other two batches of the cheese. From epidemiological evidence presented, they believed that *Eberthella typhosa* probably survived in a commercial cheese for 63 days at 60° F. (15.5° C.). Ranta and Dolman (32) found the survival time of *Eberthella typhosa* in ground Cheddar cheese to be one month at room temperature and 17 weeks in a refrigerator.

Campbell and Gibbard (8) in order to determine the longevity of *E. typhosa* in cheese experimented with three different types of *E. typhosa* which they identified as phage types C, F, and M. They were all smooth Vi forms, two of recent isolation and the other a rejuvenated Rawlings strain. These various strains were inoculated into a high-quality raw milk when the acidity had reached 0.2 percent. Twenty cheeses were made on a commercial scale, 10 from phage type F, 6 from type M, and 4 from type C. Half of each type was stored at 40–42° F. and 58–60° F. respectively, and viability determinations made. *E. typhosa* died out in the majority of the cheeses stored at the higher temperature within three months, although in two out of the 10 cheeses viable organisms were isolated at the end of 196 days. At the lower storage temperature, 40–42° F., *E. typhosa* remained viable in 7 out of 10 of the cheeses for more than 10 months. In the other three they died out after 182, 210, and 252 days respectively. At the conclusion of the experiment all the cultures isolated were tested sero-

TABLE 1

SHOWING CERTAIN DATA RELATIVE TO EPIDEMICS CAUSED BY CHEESE OVER A 10-YEAR PERIOD—1935 TO 1944 INCLUSIVE

(Data compiled from U. S. Public Health Reports)

Year	State	Disease and organism	No. of cases	No. of deaths	Cheese made from raw or past. milk	Source of contamination	Kind of Cheese: Domestic: Cheddar	Cottage	Roquefort	Longhorn	Munster	Imported: Colby	No name	Romano	Pecorina
1935	Idaho	Food poisoning	12	...	...		1	..	..	..	..	..	..	..	..
	Indiana	Gastroenteritis	8	0	...		1	..	..	..	..	..	..	..	..
	Illinois	Food poisoning	51	...	...		1	..	..	..	..	..	..	..	..
1936	None reported														
1937	Minnesota	Food poisoning (Staph. albus)	37	0	Raw and past.	Unpast. cream, also other ways	..	1	..	..	..	..	..	..	..
	Washington	Food poisoning (Staph. aureus)	6	0	...		1	..	..	..	..	..	..	..	..
	Illinois	Diarrhea	11	0	...		..	..	..	..	..	..	..	1	..
	Illinois	Diarrhea	24	0	...		..	..	..	..	..	..	..	..	1
1938	Idaho	Food poisoning (tyrotoxin)	17	0	Raw	Fecal contamination	1	..	..	..	..	..	..	..	..
1939	California	Food poisoning	4	0	...		1	..	..	..	..	..	..	..	..
	Mass.	Gastroenteritis (Staph. and Strep.)	10	...	...		..	..	..	..	..	..	1	..	..
1940	California	Food poisoning	6	...	...	Home-made, Mexican style	..	1	..	..	..	..	..	..	..
1941	Illinois	Food poisoning	10	0	...	Prob. by employee with intestinal disturbance	..	..	..	1	..	..	..	..	..
	New York	Typhoid fever	19	1	Raw	(Suspected carrier)	1*	..	..	..	..	..	..	..	..
1942	New Jersey	Gastroenteritis	8	0	...		1	..	..	..	..	..	..	..	..
1943	Maryland	Food poisoning	6	0	...		..	..	..	..	1	..	..	..	..
	Kentucky	Food poisoning	86	0	...		..	1	..	..	..	..	..	..	..
1944	Michigan	Food poisoning (Proteus morgani)	104	0	...		1	..	..	..	..	..	..	..	..
	New York	Food poisoning	5	0	...		..	..	1	..	..	..	..	..	..
	Virginia	Gastroenteritis (Staph. aureus)	71	0	...		..	..	..	..	..	1	..	..	..
	Cal. & Nevada	Typhoid fever	83	4	Raw	Unripened	1	..	..	..	..	..	..	..	..
	Indiana	Typhoid fever	246	13	Raw	Green	..	..	..	..	..	1	..	..	..
		Totals	824	18			10	3	1	1	1	2	1	1	1

* Cheese curds.

logically and biochemically for identity and found to be the smooth Vi type and had not reverted to the rough or M form. As the authors point out this may be of considerable epidemiological significance.

Brucellosis and Cheese

There is no record in the literature of undulant fever caused from eating cheese made from cow's milk. The literature shows that so far no *Brucella abortus* organisms have been isolated from any sample of commercial cheese. Carpenter and Bock (7) did not find *Brucella abortus* or *Brucella melitensis* in 72 samples of various kinds of imported cheese such as Swiss, Roquefort, Reggiano, Gorgonzola, and Edam and 10 samples of domestic cheese. Smith (42) examined 63 samples of cheese of which 28 were foreign, 23 from England, 6 from the colonies, and 6 local. They include a wide variety of cheese such as Cheshire, Cheddar (red and white), Bel Paese, Convalli, Camembert, Double Gloucester, Roquefort, Gouda, Stilton, Edam, Wenslet, Gorgonzola, Chilveran, Parmesan, and cheese prepared locally in Scotland. The cheese after mincing and adding saline was injected into guinea pigs in 5 ml. amounts.

More recently Gilman *et al.* (18) have made an extensive study of the occurence and survival of *Brucella abortus* in Cheddar and Limburger cheese. They first made a preliminary study on cheese milk in New York State. Then studies were made on cheese made from milk inoculated with recently isolated cultures from naturally infected milk and on cheese made from milk produced by reacting cows whose milk was heavily infected with *Brucella abortus*. Finally, they conducted a survey to determine the contamination naturally occurring in milk of cheese-producing areas (in New York and Wisconsin where brucellosis had not been eradicated from the dairy herds) in order to determine the survival of *Brucella abortus* in naturally-infected commercial Cheddar cheese. A total of 59 vats of commercial cheese milk of which 11 were positive for *Brucella abortus* showed that of the nine Cheddar cheeses from the nine positive vats (two cheeses made from the positive milk could not be located), all were negative on first examination after storage at 1.1° to 2.7° C. for periods varying from 41 to 84 days. Likewise, they found that Limburger cheese made from milk positive for *Brucella abortus* was negative on the first test after 57 days storage. Since no *Brucella abortus* organisms were recovered from any sample of commercial cheese and since Cheddar cheese has not been proved to be a carrier of undulant fever, they feel that an aging period of 60 days is reasonable assurance against the presence of viable *Brucella abortus* organisms in Cheddar cheese.

Thompson (48) in his article on ice cream cites Voillé to the effect that *Brucella abortus* may be isolated from Roquefort cheese for as long as two months. Gilman *et al.* (18) call attention to the fact that since Roquefort cheese is made from ewe's milk, it is probable that the organism in question was of the *melitensis* rather than the *abortus* variety.

While it is apparent from the foregoing discussion that cheese made from unpasteurized cow's milk containing *Brucella abortus* is not a serious source of infection for undulant fever, this is not true of cheese from unpasteurized goat's milk. On the contrary cheese made from unpasteurized goat's milk is highly infective for man especially if consumed shortly after it is made. Eyre (12) could not isolate *Brucella melitensis* after 48 hours from cheese made from goat's milk due to the growth of lactic acid bacteria. In Brynza cheese held at 11° to 14° C., prepared from milk inoculated with *Brucella melitensis* after pasteurization, Versilova (54) found viable organisms

which were infective for guinea pigs after 45 days. Peres and Granon-Fabre (31) recovered *Brucella melitensis* by guinea pig inoculation from cheese made from naturally infected goat's milk which had aged 20 days but not from the samples aged 6, 10, or 17 days. Veloppe and Jaubert (55) reported 14 cases and 2 deaths of undulant fever due to fresh cheese. According to them it is common practice to mix goat's, sheep's, and cow's milk for cheese making. They concluded that cheese infected with *Brucella melitensis* is harmless after 30 days. Stiles (44) found 8 out of 19 samples of cheese positive for *Brucella melitensis* when injected into guinea pigs. Four samples of the Feta type and three of the Romano type were made from raw goat's milk and the other, imported from Mexico, was a yellow cream cheese supposedly made from cow's milk.

Streptococci and Cheese

Hucker and Marquardt (21) made studies with *Streptococcus pyogenes* which had been isolated from milk which was responsible for an outbreak of septic sore throat. In a batch of cheese which had been made from milk seeded with the above streptococci and cured at 40° F., the organisms had increased greatly in number at the end of 160 days. In a similar batch cured at 60° F., relatively few organisms remained after 85 days. An examination of cheese known to have been made from milk containing *Strept. pyogenes* showed that this organism was present in the cheese. Yale and Marquardt (57), working with two cultures of *Strept. pyogenes* Rosenbach belonging to Lancefield's Group A which had been isolated from two outbreaks of septic sore throat caused from milk, found that the organisms died out very rapidly (within 24 hours) in cottage cheese. The organisms survived between 28 and 51 days in one lot and between 9 and 14 days in another lot of Limburger cheese. In Cheddar cheese cured at 45° F. (7.2° C.) the organisms survived for over 18 weeks and between 9 and 11 weeks in a duplicate cheese cured at 62° F. (16.6° C.). In a second lot of cheese cured at 50° F. (10° C.) the organisms survived less than 18 weeks. They concluded that the variety of cheese, its moisture and salt content, and the curing temperature are some of the important factors affecting the survival of these organisms in cheese.

It is interesting to note that to date, no epidemics of septic sore throat traced to cheese have been reported in the literature, despite the fact that there doubtless is considerable cheese made from milk so infected.

Tuberculosis and Cheese

The prevalence of bovine tuberculosis is gradually disappearing under our present system of eradication and control. However, we should not be lulled into a false sense of security but should remember that tuberculosis still ranks seventh among the first 10 causes of death in man. We should still continue our first line of defense, tuberculin testing as well as our second, pasteurization. As early as 1909 Mohler *et al.* (28) showed that *Mycobacterium tuberculosis* would live in cheese from 33 to 261 days. They quote previous work in which viable tuberculosis bacteria were found in soft cheeses such as cottage and others from 14 days to two or more months, in Emmenthaler to about the fortieth day, and in Cheddar after 104 days. Harrison (19) earlier had reported results of seeding milk with *M. tuberculosis* which was made into Swiss Emmenthaler cheese where they remained viable for 28 days and into milk which was made into American Cheddar where they remained viable for 104 days. Earlier work had shown that the tubercle bacillus would remain viable for some time since Galtier (15) in 1887 inoculated milk with *M. tuberculosis* and found it in the cheese after 70 days and Heim (20) in 1889 found

it after 14 days but not after four weeks.

In 15 samples of cottage cheese examined by Hormann and Morgenroth (22), 3 contained *M. tuberculosis* and 3 out of 5 samples of cottage cheese examined by Rabinowitch (36) contained it. Harrison (19) was able to produce tuberculosis in three samples of soft cheese purchased in the market in Berne.

In this country Schroeder and Brett (41) purchased from retail dealers in Washington, D. C., 59 samples of Cheddar, 32 of Neufchatel, 31 of cottage, 131 cream, and 3 miscellaneous varieties of cheese and examined them for infective *M. tuberculosis*. They found one cottage and 18 cream cheese samples which were infective for animals.

Botulism and Cheese

Milk and milk products in general have never been a source of botulism. Although milk and its products are an excellent source of food for bacteria, yet the physiological conditions evidently are not to the liking of *Clostridium botulinum*, this despite the fact that the normal habitat of his organism is the soil and there are many chances for contamination from this source.

It is well known that *Cl. botulinum* does not flourish in an acid medium. The limiting pH for its growth is around 4.5. Nevertheless there are records of botulism occurring in supposedly acid foods such as tomato catsup, juice, and relish, in dill pickles, apple sauce, green tomatoes, and apricot butter. All except one of these, tomato catsup, were home-canned where the method of preparation may have neutralized a part of the acid so as to have brought the acidity within the pH growth range of the organism. The commercial preparation of the catsup in question may likewise have been faulty so as to have permitted the growth of *Cl. botulinum*. Thus in the case of the home-canned cheese listed in the following paragraph as causing five outbreaks of botulism, the cheese may have been green and canned within a short time after it was made so that there was no opportunity for acid development.

Dack (9) gives a table prepared by H. F. Meyer in which is listed foods which have been involved in 359 outbreaks of botulism in the United States between 1899 and 1941. Home-canned cheese is listed as causing 5, and commercially packed canned milk as having caused 2 of these outbreaks. Nevin (29) reports an outbreak with 3 deaths traced to home-made cottage cheese from which *Cl. botulinum* type B was isolated. She seeded fresh sterilized and unsterilized cottage cheese with a loopful of a 48-hour broth culture of the above organism and injected 1 ml. portion into guinea pigs with fatal results. Controls of market cheese were negative.

Escherichia coli in Cheese Poisoning

Since *Escherichia coli* is omnipresent in the barn and on the cows, there is likelihood of it getting in the milk for cheese making. That a great deal of low grade milk is so infected there can be no doubt. It is obvious that *E. coli* cannot be either very pathogenic or toxigenic under the conditions prevailing in cheese-making or else there would be many outbreaks attributed to it. Timmerman (47) likewise discounts the role of *E. coli* in cheese poisoning since it is present in about 60 percent of the samples of cheese and causes no poisoning. He is of the opinion that a larger number of cases of food poisoning are caused by staphylococci than is generally believed, and that poisonings that develop after the eating of cheese are frequently due to them.

Influence of Acid and Temperature

Conditions in the early stages of cheese manufacture favor the growth

of most bacteria since there is moisture, sufficient heat, and plenty of food. However, certain types such as the lactic acid-producing bacteria find the conditions more suitable than other types and soon create an environment unfavorable to the growth of most other bacteria. Furthermore, both the pressing of the curd which greatly reduces the water present and the storage temperature during aging are inimical to bacterial growth. Then there is the time factor and the growth curve of bacteria to consider. Under the most favorable circumstances the bacterial growth curve reaches its peak in several days after which there is a rapid and then a steady but constant decrease in the number of bacteria. Just how long it takes them to die out depends upon how favorable or unfavorable the environment. For example high acidity and high temperature are very destructive. More so than any other combination of these two factors such as high acidity and low temperatures or low acidity and high temperatures. In the case of *Brucella abortus* in cheese, Lerche (24) found that in cheese such as Landekäse which is an acid curd-type cheese made by the natural souring of milk, the organism died promptly. However, in cheeses made by the rennet process such as Frühstückkäse and Weisskäse, *Brucella abortus* persisted for 24 days. Drescher and Hopfengärtner (11) inoculated milk with *Brucella abortus* and made several types of cheese which were sampled at intervals from near the rind and near the center. The viability of the organisms were determined by culturing and guinea pig inoculations. The cheese was stored at 4 to 5° C. In Emmenthaler cheese, the organism survived 49 days in both the rind and central portion. In Dilikatkäse they recovered the organism from the rind and central portion in 35 but not 42 days while in Tilsiterkäse they found the organism to remain viable in the inner part of the cheese for 91 days but for only 35 days in the rind by both methods. However, by the inoculation method they found it at the end of 49 days in the rind.

The longevity of *E. typhosa* in milk and milk products has long been a subject of investigation due to the large number of epidemics which it has caused in these products. One of the early investigators, Barthel (2), held that "Typhoid bacteria increase in milk and remain alive for 25 days without a decrease in number due to the formation of acid in milk" and "they themselves contribute to this acid production. In butter, typhoid bacteria are found after 10 days, especially in butter which is strongly acid, as the enclosed sour brine is a good nourishing medium." However, that this opinion was not general is indicated by the work of Bassenge (3) and Behla (4) who found that when milk, buttermilk, etc., reaches 0.4 percent lactic acid, the typhoid bacilli are killed within 24 hours. Northrup (30) investigated the influence of the natural production of lactic acid in milk by various organisms on the destruction of *E. typhosa*. She found the minimum acidity produced by *Bact. lactis acidi (Strept. lactis)* which would destroy *E. typhosa* to be +37° acid (0.55 percent lactic acid) in lactose broth which corresponded to +80° (1.2 percent lactic acid) in milk and +28° acid (0.42 percent lactic acid) in whey, while for *L. bulgaricum* it was +53° (0.8 percent lactic acid) in lactose broth which corresponded to +208° (3.12 percent lactic acid) acid milk and to +66° (1.0 percent lactic acid) acid in whey. She recognized that there were many other factors entering under natural conditions which could influence the results such as temperature, character of initial microflora, associative action, metabolic products, and antibiotic action which could either hasten or retard the destructive action of the lactic acid bacteria.

Yale and Marquardt (57) in their

work with *Streptococcus pyogenes* found that these organisms died out within 24 hours in cottage cheese at pH of 4.57 to 4.32 while in Limburger cheese where the pH values ranged from 6.6 on the interior to 4.85 near the surface the maximum survival of the organisms was between 28 and 51 days in one lot and between 9 and 14 days in another lot of this cheese. However, in Cheddar cheese where the pH values did not drop below pH 5, the *Strept. pyogenes* remained viable for 18 weeks when the cheese was cured at 45° F. and between 9 and 11 weeks in a duplicate cheese cured at 62° F. Of course there were other factors operative such as salt and moisture in addition to temperature in these cheeses. Nevertheless acidity played an important role in the viability of the bacteria.

Versilova (54) likewise found acidity and temperature were important factors in determining the viability of *Brucella melitensis* in sheep's milk. At a pH of 6.0 to 6.8, it remained viable for 22 to 40 days in sheep's milk held at 11° C. whereas at pH 4.0 to 5.0 it remained viable 15 but never more than 30 days. However, in milk kept at 37° C. it frequently died out the first few days.

More recently Campbell and Gibbard (8) in studying the longevity of various smooth type strains of *E. typhosa* in experimental commercial cheese found the temperature at which the cheese was held to be more important than the acidity on the viability of *E. typhosa*. They state, "It was expected that the acidity of the cheese might have played some part in the death of *E. typhosa* and, although it is true that the cheese held at high temperature developed acid more quickly than those held at low temperature, there does not seem to be any correlation between acidity and viability of inoculated bacteria in different cheese."

It is unfortunate that Campbell and Gibbard (8) did not record pH values as well as titratable acidity since this would have been a better indicator. However, from their table I, acidity did affect the viability of *E. typhosa* because the acidity for duplicate cheeses made from the same batch showed acidities consistently higher for those cheeses held at the higher than at the lower temperatures for both November 25, 1942, and January 23, 1943, approximately 5 to 6 and 7 to 8 months respectively after the cheese was made. It was not until approximately 10 to 11 months later, April 13, 1943, that the acidities for the cheeses held at the two temperatures were practically equal. It would appear from these and other data that the higher acidities developed at the higher temperatures was an important factor in killing off the organisms.

MINIMUM SANITARY REQUIREMENTS

A committee representing the Wisconsin Cheesemakers' Association, Wisconsin Milk Producers' Association, the University and the State Department of Agriculture of Wisconsin, and the National Cheese Institute framed acceptable minimum sanitary requirements * for cheese factories and recommended their official adoption by Wisconsin as well as by other states producing cheese.

As Freidel and Yale (14) point out the cheese industry itself is deeply concerned with proper public health protection for cheese and feels that all concerned would benefit if all states would adopt uniform regulations. Since cheese is so widely distributed, this would make compliance with state regulations much easier for the industry.

PASTEURIZED MILK FOR CHEESE

As a result of the long list of epidemics and food poisonings in which cheese was either directly or indirectly implicated, it became evident that additional safeguards were necessary in order to produce cheese free of these

agents. Pasteurization was the only logical answer. In addition to pasteurization as a health measure, it has been shown by the Bureau of Dairy Industry, U. S. Department of Agriculture, that cheese made from a good grade of pasteurized milk to which has been added a good starter is more uniformly high in quality, and furthermore, that it is possible to ripen such cheese at 60° F. instead of 50° F., thereby reducing the curing time of six to eight months to three to four months. Cheese so made and cured develops more and a better flavor than cheese made from unpasteurized milk and held at 50° F. for six to eight months. Therefore, there are two most excellent reasons now for pasteurizing milk for cheese—an economic as well as a health consideration.

STATE LAWS REQUIRING PASTEURIZATION OF MILK FOR CHEESE

California in 1944 was one of the first states to pass a law requiring that all cheese sold to the retail trade shall be pasteurized or made from pasteurized cream, milk, or skim milk which has been pasteurized, except cheese which has been allowed to ripen or cure for a minimum period of sixty days. Further requirements are made for labeling the variety, grade, factory number, state of origin, and date of manufacture. This law resulted from an outbreak of typhoid fever caused by green cheese in which there were 79 cases in nine counties in California and four in Nevada.

Colorado likewise passed a similar state law which became effective January 1, 1945, which requires that all cheese shall be pasteurized or made from pasteurized cream, milk, or skim milk except cheese which has been allowed to ripen for a minimum period of 120 days or longer if deemed necessary. In the same year New York amended the State Sanitary Code to require that cheese be pasteurized or that it be made from pasteurized cream, milk, or skim milk or had been allowed to cure or ripen at a temperature of not less than 35° F. for a period of not less than 60 days, from date of manufacture.

In the meantime our good neighbor, Canada, had taken similar action to stop the numerous outbreaks of disease traced to cheese by passing a regulation which became effective August 1, 1945. This regulation requires:

1. Every manufacturer of cheese by the Cheddar or other process from raw or pasteurized milk that yields a hard-pressed cheese shall mark or brand within 24 hours after removal from the press every merchandising unit of such cheese correctly and distinctly with the date of manufacture indicating the day, month, and year when such cheese was put into press.

2. No person shall cut any Cheddar or other hard-pressed cheese made from raw or pasteurized milk for sale or consumption as such in Canada within a maturing period of 90 days from the date of manufacture. Throughout the first 10 days of said maturing period of 90 days the temperature of storage shall be maintained at not less than 58° F. and throughout the remainder of the period, at not less than 45° F.

GENERAL DISCUSSION

A review of the literature shows that cheese has caused a great deal of food poisoning since the cause of food poisoning began to be investigated scientifically about the beginning of the nineteenth century. At first the search was for a chemical poison produced biologically during putrefaction. Thus there was isolated a class of basic chemical compounds known as ptomains such as amylamin, putrescin, cadaverin, neurin and tyrotoxicon, some of which were poisonous and some non-poisonous. However, when Pasteur announced the germ theory of disease, research workers devoted their efforts to finding a bacterial rather than a chemical cause for the effects

noted. This resulted in finding that the cause of most food poisoning was due to specific bacteria or groups of bacteria such the *Salmonella, Eberthella,* staphylococci, streptococci and various strains of *Cl. botulinum* such as A, B, C, D, and E. These groups of bacteria acted principally in one of two ways either by establishing themselves in the intestinal tract or by producing a soluble thermostable toxin on the contaminated food which was responsible for the illness.

Experiments on the longevity of pathogenic bacteria in cheese show that they remain viable for varying periods of time. Two States, California and New York, have adopted a 60-day and Colorado a 120-day holding period for cheese made from unpasteurized milk or cream. Canada requires a 90-day holding period for cheese made from either raw or pasteurized milk. In view of experimental evidence, it would appear that the 60-day holding period is too short a time.

There are conflicting results on the longevity of *E. typhosa* in cheese. Experimental work on a commercial scale by two groups of investigators show different results for cheese. Wade and Shere (56) showed the longest survival time for *E. typhosa* to be 36 days whereas Campbell and Gibbard (8) found them present in some cases and under certain conditions after 10 months. The evidence would indicate that the *Salmonella* group of bacteria most likely would die out within the 60-day period. The records show that cheese does not appear to be a very serious source of undulant fever due to *Brucella abortus* but that *Brucella melitensis* is, especially cheese made from raw, unpasteurized, ewe, or goat milk. Streptococci isolated from septic sore throat persisted in Cheddar cheese for as long as 18 weeks but died off within the 60-day period in cottage and Limburger cheese. Despite this fact, there is no record in the literature of an epidemic of septic sore throat due to eating cheese.

M. tuberculosis persists for a long time in cheese according to all experiments to determine its viability in cheese. Cheese does not appear to be a serious source of botulism since very few epidemics have been traced to *Cl. botulinum.* What few there have been have been traced to home-canned cheese where doubtless the conditions of manufacture precluded the possibility of the formation of sufficient acid or other abnormal conditions not found in commercial practice.

SUMMARY AND CONCLUSIONS

Cheese has caused a great many epidemics of disease. The organisms most commonly associated with cheese-borne infections are *E. typhosa,* the *Salmonella* group such as *Salmonella aertryke, Salmonella suipestifer,* and *Salmonella schottmülleri,* of the *Brucella* group *Brucella melitensis,* and to a lesser extent *Clostridum botulinum,* and streptococci. There are no reports in the literature of undulant fever due to *Brucella abortus* or septic sore throat due to streptococci caused by eating cheese despite the fact that these organisms surely must be present at times in raw milk made into cheese. There is no evidence that *Escherichia coli* is a health hazard although it is an economic one in cheese.

The conflicting results regarding the viability of *E. typhosa* in the experiments with commercially prepared cheese may be due to the type and strain of *E. typhosa* used by the different investigators. Wade and Shere used a culture of typhoid bacilli which they isolated from a 57-year-old woman who had had typhoid fever in 1900 which was about 27 or 28 years previous. So it may have been an R or rough strain of the organism. Campbell and Gibbard used a smooth Vi strain which might be more resistant than the rough strain.

From all the available information it would seem that the 60-day holding period is too short a time. The cheese does not have time to ripen and there

may also be too many viable pathogenic bacteria present at the end of this time. A 90-day holding period should be the minimum time required. This gives the cheese time to ripen, and practically all pathogenic bacteria should have died or have become attenuated by the end of this time. A 120-day holding period such as Colorado requires is much better and safer for cheese made from raw unpasteurized milk.

Pasteurization is the only safe procedure to recommend or require. It is not only a safe procedure but also an economic one since cheese made from pasteurized milk or cream can be ripened in half the time because it can be ripened at a higher temperature, 60° F. instead of 40° F. or lower. Furthermore, if pathogenic bacteria are present, they will die more rapidly at the higher than at the lower temperature in cheese.

A combination of pasteurization and a 90-day holding period would be more nearly ideal as well as economically sound since it would produce a safe as well as a mature cheese.

REFERENCES

1. Baker. *Michigan State Board of Health Report,* 122–128 (1884).
2. Barthel, Chr. Bakteriologic des Meiereivesens, p. 116. Cited by Northrup (30).
3. Bassenge. *Deutsche. Med. Mach.,* 29, 675, 697 (1903).
4. Behla. *Handbuch. d. Fechnischen Mykologie.,* 2 Lafar p. 36.
5. Bowman. *Canadian P. Pub. Health,* 33, 541 (1942).
6. Blix and Tesdal. *Norsk. Mag. f. Laegevidenskapen,* 89, 689–707. Cited by Tanner, *Food-Borne Infection and Intoxications,* p. 225, 1933.
7. Carpenter and Book. *Amer. J. Pub. Health,* 18, 743 (1928)
8. Campbell and Gibbard. *Canadian J. Pub. Health,* 35, 158 (1944).
9. Dack. *Food Poisoning.* University of Chicago Press, Chicago, Illinois, pp. 42–43 (1943).
10. Dolman. *Canadian J. Pub. Health,* 32, 183 (1941).
11. Drescher and Hopfengärtner. *München. Tierärztl. Wchnschr.,* 81, 465, 485, 496, 507, 519 and 533 (1930).
12. Eyre. Part IV, pp. 25–27, Report 1905–1907, Great Britain Comm. for the Investigation of Mediterranean Fever.
13. Foley. *11th Ann. Rept. Prov. Bur. Health,* 1932–1933, p. 25, Montreal, Canada.
14. Freidel and Yale. *J. Milk. Tech.,* 8, 49 (1945).
15. Galtier. *Comp. Rend. Acad. Science,* 104, 1333 (1887).
16. Gauthier and Foley. *J. Milk Tech.,* 7, 211 (1944).
17. Graham-Stewart and Associates. *Brit. Med. J.,* 1, 1934 (1928).
18. Gilman, Dahlberg, and Marquardt. *J. Dairy Science,* 29, 71 (1946).
19. Harrison. *19th Ann. Rept. Bureau of Animal Ind.,* 217, 1902. [Also *Ann. Agr. Suisse,* 1, 321 (1930).]
20. Heim. *Arb. a. d. Kaiserl. Gesundheit,* 5, 294 (1889).
21. Hucker and Marquardt. *10th Ann. Rept. N. Y. Assn. Dairy Milk Inspectors,* p. 171, 1936.
22. Hormann and Morgenroth. *Hyg. Rund.,* 8, 1081 (1898).
23. Jordan. *Food Poisoning.* University of Chicago Press, Chicago, 115 pages, 1917.
24. Lerche. *Ztschr. f. Infektionskrank.,* 38, 253 (1931).
25. Levin. *J. Lab. and Clinical Med.,* 2, 761 (1917).
26. Linden, Turner and Thom. *Pub. Health Repts.,* 41, 1647 (1926).
27. Macaulay. *Lancet,* II, 1012 (1922).
28. Mohler, Washburn and Doane. *26th Ann. Rept. Bureau of Animal Ind.,* 187, (1909).
29. Nevin, J. *Infectious Diseases,* 28, 226 (1921).
30. Northrup. *Mich. Agr. Exp. Station.* Tech. Bull. 9, 1911.
31. Peres and Granon-Fabre. *Bull. Soc., Sci. Vet. Lyon,* 39, 110–113 (1936). Abstract in Veterinary Bull. (Weybridge), 7, 414 (1937). Cited by Gilman *et al.* (18).
32. Ranta and Dolman. *Canadian J. Pub. Health,* 32, 73 (1941).
33. Reed. *J. Amer. Med. Assn.,* 21, 354 (1893).
34. Rev. in *Bull. Hygiene,* 2, 825 (1927).
35. Rich. *Am. J. Pub. Health.,* 13, 210 (1923).
36. Rabinowitch. Part I, 123—G. Cornet's *Die Tuberkulose* (1907).
37. Rowland. *British Med. J.,* No. 1799 (1895).
38. Savage and White. *Med. Research Council. Spl. Rpt. Series,* 92, 1925, p. 90.
39. ——— *Ibid.,* 91, 1925.
40. Schytte and Tesdal. *Norsk. Mag. f. Laegevidensk.,* 89, 689 (1928).
41. Schroeder and Brett. *J. Amer. Vet. Med. Assn.,* 52, 674 (1917).
42. Smith. *J. Hygiene* (Cambridge), 34, 242 (1934).

43. Swanner, R. O. Unpublished Report N. Y. State Department of Health, 1939. Cited by Yale and Marquardt (57).
44. Stiles. *Rocky Mountain Medical Jour.*, 42, 18 (1945).
45. Sternberg. *Report Michigan State Board Health*, 1885.
46. Spica. *Chem. Absts.*, 10, 1559 (1916).
47. Timmerman. *Nederl. tijdschr. v. genesk.*, 81, 443 (1937). Abst., *J. Amer. Med. Assn.*, 109, 1590 (1937).
48. Thompson. *Canad. Med. Journ.*, 29, 9 (1933).
49. *U. S. Naval Bull.*, 25, 484 (1927).
50. Vaughan. *Ztschr. f. physiol. Chem.*, X, 146 (1886).
51. Vaughan and Novy. *Ptomains and Leucomains.* Lea and Febiger, Philadelphia, 1896, p. 83.
52. Vaughan and Novy. *Ibid.*, pp. 94–97.
53. Vaughan and Perkins. *Arch. f. Hyg.*, 27, 308 (1895).
54. Versilova. *Brucellosis in Sheep*, pp. 407–417. Moscow: Viem. Publ. Dept. Abstract in *Veterinary Bul.* (Weybridge), 7, 414 (1937). Cited by Gilman *et al.* (18).
55. Veloppi and Jaubert. *Rev. Gen. de Med. Vet.*, 44, 513 (1925). Cited by *Gilman et al.* (18).
56. Wade and Shere. *Amer. J. Public Health*, 18, 1480 (1928).
57. Yale and Marquardt. *J. Milk Tech.*, 3, 326 (1940).

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GERMAN CONTINUOUS BUTTER CHURN

A novel German continuous buttermaking machine, captured in Germany by a Quartermaster Corps Intelligence Team and brought to the United States for testing.

A novel German continuous buttermaking machine has been brought to the United States for testing. Results of the test will be made available to American industry after research is completed, in about three to six months. The machine will not be displayed publicly until then.

Continuous buttermaking machines have not been used commercially in the United States, though two companies have developed experimental models and set up pilot plants. The German machine was designed to produce 1,500 pounds of butter per hour, according to the Quartermaster Corps Intelligence Team that captured it. It occupies less space than American churns of similar capacity.

The machine was produced by the Roth Moelkerei Maschinenfabrik, manufacturers of dairy equipment in Stuttgart.

Testing will be conducted by the Research Committee of the American Butter Institute, under contract with the Quartermaster Corps Food and Container Institute (formerly the Subsistence Research and Development Laboratory).

Experiments on application of the machine to American dairy plant operation will be conducted under the supervision of Dr. H. A. Ruehe of the Department of Dairy Husbandry of the University of Illinois in the Beatrice Creamery Company plant at Champaign, Ill.

The machine will be closely restricted during the tests, to make sure that all dairy and equipment companies have an equal opportunity to use the information. Findings will be reported to the Quartermaster Corps and turned over to the Office of the Publication Board for general release.

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