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Milk-borne diseases, epidemiology and control

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MILK-BORNE DISEASES
EPIDEMIOLOGY AND CONTROL

by
John D. LeMar

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INTRODUCTION

It has been said that the most important single food to which we have access is milk. Man, seeking for an easily accessible source of food, early learned the value of the milk-producing animal. The origin of the practice of using animal milk for food is lost in antiquity. We can only say that it was well established at a very early date and is referred to frequently in the art and the literature of ancient civilizations.

However, the very factors which made for the introduction of animal into the human diet also provided for a very real threat to the life and health of its users. As a culture medium for bacteria its excellence is equaled by few substances, perhaps surpassed by none. This becomes particularly true when milk is collected and held in storage, for then the all-important factors of time and temperature and opportunity for contamination really come into play. All factors being favorable, the multiplication of bacteria in milk can go on to staggering proportions. When these organisms are pathogenic for human beings, milk-borne disease becomes possible.

It is not known just when milk was first suspected as being an agent for the transmission of disease.

Suppositions and suspicions must have been entertained by medical observers comparatively early, but they could be nothing but suspicions until the pioneer work in bacteriology in the last century opened the way to a clearer understanding of the causation of disease.

It is the purpose of this thesis to discuss the milk-borne epidemics of the United States and methods for their prevention. It is obviously impossible to discuss all of the epidemics (there have been more than 1100), but the general features of each particular milk-borne disease will be described, together with a description of one or more type epidemics which illustrate salient points. This work is necessarily a copy of the work of many authors and is in no sense original. Certain mistakes are inevitable, for there are numerous discrepancies in the literature. The numbers of epidemics, as listed here, certainly represent an underestimate.

Acknowledgements must go to Dr. C. W. M. Poynter and to the members of the library staff for suggestions and for aid in obtaining data. Dr. H. A. Harding of the Dairy Research Bureau and Mr. Harry Iddings of the Roberts Dairy Company have contributed much in the way of valuable information. Dr. Thomas Parran

and Dr. Leslie C. Frank of the United States Public Health Service have generously supplied me with much information concerning the prevention of milk-borne epidemics and the application and functions of the U. S. Public Health Service Milk Ordinance and Code.

The problem, as Armstrong and Parran (1927) have pointed out, is peculiarly an American one, and for that reason this discussion has been limited to the diseases occurring in this country.

MILK-BORNE EPIDEMICS.

General Considerations.

Milk-borne epidemics have long been known to have certain common characteristics. The health officer has been able, in numerous instances, to diagnose such an epidemic by these characteristics even before he has had an opportunity to study the situation. Armstrong and Parran (1927) list them as follows:-

1. The outbreak is often explosive in onset - but not always so.
2. The percentage of cases on the incriminated milk supply is greater than the percentage of population using that supply.
3. Cases occur among users of milk, ice cream, etc; therefore children, women and members of

well-to-do families often suffer higher attack rates than men and members of poorer families.

4. Multiple simultaneous cases often occur in the same household.
5. The incubation periods of a given disease may be shortened.
6. When the infected milk supply is stopped, the outbreak subsides.

There are special features of the different diseases which will be discussed in their proper places.

Interest in milk-borne diseases in the United States began about 1880. Trask (1909) discussed the problem and reported a number of epidemics. His compilations, together with those of earlier authors brought the total number of epidemics to 179 at that date. Armstrong and Parran (1927) completed the compilation to January 1, 1927, bringing the total number to 791. They confined their study to the United States because, as they state, the habit of using uncooked milk and its products is more common in this country than elsewhere and because the data from other countries is so incomplete as to be almost useless. According to these authors, outbreaks of milk-borne disease may be listed according to years in the following manner:-

Years	Outbreaks
1881-1885	3
1886-1890	14
1891-1895	26
1896-1900	33
1901-1905	60
1906-1910	145
1911-1915	238
1916-1920	130
1921-1925	130
1926	12
Total	791

Thus the incidence of milk-borne outbreaks rose steadily to a peak in 1914, during which year 55 epidemics were reported. Then came a sharp reduction, going as low as 8 for 1919, followed again by a rise to another peak of 28 epidemic in 1921 and still another in 1924 of the same number. These authors believe that the general reduction since 1914 are due to betterment of the milk supply over the country.

Harding (1936), obtaining his data from the U. S. Public Health Service, the Health Departments of various states and from health officers directly, gives the following list of epidemic occurring since the work of Armstrong and Parran:-

Year	Epidemics
1927	36
1928	47
1929	50
1930	48
1931	34
1932	33
1933	42
1934	45
1935	38
Total	415

This brings the grand total to 1206 epidemics listed in the United States up to January 1, 1936. No one seems to know why more than a third of the reported epidemics should have occurred in the last ten years. Possibly more efficient reporting and greater interest in public health matters has led to a more diligent search for such epidemics. Interestingly enough, the locations of these epidemics has shifted from large cities to the smaller towns and the rural sections. Harding has often discussed this and in his report on the 1935 epidemics (1936) he states, "There is the usual relation between location of the milk borne epidemic and the size of the community in which it occurs."

"Seven of these outbreaks occurred in such small communities that the population is not given. Seven more were in communities of less than 1000 people. Fourteen more, or a total of 28 epidemics, were in communities of less than 10,000 -- in groups so small that little or no local supervision of health matters was possible. Put in another way, 74% of these epidemics occurred in communities so small that the milk supplies were not under local health control."

Armstrong and Parran (1927) state that in the 612 milk-borne epidemics studied by them, 42,327 individuals were affected. There were 410 deaths. They admit that the data is incomplete. Since then the year attack rate has varied, according to files of the U. S. Public Health Service, but average about 1500 per year. The largest number of people afflicted in one year (recently) was 2,589 in 1929, the lowest available figure being 638 for 1932. There is no uniform death rate, it varying from less than 1% to nearly 10% in different years.

Rosenau (1929) listed the number of bacterial diseases transmittable by milk as twelve. They are :- typhoid fever, paratyphoid fevers, tuberculosis, food infections, diphtheria, scarlet fever, septic sore throat, undulant fever, foot and mouth disease, diarrhea and dysentery, epidemic arthritic erythema and

anterior poliomyelitis. Armstrong and Parran (1927) add botulism, epidemic appendicitis and parotitis, a dengue-like syndrome. "Milk sickness" may be classed here, according to Rosenau (1928), though the disease quite probably is not bacterial in origin.

A general discussion of the sources of milk contamination may be given at this point. The opportunities for infection of a milk supply are three fold, namely, via the infected milk-producing animal, during the handling of the milk previous to its delivery to the milk plant, and during the processing and the delivery of the finished product. Infection of the milk-producing animal may be of two types: in the first a given disease may be primarily present in the animal, as in bovine tuberculosis foot and mouth disease, undulant fever and perhaps some others. In such cases the infection of the milk supply is incidental and the manifestations of the disease in man are entirely secondary. In the second type of animal infection, the animal may acquire a disease which is primary in man thereby act as a distributing agent for a disease which probably does not occur in cattle in the natural state. Septic sore throat, scarlet fever and diphtheria are the chief diseases falling into this category. Specific examples will be

given during the discussion of each disease.

While the carrier of a disease and the active case may infect cattle, they really come into their own as purveyors of disease when they act as milk handlers, dairy attendants and milk distributors. Typhoid fever, the paratyphoid fevers, diarrhea and dysentery, and also poliomyelitis, diphtheria, scarlet fever, septic sore throat and undulant fever may be transmitted this way.

The indictment, states Rosenau (1929), is strong against raw milk as the chief avenue of milk-borne infection. According to this author, no diseases can be traced to properly pasteurized milk. Armstrong and Farran (1927) found that raw milk was incriminated in 179 outbreaks; pasteurized milk or its products were incriminated in 29 outbreaks, certified milk in 3, ice cream in 36, butter in 3 and cheese in four. In 356 outbreaks the agent of transmission was not stated. The incidence of raw milk borne epidemics in later years is more appalling. Harding (1930-1936) states that in 1929, 45 of the 50 epidemics were traced to raw milk, while 45 of the 48 epidemics of 1930 were traced to the same source. In the latter group two were due to both raw and pasteurized milk, and one was traced to pasteurized milk alone. In 1931, 31 of

34 epidemics were traced to raw milk, while in 1932 the figure was 31 of 33 epidemics. In 1933 all of the 42 epidemics occurred after the use of raw milk. In 1934, 44 of 45 epidemics occurred after the use of the same, the single exception being probably transmitted via the same agent. The score was no better in 1935, for 31 of the 36 epidemics were transmitted through raw milk and the remainder through improperly-pasteurized milk or mixed supplies. The case against raw milk is clear.

Rosenau (1929) states that while the sole function of milk is food and although it is made precisely for the homologous species and was intended to aid in the preservation of the species, it has probably been responsible for more sickness and death than all other foods combined.

The case cannot be laid at the door of milk, as such, but the blame must be placed on our methods of handling this natural product.

SPECIFIC MILK-BORNE DISEASES.

I. Typhoid Fever

Since the earliest studies were begun on milk-borne epidemics, this disease has led the list in the number of outbreaks. Trask (1909) described 107 of

these, while previous investigators had listed 27. To these the Armstrong and Parran (1927) series added 479, bringing the total to 613 at the close of 1926. Later compilations by Harding (1936) brought the total to 836 milk-borne typhoid fever epidemics up to 1936. The 613 epidemics occurring between 1881 and 1927 comprised 77.5% of all the reported milk-borne epidemics. The 479 typhoid fever epidemics occurring between 1907 and 1927 (milk-borne) affected 14,968 individuals and caused 219 deaths.

Since 1926 the outbreak-percentage of typhoid fever has been maintained with remarkable constancy. This is best shown in Table 1.

Table 1.

Year	Total Outbreaks (all kinds)	Typhoid Outbreaks	Typhoid %
1881-1926	791	613	77.5
1927	36	25	69.0
1928	47	26	55.0
1929	50	29	58.0
1930	48	30	63.0
1931	34	22	65.0
1932	33	23	70.0
1933	42	25	62.0
1934	45	27	60.0
1935	38	16	42.0

Thus there was a total of 836 typhoid fever epidemics in a total of 1206 of all kinds, a general percentage of 69.3%.

The national percentage is closely approximated by figures from comparatively small areas, notably Massachusetts. Bigelow and Forsbeck (1927) studied the outbreak percentage of typhoid fever in this state. According to their figures, typhoid fever constituted 69.45% of the epidemics reported during the period of 1907-1914. From 1915 to 1918 the percentage fell to 57.14%, but rose to 70.59% during the period of 1919-1923. Instead of improving during the next period, 1924-1926, it rose further to 77.78%. The persistence of these percentages is a thing difficult to explain.

Bigelow and Forsbeck (1927) discuss another interesting angle; from 1907 to 1914 about 9.43% of all cases of typhoid fever could be traced to infection via milk. From 1915 to 1918 the percentage declined to 7.83%, but little difference could be seen during the 1919-1923 period, the percentage declining but 0.5%. But during the 1923-1926 period the percentage again declined to 4.1%.

The case percentage distribution of typhoid fever in relation to other milk-borne diseases showed no such encouraging change however. In the period from 1907-

1914 typhoid fever comprised 29.13% of all cases of milk-borne infections. This rose during the period of 1916-1918 to 32.36%, to be followed by another sharp rise during the 1919-1923 period to 62.40%. There was a fall to 50.0% during the period of 1924-1926 inclusive.

Armstrong and Parran (1927) studied the monthly distribution of the 479 typhoid fever epidemics reported by them. Outbreaks were scattered throughout the year, but the peak was reached in August and September. The rise to and the fall from this peak was sharp. The authors attempt to explain this by saying that the opportunities for contamination are greater during this time, since cases of typhoid fever from general sources are somewhat more numerous at this time than at others. Furthermore, they state, extra help must be hired about the dairy during the summer, thereby possibly introducing carriers who would not otherwise come in contact with the milk. Flies are more numerous at this time of year, and heat of summer makes proper cooling of milk difficult. The authors mention an epidemic which occurred at the State College, Pullman, Washington, in which 60 cases developed among students at a boarding house. The same milk was used by two other boarding houses but no cases resulted in them. The only difference found was that the first boarding house kept the milk at room

temperature, while the others kept their supplies in the refrigerator.

Throughout the history of milk-borne typhoid fever, raw milk has been the chief medium of infection. In the series of Armstrong and Parran (1927) milk was incriminated in 444 instances, 133 of these being raw milk. Pasteurized milk was involved in only 21, while the character of the milk was not stated in 290. Even where wasteurized milk has been involved, investigation usually reveals that the milk was contaminated after heating, or that the pasteurization process was faulty or not actually done.

The source of infection was traced or the probabilities established in 373 of the outbreaks listed by Armstrong and Parran. First and foremost was the carrier on the farm, among the milk handlers and in the distributing plant. These authors list 162 outbreaks traced to this source. In most instances the carrier was not even suspected until a disastrous outbreak had occurred. Harding (1934), quoting from Health News for July 2, 1934, states that 307 carriers of this disease were discovered in upstate New York in the ten year period between 1924 and 1934. Sixty-eight percent of these were discovered during epidemiological studies incident

to outbreaks. Sporadic cases often warn the health officer that a carrier is about. Harding has long been an advocate of careful examination of milk handlers to detect carriers.

This same author (1936) quotes the 1935 Kentucky Public Health Manual on the carrier problem. In this state all milk handlers were required to have a certificate of health, renewable each year, to the effect that they were not carriers of typhoid or paratyphoid bacilli. This has been carried on for ten years, and 32,200 examinations have been made, with incidence of positive stool cultures being 0.5%. All those having a positive stool culture have been required to submit daily stool samples for 10 days. If no more positive cultures develop, the individual is given a provisional certificate of health for 3 months, after which time he just again submit more samples. Only about 15.8% of those which were found to be positive on a single examination actually proved to be carriers, i.e., one carrier was found for each 1464 milk handlers. A dealer employing twenty persons might expect to have one who occasionally discharges typhoid bacilli. Many actual carriers do not constantly expel the organisms, a fact which makes it more difficult to discover them.

Miller (1934) states that apparently about 2% of

those convalescent from typhoid fever become chronic intestinal carriers, a smaller number becoming urinary carriers.

However typhoid carriers have not necessarily had a recognizable form of the disease. The Armstrong and Parran (1927) studies include six outbreaks due to carriers who had not had clinically recognizable typhoid fever. These same authors found that in 32 outbreaks in which carriers were discovered, 28 were fecal carriers, three urinary carriers and two were mixed. The Widal reaction and the routine bacteriological examination of both feces and urine have been used in the search for carriers. A history of previous typhoid fever should always label the individual as a suspected carrier until reliable examinations have proved otherwise.

The plight of the carrier is not an enviable one. Without cure he is a constant menace, yet a cure cannot be assured him. Since the seat of the bacillary discharge is often the gall bladder, cholecystectomy would seem advisable. Yet this procedure cures but 75% of those submitting to it and the operation is a formidable one with a mortality of 15%.

The active case of typhoid fever constitutes the second most important source of milk infection, account-

ing for 134 outbreaks in the Armstrong and Parran series. It could easily be handled if illness among milk handlers was quickly reported and investigated by health authorities.

Exchange of bottles from dwellings housing active typhoid cases have accounted for a few epidemics. Characteristically, according to Armstrong and Parran (1927), infection through this source causes sporadic cases on a given route and not infrequently scattered cases on other routes. The use of contaminated water in washing equipment has been incriminated in some instances, while soilage of cows in polluted water has given rise to four outbreaks. Infected cream, butter and ice cream have also been implicated in a few instances.

The milk-borne outbreaks of typhoid fever have certain well established features, according to Rosenau (1929) They are:-

1. There is special incidence of the disease on the tract of the implicated milk supply, the outbreak often being localized to the route of the milk wagon.
2. The homes of the better class are invaded and often these suffer the most.

3. The milk supply is usually raw.
4. The incidence is higher in women and children than in typhoid epidemics from other sources.
5. The incubation period of the disease may be shortened due to massive dosage of the pathogenic organisms.
6. More than one case will occur in a given home. Frequently the appearance of several cases simultaneously in a house is the first indication of an epidemic.
7. Clinically the disease is often mild, possibly because there may be some attenuation of the organisms due to multiplication in milk. This does not occur in water.
8. Outbreaks are usually small.

There have been some notable exceptions to the last statement. One of these, the Montreal epidemic, has been chosen as the type epidemic.

The Montreal Epidemic.

The city of Montreal was visited by a typhoid epidemic between March 1 and June 28, 1927. The epidemic appeared suddenly and ran nearly unchecked for this period. It was the largest one in history, involving 5,014 individuals and causing 488 deaths.

The affair was investigated by a special board of the United States Public Health Service (1927) and the report of this body forms the source of information given here.

The epidemic began about February 15th and rapidly built up to a peak about March 5th. Its incidence of cases remained high until March 18, then subsided sharply until April 20th when a recrudescence began. This was sudden and reached the peak on May 2nd. It remained high until May 6th, then fell off to a fifth of its former level by May 15. From then on the numbers of patients among those using the implicated milk declined while the number of secondary cases mounted.

Investigation indicated that a preponderance of the cases occurred among users of the milk of Montreal Dairy Company Ltd. Of a thousand investigated cases, 90% of the patients were users of this milk and of other dairy products from this plant. Furthermore, in the various institutions of the city inmates using this milk developed many cases while users of other brands of milk had no increase over the usual number of cases. It became evident early in the investigation that the water supply was not implicated, since an adjoining town used the same milk supply but a different water supply, and still had many cases.

The local health officer recognized the situation and did his duty as best he could. Because of entirely inadequate help the department was not able to offer much aid.

32% of the cases occurred in children under 10 years of age, a significant fact according to many health authorities. Investigation of the milk supply indicated that it had its origin at 1200 to 1500 farms. It was estimated that the milk was exposed to 20,000 people. The milk was taken to collecting stations in some instances and it was found that one of these used water pumped from a contaminated river to rinse the equipment and utensils. Many of the producing farms were unsanitary and some of the milk came from uninspected farms.

Inspection of the dairy personnel disclosed that the foreman was a carrier of typhoid bacilli. He had had the disease 20 years previously. As far as he knew he had never caused any cases elsewhere. It did not seem likely that he could have been the source of the infection, since he did not handle any milk personally. Curiously enough, the man who replaced him developed an illness which was probably typhoid fever. He promptly disappeared and could not be traced.

Inspection of the plant failed to reveal anything faulty about the pasteurizing apparatus and methods nor of the bottling and delivery units. Company records suggested, however, that more milk was delivered to the plant than was pasteurized. Later examinations showed that at about the time of the recrudescence, the counts of the pasteurized milk were low (1000 per cc) but that B. coli was present in the milk. It was discovered that a pipe line to the bottling machine was connected to the feed line of the milk and cream pasteurizers. When this was blocked off the colon bacilli disappeared, indicating that the valves were leaking.

The board, in its final analysis, came to the conclusion that the bulk of the infection must have occurred at the farms and that a considerable portion of the milk got through the plant without pasteurization. They recommended a more efficient health department, closer check on pasteurization equipment and water supplies to collecting stations, and medical supervision for the dairy employees. It was also stated that every plant needs a reliable trained man in charge of plant sanitation.

II. Paratyphoid Fevers.

Milk-borne outbreaks of these diseases have been comparatively rare, only 7 being reported by Armstrong and Parran (1927). These caused 434 cases, with 15 deaths. Two outbreaks were traced to active cases on farms, three to carriers.

The 1929 Annual Report of the Surgeon General of the U. S. Public Health Service listed 2 epidemics in 1927. None were reported in 1928.

Harding (1930-1936) has listed 1 epidemic in 1929, with 38 cases and 1 death, one in 1931, with 22 cases and no deaths, 1 in 1933 with 17 cases and no deaths. There was 1 epidemic in 1934 with 400 cases and no deaths and in 1935 there were 2 epidemics with 50 cases and no deaths. All of these occurred in users of raw milk. Carriers were incriminated in 4 of these outbreaks, active cases in the others. In all respects these diseases behave epidemiologically as typhoid fever. No doubt some confusion between these and true typhoid fever has occurred.

The single epidemic occurring with Certified milk was reported by Williams (1925). Sixty infants and children were involved. There were no deaths. The epidemic occurred in New Rochelle, N.Y. in 1924. The onset was sudden, the cases appearing between March 5 and May 8. When the milk of the incriminated dairy

was stopped on May 7, no more cases appeared. Every patient used the milk. A number of adults using the milk reported milk gastric disturbances. One interesting feature was that the single diagnosed adult case occurred in a 40 year-old individual who was on a Sippy diet. Examinations of the milk handlers showed that one of them was a carrier of *Salmonella schottmulleri*.

A small epidemic occurring in Ames, Iowa was reported by Levine and Ebersson (1916). In this epidemic it was found that the wife of the milk dealer was a carrier. She washed and filled the bottles, which were not sterilized. 90% of the patients drank the milk of this dealer. 70% were in children under 14 years of age.

A relatively large epidemic was reported by Wade and McDaniel (1924). This occurred in patrons of the Union Cafeteria, University of Minnesota, at Minneapolis. The epidemic began March 4, 1921 and ran until April 13th of the same year. There were 106 cases with 2 deaths. 78% of the cases developed between March 14 and March 21. The list of cases included 84 students, 1 member of the faculty, 7 employees of the Cafe and 14 outsiders. Investigation of the epidemic proved that 103 of the cases had eaten at the restaurant within

two weeks of the onset. One hundred had consumed milk as a beverage or on cereals or in coffee. The incubation period of those who had eaten but one meal at the Cafe was 2 to 11 days. More than a third of those infected had been immunized against the disease 3 or 4 years before this epidemic occurred.

Investigation of the possible means of transmission quickly ruled out all foods but milk. This milk, delivered in 50 gallon bulk lots daily, was pasteurized, but was poured out into pitchers by kitchen attendants before being served. When the stools of these attendants were examined it was found that four were carrying *B. paratyphosus B.* One of these had had an acute gastro-enteritis between March 15 and March 20. It was not possible to actually trace the epidemic to these individuals, though the implication was strong. The authors of this report believe that that epidemic strikingly indicates the necessity of routine examinations of food handlers. Furthermore they feel that all milk to be consumed uncooked should be pasteurized and served in the original containers.

III. The Streptococcal Diseases.

A. Septic Sore Throat

This disease, say Armstrong and Parran (1927), is probably always milk-borne. In character it resembles other milk-borne diseases, i. e., the onset of an epidemic is explosive and is confined to the route of the infected milk supply. There is one feature which is very important, the fact that of all the diseases which are transmitted through milk, this one affects more individuals than any other. The Armstrong and Parran series include 42 outbreaks affecting 21,045 people and causing 139 deaths. Raw milk was responsible in 19 outbreaks, pasteurized milk in 3, certified milk in one and ice cream in one. Scamman (1929) reported 45 epidemics through 1928, affecting 22,431 individuals and causing 187 deaths. The 1929 Annual report of the Surgeon General of the U. S. Public Health Service reports that there were 1,080 cases in 9 epidemics during the years 1926, 1927 and 1928.

Harding (1930-1936) has carefully collected information on epidemics since 1928. He reports that in 1929 there were 9 epidemics with 939 cases and 13 deaths. During the next year there were 9 epidemics with 1,116 cases and 7 deaths. In 1931 there were 6 epidemics with 993 cases and 8 deaths. In this particular year the number of cases for all epidemics was 1,364, with the total deaths standing at 24.

The number of epidemics dropped to 3 in 1932, with 149 cases and 3 deaths. In 1933 there were 7 epidemics of septic sore throat with 515 cases and 5 deaths. The total epidemics of the year were 42, total cases 1,348.

In 1934 there were 8 outbreaks of septic sore throat with 557 cases and 13 deaths. Nine outbreaks occurred in 1935 with 1000 cases and 7 deaths.

Thus in each year epidemics of septic sore throat comprise a relatively small number of the outbreaks, yet make up a large number of the total cases for all epidemics.

Raw milk is incriminated in most instances. As to source of infection, active cases on the dairy farm were the most frequent source in the Armstrong and Parran series. Cases on the farm combined with bovine mastitis was the second most important source, while isolated bovine mastitis and human carriers were less important. Scamman (1928) found that 55% of the outbreaks in his series were traceable to an infected milk handler. Thirty percent were traced to the combination of infected handler and infected dairy herd.

It has only recently been understood how bovine mastitis could be related to this disease. Such mastitis, or "garget", is extremely common in many

herds. The organisms usually found is *Streptococcus mastitidis*, which is non-pathogenic for humans.

Recent work in final hydrogen ion concentration in glucose broth, the fermentation of synthetic carbohydrates and the hydrolysis of sodium hippurate have made it easy to differentiate this organism from others.

Jones and Little (1928) threw valuable light on the problem when they demonstrated that udders may be infected with hemolytic streptococci which are pathogenic for humans. The organisms usually credited with causing septic sore throat has been known as *Streptococcus epidemicus*, and its differentiation from other hemolytic streptococci has been based largely on its capsule forming tendencies.

Brooks (1932) and many others have confirmed the work of Jones and Little. Steadily the evidence has been accumulating until the public health importance of bovine mastitis is well recognized. Thus Frost and Carr (1927) reported an epidemic of 63 cases in Madison, Wis., in which the only known source was three infected cows in a herd of thirty. One of these produced 36 million streptococci per cubic centimeter of milk.

Curiously enough, the states in which public health control has long been active, New York and

Massachusetts, have also lead the country in the number of septic sore throat epidemics. In discussing this Brooks (1933) found that up to 1933, 63% of the epidemics of this disease occurred in these two states. He believes that many epidemics are missed in other states, either due to "missed" cases, or to lax reporting. Possibly, he admits, other states do control it by adequate milk control, and the disease may be actually more prevalent in these states than elsewhere. The author believes, however, that more cases would be found if more states had an adequate public health administration.

The differentiation of true septic sore throat from true scarlet fever without rash is not easy. Indeed, Williams, Gurley, Sobele and Castelda (1932) state that hemolytic streptococci of the so-called scarlet fever type and the septic sore throat type may be found together in the same epidemic. Furthermore, they state, streptococci of the scarlet fever toxigenic type have been isolated from clinical septic sore throat, and vice versa. They found that a greater variety of strains of streptococci were to be found in a septic sore throat epidemic than in a milk-borne

Swift, Lancefield and Goodner (1935) reviewed the present methods of streptococcal classification

and concluded that the serological grouping devised by Lancefield (1933) was of particular value in dealing with epidemiological problems. The close relationship between epidemic septic sore throat and erysipelas adds another confusing angle.

There have been many references to epidemics of septic sore throat, but two are particularly interesting. The first of these was the famous epidemic which occurred in Chicago in December, 1911, and January 1912. This was reported by Capps and Miller (1912). The epidemic was explosive, most of the cases occurring about Christmas and New Years' Day. It soon became evident that most of the victims were drinking pasteurized milk from Dairy X. 85% of those with severe symptoms were users, as were 75% of the 19 who died. The dairy supplied milk to better class homes on the North and South divisions of the city. On one route, 51% of the households were affected. The morbidity ratio of X users to others was 14 to 1.

Of 153 nurses using X milk, 52% developed sore throat, while in another group of 721 nurses using the milk of other dairies only 4.8% developed sore throat. In one hospital the children were supplied with milk from Dairy X, but the milk was specially pasteurized after being delivered to the hospital.

Not a single case developed among the children, while internes and nurses in the same hospital had many cases. At Batavia, Ill., site of the pasteurizing plant and center for the herd, consumers of X milk had 3.6 times as many cases of sore throat as did users of other milk.

The milk had been pasteurized, the "flash" method being used. It was found that this was inefficient, the temperature not reaching the required level on December 17, 19, 28 and 30, and on January 7 and 11. The first wave of the epidemic appeared about December 21, reaching a peak on December 25th. The next peak came January 10-17. Allowing 2 days for delivery and 2 to 4 days for incubation, the outbreaks are seen to correspond to pasteurization failures.

Another significant point was noted by veterinarians of the area. They noticed that there was an unusual amount of bovine mastitis about at this time. The infection usually started at the tip of the teat and ascended into the lactiferous ducts and glands. At the same time, eleven of the milk farms had 28 cases of pharyngitis among handlers, and 8 of the 11 farms had mastitis in the herds.

It is estimated that 10,000 cases occurred in this epidemic.

The second interesting epidemic assumed the nature of a carefully controlled experiment, quite by accident. The epidemic occurred in May 1936 in Bergen County, N.J. and involved 175 cases with 7 deaths. The information given here is quoted from Harding (1936), who in turn quotes the report given by McDonald before the International Association of Dairy and Milk Inspectors.

Four hundred fifty quarts of milk were sold daily from a herd of 35 cows. Of these, 275 quarts were sold as raw milk, the rest being pasteurized at the farm. The outbreak appeared explosively during the first week of May, 1936, ran unabated until May 16th, then suddenly disappeared. No cases developed among those using the pasteurized milk, all cases being confined to those using raw milk. It was later found that on May 15th a cow was removed from the milking line because of an abnormal udder. This cow had been milked in April by a milker who had had a sore throat and by a second man who developed a sore throat a few days later. Evidently this animal was the source of the epidemic.

B. Scarlet Fever

This disease has long been associated with milk, though epidemics through this medium have not been

exceptionally numerous. Armstrong and Parran (1927) reported 40 epidemics occurring up to and including 1926. These involved 3,939 cases with 20 deaths. The Annual Report of the Surgeon General of the U. S. Public Health Service for 1929 reports that five epidemics each have occurred in 1927 and 1928, involving about 350 cases and causing 6 deaths. Harding (1930-1936) has collected data which show that 11 epidemics occurred in 1929, with 1,052 cases and 1 death. In 1930 there were 2 epidemics with 42 cases and no deaths. One epidemic was reported for 1931, with 9 cases and no deaths. In 1932 there were 6 epidemics with 148 cases and three deaths. Three epidemics occurred in 1933 with 238 cases and 4 deaths. The number of epidemics dropped in 1934 to two, with 39 cases and 1 death, and in 1935 there were also two epidemics with 65 cases and no deaths. The great majority of these occurred in cities of less than 10,000 inhabitants and in rural sections.

Armstrong and Parran (1927) quote Clark as saying that while ordinary scarlet fever shows an adult-child case ratio of 1 to 48, this is changed in milk-borne epidemics of this disease to 1 to 1. These authors feel that this is not so commonly observed now in this country. But Godfrey (1929) studied 21 outbreaks and found that 57.1% of the 1,362 cases

were in individuals more than ten years old.

Raw milk has always been the chief avenue of infection, while the active case has proven to be the chief agent of infection of the milk supply. Scamman (1929) found that 82% of 56 epidemics reviewed by him were traceable to diseased milk handlers. However the milk cow may become infected from the handler and discharge the organisms in the milk. Jones and Little (1928) state that while the usual view is that the milk supply is infected directly by the infected handler, this is not always the case. Streptococci isolated by them from infected udders matched in all respects the accepted characteristics of human type streptococci. The toxins produced by these organisms were neutralized by known scarlet fever antitoxin. Another feature of their work was the discovery that scarlet fever streptococci are somewhat inhibited by fresh cows' milk, thus, they say, casting doubt on heavy infection of milk by human carriers. Artificial infection of teats and udder was accomplished by them, using streptococci isolated from various human infections.

A typical milk-borne epidemic of scarlet fever has been described by Wilkinson (1931). Sixty-six cases of scarlet fever appeared with explosive suddenness, to-

gether with one hundred cases of severe sore throat. It was found that the epidemic occurred in 20 of 54 homes using the milk of one dairy; all of these were on the route of one man who had had a "rough throat" for a few days (desquamation took place on this man about three weeks later). Only 29 homes in a group of 236 were affected on another route of the same dairy. The milk was the same but the deliverer and handler were different than on the first route.

An epidemic transmitted by ice cream was reported by Ramsey (1925). This was in Flint, Michigan, where about 6 cases of scarlet fever appeared per week under ordinary circumstances. Then suddenly 41 cases appeared within one week. Altogether there were 94 cases. Three deaths occurred. Investigation showed that 81.91% of the cases had eaten the ice cream of one manufacturer. It was also discovered that this manufacturer, who made all the ice cream in the plant, had scarlet fever, and had worked for three days after the onset of his illness. The organisms isolated from this individual and from the ice cream gave positive intradermal filtrate reactions in known positive Dick reactors.

Not uncommonly clinical scarlet fever and septic sore throat have occurred together in the same milk-borne epidemic. Williams, Gurley, Sobeles and Cast-

elda (1932) studied hemolytic streptococci isolated from several epidemics and found that scarlet fever streptococci and the strain commonly found in true septic sore throat - Str. epidemicus Davis - are sometimes found together. Recent studies on streptococci show that there is much to learn about the pathogenic potentialities of these organisms.

III. Diphtheria

Up to January, 1927, twenty-six milk-borne epidemics of diphtheria had been reported, affecting 971 individuals and causing 6 deaths, according to the Armstrong and Parran series (1927). Two epidemics each occurred in 1927 and 1928, causing 48 cases. None were reported for 1929 and 1930, but Harding (1932) states that there was one epidemic in 1931, involving 22 cases with no deaths. Again in 1933 two epidemics appeared, with 19 cases and 3 deaths reported, and in 1934 a single epidemic of 9 cases was reported. There were no deaths.

As in other milk-borne epidemics, raw milk has been the chief infecting medium. Eight of the epidemics reported in the Armstrong and Parran series were transmitted this way; one was through certified milk, one through pasteurized milk and one each through ice cream

and butter. All of the four epidemics described by Harding (1932, 1934, 1935) were transmitted via raw milk.

The active case and the carrier are the chief sources of milk supply contamination. An epidemic occurring in Lincoln, Nebraska was described by Waite (1914). There was a sudden increase in the number of cases occurring in that city in April, 1913. The increase began on April 21, with 9 cases appearing simultaneously, and reached a peak on April 24th when 32 new cases were reported. Altogether 110 cases appeared. It was found that these patients had been drinking the milk of one dairy. Further investigation showed that one of the dairymen, who started working at the dairy April 15th, had consulted a physician for sore throat on April 13th. On April 24th, the throats of this man and his wife contained virulent diphtheria bacilli. An interesting feature is the fact that only 6 other cases appeared in the whole city during this time.

The source of infection is not always traced to infected human throats however. Henry (1920) reports two outbreaks occurring close together in Williamstown, Mass. There was a sudden outbreak in August, 1920, involving 13 cases. Five of these occurred in one boarding house. The kitchen helpers could not be

incriminated. The health officials then suspected the milk supply, since all of the first 21 patients drank raw milk from this source. None of the dairymen were throat carriers, but one girl milker was found to have a diphtheritic infection of a finger. The bottles used here had not been sterilized and "wet-milking" was permitted.

The second outbreak came a month later and was traced to a diphtheritic teat infection in one of the cows. A milker was also found with a diphtheritic hand infection.

McSweeney and Morgan (1926) report a similar situation in England. Seven cases of diphtheria developed in six days in a district supplied by one dairy. It was found that a daughter in the dairyman's family was a throat carrier. It was also found that a number of cows had teat infections, apparently superimposed on cowpox. The shallow ulcers on the teats contained virulent diphtheria bacilli.

The largest epidemic on record is quoted by Armstrong and Parran (1927). The epidemic appeared in Newport, R. I., in July, 1917. Four-hundred two cases appeared, with 50 secondary cases. The only common source of infections was a supply of ice cream. It was discovered that there were two active diphtheria

cases and four carriers on the farm supplying the milk from which the ice cream was made.

IV. Malta Fever and Undulant Fever.

These related diseases have only recently been recognized in human beings. Both are caused by organisms of the genus *Brucella*. Grayson and Hastings (1934) have correlated the findings of earlier investigators and state that true Malta fever in humans is caused by *Brucella melitensis*, the caprine strain. Undulant fever, on the other hand, is caused by bovine or porcine strains of *Brucella abortus*. These are only to be differentiated by cultural reactions and delicate agglutination tests.

Evans (1927), one of the earliest and most productive workers in the field, was one of the first to discuss human infection with *Br. abortus*. According to her, infection with this strain is clinically indistinguishable from true Malta fever. The bovine and caprine strains are very closely related. This author also states that the diseases are often confused with malaria, tuberculosis, acute rheumatism, typhoid fever and tularemia. True *Br. melitensis* is transmitted mainly through goats' milk. Twenty cases are discussed, 8 of which are proven to be of raw milk origin.

Armstrong and Parran (1927) report only one epidemic of true Malta fever. Thirty cases appeared in Phoenix, Arizona, in 1922. These authors, quoting Lake, state that 27 of these used goats' milk from the same source, which the other three may have done so.

Five milk-borne outbreaks of Undulant fever occurred in the U. S. in 1928, according to the 1929 Annual Report of the Surgeon General of the U. S. Public Health Service.

Carpenter and Boak (1928) found *Br. melitensis* variety *abortus* in the udders of 6.08% of 378 cows examined in certain certified dairies. Twenty percent of the animals carried blood agglutinins for this organism. When strains were inoculated into butter, they remained viable for 30 to 80 days.

These findings lead to many investigations, and King and Caldwell (1929) reported that 851 patients and 156 staff members of a sanatorium using Grade A raw milk carried blood agglutinins for *Br. abortus* in titers of 1-15 or higher. Cows in the dairy with an agglutinin titer of 1-60 or lower did not seem to discharge the organisms in the milk, but 23 of 56 cows with a titer of 1-120 or more were discharging the organism. To these authors, presence of blood aggl-

lutinins meant infection.

King (1929) also stated that 20% of the raw market milk examined by him was infected with *Br. abortus*. The disease, he says, is widely prevalent in the United States. Ingestion of infected milk is an important source of infection in humans, and the control of the disease must be brought about by either universal pasteurization of all milk or a new code of Certification.

Hasseltine and Knight (1931) reported a small outbreak of the disease in Pitman, N.J. Six cases appeared rather suddenly, all persons involved being consumers of raw milk from one dairy. Examination of the herd showed that 24 of 42 animals were infected. The Department of Health immediately imposed a ban on raw milk and raw milk products. The dairyman put in a pasteurizing plant and no new cases appeared.

More significant, perhaps, is the work of Johns, Campbell and Tennant (1932) who blood-tested 100 inmates of an epileptic hospital which was supplied with raw milk by a herd of cows infected with *Br. abortus*. Twenty-six of the patient had the disease clinically. Twenty-three patients had a blood titer of 1-80 or more. All active cases were ambulatory. The final results of the investigation illustrated that 21% of the patients had active infection, 38% showed evidence of past infection and 40%

were negative.

That the disease is also active in England is shown by the work of Pullinger (1934), who found evidence of infection in 70 of 101 samples of milk from 45 tuberculin-tested herds. Fifty-three of 63 1000 gallon rail tanks were also contaminated by the organism. Other herds showed comparable results.

Cameron and Wells (1934) found that 26 cases of undulant fever studied by them could be traced to 15 herds, the composite samples of which contained agglutinins in the milk serum. More than 20% of the animals were infected. No cases were traceable to herds in which composite samples showed no evidence of infection (Maryland). Cases in Hagerstown, Md. declined after infected cattle were eliminated.

Still another significant study was made by Stone and Bogen (1935) of patients in a tuberculosis sanatorium. About 6% (66) were found to have sufficient clinical manifestations to warrant a diagnosis of undulant fever. All had been exposed to infected milk.

The milk was then pasteurized and the herd cleaned up. None of those exposed to milk after this developed a positive blood agglutinin titer. The blood titer in those already infected declined rapidly when the source of infection was removed, and 25 of those originally

having a positive blood titer lost it completely.

Harding (1933) lists a table showing the increase of reported cases during the years 1922-1932. It is intended to imply that all of these are the result of milk-borne infection, though probably a large proportion of them are so.

Year	Undulant fever reported cases
1922	1
1923	0
1924	2
1925	24
1926	42
1927	217
1928	649
1929	1,301
1930	1,450
1931	1,545
1932	1,407

Further reports by Harding (1933-1936) give the following figures for later years.

Year	Undulant fever reported cases
1933	1,933
1934	2,010
1935	1,936

Whether these figures mean that the disease is

rapidly spreading through this country or whether it is merely the result of more efficient diagnosis is a matter of speculation. The case incidence per 100,000 population, according to Harding (1936) is highest in Vermont (7.5), Kansas and Iowa, in that order, while Nebraska is 40th on a list of 45 states.

V. Bovine Tuberculosis.

This disease, once regarded as innocuous to humans by no less an authority than Robert Koch, exists primarily in the cow. The bacilli may be discharged in the milk when tuberculous mastitis exists, or it may gain entrance to the milk by fecal contamination.

Raw milk supplies in many places have been tested for living tubercle bacilli, with varying results. One such series was reported by Tonney, Which and Danforth (1927). They reviewed the literature and found that of 16,700 specimens of milk examined by 46 authors from 1893 to 1925, 8.66% (1,448) contained living tubercle bacilli of animal origin. These authors then undertook to study the situation in Chicago in 1923, 1924 and 1925, because, they felt that pasteurization had nearly replaced attempts to keep cattle healthy and because existing ordinances prevented authorities from enforcing tuberculin-testing. They

took 329 samples throughout the whole Chicago area, centrifuged them at high speed and inoculated guinea pigs with 1 cc of the sediment. Seventy-one died in less than thirty days, too short a time to be significant. Of the remaining 258, 3.5% developed tuberculosis. In one county alone, 6.8% of the samples contained living tubercle bacilli. The authors estimate that 15 million quarts of raw milk per year were being contaminated with the organisms and that 6,250,000 of them came from this one county alone. After their findings were made public satisfactory ordinances were out in force.

Pullinger (1934) studied the situation in England and found that about 1% of the samples coming from 45 tuberculin-tested herds contained living bovine tubercle bacilli. The incidence jumped to nearly 25% in samples taken from nontested herds. Furthermore this author checked the milk in 63 1000-gallon rail tanks and found that every one of them were contaminated with the living bacilli. When milk was taken from cows with tuberculous mastitis, dilution a million times with clean milk was not sufficient to make the samples non-infectious for laboratory animals. Since neither tuberculin-testing nor pasteurization have been generally practiced in the

British Isles it is easy to account for their large incidence of bovine tuberculosis in children.

Dr. Harding (1935) quotes Dr. M. P. Ravenel as saying that human infection with bovine tubercle was -proven in the United States in 1902 and promptly confirmed in Europe. It has long been accepted that the bovine strain did not attack the human lung, but with developments in the line of tubercle bacillus typing, it has been discovered that this is not true. Ravenel quotes the State Serum Institute of Copenhagen as saying that they had found 26 patients suffering pulmonary tuberculosis caused by the bovine strain. Ravenel deplores the tendency in the United States to accept the work of others. He says that we do not know how many of our cases are caused by the bovine strain.

Soper (1934) states that while there is not doubt that pulmonary tuberculosis in man can be caused by the bovine organisms, such occurrence is rare. He quotes the work of Kossel, who found only 5 such cases in a total of 811. For this reason the disease is liable to be benign, though it may develop into tuberculous meningitis or generalized miliary tuberculosis.

Harding (1932) states that the case percentage of bovine strain infection in New York was 8%, it was

24% in England and 55% in Scotland (in children under 16). The bovine strain was responsible for cervical adenitis in 90% of the children tested.

This same author (1931) also quote Dr. T. C. McVeagh, of Honolulu, Hawaii, who states that in the islands it was found that 80% of the bone tuberculosis was due to bovine tubercle bacilli. Twenty-eight percent of the hospital beds for crippled children there were occupied by suffers of bovine type tuberculosis.

Recently Price (1932) carefully studied the type of tuberculosis occurring in 220 juvenile patients. Of the group, 13.6% (30) of the non-pulmonary cases were due to the bovine strain. All had been drinking raw milk. He also noted that the generation of children who have been brought into the world since general pasteurization of milk was put in force have failed to develop the disease. He considers this excellant evidence that the disease is milk borne and that pasteurization is essential for its control.

Significant are the figures compiled by Kelly and Weber (1924) in which it was proven that the death rate from non-pulmonary tuberculosis in Massachusetts showed little change in the years previous to 1910. In that year pasteurization was extensively introduced into the state; the death rate from non-pulmonary tuberculosis

declined nearly 75%. Thus the death rate from non-pulmonary tuberculosis per 100,000 population from 1905 to 1909 was 57.7. In 1920-1923 it was 15.0

In a recent personal communication Dr. Harding stated that milk-borne bovine tuberculosis does not occur in epidemics in the sense that other diseases do, though he has seen a few instances where several members of a family have been infected by the milk of a tuberculous family cow.

Much credit is due Dr Harding for his energetic campaign against this disease. His summary of Tuberculosis in Cattle and Humans (1934), his extensive quotation and comments on the Presidential Address of Dr. Charles H. Mayo before the Minnesota Public Health Association, and the more recent discussion of Bovine Tuberculosis in the U.S. (1935) have been his latest contributions.

VI. Miscellaneous Diseases.

A. Dysentery and Diarrhea.

Comparatively few epidemics of this character have occurred in the United States. Only six were reported by Armstrong and Parran (1927), involving 92 cases with 5 deaths. Harding (1929, 1930, 1931, 1935) in his yearly surveys of state and city health departments

found one epidemic reported in 1929, with 8 cases and no deaths, one in 1930 with 64 cases and 2 deaths, one in 1931 with 65 cases and no deaths and one in 1935 with 131 cases and no deaths. In the instances where the source was known, a carrier was implicated.

Armstrong and Parran (1927) discussed an epidemic which occurred in Nevada in 1914. The outbreak was explosive in character, the first recognized case occurring on the dairy farm. Twenty-eight cases were investigated and it was found that 20 of these occurred in children less than 5 years old. Investigation showed that the milk utensils were stored beneath the water closet and that both utensil and closet were exposed to flies.

B. Gastroenteritis.

This condition is separated from the above diseases because of the difference in etiological agents. One 3 epidemics were reported by Armstrong and Parran (1927) with 107 cases and no known deaths. Since then the outbreaks have been somewhat more common. The Annual report of the Surgeon General of the U. S. Public Health Service (1929) lists two epidemics, with 104 cases, occurring in 1927 and 1928.

Harding (1930, 1931, 1932, 1933, 1935) reports that

1 epidemic occurred in 1930, with 68 cases and 6 deaths. In 1931 another outbreak appeared with 13 cases and no deaths, and another appeared in 1932 with 32 cases and no deaths. In 1933 there were 3 outbreaks, with 125 cases and no deaths, and in 1935 five outbreaks occurred with 219 cases and no deaths.

Four epidemics were traced to cows with udder infections, two to active cases and the source of the others is unknown. Raw milk, cream or ice cream, and cheese, were the chief avenues of infection.

Armstrong and Parran (1927) quote an account from Health News, New York State Department of Health, March 1924, of an outbreak in which 82 of 132 children in a school drank raw milk for one source and became violent-ill in less than two hours. There was nausea and vomiting, gastralgia, diarrhea, drowsiness and prostration. The infection was short-lived, for all but twenty were apparently normal the next day. A non-hemolytic streptococcus was isolated from the milk and from the udder of one of the dairy cows.

Linden, Turner and Thom (1926) report 2 epidemics traced to cheese. The first occurred in Maine and involved 9 persons. A streptococcus was isolated from the cheese and from the patients. When fed to cats, a similar syndrome was produced and the streptococcus was recovered from the infected animals.

The second outbreak occurred in Kansas city, Kansas, where 22 persons became ill after eating cheese. The same sort of an organism was isolated. The organisms was somewhat heat resistant, since a pasteurizing temperature (experimental) of 142 degrees F. affected it but did not destroy it.

C. Anterior Poliomyelitis.

Three milk-borne epidemics of this dreaded disease have been reported in the literature.

The first of these appeared at Spring Valley, N.Y. in 1916 and was reported by Dingman (1916) Eight cases appeared in two days. Investigation revealed that all drank milk from one source. It was found that a child on the dairy farm had developed the disease 16 days before the onset of the epidemic.

The second outbreak appeared in and around Cortland, N.Y. in December, 1925. It was reported by Knapp, Godfrey and Aycock (1926) Eight cases appeared during the course of 11 days. All drank milk from one farm. It was also learned that there had been a sudden outbreak in the same area three months before with 4 cases and three deaths. Of the latter eight, five were regular customers of the dairy, one ate at a

factory and drank milk from the same farm and one ate food salvaged from a local restaurant. This food included various substances prepared from the incriminated milk.

The dairy in question sold about 4% of the milk sold in the city and bought milk from a number of other producers. Among these sources was a farm on which a boy of 16 was employed as a milker. This boy had become ill on December 7th, but kept working until December 19th, on which day his left arm became paralyzed. All cases drank milk from this farm. Two cases appeared later.

The third outbreak occurred in England in 1926 and involved 72 cases. The onset of the epidemic was explosive, 58 cases occurring in 10 days, according to Rosenau (1928). Fifty cases occurred among the users of milk from one dairy, 19 others among consumers of milk from another distributor who bought milk from the first.

D. Epidemic Arthritic Erythema

This disease, a rare occurrence, was first described in epidemic form by Place, Sutton and Willner (1926). Sixty cases of the disease appeared suddenly

in Haverhill, Mass., in January 1926.

A short description of the disease would not be out of place here. Chills, vomiting, intense headache and prostration were the first symptoms to appear. The fever rose rapidly to 103-105, then fell on the 3rd or 4th day, then recurred and remained remittant for some time. An eruption appeared in 1-3 days on the extremities, particularly on the extensor surfaces and about the joints. This was blotchy, irregular, maculopapular and dull red in color. The eruption increased for 1-2 days, then faded, followed by desquamation. Joint symptoms, such as swelling, pain and effusion appeared on the 3rd or 4th day of the disease. The epidemic was confined to a small area occupied by Lithuanian mill workers, all of whom drank raw milk from one dairy. A gram's-negative rod was recovered in the joint fluid. The actual source was not traced, though all evidence pointed to the milk as the avenue of transmission.

Four hundre cases of a dengue-like syndrome appeared in Chester, Pa., in 1925. Some authorities have regarded this as epidemic arthritic erythema (nosenu 1928). The epidemic appeared on one milk route, and cases were discovered on the dairy farm supplying the milk. Armstrong and Parran (1927) seem to think that

this was streptococcal in nature.

E. Appendicitis and Parotitis.

A sudden outbreak of these two diseases appeared at Culver Military Academy, Culver, Ind., in 1915. This epidemic was reported by Rosenow and Dunlap (1916).

Eight cases of appendicitis appeared in 12 days. Only 7 other cases appeared throughout the year. A viridans strain of streptococcus was isolated from the appendices of these patients and from the milk and associated dairy products used by them. Fifty percent of six rabbits inoculated with these organisms developed appendiceal lesions.

During the same epidemic a viridans streptococcus was isolated from Steno's Duct of 34 cadets developing parotitis. A similar organism was found in associated dairy products. Seventythree percent of the rabbits inoculated with cultures from the patients developed parotid lesions, while 30% of those inoculated with the organisms from dairy products developed the disease.

F. Botulism

Only three cases of this disease are listed in the Armstrong and Parran series (1927). These authors, quoting Nevin and Mann (in the New York State Department

of Health report for 1915) state that cottage cheese was blamed and that the organism was isolated from the cheese. All the patients died.

G. Foot and Mouth Disease.

Rosenau (1928) states that this disease, though primary in cattle, occasionally occurs in children after the ingestion of milk from infected animals. Fever, vomiting, heat and dryness of the mouth, accompanied with an eruption of pea-sized vesicles in mucous surfaces and about the fingers, are the most commonly observed symptoms. No descriptions of epidemics were available .

H. Milk Sickness.

This disease is probably not of bacterial origin, but because it is traceable to milk it may be included. It is primarily a disease of cattle. The pioneers in this country suffered much from it. Of historical it caused the death of the mother of Lincoln. The disease is rare now.

Armstrong and Parran (1927) do not list any outbreaks of the disease. Harding (1934) lists two outbreaks in 1933, with 10 cases and 1 death. According to Rosenau (1928) the disease may be bacterial but

probably is due to poisoning of milk cows by the rayless goldenrod, *Aplopappus heterophyllus*. The disease disappears in sections where the sod is broken and timber land cleared.

THE CONTROL OF MILK-BORNE DISEASES.

The safety of a milk supply, say Topley and Wilson (1936), depends upon its freedom from pathogenic bacteria. These organisms come from one of three sources. First there is the infected udder of the dairy cow. From this comes bovine tubercle bacilli, *Brucella abortus*, and some streptococci and staphylococci, and under special circumstances, such organisms as the diphtheria bacillus.

The infected human nasopharynx is the second source of contamination of milk supply and from this source the organisms of the streptococcal diseases and diphtheria are usually distributed.

Thirdly, contamination of the hands of workers and of the water supply by infected excreta may lead to the infection of a milk supply. Typhoid fever, the paratyphoid fevers, dysentery and food poisoning may be transmitted in this way. No raw milk can ever be regarded as completely safe for human consumption,

these authors point out.

Rosenau (1928) put it in even stronger fashion. He says that no one should drink raw milk unless it is guaranteed by the health officer that the same is safe, and no health officer would give such a milk a safe bill of health.

Milk-borne epidemics have been recognized for more than fifty years in this country, yet we still see 30 to 50 outbreaks per year, a number comparable to the number seen at the turn of century.

A review of the list of diseases which are traceable to milk at once indicates that here is a situation about which something must be done. Obviously control of the situation must be brought about by simultaneous attacks upon the various sources of infection.

All health authorities now recommend the routine practice of heating milk to a temperature which in no way damages the milk and for a time which brings about the destruction of the pathogenic organisms which might be contained in it, i. e., pasteurization.

Pasteurization, says Rosenau (1928) ordinarily reduces bacterial numbers in milk 99%. By common consent the thermal death-point of the tubercle bacillus has become the standard of pasteurization

efficiency. In most instances these organisms die within 30 minutes if exposed to temperatures of 136 degrees F., and are always killed if the temperature is raised to 140. In handling large volumes of milk, however, the temperature is raised to 142-145 in order to give a satisfactory factor of safety.

Rosenau (1928) also states that milk heated to 145 degrees F. for 30 minutes undergoes no changes. Higher temperatures do cause changes, as decomposition of protein, loss of organic phosphorus, precipitation of calcium and magnesium salt, etc. Carbon dioxide is driven out and the emulsion is disarranged. Quick boiling does not affect milk calcium as much as does high temperature pasteurization for longer period. The amount of visible cream or "creaming ability" is affected at temperatures of 146 degrees F. and above.

Prucha (1927) studied the effect of pasteurization of milk upon milk flora. He found that when the temperature was maintained at 140 degrees, the counts of milk dropped from 16,000,000 to 24,000 and from 100,000 to 17,000. But when the temperature was raised 5 degrees, the counts of milk containing 5 million bacteria per cc were lowered to 1 million in 10 minutes. No known pathogenic organisms can survive efficient pasteurization, except, possibly, those which

may exist in spore form.

Pasteurization is accomplished by one of two general processes, according to Putnam (1929); either the holding method or the "flash" method may be used.

In the former the milk is heated to 142-145 degrees F. and held there for 30 minutes. Vats, either large or small (pockets), the continuous flow method or the in-bottle method may be used. The "flash" method consists of heating milk to 160 degrees F. for 15 seconds and then immediately cooling it. This method has been condemned by many health authorities on the grounds that too often the heating has been uneven.

The process, technical as it is, is not without its difficulties. North, Park, Moore, Rosenau, Armstrong, Wadsworth and Phelps (1925) undertook the most extensive study of commercial pasteurization ever done in this country to locate the most common engineering defects. They list these as: dead ends (of pipe), in which milk is not heated thoroughly, valve leakage, foam and splash in pasteurizers and defective continuous flow regulators which allow the milk to flow through the pasteurizer at a too rapid rate. Defective thermometers were also discovered in many cases. These authors also insist on accurate record being kept in the dairy.

Pasteurization is of paramount importance and

saves many lives. But, says Rosenau (1928) it does not render filthy milk less so. It implies precaution, protection and prevention. It represents the best insurance against the disease for both the consumer and industry. Pure milk is better than purified milk. Even clean milk should be pasteurized, for no method of control and no inspector can see missed cases and carriers.

According to the Preliminary Report of the Committee on Milk Production and Control (1931), pasteurization should be required where ever practicable. It is not intended to replace sanitary production and clean and wholesome milk, but to provide a factor of safety. There should be inspection of the farm and plant, examinations of the milk and finally pasteurization.

Walker (1928) states that there are three general types of control in enforcement of ordinances and statutes, once these legal standards are put in force. Licensing of the dealers, grading of milk and the invoking of penalties are the most valuable means.

The United States Public Health Service has compiled a model ordinance and code, embodying the most modern thought in dairy sanitation. This has been approved by the Service and by the Bureau of Dairy Industry (U. S. Department of Agriculture).

Also the U. S. Public Health Service has created the Public Health Service Sanitation Advisory Board, a group of experts who are qualified to deal with all aspects of milk production and distribution. The Board consists of eleven men from the Public Health Service, the health departments of six states, the U.S. Department of Agriculture, the Certified Milk Producers Association of America, the Dairy & Ice Cream machinery & Supplies Association and from the International Association of Milk Dealers. A brief discussion of this ordinance and code is presented herewith.

The Milk Ordinance and Code of the U. S.
Public Health Service.

As the name implies this treatise is divided into two parts. The first, the ordinance proper, sets forth the conditions under which milk may be produced, handled and sold. The second part, or the milk code, is provided for explanation and interpretation of various parts of the Ordinance. There were devised for adoption by city, county, district and state governments. With it is included a short enabling form by which a governing body may adopt the Ordinance and Code. This places the Ordinance in force and makes special provision for the fixing of penalties and for the

the repeal of parts of previous ordinances which are in conflict with it.

Because of its length, the ordinance and code cannot be given in full. However a review of the various sections and the interpretations of these will present the most pertinent facts of modern milk production, handling and control:-

Section 1: this deals expressly with definitions. Milk must contain not less than 8% of milk solids not fat and not less than $3\frac{1}{2}$ % of milk fat.

Cream must contain 18% butter fat or more.

Skimmed milk contains less than $3\frac{1}{4}$ % butter fat.

Pasteurization is defined as the process of heating every particle of milk or milk products to an arbitrary temperature of 142 degrees F. for 30 minutes. This is the holding process of pasteurization. The term also applies to heating of every particle of milk to 160 degrees F. for not less than 15 seconds, or any other method approved by the state health authorities.

Adulterated milk and milk products, milk producer, milk distributor, dairy or dairy farm, milk plant and health officer are also defined. The average bacterial plate counts are listed as the logarithmic average of plate counts of the last four consecutive samples taken upon separate days. Average reduction time is taken

to mean the arithmetic averages of the reduction times (in methylene blue) of the last four samples taken on consecutive days.

Grading periods refers to the period of time the health officer designated, but it must not exceed 6 months. According to the Code, 3 months is a good period, though more expensive than the 6 months period. The grades are to be announced regularly.

Bactericidal process refers to the destruction of bacteria by any method or substance which the health officer believe effective and which is satisfactory for use in equipment and which does not threaten the health of the individual. Several bactericides are listed as complying with the ordinance. The first is calcium hypochlorite. The stock solution may be made of 12 ounces of the chemical to a gallon of water, and a teaspoon of this to each gallon of rinse water is considered to be an effective germicide for hands and udders. Sodium hypochlorite seems to be as effective as the calcium salt and is used in the same way. These instructions are designed to give a solution containing 100 parts of available chlorine as hypochlorite per million. The inspector must see to it that the dairy is using hypochlorite solutions of the proper strength. The Code gives detailed directions for the

application of the orthotolidin test for chlorine. No other form of bactericide is permitted, unless the inspector is satisfied that it is satisfactory from all viewpoints.

Section 2: this deals explicitly with the prohibition of the sale of adulterated, misbranded or ungraded milk or milk products. It also makes it unlawful for a person to possess such milk or milk products except in a private home. According to the code this section is to be used in preferring charges against those who are guilty of these things.

Section 3: this section makes it unlawful for anyone to sell milk without a permit from the health officer. Such a permit must be displayed on the delivery vehicle and may be revoked by the health officer if the permittee becomes a menace to public health. This is a registration device and gives the health officer a method of controlling the sale of milk.

Section 4: this provides for the labeling of all milk and milk containers as to name of contents, the grade of the contents, whether or not the product is pasteurized and the name of the producer or the pasteurizing plant. In the case of Vitamin D milk, the designation must also be included and also the source of the vitamin. The health officer has control

over the size, color and wording of the labels. This section also provides that all establishments serving milk or milk products must display a notice stating the lowest grade of milk or milk product served. The idea of this is to encourage the consumer to buy by grade and thus gradually force out the lower grades.

Since many cities do not specify that all milk be of high grade, this section is of particular value. It also enables the health officer to see to it that all degraded milk is so marked. It also prevent the dealer using any distinctive terms upon his label, as "natural Milk." This requirement was made because the term is misleading and may militate against the use of pasteurized milk. In as much as "cows milk was intended for calves, it cannot be regarded as natural milk for human babies (Committee on Milk of the 1932 Conference of State and Provincial Health Authorities). The health officer should see to it that the proper grades for various brands be correctly displayed in all places in which milk is sold or served; this is to be on placard or menu card.

Section 5: this important section deals with the inspection of farms and milk plants by the health officer. In case of violations he may make a second inspection within 3 days and the second inspection

is to be used in determining the grade of the product. One copy of the report is to be posted in a conspicuous upon an inside wall of one of the farm or plant buildings and the second one filed with the health department. Practically, it is desirable to inspect the farm several times during the grading period, and the dairy plant should be inspected at least every 2 weeks. If one or more violations are discovered on two successive inspections, the plant or farm is to be immediately degraded. Strict enforcement is said to make for a better and friendlier relationship between the health officer and the dairy industry.

Section 6: this deals with the examination of milk and milk products. During each grading period at least four samples of milk are to be taken and tested, the samples being collected on separate days. Samples of other milk products and of milk as sold in stores, restaurants, etc., are to be examined as often as the health officer deems necessary.

The methods of examination should include bacterial plate counts, reductase tests and such other chemical and physical examinations as the health officer desires. This may include bioassays of Vitamin D content in Vitamin D milk. The results, should they

fall outside of the requirements of the grade in which the milk or milk product was formerly classed, are to be sent to the producer or distributor immediately. Then, in less than a week, fresh samples may be taken. This time gives the operator a chance to correct anything not in order. Furthermore, the Code emphasizes that samples to be used for grading purposes should be taken while the milk is still in possession of the dairyman. Any other practice would be unfair.

The technical details of the bacterial counts and of the methylene blue reduction test are fully described in Standard Methods Of Milk Analysis (1927) and need not be described here. The recommended method of recording the counts is to use the logarithmic average. Tables are listed in the Code for determining this figure. Reduction time is to be recorded in arithmetic averages and grading is to be based on these figures.

Section 7: At least once every six months the health officer shall announce the grades of all milk and milk products which are to be consumed within the the city or district. A series of standards are given in this section. They are:-

I. Vitamin D Milk - this shall be only of Grade A raw quality, or certified or grade B pasteurized milk.

II. Certified Milk:- this is milk which meets the requirements of the American Association of Medical

Milk Commissioners and is produced under the supervision of the Medical Milk Commission of the Medical Society of the county and of the State Board of Health, or of the city or county health officer. The standards are fully described in Methods and Standards for the Production of Certified Milk (1936) and will not be discussed here.

III. Grade A Raw Milk: this is milk the average bacterial counts of which do not exceed 50,000 per cubic centimeter, and the reduction time of which is not less than 8 hours. This must be produced under sanitary requirements which are extensive and exact. Twenty-six items are included. For purposes of discussion these may be conveniently divided into several groups.

The first of these deals with the dairy cow. Physical examinations and tuberculin-testing of the herds by a licensed veterinarian approved by the State Livestock sanitary authority must be done at least once a year. The standards for this are to be those approved by the U. S. Department of Agriculture, Bureau of Animal Industry. Such other examinations as the health officer deems necessary may be made.

In the second group, requirements are set forth as to the dairy barn, its lighting, ventilation, cleanli-

ness of floors and walls, the cow yard and manure disposal, and the milk house and its construction and cleanliness.

One or more toilets must be provided on the farm and they must be of such location and construction as to not pollute the surface soil or the water supply. Furthermore the water supply must be adequate and safe.

Utensils must be approved design, properly cleaned and disinfected before use and handled in such a manner that the surface with which the milk comes in contact is not contaminated during milking or during storage.

In the third section, provision is made for cleaning the cows' bellies, flanks and tails and udder and teats before milking. Any abnormality in the appearance of the teat or udder or of the milk itself is to be noted and milk from such sources discarded. The milker must wash his hands, disinfect them and dry them before milking. Wet-hand milking is prohibited. The milker's outer clothing must be clean.

Each pail of milk must be removed immediately after being withdrawn and cooled to 50 degrees F. or less within 1 hour; the milk must be held at that temperature until delivery. If it is to be delivered to a plant or receiving station this must be done within 2 hours, or the milk must be cooled and kept cool until it can be

delivered.

As for the personal health of the workers, they must submit to any examinations of any kind which the health officer thinks necessary. Active tuberculosis, diphtheria, typhoid and the paratyphoid fevers are the diseases usually sought for. According to the Code any person having a positive Widal, in the absence of recent typhoid immunization, should be excluded.

IV. Grade B Raw Milk

This is milk the average bacterial counts of which at no time exceed 200,000 per cubic centimeter, and the reduction time of which is not less than six hours. The sanitary requirements are similar to those of Grade A Raw Milk, but less strict. For example, tight wooden floors and gutters may replace wooden ones, and white-washing of the barn is not required. The milk may be cooled to 60 degrees F or less, instead of 50. Personal health examinations of employees are not required. This allows the production of a milk far better than that produced in uncontrolled communities, but not as good as Grade A Raw Milk.

V. Grade C Raw Milk

This milk must at no time have more than 1,000,000 bacteria per cc., nor have a reduction time of less than $3\frac{1}{2}$ hours. The sanitary requirements are even less

strict than above.

VI. Grade D Raw Milk

This does not meet the requirements of Grade C Raw Milk and must be labeled "cooking only."

VII. Grade A Pasteurized Milk

This is Grade A Raw Milk or Grade B Raw Milk which has been pasteurized, cooled and bottled in a milk plant conforming with certain sanitary requirements and the bacterial plate counts of which never exceed 30,000 per cubic centimeter. Twenty-three items of sanitation are included in the Ordinance and interpreted in the Code. They may be discussed in several general groups.

The first group deals with the floors, walls and ceilings, the doors, windows, lighting and ventilation facilities of the rooms in which the milk is handled. Cleanliness, water-tight floors, proper screening and good lighting and ventilation are the essentials.

The second group of requirements deals with the placing of the rooms for various operations in the plant. Pasteurizing, cooling and bottling shall not be done in the same room as the washing and bactericidal treatment of miscellaneous containers and equipment. Furthermore cans are not to be unloaded in either of these rooms because of the opportunities for contamination, especially by flies. There should be no bypass around the

pasteurizer.

Toilet rooms should be separate from other parts of the plant, be properly cleaned and screened, and kept in a good state of ventilation and repair. Warm running water, soap and individual sanitary towels are to be provided for hand washing purposes. Furthermore the water supply must be safe and sufficient.

All piping and fittings should be large enough to be easily cleaned with a brush and should be smoothly finished and not easily corroded. The same applies to all other containers and equipment.

All trash and wastes are to be disposed of via a public sewer, covered garbage cans or other approved means.

One of the most important requirements is that all equipment must be subjected to some sort of bactericidal treatment immediately before use. All demountable apparatus must be taken down at least once a day for cleaning. The assembled equipment must be sterilized by hot water, steam or chlorine solutions. If hot water is used, it must be circulated at least 5 minutes after the temperature at the outlet has reached 170 degrees F; if steam is used, it must be circulated for at least 5 minutes after the temperature at the outlet has reached

200 degrees F. With chlorine solutions, it must be of the required strength as it escapes from the outlet and must be pumped through the system for at least 5 minutes. Special provisions are given for other pieces of equipment.

Equipment must be handled in such a way as to avoid contamination, while bottle caps and other sealing devices are to be purchased and stored in sanitary containers.

The requirements and standards and the engineering details of the pasteurizing equipment are too extensive to be described here. Suffice it to say that dead ends are eliminated, mercury column type thermometers and automatic recording thermometers must be used, and leak-proof and leak-protector valves must be installed. The accuracy of the temperature control in holders and the heating of the foam which collects on the top of milk in vat or pocket type pasteurizers are important features and must be checked. Vat and pocket type pasteurizer covers must be tight. All holders in which the milk must be preheated must be preheated themselves to the pasteurizing temperature before the milk enters them. Milk and cream which have been pasteurized must be cooled immediately to 50 degrees F. or less and

maintained there; there are special qualifications for cooling apparatus.

Bottling of the milk is to be done by machine of approved design. Capping is never to be done by hand. These features are very important from the public health point of view, since it is at these points that pasteurized milk is most easily infected. All overflow milk is not to be sold for human consumption.

All milk handlers and workmen who come in contact with milk must furnish such information as submit to such examinations as are necessary to prove that they are free from transmissible diseases. These employees must also wear clean clothing at all times and keep their hands clean.

VIII. Grade B Pasteurized Milk

This is Grade C Raw Milk which has been pasteurized, cooled and bottled in a plant conforming to the requirements given for Grade A Pasteurized milk. The bacterial plate count must never exceed 50,000 per cc. after pasteurization and before delivery.

IX. Grade C Pasteurized Milk

This is pasteurized milk which does not meet the standards of Grade B Pasteurized Milk and must be labeled "cooking only."

Section 8:

Grades of Milk and milk products which may be sold; two wordings are supplied in this section, since some communities prefer to improve their milk supplies by grading and degrading, while others prefer to refuse permission to sell any milk except that in a definite grade or grades.

Section 9: In this section the health officer is given authority to degrade any milk at any time if it is evident that said milk no longer belongs in the former grade. The dairyman may apply at any time for regrading. In such a case the health officer may take new samples (not more than two per week) and if the last four indicated that the grade may be raised, this is done at once. If degrading was done because of violations of items in Section 7, other than because of average plate counts, reduction time or cooling temperatures, the application must be accompanied with a statement that a correction has been made.

Section 10: the sale of dip milk is prohibited, since this is a menace. No producer or distributor may transfer milk from one can or container to another except in a bottling room or milk room especially provided for that purpose. All milk must be placed in a final container before being delivered and all rest-

aurants, etc,m must sell the same in the original containers. All containers must be cleaned before being returned to the distributor. Delivery of milk to and receipt of containers from a quarantined residence must be subject to requirement of the health officer.

Section 11: this provides that milk from outside the city limits may not be sold within the city limits unless these requirements are met, provided the health officer in the outside district is doing his job.

Section 12: the health officer must be notified of any infections, contagious or communicable diseases which occur upon the farm or in the milk plant of any producer or distributor.

Section 13: All dairies to be constructed in the future must conform to the Grade requirements of the Ordinance and Code.

Section 14: if suspicion arises as to the possibility of a milk handler transmitting infection, that person is to be excluded from milk handling, the supply is to be excluded from distribution and use, and adequate medical and bacteriological procedures for examination of the person and his associates are to be instituted.

Section 15: this and section 16 provide for the enforcement and the penalties for violation of the

Ordinance.

Section 17: this provides for the repeal of all past ordinances and parts of ordinances which conflict with this one.

Section 18: If any particular part of the ordinance is found to be unconstitutional or invalid for any reason, the remainder is unaffected.

A personal communication from Dr. Leslie C. Frank, Senior Sanitary Engineer in Charge, Office of Milk Investigation, U. S. Public Health Service, states that 694 American Communities have thus far adopted the Standard Milk Ordinance (March 1937).

The results of the actual operation of the standard ordinance in Missouri have been recorded by Clark and Johnson (1931). According to these authors, high infant mortality in that state brought requests by unofficial civic organizations (Commercial Clubs and Parent-teachers Associations, Etc) for information regarding the quality of their respective milk supplies. Investigation showed that there were no milk ordinances in many cities and unsatisfactory enforcement of existing ordinances in others. The work was begun in 1923, with a survey and a report. No follow-up work was done. It

became evident that the following was needed for a satisfactory milk sanitation program:

1. Frequent advisory assistance to the cities.
2. An ordinance designed in such a way that gradual improvement of the sanitary quality of the milk could be effected without placing undue burden upon dairymen, and of such a type that it would appeal to the average councilman as being fair to all concerned.
3. An ordinance was needed which could be adequately enforced without too much recourse to the courts.
4. Adequate state personell were needed to advise and assist the local milk inspectors.

In 1925 the U. S. Public Health Service Standard Milk Ordinance and Code were adopted by the State Board of Health. This was done because the previous program had not solved the problem milk-borne typhoid fever was on the increase, and because the Standard Ordinance as its program of enforcement constituted a remedy for most of the difficulties already encountered. Furthermore it was the most effective method available.

The Ordinance was easily passed in many cities and easily enforced. It was effective in securing a reasonably rapid improvement in milk quality and it

promoted the per capita consumption of milk.

Two men were assigned to the work in 1928, one being from the Public Health Service. Letters were sent to various cities to learn if they would be interested in the plan. One third of the state program was devoted to interesting other cities in passage of the ordinance, one-third to training of city milk inspectors, while the remaining parts were spent in making surveys of the work of the Ordinance and to special problems.

Improvement of the retail raw milk supplies (in regard to cows, dairy equipment and methods, and employees) is shown by the fact that these rose from 56% compliance to 85.8% compliance to the Ordinance. It was most marked in 19 cities which had spent 6 months or more under the ordinance.

Previous to the passage of this Ordinance, not a single city had practiced routine inspection of sources of raw milk to plants. There was an improvement of from 39.9% to 75.8% average compliance to the ordinance in 17 cities.

The average ratings of pasteurization plant sanitation improved from 52% to 83% compliance. Much of the former low rating was due to the use of old and faulty equipment.

There was an increase of 18% in milk consumption.

The authors state that the help of Chambers of Commerce, Parent-Teachers Associations and other civic organizations can be a great power. Once the ordinance is passed it must receive the support of city officials and citizens, and the latter are best approached through these groups.

Success in a given community is proportionate to the qualifications of the inspector and the support and direction he receives from his superiors. The plan works best when backed by the State Health Board. Enforcement may be made possible in small communities by the grouping of several under one inspector. The sanitary inspector of the county health unit is the logical individual to enforce the program in small communities with a county health unit.

The Ordinance and Code, comprehensive and constructive as it is, is not without its critics. Many of the criticisms are of minor thing, however. None, perhaps, is better qualified to judge the ordinance than Dr. H. A. Harding, Chief of the Dairy Research Bureau. A query addressed to him brought a reply which may be quoted in part:

"I note your comment on the Standard Ordinance of the U. S. Public Health Service. I have come upon various cities in the South where this ordinance was in use and I think quite uniformly the attitude of all parties was favorable to the ordinance provided it was enforced.

"The satisfactory working of this ordinance practically required the delivery of the milk from the farm twice per day. Where such twofold delivery does not fit into the situation there would undoubtedly be considerable trouble in making the thing work. My own criticism to the ordinance is that it depends very largely upon the bacterial plate count for its grading purposes. This is a rather weak reed to lean on because it really does not indicate anything in which we are interested as milk consumers or milk handlers.

"In the days when milk coming from the farms had a germ content of a few million the bacterial plate count undoubtedly served a useful purpose in developing milk of a better keeping quality. It may still have usefulness in this connection in your market. However, in many of the markets the general quality of milk coming as such is that the bacterial plate count is a rather clumsy way of controlling the situation.

"In connection with the Standard Ordinance there is a provision that the methylene blue may be used in place of the plate counts in classifying the raw milk. This is a very distinct improvement because the methylene blue test is a workable means for this purpose under any ordinary conditions." (March 1937)

Frank (1935) discussed the Ordinance and Code in comparison to other existing attempts at milk control and stated that "the national milk control program recommended by the Public Health Service offers a solution not only for the problem confronting the milk consumer, namely, his present uncertainty in most areas as to when he is receiving and when not receiving an approved milk supply, but also offers the most sensible and practicable solution of the problem of the milk industry, namely, its present inability to dispose of enough of its product at a sufficiently attractive price."

And again, "The following conclusions therefore become immediately apparent:

(a) Every American municipality should exert itself to the utmost to deserve a 90 percent rating and thus deserve inclusion in the 90% list published by the Public Health Service. ---

(b) Every milk distributor should demand early adoption and strict enforcement of the Public Health

Service Milk Ordinance in order that his products may attain the consumer prestige which would accompany the inclusion of this city in the federally approved list.

(c) Having secured admission to the approved list a municipality should then organise an educational program which will repeatedly call to the attention of every milk consumer in the city the food value and the safety of milk. The milk distributors could well afford , either individually or as a group, to distribute to all milk consumers such articles as "What Every Person Should Know About Milk," which appeared in the Public Health reports in December 1934, and is now available in reprint form at a price of \$5.00 per thousand. ---"

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