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THE PASTEURIZATION OF MILK

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WITH A FOREWORD BY

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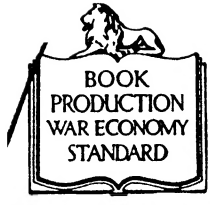

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FOREWORD

A word is necessary on the origin of this volume. For some years before the war many persons had been becoming increasingly uneasy over the frequency of milk-borne disease, especially tuberculosis. The results of inquiries into the facts by unofficial bodies had done nothing to alleviate their suspicions. They, therefore, welcomed the appointment by the Prime Minister in 1932 of the Committee on Animal Diseases of the Economic Advisory Council. The report of this Committee in 1934 contained a number of very important but disturbing facts, including the statement that about 40 per cent. of the cows in this country were infected to a varying extent with tuberculosis, that at least 0·5 per cent. were actually excreting virulent tubercle bacilli in their milk, and that over 2,500 deaths in human beings occurred annually from tuberculosis of milk-borne origin. Among other measures they recommended the introduction of some measure of compulsory pasteurization. These findings, which had been accepted only after most careful scrutiny of the evidence, could not be ignored, and various steps were taken by the Government. It was not, however, until the Government's proposals for the milk industry were made in the White Paper on Milk Policy, published in July, 1937, that legislation involving the principle of compulsory pasteurization was suggested. In the White Paper it was proposed that, subject to certain conditions and exceptions, Local Authorities should be enabled to apply for an Order making compulsory the efficient pasteurization of milk sold by retail in their areas. Clauses to this effect were included in Part VII of the Milk Industry Bill which was introduced in November, 1938, and, though hedged about with many restrictions, they would have gone some way towards providing a safe milk supply for the population. The Bill, however, met with so much opposition that it was withdrawn after its first reading.

Subsequently, the Ministry of Health and the Ministry of Agriculture were approached by various bodies urging them to take up this matter again. In response to one of these deputations, Mr. Walter Elliott, then Minister of Health, said that there was need for further education of public and parliamentary opinion. He had therefore asked a suitably qualified medical man to undertake the collation and review of the existing evidence on the whole subject of pasteurization and the safety of milk supplies, and he proposed to publish the result.

The present report was prepared by Professor G. S. Wilson and handed to the Ministry of Health in August, 1939. The circumstances of the war having caused the question of publication to be deferred, Professor Wilson asked the Ministry for permission to publish his report privately. Consent was readily given, the report has been brought up to date and, with suitable modifications, now appears in book form. I hope it will be widely read.

WILSON JAMESON.

MINISTRY OF HEALTH,
January, 1942.

PREFACE

The aim of the present volume is to describe the chief physical, chemical, and bacterial changes brought about by the low-temperature pasteurization of cows' milk; to furnish a critical review of the evidence relating to the effect of pasteurization on the nutritive value of the milk for animals and for human beings; to record the frequency with which raw milk is infected with various pathogenic micro-organisms and the amount of disease for which it is responsible in Great Britain; and to inquire into the effect on the health of the community that has resulted in various parts of the world from the replacement, either naturally or under compulsion, of raw by pasteurized milk.

To accomplish this task without falling victim to the Scylla of scientific pedagogy on the one hand or the Charybdis of undocumented statement on the other has been by no means easy. Unfortunately pasteurization is a subject on which many persons find it difficult to form an unbiassed judgment. It is not infrequently regarded with such intense prejudice that no reasoned statement of the evidence is listened to, much less given adequate consideration. This attitude is the direct antithesis of that usually adopted by scientific workers, namely that of a dispassionate review of the whole of the available evidence with conclusions expressed in the form of cautious understatement.

In view of this difference in mental approach, it is useless for men of science to expect that a careful presentation of the facts will make any impression on the minds of those who, whether from unreasonable conviction or from a repressed suspicion of the weakness of their own case, are prepared to resort to half-truths, unverifiable assertions, or, as experience has shown, actual falsehoods in support of their contentions. No matter how clear and well documented the evidence may be, there are always those whom Shakespeare must have had in mind when he made Cicero say:

> "Men may construe things after their fashion,
> Clean from the purpose of the things themselves."

Much of the current prejudice is doubtless due to lack of education and to false propaganda. The majority of persons who distrust or oppose pasteurization do so because their information is incomplete. Many of them have a partial knowledge of the process, which they may dislike, but few of them are thoroughly acquainted with all the

relevant facts, on which alone a rational opinion of its merits and demerits can be based.

To those interested in preventive medicine this is no new experience. Indeed, each new step in public health legislation in this country has involved a fight against the forces of prejudice and ignorance. In this connexion a passage may be quoted from Sir Edward Mellanby's introduction to the Report of the Medical Research Council for the year 1937–1938.

"It is not the duty of the Medical Research Council to guide the public on the importance of medical discovery in its practical applications to human needs. Their task is wholly to promote discovery and to announce it to the world when it is made. They cannot but view with regret, however, the delay which so often occurs between medical discovery and its application, especially when the discovery is one that provides an immediate opportunity of preventing or even eliminating disease. What the ultimate solution of this difficulty will be is not obvious, but with the present increase in knowledge of methods of preventing disease, a better method of informing and educating the public becomes imperative."

It is with this object in view that the present volume has been compiled. Its aim is solely to set down the relevant *facts* about pasteurization and to draw from them such conclusions as are clearly justified. No opinions in favour of pasteurization are quoted except those expressed by representative medical organizations after reviewing the facts ; but adverse opinions receive considerable attention. Some of the earlier evidence has already been fully reviewed by other workers, and is therefore quoted here quite briefly. Recent work, however, is treated at greater length, and experiments dealing with highly disputed subjects, such as the comparative nutritive value of raw and pasteurized milk, are described in some detail. With the exception of figures on the extent of pasteurization and of an experiment carried out to determine whether it is possible to detect a cooked flavour in pasteurized milk, no completely new series of observations are recorded. Data, however, particularly those afforded by the Registrar-General, have been frequently analysed and used to test the truth of particular theses.

For the sake of convenience a summary of the facts and conclusions to be drawn from them is provided at the end of each chapter, and a general summary of the more important facts and conclusions is reproduced at the end of the volume. If any ambiguity should arise on the facts or any doubt should be felt about the justice of the conclusions, reference may be made to the body of the book where the original observations are recorded.

It may be emphasized once more that the purpose of this volume is to present a conspectus of our knowledge on the medical and scientific aspects of pasteurization. That other factors must be taken into consideration before this knowledge can be applied in practice is fully realized; but whatever the verdict of the economist or the politician may be on the advisability of introducing compulsory pasteurization, no one, it is hoped, who approaches this question with an open mind will have any doubt after reading this book that the need for pasteurization is fully justified by all the available scientific evidence, and is supported by such a wealth of authoritative medical opinion as has seldom before been united in favour of any measure designed to protect and foster the public health of the nation.

The writer would like to express his thanks to all those who have helped him in one way or another in the compilation of this volume. He is specially indebted to Dr. J. M. Hamill and to Mr. P. N. R. Butcher, of the Ministry of Health, for supplying him with much useful information and for discussing many of the difficulties involved; to Dr. H. E. Magee, of the Ministry of Health, Miss Margaret Hume, of the Lister Institute, and Miss S. T. Widdows, of the London School of Medicine for Women, for help in the construction of Table XIX dealing with the composition of cows' milk and human milk; to Dr. A. C. Dahlberg for permission to reproduce the figure on p. 145; to Sir William Savage, late County Medical Officer of Health, Somersetshire, for references to milk-borne epidemics; to Mr. G. S. Reid Chalmers, Divisional Inspector of the Ministry of Agriculture and Fisheries, for information on the practical working of the various Orders dealing with the control of animal disease; to Dr. A. Bradford Hill and Mr. W. T. Russell, of the Department of Epidemiology and Vital Statistics in the London School of Hygiene and Tropical Medicine, for advice in the handling of certain statistical data; to Miss Irene Maier for her constant help in the preparation of the manuscript; and finally to Sir Wilson Jameson, Chief Medical Officer, Ministry of Health, not only for his kindness in consenting to write a foreword to this volume, but for his counsel and assistance while he was still Dean of the London School of Hygiene and Tropical Medicine.

G. S. W.

May, 1942.

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THE PASTEURIZATION OF MILK

CHAPTER I

THE CONSUMPTION OF COWS' MILK

The Nutritive Value of Milk. There is ample evidence, not of the vaguer sort, but based upon sound experiment and observation, that cows' milk is an exceptionally valuable food for the human species throughout the whole period of growth (see, for example, the reports of Corry Mann 1926, Leighton and McKinlay 1930, and the Milk Nutrition Committee, Report 1938*b*, 1939*c*). It contains fat, carbohydrate, and protein for the supply of energy; it contains all the essential vitamins; and it contains an abundance of mineral salts, particularly those of calcium and phosphorus. The high biological value of its protein, and its content of vitamins and mineral elements, combine to render it an excellent food for the growth and maintenance of the body tissues.

Its value, however, must not be overstated. It is not a perfect food, and neither infants nor children can thrive on it indefinitely. It differs both qualitatively and quantitatively from human milk; it is deficient in iron and copper, both of which are required for the formation of hæmoglobin; the anti-scorbutic vitamin C is present in only small quantity; and vitamin D, which in combination with calcium and phosphorus plays an important part in the development of bone and the prevention of rickets and dental caries, is practically absent during the winter months. Nevertheless it is a food of outstanding value which is coming to be regarded by many as the key to proper nutrition in childhood.

The Desirable Level of Milk Consumption. There is general agreement among nutritional experts that milk should constitute the foundation of a well-balanced diet for the growing child. The exact quantity to be given at different ages is subject to a certain amount of discussion, British investigators on the whole favouring rather larger amounts than their colleagues on the Continent. As examples, however, may be quoted the recent pronouncements of three authoritative bodies.

The Committee on Nutrition appointed by the British Medical Association (Report 1933*a*) regarded one pint of milk per day as the

basal primary requirement of diets for children between the ages of 1 and 5, and half a pint for children between 5 and 10 years of age.

Since their report, further studies have been conducted, the conclusion of which is to point to the desirability of even larger quantities of milk for growing children. The Technical Commission of the Health Committee of the League of Nations appointed to inquire into the problem of nutrition (Report 1936*a*) recommended, for example, that 75–100 per cent. of the total energy requirements during the first years of life, decreasing to about 50 per cent. at 3 to 5 years, and to about 25 per cent. at the age of puberty, should be provided by milk. In terms of volume these figures correspond to $1\frac{1}{4}$–$1\frac{3}{4}$ pints of milk per day for children between the ages of 1 and 3, and $1\frac{3}{4}$ pints for children between the ages of 3 and 14. The Commission is also in favour of allowing $1\frac{3}{4}$ pints per day for pregnant and nursing women.

In rather more general terms the Advisory Committee on Nutrition appointed by the Ministry of Health (Report 1937*a*) expresses the opinion that the desirable amount of milk for children is from one to two pints per day, for expectant or nursing mothers about two pints per day, and for other adult members of the community half a pint per day.

The Present Level of Milk Consumption. When we inquire into the actual amount of milk consumed in this country, we find that it is considerably less than that regarded as desirable by the nutritional experts. Exact figures are impossible to obtain, but those furnished by the League of Nations (Report 1936*b*) may be accepted as the nearest approximations available (Table I).

It will be noted that the average *per caput* consumption of milk and cream in Great Britain during the years 1930 to 1931 was below that in the Netherlands, Denmark, New Zealand, and the United States of America, and less than half of that in Norway, Switzerland, and Canada. On the other hand, the consumption of butter was higher than that in any other European country listed in the table. Together with a moderate consumption of cheese, this brings the total dairy produce consumed in Great Britain up to the equivalent of nearly two pints a day. This figure, however, is justifiably open to criticism, since neither butter nor cheese contains all the constituents of the original milk.

The important item is liquid milk. The figure of half a pint a day refers to 1931. Since then the sale of liquid milk has increased and is estimated to have risen by about 13·5 per cent. between the years 1933–1934 and 1937–1938. Taking the total liquid milk sales for the year 1937–1938 as 752·4 million gallons, this gives an average liquid milk consumption in England and Wales of 0·42 pint per head

TABLE I

ESTIMATED AVERAGE CONSUMPTION PER HEAD PER DAY OF MILK AND CREAM, BUTTER AND CHEESE, IN THE YEARS 1930–1934 (REPORT 1936*b*, *c*)

Country.	Milk and Cream [1] (pints).	Butter (oz.).	Cheese (oz.).	All Dairy Products (Liquid Milk equivalent in pints).
Italy	0·15	0·10	0·46	0·53
Poland . . .	—	—	—	1·10
France	0·50	0·58	0·55	1·51
Germany . . .	0·50	0·72	0·57	1·73
Belgium . . .	0·37	0·90	0·28	1·75
Netherlands . .	0·66	0·72	0·61	1·75
United States .	0·86	0·77	0·19	1·78
Great Britain . .	0·50[2]	0·96	0·42	1·95
Denmark . . .	0·79	0·78	0·53	2·02
Norway . . .	1·11[3]	0·58[3]	0·63	2·15
Australia . . .	0·48	1·29	0·18	2·23
Switzerland . .	1·27	0·63	0·81	2·45
New Zealand . .	0·61	1·65	0·37	2·78
Canada. . . .	1·16[3]	1·34	0·15	2·92

[1] Including condensed and evaporated milk in Australia, the United States, and Great Britain; consumption of these products is unimportant elsewhere.
[2] 1930–1931 only. [3] 1935 only.

per day (Report 1939*a*). The discrepancy between this figure and the figure of 0·5 pint in Table I is probably due to the fact that the latter figure refers not only to England and Wales but also to Scotland, where the average consumption appears to be rather higher (Leighton and McKinlay 1934), and includes condensed and evaporated milk and cream as well as liquid milk.

Taking, however, half a pint per head per day as an average figure, it is seen that the consumption of liquid milk in this country is very much lower than that recommended by the nutritional experts.

Reasons for the Present Low Level of Milk Consumption. Cows' milk constitutes one of the most important articles of our dietary. It is also one of the most dangerous. This statement is no exaggeration, and abundant evidence will be brought in the following pages to justify it. Put quite briefly, udder disease of different types is so prevalent among cattle in this country that the milk as it comes from the cow is frequently infected with pathogenic bacteria. In addition the milk is occasionally exposed to infection from the human personnel engaged in handling it, as well as to contaminated water used in washing the utensils. Some of the pathogenic micro-organisms that gain access to it, particularly those responsible for the enteric

group of diseases, are capable of multiplying rapidly in the milk (Pullinger and Kemp, 1938) which, unlike most of our common foodstuffs, is an excellent medium for the growth of bacteria. It is not theerfore surprising that, exposed as it is to infection with so many different micro-organisms coming from such a variety of sources, milk should frequently be responsible for disease in those consuming it in the raw condition.

This state of affairs is well recognized by the medical profession, and has been the subject of numerous resolutions by authoritative bodies (see p. 193) who have urged that all pathogenic micro-organisms in milk should be destroyed by pasteurization so as to render it safe for human consumption. The fact that a great deal of raw potentially dangerous milk is being retailed in this country at the present time, and that in several districts a supply of safe milk is unobtainable, has a prejudicial effect on the total amount consumed. Many Medical Officers of Health are placed in the invidious position of being asked to approve of raw milk from an unsatisfactory source for distribution to schoolchildren, and of having to decide whether it is better to let them run the risk of contracting infection, or to deprive them of milk altogether.

That the retail price and other factors play a considerable part in determining how much milk shall be drunk is not disputed. This inquiry, however, is concerned with the health aspects of the problem, and the effect of the present lack of confidence of the medical profession in the milk supply is undoubtedly one important reason why the consumption of liquid milk in this country is so low. As the authors say in the introduction to one of the League of Nations Reports (Report 1937*b*), " Not until municipal authorities exercise the same care over the milk supply as they do over the water supply, and not until the public is educated to understand that milk is an article of their dietary which has to be treated with special caution, will it be justifiable on hygienic grounds to recommend the unlimited consumption of cows' milk." The present report will be devoted to ascertaining how far pasteurization can justify the demands of the nutritional and bacteriological experts for " more milk " and " safe milk."

Summary

1. Cows' milk is a food of exceptional value for the human species. It contains a well-balanced mixture of fat, carbohydrate, and protein ; it is particularly rich in mineral salts ; and it contains all the important vitamins.

2. Cow's milk, however, is not a perfect food for the human infant. It differs both qualitatively and quantitatively from human milk

and requires to be supplemented with iron and perhaps copper, with vitamin C, and during the winter months with vitamin D.

3. The *per caput* consumption of liquid milk and cream in Great Britain is considerably less than that recommended by authoritative bodies of dietitians, and is probably not much greater than half a pint per day.

4. One reason for this state of affairs is believed to be the unsatisfactory nature of our raw supply, which is more frequently infected with pathogenic bacteria and more likely to give rise to disease than any other common article of our dietary.

5. Not until safe milk is made generally available, will the medical profession be able unreservedly to recommend its use.

CHAPTER II

MODE AND EXTENT OF INFECTION OF RAW MILK WITH PATHOGENIC BACTERIA

Milk may be contaminated with pathogenic micro-organisms derived from cattle, from the human personnel, from water, or from rodents. It will be convenient to deal with these in order.

Incidence of Disease among Cattle

Tuberculosis. Bovine tuberculosis is very prevalent in Great Britain. It appears on the whole to be commoner in the northern half of the kingdom, including Scotland, than in the southern, but there is considerable variation between different counties in any one area.

Apart from the avian type of bacillus, which is not common and which gives rise only to a localized retrogressive form of disease, tuberculosis in cattle is due to the bovine type of tubercle bacillus. The disease affects particularly the lungs and pleura, though the abdominal organs, and less frequently the udder, may be invaded. Infection occurs usually, either by inhalation or by feeding, from another cow ; but it may sometimes result from contact with pigs, or very occasionally from human beings whose lungs are infected with the bovine type of tubercle bacillus. The organisms are discharged by the diseased animal in the cough spray, in the fæces, and, if the udder is infected, in the milk.

The incidence of infection increases with the age of the animal. It is uncommon in calves, fairly common in bullocks and heifers, and very common in cows—particularly in older animals. Table II, compiled by Delépine (see Cobbett 1917) on the basis of tuberculin tests controlled by post-mortem examination, will make this clear.

Though infection with the tubercle bacillus is very common in cattle, only a proportion of infected animals develop manifest disease. In the remainder the infection lies latent, ready to flare up under unfavourable environmental conditions. In this state its detection during life is possible only by means of the tuberculin test.

The exact frequency of tuberculosis in cattle in this country is not known with certainty. There is a general consensus of opinion, however, based on numerous records of individual observers, that about 40 per cent. of cattle react positively to the tuberculin test (Report 1932*a*, 1934). This test merely indicates that the animals are *infected*

TABLE II

FREQUENCY OF TUBERCULOSIS IN CATTLE CLASSIFIED ACCORDING TO AGE (AFTER COBBETT, 1917)

Age.	Number examined.	Percentage Tuberculous.
0–1 years	29	3·4
1–2 years	68	13·2
2–3 years	112	24·1
3–5 years	51	23·5
5–9 years	94	48·9
9–13 years	25	76·0

with the tubercle bacillus. It does not necessarily mean that they are *infective* to other animals or to human beings; indeed, the majority of cattle reacting to this test are suffering from only a latent form of the disease.

This figure of 40 per cent. is borne out by the result of abattoir inspections. Of 55,318 cows slaughtered at various centres in Great Britain, including Dublin, 39·5 per cent. were found on post-mortem examination to contain macroscopic lesions of tuberculosis (Report 1932*a*).

The incidence of *udder tuberculosis* is not easy to assess. In its early stages it cannot be detected by clinical examination, even though tubercle bacilli are being excreted in the milk. Moreover, there is reason to believe that it is frequently overlooked in animals that are suffering from pulmonary tuberculosis and are condemned for this cause (see Pallaske 1939). Its diagnosis depends to some extent on the skill, experience, and care of the veterinarian. Without a bacteriological examination of the milk of every cow reacting to the tuberculin test, it is impossible to ascertain the real frequency of udder involvement. The available figures suggest an incidence among milch cows of between 0·2 per cent. (Report 1932*a*) and 2·0 per cent. (see Report 1931*a*). Probably the figure of 0·5 per cent. quoted in the Cattle Diseases Report (Report 1934) may be taken as an approximation to the truth.

The number of tubercle bacilli excreted in the milk varies greatly. In the early stages they are very few, but in advanced cases of udder involvement they run into hundreds and thousands, and even more, per millilitre. Ostermann (1908), for example, found that milk could be diluted 50,000 times and still be infective. Positive results in even higher dilutions were obtained by Ostertag and by Bongert (see Ostermann 1908). On the average he estimates that 1 ml. of milk from a cow with a tuberculous udder contains about 1000 infective doses for

a guinea-pig. The number of living tubercle bacilli in an infective dose varies somewhat. It is known that a single bacillus may cause infection, but the minimal certain infecting dose is probably rather over 10 bacilli (Schwabacher and Wilson 1937). Adopting a conservative average of 5 bacilli per infective dose, it follows that milk from a cow with a tuberculous udder commonly contains somewhere about 5,000 virulent tubercle bacilli per millilitre. Pullinger (1934), it may be noted, found that two out of three milks from tuberculous udders could be diluted 1 in 1,000,000 times and still prove infective for guinea-pigs. The average number of tubercle bacilli excreted by these animals must therefore have been in the neighbourhood of 5,000,000 per millilitre.

Contagious Abortion. This disease is almost as widespread in Great Britain as tuberculosis, and causes even greater financial loss. Its exact frequency is difficult to ascertain, but Priestley (1934) estimates that 20 per cent. of the cows are infected. Large herds are more often attacked than small.

When introduced into a fresh herd the disease spreads rapidly, assuming epidemic proportions. If no new animals are imported, the severity of the infection dies down, and the disease becomes endemic. In this state it may remain for years. The introduction of fresh animals may be followed by a recrudescence of the epidemic state.

Cows infected for the first time usually abort at an early stage. At their next pregnancy they abort either late or not at all, and in subsequent pregnancies they generally carry their calves to full term. The disease is, however, often accompanied by sterility, and the animals after aborting may fail to become pregnant again.

The causative organism, *Brucella abortus*, is discharged from the vagina at the time of abortion or premature calving and for some days later.

Besides invading the uterus, it often gives rise to a mild form of mastitis, not detectable by clinical examination, and is excreted in the milk. Both cows that have aborted and those that have calved normally may be affected. The udder is invaded far more frequently than in tuberculosis. Judging from the fact that *Br. abortus* is found in the milk of individual herds three or four times as often as the tubercle bacillus, it is probable that an average of at least 2 per cent. of milch cows in Great Britain are excreting *Br. abortus*.

The number of organisms in the milk fluctuates from day to day, but is usually greatest soon after abortion or calving, gradually diminishing as lactation advances. In the first week or two there may be as many as 200,000 per millilitre, but later the usual number is of the

order of 10,000–20,000 per millilitre (Karsten 1932). Many cows cease to discharge the organism in their milk after a few weeks; but others may remain infected for a year or two, or even permanently. Human beings, who become infected either by contact with aborting animals or by the consumption of infected milk, may develop undulant fever.

Mastitis. Inflammation of the udder is one of the commonest diseases of cows. Indeed, pathological examination shows that practically all cows which have passed through one or more pregnancies are suffering from it to some extent. The disease may be acute, subacute, or chronic. It is common for more than one-quarter of the udder to be attacked. Infection normally occurs through abrasions of the teats or *via* the teat canal, and is spread by the hands of the milker, but some forms are the result of internal infection. The condition is very persistent, and it is doubtful if complete recovery ever occurs.

Most commonly the disease is due to a type of hæmolytic streptococcus known as *Streptococcus agalactiæ*. So far as is known, this organism is not infective for man. Nevertheless, the milk from cows suffering from this type of mastitis is not satisfactory for drinking purposes. It contains large numbers of pus cells, often exceeding a million per millilitre, and in the acute stage even blood. Moreover, there is a suggestion that toxic substances may be present in " mastitis " milk which may give rise to gastro-intestinal disturbances in susceptible human subjects (see Brooks and Tiedeman 1937). The milk itself may be normal or greatly changed in appearance, according to the stage and severity of the infection.

Very occasionally mastitis is due to infection with *Str. pyogenes* derived from the nasopharynx of a milker or other person handling the milk (see Report 1937*b*, 1937*e*, Bendixen and Minett 1938). This organism is responsible for scarlet fever and septic sore throat. Its presence in the milk is of the greatest concern, and it is fortunate that mastitis caused by this organism is of infrequent occurrence.

Sometimes mastitis may be due to infection with *Staphylococcus aureus*. When ingested in the milk this organism does not appear to be directly pathogenic for human beings. If, however, the milk is left under suitable conditions, the organisms may grow and form a potent toxin capable of giving rise in man to acute gastro-enteritis.

Salmonella Infections. Infections with organisms of the Salmonella group are not infrequent in cattle. They give rise most commonly to inflammation of the intestine, resulting in diarrhœa; but other organs may be invaded, and death may occur through septicæmia. In young animals the infecting organism is generally of

the *Bacterium enteritidis* var. *dublin* type, while in older animals *Bact. typhi-murium* and *Bact. enteritidis* are more frequent. The organisms may gain access to the milk by fæcal contamination, but occasionally the udder is invaded and the organisms are discharged directly. In human beings they give rise to acute gastro-enteritis or sometimes, as with the *dublin* type, to a paratyphoid-like fever.

Other Diseases. In addition to those already mentioned, numerous diseases occur that may render the milk of cows undesirable for human consumption, such as foot-and-mouth disease, cowpox, anthrax, suppurative and septicæmic infections, Johne's disease, and other conditions accompanied by diarrhœa. In some of these diseases the causative organism is excreted in the milk; in others it gains access to it from an ulcerated teat or from fæcal contamination.

Human Beings as a Source of Infection

Milk may be infected from human beings with several different types of bacteria. These organisms fall mainly into two groups, those dwelling in the nose and throat and expelled in the cough-spray, and those dwelling in the intestine and excreted in the fæces and sometimes in the urine. Generally speaking the former group of organisms, comprising the hæmolytic streptococci and diphtheria bacilli, do not multiply in milk to any appreciable extent at ordinary atmospheric temperature (Pullinger and Kemp 1937), while the latter group, comprising the typhoid, paratyphoid, dysentery, and food-poisoning bacilli, are able to multiply readily (Pullinger and Kemp 1938). For this reason direct contamination with organisms of the latter group may lead to dangerous infection of the milk, whereas direct contamination with organisms of the former group is more dependent in its results on the number of organisms originally gaining access to the milk. We may now consider briefly the various organisms in turn.

Hæmolytic Streptococci. Organisms belonging to Lancefield's Group A, now usually referred to as *Str. pyogenes*, are found in the nasopharynx of something like 6–7 per cent. of adults (Hare 1935, Frisch 1938). So far as can be determined by epidemiological inquiry, it is doubtful whether healthy carriers of this organism are more than mildly infective. On the other hand, patients who are suffering from scarlet fever or septic sore throat, even if only in the incubation period, and patients with local disease of the nose or ear caused by *Str. pyogenes*, are often highly infective. The organisms expelled from the nose and throat during coughing, sneezing, laughing, spitting, and even talking, may contaminate the milk directly, or occasionally they may find their way into the cow's udder, give rise to mastitis, and so lead indirectly to infection of the milk.

Diphtheria Bacilli. These organisms are found in the nose and throat of persons suffering from diphtheria, and sometimes in apparently healthy carriers. They may reach the milk by direct contamination from the cough-spray, or they may sometimes be implanted on the ulcerated teat of a cow.

Typhoid Bacilli. These organisms are usually present in the fæces of patients suffering from typhoid fever, and sometimes in the urine. About 3–4 per cent. of patients who have recovered from typhoid fever may continue to excrete these organisms for months or years. Since there are about 2,000 cases of typhoid fever notified annually in England and Wales, of which about 10 per cent. prove fatal, it follows that among those that recover about 70 will remain as permanent carriers. Assuming that the average length of time during which these carriers remain infected is 10 years, it may be calculated that at the present time there are probably about 500–1000 healthy carriers of typhoid bacilli in England and Wales, or roughly 1 in 60,000 of the population. Their distribution is, of course, irregular, and after an outbreak of typhoid fever the proportion of carriers in the affected locality will greatly exceed that in the country as a whole.

Typhoid carriers are of the greatest danger. Unless their standard of personal hygiene is of an unusually high order, their fingers are liable to be contaminated with urine or fæcal material, and may cause direct infection of the milk. Moreover, their dejecta may find their way into water used for drinking or washing purposes and cause indirect infection of the milk. Typhoid patients, except those in the ambulant stage, are less likely to be concerned in handling milk, but their dejecta, unless properly sterilized, may lead to infection of the water supply and utensils, and consequently of the milk itself.

Paratyphoid Bacilli and Dysentery Bacilli. These organisms behave more or less like typhoid bacilli, and may gain access to milk directly from imperfectly cleansed fingers or indirectly through water.

Food-Poisoning Bacilli of the Salmonella Group. Organisms such as *Bact. typhi-murium*, *Bact. enteritidis*, *Bact. thompson*, and *Bact. newport*, which are responsible for acute food-poisoning in man, may gain access to the milk in the same way as typhoid bacilli. Chronic carriers, however, of the food-poisoning organisms, do not appear to be common, and infection of milk is more likely to occur from the fæces of convalescents or of patients suffering from a mild attack than from healthy carriers.

Water as a Source of Infection

Water may be contaminated from the urine or fæces of human patients or carriers infected with typhoid, paratyphoid, or dysentery

bacilli. Salmonella organisms of the food-poisoning type may gain access to it from the fæces of human patients or carriers, or from the fæces of cattle, pigs, and rodents. The contamination of our rivers is now so great that many of them are bound to be infected from time to time, or in some instances almost continuously. Fortunately, however, water from the larger rivers is usually employed for the supply of large towns and is properly filtered and chlorinated. The main risk of infection of milk from water occurs in districts where small surface supplies and shallow wells are used for dairy purposes without adequate purification. In these areas the conservancy system is often unsatisfactory, so that adventitious contamination of the water supply from excretal material is liable to occur.

Contaminated water may infect milk in various ways. It may be used for washing the utensils or for watering the milk. If it is supplied to the cows for drinking purposes, or if it is used for washing their udders, it may drip into the milk vessels. Some organisms, like *Bact. enteritidis*, *Bact. dublin*, and *Bact. typhi-murium*, may cause an intestinal or systemic infection of the cow, be discharged in the fæces, and thus find their way into the milk. There is a slight suggestion that even typhoid bacilli may infect cows occasionally and be excreted in the fæces (Shaw 1937), but the evidence is quite inconclusive.

Rodents as a Source of Infection

Rodent typhoid is a very common natural disease of rats and mice. Its spread is often intentionally encouraged by the use of such preparations as Ratin and Liverpool virus. The causative bacillus is usually *Bact. typhi-murium* or *Bact. enteritidis*. Both of these organisms are infective for cattle and for man. The disease is characterized by an enteritis, usually accompanied by septicæmia and generalized infection of the tissues. The organisms are discharged during the illness in the urine and fæces. The mortality varies greatly, but usually a moderate proportion of the animals recover. Many of these become temporary or permanent carriers, and are liable to infect milk, milk-vessels, water, and other objects to which they are able to gain access. Infection from this source is usually very difficult to trace.

Rats frequently carry an organism, usually referred to as *Streptobacillus moniliformis*, in their nasopharynx, which gives rise to rat-bite fever in man. A very similar, if not identical organism, known as *Actinobacillus actinoides*, gives rise to broncho-pneumonia in calves (see Dienes and Edsall 1937). At least two outbreaks of milk-borne disease caused by *Str. moniliformis* are on record (Parker and Hudson 1926, Place and Sutton 1934), and it must be assumed that infection had gained access to the milk either from rats or from cattle.

Extent of Infection of Raw Milk Supply with Pathogenic Bacteria

Tubercle Bacilli. In estimating the extent of infection of the raw milk supply with tubercle bacilli it is necessary to distinguish between (1) the milk of individual cows, (2) the mixed milk from the cows in individual herds, and (3) the mixed milk from several herds, usually referred to as " bulked milk." It has already been stated that something like 0·5 per cent. of milch cows in this country are suffering from tuberculous mastitis and are excreting tubercle bacilli in the milk. Attention can therefore be directed to the mixed milk of individual herds and to bulked milk.

The milk coming into our large towns often consists of both types. The former type, representing the composite milk of individual herds, arrives in churns. The latter type, or bulked milk, is now usually filled at receiving depots in the country into glass-lined tanks capable of holding 500 to 3,000 gallons of milk. On arrival in the towns samples are taken at intervals by the local authorities and examined for tubercle and sometimes for *abortus* bacilli, particular attention being paid to the churn milk coming from individual farms. The results of these examinations are generally recorded in the annual report of the Medical

TABLE III

Proportion of Raw Churn Milks from individual Farms entering various Towns during the Ten Years 1928–1937 found to contain virulent Tubercle Bacilli

Town.	No. of Samples examined.	Percentage containing Tubercle Bacilli.
London	18,910	9·3
Brighton	680	9·3
Birmingham	15,350	8·1
Liverpool	9,053	6·1
Manchester	8,803	13·2
Sheffield	9,275	7·9
Newcastle-on-Tyne	3,813	3·7
Glasgow	3,438	8·5
Edinburgh	2,446	8·0
Aberdeen [1]	1,741	9·8
Total	73,509	
Weighted Mean		8·6

[1] No record for years 1932, 1933, or 1934.

Officer of Health. By collecting these data from different towns over a suitable period it is possible to obtain an idea of the extent of infection of the milk.

Churn Milk from Individual Herds. An inquiry made some years ago (Report 1932*a*) showed that of 69,901 samples of churn milk from individual farms entering London and various county boroughs situated in different parts of Great Britain during the years 1918 to 1930, 6·7 per cent. were found to contain virulent tubercle bacilli. In Table III similar data have been collected for the years 1928 to 1937 inclusive.

There is a considerable difference between the extent of infection of the milk entering different towns, but it will be seen that on an average about 8·6 per cent. of the milk contained tubercle bacilli. The figures given in the table do not represent the extremes. For instance, Pullinger (1934), who examined milk samples from 105 non-tuberculin-tested herds in Cheshire, found that 21 per cent. contained tubercle bacilli, whereas of 104 samples from similar herds in Somerset only 2 per cent. proved to be infected.

TABLE IV

PERCENTAGE OF RAW CHURN MILK FROM INDIVIDUAL FARMS ENTERING VARIOUS REPRESENTATIVE TOWNS DURING THE YEARS 1921–1937 FOUND TO CONTAIN TUBERCLE BACILLI

Year.	London.	Birmingham.	Manchester.	Glasgow.
1921	3·5	6·0	12·1	3·9
1922	2·6	3·5	8·6	2·6
1923	4·1	7·7	11·1	7·9
1924	5·0	8·6	9·5	3·0
1925	4·6	7·6	8·2	0·0
1926	4·5	8·8	10·3	1·9
1927	7·8	7·1	11·1	2·0
1928	8·9	9·1	17·6	5·5
1929	7·5	6·7	12·6	3·6
1930	9·1	6·2	14·3	4·1
1931	10·2	8·0	16·2	13·2
1932	10·9	8·9	14·1	9·5
1933	8·4	6·4	12·0	7·7
1934	9·8	6·4	12·0	8·1
1935	12·6	8·0	13·1	9·9
1936	9·3	10·1	12·0	11·9
1937	7·0	10·2	10·1	9·7

Table IV is included to show that the infection of the milk supply coming into our large towns does not seem to be decreasing in frequency.

The figures that have been quoted so far refer to samples of raw milk coming from graded and ungraded producers, excluding Tuber-

culin Tested milk. Lest it should be thought that the infection of the milk is derived almost exclusively from dirty farms, it may be well to compare figures for the degree of infection of ungraded and of graded raw milk. An examination of 5,274 samples of Grade A milk, corresponding to the present Accredited milk, carried out in different parts of Great Britain during the years 1930–1935, showed that 5·6 per cent. were infected with tubercle bacilli, while of 37,920 specimens of ungraded milk 7·4 per cent. contained tubercle bacilli (Report 1936*d*).

More recently figures have been published for milk coming into London from individual producers (Report 1937*d*); these are given in Table V.

TABLE V

PROPORTION OF RAW MILK FROM GRADED AND UNGRADED PRODUCERS CONTAINING TUBERCLE BACILLI COMING INTO LONDON IN 1937.

Grade.	No. of Samples examined.	No. containing Tubercle Bacilli.	Percentage containing Tubercle Bacilli.
Tuberculin Tested . . .	67	1	(1·5)
Accredited	354	34	9·6
Ungraded	1,508	100	6·6

It is seen that the average degree of infection of Accredited milk was rather higher than that of ungraded milk.

Again in Manchester during the three years 1935 to 1937 12·1 per cent. of all milk samples and 13·6 per cent. of Accredited milk samples were found to contain tubercle bacilli.

Too much attention should not be paid to these actual figures, since the average size of the herds producing different types of milk may not be identical. There is no evidence, however, to suggest that Accredited is less likely to carry tubercle bacilli than ordinary raw milk.

Bulked Milk. Figures for bulked milk are of less interest, since they must depend to a considerable extent on the degree of bulking. Other things being equal, the chances of finding tubercle bacilli in the mixed milk from 100 farms must be greater than in that from 20 farms. Milk arriving in London, for instance, in 3,000-gallon tanks, representing the mixed milk of perhaps 50 farms or so, is almost invariably infected with tubercle bacilli (Report 1933*b*, Pullinger 1934). Sometimes the degree of infection is quite heavy. Thus in 7 tanks tubercle bacilli were found in 50 ml. of milk, in 5 tanks in 5 ml. of milk, and in 3 tanks in 0·5 ml. of milk (Report 1933*b*). Pullinger (1934) was sometimes able to demonstrate tubercle bacilli in as little as 0·25 ml. of tank milk.

Assuming that the average yield of milk per cow is 3 gallons a day, it follows that milk from about 1,000 cows is required to fill a 3,000 gallon tank. Assuming further that 0·5 per cent. of milch cows are suffering from tuberculosis of the udder, five such cows will be expected in a total of 1,000. If each of these cows contributes 3 gallons a day, there will be a total of 15 gallons of tuberculous milk. Further, if examination shows the presence of tubercle bacilli in 1 ml. of the bulked milk, there must be at least 13·5 million infective doses in the tank. Assuming an average of 5 organisms in each infecting dose (see p. 8), it follows that the tank must contain about 70 million virulent tubercle bacilli. This figure corresponds to an average of 14 million tubercle bacilli excreted by each tuberculous cow, or approximately 100 tubercle bacilli per millilitre of tuberculous milk.

Brucella abortus. The frequency of infection of raw milk with *Br. abortus* is even greater than that with tubercle bacilli. This is due not to there being a greater number of animals infected with *Br. abortus* than with the tubercle bacillus, but to the fact that the udder is more often involved in contagious abortion than in tuberculosis.

There is no need to spend time endeavouring to obtain a precise estimate of the extent of infection of the milk with *Br. abortus*, since nearly all observers agree that this organism is very common. Thus Beattie (1932) found 34·9 per cent. of positive samples of mixed milk in Edinburgh, Smith, J. (1932) 28·3 per cent. in Aberdeen, Morgan (1932) 37 per cent. in Cardiff, Priestley (1932) 22·3 per cent. in Burnley, and Rowlands (1933) 17 per cent. in Bangor. It is difficult to say how much of this milk came from individual herds and how much was the mixed milk of several herds.

The more recent observations of Pullinger (1934), however, do not suggest that these figures are unduly high even for the milk of individual herds. Thus Pullinger found *Br. abortus* in 37 per cent. of milks from 105 herds in Cheshire and in 19 per cent. of milks from 104 herds in Somerset. Examining 101 samples of milk from 45 Tuberculin Tested herds, he found no fewer than 69 per cent. to contain this organism.

It may safely be concluded that something like 20–40 per cent. of farms in this country are sending out milk infected with *Br. abortus*. Bulked milk must be nearly always infected.

Mastitis Streptococci. Though the organisms commonly responsible for mastitis do not appear to be pathogenic for man, their presence in milk is highly undesirable, accompanied as they usually are by considerable amounts of pus and sometimes even of blood. As has already been pointed out, the disease is extremely common, and the great majority of the farms in this country must be sending out milk containing greater or smaller numbers of mastitis streptococci.

This applies not only to ordinary farms, but to the farms of graded producers. Thus Pullinger (1935) found hæmolytic streptococci in 24 out of 42 samples of milk coming from 16 producers of Certified milk, and in 42 out of 54 samples of milk coming from 24 producers of Grade A Tuberculin Tested milk. Of 58 samples of bulked milk collected from rail tanks, every one proved to be infected with these organisms.

Other Pathogenic Bacilli. No routine examination is made for pathogenic organisms in milk other than those that are commonly present in the udder of the cows themselves. How frequently streptococci of the scarlatinal type, diphtheria bacilli, and members of the enteric, dysentery, and Salmonella groups gain access to milk is unknown. Attention is drawn to the presence of these organisms only by the occurrence of an epidemic in the human population. Considering the frequency of sore throat, scarlet fever, diphtheria, and so on, it would be surprising if milk did not become infected fairly often with one or other of the organisms responsible for these diseases. The probability is that, either they reach the milk in too small numbers to be dangerous, or that the milk is consumed by relatively few persons with the result that only sporadic cases of disease occur, the milk-borne origin of which is not even suspected. This aspect of the problem will be discussed more fully in the next chapter.

Summary

1. Milk may be infected with pathogenic organisms derived from cattle, from human beings, from water, or from rodents.

2. Tuberculosis is widespread among cattle in Great Britain. It is estimated that about 40 per cent. of cattle react to the tuberculin test; that about 40 per cent. of cattle slaughtered in the abattoirs contain macroscopic lesions of tuberculosis; and that about 0·5 per cent. of milch cows suffer from udder tuberculosis and are excreting tubercle bacilli in their milk.

3. Contagious abortion is likewise very common. Probably about 20 per cent. of cows are infected, and something like 2 per cent. are excreting *Br. abortus* in their milk.

4. Mastitis is even commoner than tuberculosis or contagious abortion, and affects in some degree nearly every cow that has passed through more than one or two pregnancies. The organism usually responsible, namely *Str. agalactiæ*, does not appear to be directly pathogenic for man. *Str. pyogenes*, however, which occasionally infects the udder from human sources, renders the milk highly dangerous.

5. Salmonella infections are not uncommon in cattle, but no reliable figures are available for their frequency in this country. Milk

may be infected with these organisms either from a focus of infection in the udder, or from contamination with the fæces.

6. Human beings suffering from scarlet fever, septic sore throat, or diphtheria may infect milk directly through their cough-spray.

7. Human patients or carriers who are excreting typhoid, paratyphoid, dysentery, or Salmonella organisms of the food-poisoning type in their fæces or urine may infect milk directly through their imperfectly cleansed fingers.

8. Water that is contaminated with the types of organisms mentioned under (7) may lead to infection of the milk in various ways.

9. Rats and mice frequently suffer from rodent typhoid due to certain organisms of the Salmonella group which are infective both for cattle and for man. Milk may be contaminated directly or indirectly from the urine and fæces of these animals. Milk may also be infected with the organism of rat-bite fever, which is a common inhabitant of the nose of rats.

10. Examination of records shows that about 5–10 per cent. of farms in this country are sending out milk infected with virulent tubercle bacilli, about 20–40 per cent. with *Br. abortus*, and a much higher proportion with mastitis streptococci.

11. The available figures suggest (*a*) that Accredited milk is just as heavily infected with tubercle bacilli as ungraded milk; and (*b*) that Tuberculin Tested milk is just as heavily infected with *Br. abortus* as ungraded milk, if not more so.

12. Bulked milk reaching our large towns in 2,000- and 3,000-gallon tanks appears to be almost invariably infected, often heavily so, with all three organisms, namely tubercle bacilli, *Br. abortus*, and mastitis streptococci.

CHAPTER III

EXTENT OF MILK-BORNE DISEASE IN THE HUMAN POPULATION

In the previous chapter an attempt has been made to give some idea of the frequency with which raw milk in Great Britain contains pathogenic organisms. The present chapter will be devoted to considering the total amount of disease in the human population that results from the presence of these organisms in the milk supply.

Sporadic and Epidemic Milk-borne Disease. Broadly speaking milk-borne disease may be divided into two types—sporadic and epidemic. *Sporadic* cases of disease are caused chiefly by organisms which are frequently present in the milk, but which have a low degree of pathogenicity for human beings. Such organisms are the tubercle bacillus and *Br. abortus*. Persons ingesting these organisms frequently become infected with them, as can be shown by tuberculin and brucellin skin tests, but do not usually develop any obvious signs of disease. Fortunately only a very small proportion of persons who drink milk containing these organisms suffers from actual clinical tuberculosis or undulant fever.

Epidemics of disease, on the other hand, result from the presence in the milk of organisms having a high degree of pathogenicity for human beings. Such organisms are *Str. pyogenes*, the diphtheria bacillus, the typhoid bacillus, and the paratyphoid, dysentery and food-poisoning groups of bacilli. Persons ingesting any of these organisms run a high chance not only of getting infected but also of developing clinical manifestations of disease. The proportion of persons ingesting the infected milk who become ill varies with the different organisms, the age and sex distribution of the population and other factors, but quite frequently 70, 80, or 90 per cent. of those at risk contract the disease.

There is, of course, no clear line of demarcation between sporadic and epidemic milk-borne disease. Occasionally, for instance, tuberculosis (see Stahl 1937, Gnosspelius 1939) and undulant fever (see p. 33), which are characteristically sporadic diseases, may occur in a minor epidemic form following the consumption of heavily infected milk; while diseases like scarlet fever, septic sore throat, diphtheria, enteric fever, dysentery, and acute food poisoning, which are characteristically epidemic diseases, necessarily occur sporadically if the milk is consumed by only a few persons.

The fact that some types of pathogenic organisms in milk, particularly the tubercle bacillus and *Br. abortus*, give rise to clinically detectable disease in only a very small minority of persons consuming the milk has led many members of the laity, and even of the non-medical scientific professions, to express a doubt whether these organisms are in fact responsible for disease at all. Such a doubt might have been justifiable fifty years ago, when current bacteriological teaching on the causation of disease laid undue stress on the organism and practically neglected the reaction of the host. It was then assumed that the mere access of the organism to the tissues would result in an attack of the corresponding disease. Since then, we have learnt a great deal about abortive attacks, sub-clinical infections, latent infections, healthy carriers, and so on, and we now know what many of the older epidemiologists realized long ago, that the occurrence of disease is only possible when contributory factors are at work to lower the resistance of the tissues, and hence permit the *in vivo* proliferation of the invading organism. Since the resistance of the tissues is determined in any individual by a multiplicity of factors of which we have at present only very imperfect knowledge, and since it varies greatly from one individual to another, and even from time to time in the same individual, it follows that the reaction of any individual to infection with a particular bacterium is generally quite impossible to predict. When the bacterium is highly virulent, and gains access to a non-immune population, the majority of those exposed to risk will probably develop a typical attack of the disease. When, on the other hand, the bacterium is less virulent and is circulating in a population a large proportion of which is protected by a latently or overtly acquired immunity, only isolated persons, here and there, whose resistance for some reason or other is unusually low, are likely to suffer from the disease in its characteristic form. When it is further realized that the outcome of any bacterial onslaught is determined not only by the virulence of the organism and the resistance of the tissues, but also by the numbers of organisms in the infecting dose and the frequency with which the doses are repeated, some idea will be gained of the complexity of the equilibrium that exists between any given parasite and any given host (Report 1937*b*).

Difficulties in ascertaining the frequency of Milk-borne Disease. Attention has very properly been drawn by the authors of the League of Nations report (Report 1937*b*) to the fact that milk-borne disease is rarely obtrusive and is not usually noticed unless it is looked for. Our knowledge of milk-borne disease in any country varies, in fact, with the activity of the public health and laboratory

services. Up till a few years ago practically nothing was known of the frequency of tuberculosis of bovine origin except in Great Britain, Germany, and the United States of America, and even in these countries our knowledge was, and still is, very incomplete. The recent observations, however, of Jensen (1932, 1935) in Denmark, and of Ruys (1936, 1937) in the Netherlands have brought to light the existence in these countries of a great amount of milk-borne tuberculosis that had not previously been suspected.

Undulant fever is another example. Until Evans (1918) in the United States drew attention to the similarity of *Br. abortus* and *Br. melitensis*, and until Bevan (1921–1922) in Southern Rhodesia first described the occurrence of undulant fever due to infection with *Br. abortus*, no serious suspicion had been cast on the possible infectivity for human beings of milk from cows suffering from contagious abortion. As soon as the first cases were recognized, investigators in many different countries began to look for the disease, with the result that in a few years undulant fever of milk-borne origin had been reported from practically every part of the world.

The difficulty is not confined to diseases like tuberculosis and undulant fever that are normally sporadic in their occurrence. The recognition of even epidemic milk-borne disease varies greatly in different countries, and even in different parts of the same country. In Great Britain interest was early aroused by the observations of Power and other workers in the latter half of last century. By 1881 Hart (see Parkes and Kenwood 1913) was able to compile a list of no fewer than 50 outbreaks of enteric fever, 15 of scarlet fever, and 6 of diphtheria, which had been traced to infected milk supplies. Since then interest has been greater in this than in most other countries, and our present knowledge is correspondingly fuller. In some parts of Europe the existence of milk-borne disease is practically denied, not because there is any evidence that it does not occur, but because no one has ever troubled to look for it.

In the United States the number of milk-borne epidemics that have been recognized varies greatly in different localities. For example, Armstrong and Parran (1927) obtained records of 42 milk-borne outbreaks of septic sore throat in the United States between 1908 and 1927, and Brooks (1933) obtained records of 30 outbreaks of the same disease between 1927 and 1932. Analysing the distribution of these 72 outbreaks, Brooks found that no fewer than 45 of them had occurred in two States, namely New York and Massachusetts. In 32 States there were no records of milk-borne outbreaks of septic sore throat at all. It is impossible to escape the conclusion that a great deal of milk-borne disease is simply overlooked.

Some of the reasons why milk-borne disease is not more often recognized may be briefly considered (Report 1937*b*).

" 1. No disease is exclusively milk-borne. All the diseases that may be carried by milk may also be carried by other means, such as air, food or water. In any particular disease, milk may be, and often is, an unusual source of infection, and the occurrence of cases of the disease in question does not therefore arouse an immediate suspicion that the milk is at fault. Too often, the common method of infection is taken for granted, and the possibility that the milk supply is infected is not even envisaged. Perhaps one of the most striking examples of this is afforded by the outbreak of septic sore throat in August 1935 in Copenhagen. In spite of the magnitude of this epidemic, involving as it did about 10,000 persons, no suspicion of its milk-borne origin was aroused until attention was drawn to it by a careful epidemiological inquiry. More recently in the United States three outbreaks of milk-borne septic sore throat occurred within a few months in a single State possessing an unusually efficient department of health; yet in each instance the epidemic was in progress for some weeks before its existence was discovered by the State Authorities (Defries 1938).

2. The milk-borne nature of some diseases, particularly tuberculosis, can be determined with certainty only by bacteriological examination. No distinction exists clinically between tuberculosis of human and tuberculosis of bovine origin. Since only a minority of cases of tuberculosis are investigated bacteriologically to ascertain the type of infecting organism, the origin of most cases remains undetermined.

3. The occurrence of a long incubation period and a gradual onset in some milk-borne diseases, such as tuberculosis, and undulant fever, serve to mask any direct relationship between the infection and the vehicle by which it was borne. It is, in fact, only within recent years that the very existence of undulant fever has been recognized, and that its frequent milk-borne origin has been proved.

4. When a large epidemic of an infectious disease occurs with explosive suddenness, attention is very often directed to the milk supply. But when the disease is limited to a few families, particularly if they are scattered over a wide area or are attended by different doctors, or when it affects only one or two persons, the probability that milk will receive even a passing thought is very small.

5. Sporadic cases and small outbreaks of milk-borne disease are often not reported at all, or are published only in the local medical officer of health's annual report. Without going through every such report in detail, the majority of these smaller outbreaks will be

overlooked and will never be brought to the notice of the central authorities."

For these reasons our knowledge of milk-borne disease is very defective. To ascertain its true frequency in any country would require a combined epidemiological and bacteriological investigation covering every town and village and paying attention to practically every case of infectious disease. No such investigation has ever yet been undertaken, and the records that are available are necessarily fragmentary and incomplete. Such as they are, they may now be examined.

Tuberculosis. Tuberculosis in man may be due to either the human or the bovine type of tubercle bacillus. The human type is derived almost exclusively from man, and gains access to the body mainly by the respiratory tract. The bovine type is derived almost exclusively from cattle, and gains access to the body mainly by the alimentary tract. Both types may give rise to any form of tuberculosis, but the bacteriological evidence shows that in England and Wales the human type is responsible for over 98 per cent. of all cases of pulmonary tuberculosis, and for about 70 per cent. of all cases of non-pulmonary tuberculosis. Since it is impossible clinically to distinguish between tuberculosis due to the human and tuberculosis due to the bovine type of bacillus, the number of deaths due to the bovine type can be arrived at only by indirect methods. Two such methods are available.

(1) *Calculation based on the Proportion of Samples of Tuberculous Material examined bacteriologically which are found to contain the Bovine Type of Tubercle Bacillus.* This method was used by the Cattle Diseases Committee (Report 1934). Material from cases of tuberculosis, removed either at operation or after death, is bacteriologically examined and the type of infecting organism is determined. Since this process of " typing " is time-consuming and requires skill, it is not performed as a routine. Most of it has been done by Griffith (for references up to 1936, see Topley and Wilson 1936), working originally for the Royal Commission on Tuberculosis, and more recently for the Medical Research Council. Of late years, however, other workers in different parts of the country, either alone or in collaboration with Griffith, have contributed to our knowledge of this subject (Munro and Griffith 1928; Griffith, 1937, 1938; Griffith and Munro 1933; Cumming *et al.* 1933; Walker 1934; Griffith and Smith 1935, 1938; Tobiesen *et al.* 1935 *a*, *b*; Griffith and Menton 1936; Blacklock 1932, 1935, 1936).

Having ascertained the proportion of bovine type bacilli present in the samples examined from different varieties of tuberculosis, it is possible to calculate from the Registrar-General's figures the number of deaths that occur in any one year as the result of infection with

the bovine type. The accuracy of the method depends on the assumption that the proportion of bovine infections in the cases that died was the same as in the samples of material examined. Owing to the fact that the proportion of bovine infections varies considerably in different parts of the country, being higher in the North of England than in the South, and highest of all in Scotland, and owing to the fact that the proportion of bovine infections appears to be falling steadily, this assumption is not likely to be completely justified. Nevertheless, the method affords an approximate estimate of the probable number of deaths due to infection of bovine origin.

In the Cattle Diseases report (Report 1934) the figures were worked out for 1931. The proportion of bovine infections was based on material examined during the preceding twenty-five years, and since there was reason to believe that tuberculosis of bovine origin had diminished during that time, the figures were subjected to criticism in some quarters. It is probable that the proportion of bovine infections in some of the non-pulmonary varieties of tuberculosis erred a little on the high side, but the proportion in pulmonary tuberculosis was almost certainly too low. The final figures of 2,147 deaths of bovine origin in England and Wales and 465 in Scotland were probably very near the mark.

Since 1931, tuberculosis mortality has continued to decrease, and it has therefore been thought advisable to bring the estimate of deaths of bovine origin up to date. The proportion of bovine infections has been calculated from the results of examining material during the twenty years 1917 to 1937. The necessary figures were very kindly collected and supplied by the late Dr. A. Stanley Griffith, C.B.E. The estimated number of deaths of bovine origin is set out in Table VI for England and Wales, and in Table VII for Scotland.

It will be seen that the number of deaths due to infection with the bovine type in 1937 was about 1,603 in England and Wales, and 404 in Scotland. These figures may be compared with those of 2,147 and 465 respectively for the year 1931 reported by the Cattle Diseases Committee (Report 1934).

One or two remarks may be made on the percentage figures used in calculating the proportion of bovine type infections. It will be noted, for instance, that in Table VI, the figure for tuberculosis of intestines and peritoneum at all ages is taken as 82 per cent. Though probably correct for ages under 15, this figure is almost certainly too high for all ages. Nevertheless the figure is based on the examination of tissues at all ages, and any alteration made in it would have to be pure guess work. It is thought better, therefore, to retain it. On the other hand the figure of 6 per cent. for " disseminated " tuber-

TABLE VI

CALCULATED NUMBER OF DEATHS FROM TUBERCULOSIS IN ENGLAND AND WALES IN THE YEAR 1937 DUE TO INFECTION WITH THE BOVINE TYPE OF TUBERCLE BACILLUS (FIRST METHOD).

Variety of Tuberculosis.	0–15 Years.			Over 15 years.			All Ages.		
	Total deaths.	% bovine.	Bovine deaths.	Total deaths.	% bovine.	Bovine deaths.	Total deaths.	% bovine.	Bovine deaths.
Respiratory	480	—	—	23,490	—	—	23,970	1·4	336
Central Nervous System	1,280	26	333	516	17	88	1,796	—	421
Intestines and Peritoneum	218	—	—	458	—	—	676	82	554
Vertebral Column	22	—	—	362	—	—	384	25	96
Other Bones and Joints	22	—	—	163	—	—	185	21	39
Skin and Subcutaneous Tissues	0	50	0	62	13	8	62	—	8
Lymphatic System (Abdominal and Bronchial Glands excepted)	22	82	18	31	50	16	53	—	34
Genito-urinary	12	—	—	260	—	—	272	17	46
Other Organs	3	—	—	42	—	—	45	9	4
Disseminated	437	—	—	649	—	—	1,086	6	65
Total	2,496			26,033			28,529	5·6	**1,603**

TABLE VII

CALCULATED NUMBER OF DEATHS FROM TUBERCULOSIS IN SCOTLAND IN THE YEAR 1937 DUE TO INFECTION WITH THE BOVINE TYPE OF TUBERCLE BACILLUS.

Variety of Tuberculosis.	0–15 Years.			Over 15 years.			All Ages.		
	Total deaths.	% bovine.	Bovine deaths.	Total deaths.	% bovine.	Bovine deaths.	Total deaths.	% bovine.	Bovine deaths.
Respiratory	105	—	—	2,686	—	—	2,791	5·4	151
Central Nervous System	278	28	78	96	26	25	374	—	103
Intestines and Peritoneum	43	80	34	116	50	58	159	—	92
Vertebral Column / Other Bones and Joints	8	38	3	75	21	16	83	—	19
Skin and Subcutaneous Tissues	0	—	—	8	—	—	8	69	6
Lymphatic System (Abdominal and Bronchial Glands excepted)	2	62	1	8	6	0	10	—	1
Genito-urinary	1	54	1	33	18	6	34	—	7
Other Organs	1	—	—	4	—	—	5	65	3
Disseminated	79	—	—	120	—	—	199	11	22
Total	517			3,146			3,663	11·0	**404**

culosis is almost certainly too low. Following the practice of Cobbett it has been obtained by assuming that the proportion of bovine type infections in persons dying with disseminated lesions is the same as the average proportion for all other types. This is very misleading, since (i) it includes a vast number of patients dying from pulmonary tuberculosis which, even if they are also suffering from disseminated lesions, will not be described on the death certificate as " disseminated," and (ii) it includes a number of cases of non-fatal tuberculosis in which the " typing " has been carried out during life. It would be far more accurate to base the figure for " disseminated " lesions on the findings of non-pulmonary cases coming to autopsy. What this figure would be cannot be stated, but it would undoubtedly be very much higher than the present figure of 6 per cent. On the whole it is probable that the errors due to using too high a figure for " intestines and peritoneum " and too low a figure for " disseminated " tuberculosis more or less cancel out.

One further point may be noted. Owing to the fact that a great deal of the early work of the Royal Commission and of Dr. Stanley Griffith was carried out in the South of England, it was believed for a long time that pulmonary tuberculosis in man was rarely due to infection with the bovine type. The more recent work of Munro and Griffith (1928) and of other workers already quoted on p. 23 has shown that, though the frequency of tuberculosis of bovine origin is very low in the South of England, it becomes commoner as the North is approached, and in the North East of Scotland it accounts for nearly 10 per cent. of all cases of pulmonary tuberculosis (Table VIII).

TABLE VIII

FREQUENCY OF BOVINE TYPE OF TUBERCLE BACILLUS IN PULMONARY TUBERCULOSIS (MODIFIED FROM GRIFFITH 1937)

Country.	Region.	No. of Sputa examined.	No. of Bovine strains isolated.	Percentage Bovine.
Scotland	North East Rural	317	27	8·5
	North East Urban	263	13	5·0
	Middle and South	1,471	67	4·6
England	North	888	14	1·6
	Middle	1,033	14	1·4
	South	885	5	0·56
Wales	—	203	2	1·0

It would be unjustifiable to assume that all cases of pulmonary tuberculosis caused by the bovine type of bacillus were due to milk-borne infection. In Germany Lange (1931, 1932) has brought evidence to show that in farm-workers infection may result from the inhalation of tubercle bacilli expectorated by tuberculous cattle. Most of the work in this country, however, suggests that the pulmonary disease follows on glandular tuberculosis acquired through alimentary infection, usually in childhood.

(2) *Calculation based on the Proportion of non-pulmonary Infections of Human Origin.* So far as the author is aware, this method has not been used before. It is rendered possible only by the circumstance that in London about 98 per cent. of the milk supply is pasteurized or heat-treated in some other way. It therefore follows that practically all cases of non-pulmonary tuberculosis must be infected with the human type of bacillus. The only available source of infection is constituted by patients suffering from pulmonary tuberculosis who are coughing or expectorating tubercle bacilli. In other parts of the country, where pasteurization is practised to a much smaller extent, a certain proportion of the infections will be due to the human and a certain proportion to the bovine type of bacillus. To find out what proportion of non-pulmonary infections in the rest of the country are of bovine origin, the number of infections of human origin are calculated on the assumption that the risk of any individual contracting non-pulmonary tuberculosis will be dependent on the number of patients suffering from open pulmonary tuberculosis. Since, however, the risk of infection from this source will be determined also by the density and other environmental factors of the population, a correction has to be introduced so as to standardize this variable on the basis of the London figures.

Put in another way, the argument may be stated as follows. If in London, where there is no longer any source of bovine infection, so many cases of pulmonary tuberculosis give rise to so many cases of non-pulmonary tuberculosis, how many cases of non-pulmonary tuberculosis will result from a known number of cases of pulmonary tuberculosis in other parts of England and Wales ? This figure can be readily calculated. If it is found to be below the recorded number of cases, then it is assumed that the remainder have been infected from a non-human source, namely from cattle.

In practice, since the number of cases of tuberculosis is no longer recorded by the Registrar-General, the number of deaths has to be used instead.

Two errors must be guarded against in making the calculation. (i) Even if the death-rates from non-pulmonary tuberculosis were

identical in London and in other parts of the country, the number of deaths in populations of the same size would vary with differences in age distribution. If, for example, the death-rate from non-pulmonary tuberculosis in children under 15 was the same in the rural districts as in London, the mere circumstance that the proportion of children to the total population is greater in the rural districts than in London would result in the number of deaths from tuberculosis being greater in the rural districts, even if the total populations were of the same size. For this reason it is necessary to apply the London death-rates for both sexes and for different age groups to the actual number of persons living in these groups in other parts of England and Wales, and thus obtain the number of deaths that would have occurred from non-pulmonary tuberculosis in other parts of England and Wales if the London rates, which are determined solely by infections of human origin, had been in operation.

(ii) Since the death-rates from pulmonary tuberculosis differ considerably in different parts of the country, it must be assumed that factors, such as population density, nutrition, housing, type of work, and so on, which play a part in determining the incidence of infection and the length of survival of the individual patient, operate to an unequal extent in different areas. In London, for example, the standardized death-rate from pulmonary tuberculosis for males in the year 1937 was 75 per 100,000, while in rural districts it was only 41. The chances, therefore, of dying from tuberculosis in London are much greater than in country districts. If the same factors which favour the development of pulmonary tuberculosis in our large towns also favour the development of non-pulmonary tuberculosis, it follows that a given number of cases of pulmonary tuberculosis in London will give rise to a larger number of cases of non-pulmonary tuberculosis than in the rural parts of England and Wales. For this reason, in making our calculation, it is necessary to introduce what may be termed for short a " density factor."

An example may make this clear. In London in the year 1937, the proportion of pulmonary tuberculosis deaths to the total population was 1/1418, and of non-pulmonary tuberculosis 1/11,665. Applying the first rate to the population of the aggregate county boroughs, it is found that 9,397 deaths should have occurred in these districts. In fact, 9,621 deaths occurred, giving a proportion of pulmonary tuberculosis deaths to the total population of 1/1385. The density factor for the county boroughs is therefore $\frac{1385}{1418}$ or 0·9767. In other words, owing to the rather less favourable conditions, about 2·4 per cent. more deaths from pulmonary tuberculosis occurred in the

county boroughs than would have been expected at the London rate.

If the same effect is produced on the non-pulmonary tuberculosis death-rate, then the number of expected non-pulmonary tuberculosis deaths of human origin occurring in the county boroughs would be likewise 2·4 per cent. higher than those expected at the London rate, namely 1,212 × 1·024 or 1,241. The recorded number of deaths in the county boroughs from non-pulmonary tuberculosis for the year 1937 was 1,719. It must therefore be concluded that 1,719 minus 1,241 or 478 deaths were due to infection of bovine origin.

Unfortunately this method does not enable a calculation to be made of the number of deaths from pulmonary tuberculosis of bovine origin, so that to complete our picture it is necessary to take the figure obtained by the first method, which is based on the assumption that 1·4 per cent. of patients dying from pulmonary tuberculosis are infected with the bovine type (see Table VI). It may be objected that, if it is assumed in the second method that there is no longer any source of bovine infection in London, it is unfair to apply the figure of 1·4 per cent. to the total pulmonary tuberculosis deaths in England and Wales including London. The answer to this is that the figure of 1·4 per cent. is an average figure based on examinations of material from all parts of the country. If London was excluded, then the figure for bovine infections would be higher than 1·4 per cent., and approximately the same answer would be obtained.

The results of applying this second method are shown in Table IX.

It will be seen from this method that the total number of tuberculosis deaths of bovine origin in England and Wales in 1937 was 1,916. This figure is rather higher than that of 1,603 obtained by the first method of calculation. It is interesting to note, however, that in the year 1931 the figures given by the two methods were in close agreement, namely 2,207 by the second and 2,285 by the first.

Two objections may be raised to the second method. Firstly, it is unjustifiable to assume that all tuberculosis deaths occurring in London in 1937 were due to infection with the human type of bacillus. Some of these deaths may have occurred in patients who had been infected during infancy or early childhood with the bovine type, either in districts outside London or in London itself before practically the whole of the milk supply was heat-treated. This objection is no doubt partly true, but the proportion of deaths of bovine origin must be very low. If a calculation is made on the assumption that 5 per cent. of deaths from non-pulmonary tuberculosis in London in 1937 were due to infection with the bovine type, then the effect will be

TABLE IX

CALCULATED NUMBER OF DEATHS FROM TUBERCULOSIS IN ENGLAND AND WALES IN THE YEAR 1937 DUE TO INFECTION WITH THE BOVINE TYPE OF TUBERCLE BACILLUS (SECOND METHOD).

Variety of Tuberculosis.	Area.	Population.	Ratio P.T. deaths to population.	Density Factor.	Expected Deaths of human origin at London rates × density factor.	Recorded deaths.	Bovine deaths.
Non-Pulmonary (N.P.T.)	Administrative county of London	4,094,500	$\frac{1}{1418}$	1·0	351	351	0
	Aggregate county boroughs	13,326,110	$\frac{1}{1385}$	0·9767	1,241	1,719	478
	Aggregate urban districts	14,934,030	$\frac{1}{1919}$	1·3533	1,003	1,635	632
	Aggregate rural districts	7,936,660	$\frac{1}{2328}$	1·6417	442	912	470
	Total						1,580
Pulmonary (P.T.)		1·4 per cent. of 23,970 P.T. deaths in England and Wales					336
N.P.T. and P.T.	Total						**1,916**

Note.—Since the population of England and Wales outside the administrative county of London is not recorded for 1937, the figures used in this table are based on the population for 1931. The increase in the population between these two years has been credited to the aggregate urban districts, since a comparison of the populations outside Greater London in 1931 and 1937 shows that most of the increase occurred in these districts.

to raise the calculated non-pulmonary deaths of bovine origin in districts outside London by about 5 per cent. That is to say, the number given in Table IX is likely to err on the conservative side.

Secondly, the assumption that differing environmental conditions will have the same quantitative effect on the non-pulmonary as on the pulmonary tuberculosis death-rate may be seriously questioned. Unfortunately our knowledge of the total effect, as of the mode of operation, of the various factors that influence the tuberculosis death-rate is very meagre, and all we can do at present is to assume that conditions favouring survival or death in pulmonary tuberculosis have a similar, though not necessarily, equal effect on non-pulmonary tuberculosis.

It is clear that in both methods of calculation certain assumptions have to be made, and the results cannot be expected to be more than approximately correct. In spite of the fact that the methods themselves and the assumptions on which they are based, are entirely different, it is satisfactory to note that they give results which are in fairly close agreement with each other.

SUMMARY

1. Two entirely different methods, involving different assumptions, have been used to calculate the probable number of deaths from tuberculosis of bovine origin in England and Wales.

2. The first method is the one used by the Cattle Diseases Committee (Report 1934), and is based on the bacteriological examination of tuberculous material to ascertain the frequency of the bovine type of bacillus in different varieties of tuberculosis.

3. The second method, which has not hitherto been employed, makes use of the fact that in the administrative county of London, where practically all milk is now pasteurized or heat-treated in some other way, primary infection of bovine origin is no longer possible. All patients with non-pulmonary tuberculosis must therefore be infected from human sources, namely from cases of pulmonary tuberculosis. If, in London, a given number of cases of pulmonary tuberculosis gives rise to a given number of cases of non-pulmonary tuberculosis, it is easy to calculate how many cases of non-pulmonary tuberculosis should arise from pulmonary cases in the rest of England and Wales. If this number is found to be less than that of the recorded cases, it is concluded that the remainder were infected from bovine sources. In practice, deaths have had to be substituted for cases, since the Registrar-General no longer records the number of cases of tuberculosis.

4. The results obtained show that the probable number of deaths

in England and Wales from infection with the bovine type of tubercle bacillus was as follows :

	Method 1	*Method 2*
1931	2,285	2,207
1937	1,603	1,916

The results given by the two methods are sufficiently concordant to justify the conclusion that in 1937 somewhere between 1,500 and 2,000 deaths from tuberculosis of bovine origin occurred in England and Wales.

Undulant Fever. Undulant fever is a disease caused by *Br. abortus*, *Br. melitensis*, or *Br. suis*, organisms that are natural parasites of the cow, the goat, and the pig respectively. In Great Britain *Br. abortus* is the only one of these organisms that occurs indigenously.[1] As has already been pointed out (p. 8) it is responsible for the widespread disease in cattle known as contagious abortion. Infected animals may discharge the organism from the vagina at the time of abortion or normal parturition. If the udder is invaded, as it frequently is, *Br. abortus* may be excreted in the milk for weeks or months on end. Human beings may be infected either by direct contact with aborting or infected animals, as occurs in farmers, milkers, veterinarians, and slaughterers, or by ingestion of the milk, as with most of the urban and part of the rural population. The frequency of contact and of milk-borne infection varies in different countries. In Great Britain, where over 80 per cent. of the population lives in towns, the great majority of the cases appear to result from the consumption of raw milk or cream.

As with the tubercle bacillus, *Br. abortus* has a fairly high degree of infectivity, but only a low degree of pathogenicity. That is to say, a considerable proportion of persons exposed to risk become infected, as is demonstrable by immunological tests, but only a very small proportion develop overt disease. The clinical manifestations are extremely varied, and the disease can rarely be diagnosed with certainty without the aid of laboratory methods. Partly because the disease in this country was not recognized up to a few years ago, and is largely unknown to the older practitioners, partly because it is so protean in its symptomatology, and partly because it requires bacteriological investigation for its diagnosis, there is good reason to believe that the majority of the cases are overlooked or diagnosed wrongly. Most of the cases are sporadic, but when an institution or a semi-closed community is supplied with infected milk, multiple

[1] In 1940 a herd of cattle in Staffordshire was found to be infected with *Br. melitensis* ; two other herds in the Midlands have since been shown to be similarly infected.

cases of the disease may occur. Many minor outbreaks of this type have been described (King and Caldwell 1929, Sasano *et al.* 1931, Hasseltine and Knight 1931, Dooley 1932, Johns *et al.* 1932, Welch and Mickle 1933, Stone and Bogen 1935, v. Engel 1938, Elkington *et al.* 1940, Cruickshank and Stevenson 1942).

Except in one town the disease is not notifiable, and even if it were little reliance could be placed on the figures obtained, since so few of the cases are accurately diagnosed. The frequency of the disease can be estimated only by examination of the laboratory data. Where, as in Denmark, the general practitioner is educated to use the laboratory for routine diagnosis, and where all material is forwarded to a central laboratory, the records enable a close estimate of the annual number of cases of the disease to be made. But where, as in Great Britain, only a fraction of patients suffering from pyrexia of unknown origin are thoroughly investigated, and where multiple small laboratories exist, each with a different routine, it is more difficult to make a reliable estimate of this sort. Nevertheless analysis of the available information, based on the proportion of sera from pyrexial patients found to contain agglutinins to *Br. abortus* above a certain titre, indicates that the annual number of cases in England and Wales is of the order of 400–500 (Wilson 1932).

Before passing on to epidemic milk-borne disease, it may be as well to point out that neither *butter* nor *cheese* appears to play any considerable part in the causation of tuberculosis or undulant fever. Over 80 per cent. of butter used in this country is imported, and practically all imported butter is made from pasteurized cream. Of the butter manufactured in this country the greater part is made in factories from pasteurized cream, and only a very small proportion is made on farms from raw cream. In cheese both the tubercle bacillus and *Br. abortus* die out fairly quickly, and are usually dead before the ripening process is complete. Only in farm butter and in soft cheese, i.e. unripened cheese eaten within a few days of its preparation, are these organisms likely to be found alive. The vast majority of milk-borne tuberculosis and undulant fever is undoubtedly due to the consumption of raw milk and cream.

Epidemic Milk-borne Diseases.

Space does not permit of a description of each of the common epidemic milk-borne diseases, and the present discussion must be limited to a brief account of how the milk becomes infected and to an estimate of the extent of resulting disease. The mode of infection of the milk with specific micro-organisms has already been dealt

with in the previous chapter. In the present chapter the subject is approached from the viewpoint of the disease produced. This treatment involves a certain amount of overlapping, but since some diseases are caused by more than one type of organism, it is hoped that the repetition will be excused for the sake of clarity.

Scarlet Fever and septic sore Throat. These diseases, which may occur separately or together, are due to an organism known as *Str. pyogenes*. Milk may become directly infected with the cough spray from a patient suffering from the disease, as for example in two recently recorded outbreaks (Report 1938*a*, Henningsen and Ernst 1939). Alternatively the milker may infect the udder of one of the cows with the result that mastitis develops and the milk which is secreted becomes infective. This indirect method of infection of the milk through the cow, though strongly suspected of occurring by different workers since the end of last century, has only recently received definitive proof through the work mainly of Bendixen in Denmark and Minett in this country (see Bendixen and Minett 1938).

There is at present some doubt as to which is the commoner method of infection. Since contamination of milk from a human source must be slight and occasional, and since hæmolytic streptococci do not appear to multiply in milk outside the body at ordinary atmospheric temperatures (Pullinger and Kemp 1937), this mode of infection seems more likely to give rise to small outbreaks or sporadic cases than to large explosive epidemics in which several hundred people are affected. On the other hand, if the cow's udder is invaded, the milk secreted may contain enormous numbers of hæmolytic streptococci, capable of contaminating a considerable quantity of bulked milk. Pullinger and Kemp are therefore of the opinion that probably all large outbreaks of scarlet fever and septic sore throat are due to the milk coming from a cow whose udder is infected with the specific human type of hæmolytic streptococcus. Minett (1937) holds the same view.

Diphtheria. The diphtheria bacillus, like *Str. pyogenes*, is a natural human parasite and lives in the nasopharynx. Milk may become infected (i) directly through the cough spray of a patient suffering from the disease, or from a healthy carrier, or (ii) indirectly through contamination of the milk from an ulcerated teat on which the bacillus has become implanted. As in scarlet fever, the available evidence suggests that milk contaminated by the former method is responsible mainly for small outbreaks and sporadic cases of the disease, while larger outbreaks are generally due to milk derived from an infected cow. The diphtheria bacillus is not an easy organism to demonstrate in milk, but there are, nevertheless, a number of well-authenticated

instances in which this has been achieved (Bowhill 1898–1899, Eyre 1899, Klein 1901, Dean and Todd 1902, Marshall 1907, McSweeney and Morgan 1928).

Enteric Fever, Dysentery and Cholera. These diseases may be taken together, since they are all caused by organisms whose primary habitat is the human intestine. Cholera, it may be remarked, no longer occurs in this country, and need not be specially considered here. The organisms may gain direct access to the milk from the imperfectly cleansed fingers of a healthy carrier or patient. This is a very common method. Or the organisms may be discharged in the fæces and contaminate a water supply which is used for dairy purposes, as in the large outbreak of typhoid fever at Bournemouth, Poole, and Christchurch in 1936 (Shaw 1937).

Food Poisoning. Epidemics of food poisoning traced to milk are generally found to be due to a member of the Salmonella group of bacteria, particularly *Bact. typhi-murium*, *Bact. enteritidis*, and *Bact. enteritidis* var. *dublin*. The first two organisms are responsible for rodent typhoid and are widespread in rats and mice. They give rise in cattle and pigs to enteric and other types of disease, and in man to acute gastro-enteritis. The third organism is most frequently met with in calves, in which it gives rise to diarrhœa; but sometimes it affects cows and is excreted in the milk (Tulloch 1939). Milk may be infected (i) from human cases or carriers, as in enteric fever; (ii) from the fæces of milking cows or other animals in the herd, as in the recent Wilton outbreak (Conybeare and Thornton 1938); (iii) from a small focus of infection in the udder (Poppe 1931); or (iv) possibly from the urine and fæces of infected rodents in the dairy.

A certain number of outbreaks of acute gastro-enteritis are due to staphylococcal or other forms of bacterial intoxication (see p. 38).

As has already been pointed out (p. 20) there are several reasons why our knowledge of epidemic milk-borne disease is deficient. It is important, however, to review what information we have in the form of published records. Savage (1912) collected the data that were available up to 1912. The number of outbreaks reported since then in Great Britain are summarized in Table X.

It is to be noted that the figures in Table X refer only to outbreaks traced to liquid milk or cream, and do not include the numerous outbreaks for which ice-cream, cream buns, or cheese have been responsible. Nor do they include any of the so-called toxin types of food-poisoning outbreaks which have followed the consumption of raw milk in schools (Report 1929, 1935*b*, 1936*g*), since complete proof of the bacterial nature of the toxins present was not forthcoming.

Some of the recent epidemics included in the table were very large,

TABLE X

RECORDED OUTBREAKS OF MILK-BORNE DISEASE IN GREAT BRITAIN IN 1912–1937 (EXCLUDING TUBERCULOSIS AND UNDULANT FEVER, AND OUTBREAKS DUE TO MILK PRODUCTS SUCH AS ICE CREAM, CHEESE, AND CREAM BUNS)

Disease.	No. of Outbreaks.	No. of Outbreaks in which no. of Persons affected is stated.	No. of Persons affected in these Outbreaks.
Scarlet Fever and septic sore Throat	40	34	5,331
Diphtheria	20	18	773
Typhoid Fever, Paratyphoid Fever, and Dysentery.	39	36	3,229
Gastro-enteritis	14	12	4,346
Total	113	100	13,679

such as that of the food-poisoning outbreak at Dundee in 1926, due to *Bact. enteritidis* var. *dublin*, in which there were at least 373 known cases (Tulloch 1939), that of a similar outbreak at Dundee in 1927 due to the same organism affecting about 280 persons (Tulloch 1939), that of septic sore throat at Brighton and Hove in 1929, in which 1,000 families were affected with 65 deaths (Wilkinson 1931), that of paratyphoid fever at Epping in 1931 with 260 cases, that of sore throat at Chelmsford in 1935 involving about 1,600 persons (Camps and Wood 1936), that of scarlet fever and septic sore throat at Doncaster in 1936 with 364 cases (Watson R. 1937), that of typhoid fever at Bournemouth, Poole, and Christchurch in 1936, involving at least 718 persons and causing about 70 deaths (Shaw 1937), and that of dysentery at St. Andrews in 1937 affecting about 300 persons (Report 1937*c*).

Lest the experience of this country should be regarded as abnormal, it may be noted that records are available of 760 outbreaks of milk-borne disease, excluding gastro-enteritis, in the United States during the years 1910 to 1933 inclusive (Borman, West, and Mickle 1935). These figures almost certainly underestimate the real incidence for, as the result of periodic questionnaire surveys by the Office of Milk Investigations of the Public Health Service, it was learned that 639 outbreaks occurred during the years 1923 to 1937 alone, that is an incidence of 42·6 a year (Frank 1940). This, of course, does not mean that milk-borne disease was more frequent in the latter period, but that an annual questionnaire survey of health authorities yields much more information than does an occasional survey of the literature.

In Canada there are records of 63 milk-borne outbreaks during the years 1912 to 1937 (Defries 1938).

Besides the diseases considered here, other types of infection may be borne by milk, though for one reason or another they rarely give rise to more than sporadic cases. Foot-and-mouth disease, for example, has frequently been carried to man by milk. In the 1920–1923 epidemic in Germany numerous cases of human infection occurred, and the disease in children was often very severe (Poppe 1931). Other examples are anthrax and cowpox. Two fairly large milk-borne outbreaks of rat-bite fever are also on record (Parker and Hudson 1926, Place and Sutton 1934). The milk of cattle infected with rabies, blackleg, rinderpest, tetanus, and severe mastitis is to be regarded as unfit for human consumption.

Staphylococcal Intoxication. This condition was first described by Barber (1914) in the Philippines. It is due to a staphylococcus which usually seems to be derived from the cow, and which is capable, if allowed to multiply in the milk to a sufficient extent, of forming a powerful relatively heat-resistant toxin. When ingested by the mouth, the toxin gives rise within two hours to acute nausea, vomiting, and diarrhœa, followed by extreme prostration. Sometimes, as in Barber's original case and in the outbreaks described by Crabtree and Litterer (1934), the toxin is formed in the milk itself. More usually, however, staphylococcal intoxication of this type is met with in food poisoning due to the consumption of foods, such as cream buns, layer cakes, and custards that have been made up with milk or cream.

It is to be noted that this condition is due to the ingestion of pre-formed toxin, not, as in the diseases so far considered, to the growth of the organism in the patient's tissues. The condition is one of intoxication, not of true infection. Since no satisfactory method of demonstrating the toxin in the milk has been available up till quite recently, the real extent of this disease is unknown. Some, at least, of the outbreaks of acute vomiting with rapid recovery which have been observed in the past following the consumption of particular batches of milk were probably due to this form of intoxication (Scott 1937).

Other Forms of Bacterial Intoxication. Milk from cows suffering from mastitis sometimes gives rise to acute gastro-enteritis coming on within 4 or 5 hours. Brooks (1932), for example, reports 6 outbreaks of this type in New York State alone during the years 1924 to 1931, affecting 357 persons. The ætiology of this condition is still obscure. Whether hæmolytic or non-hæmolytic streptococci or other organisms are responsible for rendering the milk toxic is not known. A great deal of work will be necessary before the mechanism

of toxin formation and the nature of the toxin itself are understood. There is reason to believe that certain non-pathogenic Gram-negative organisms, if allowed to proliferate unduly in milk, may give rise to gastro-intestinal disturbance by virtue of toxic substances, possibly such as those that have been extracted by Boivin and Mesrobeanu (1933–1935) in France, and Raistrick and Topley (1934) in this country. The whole question of specific and non-specific toxin formation in milk and cream awaits investigation.

Summer Diarrhœa. Summer diarrhœa is a disease which in the past has been responsible for an appalling mortality among infants and very young children. It occurs mainly in the months of July, August, and September, and is greatly influenced by temperature. In the two hot summers of 1911 and 1921, for instance, the diarrhœa and enteritis mortality rates for children under 2 years of age per 1,000 live births for the third quarter of the year in the Administrative County of London were 153·6 and 53·0 respectively, whereas the corresponding rates for the cool summers of 1912 and 1922 were 23·4 and 5·3. Since 1921 no major epidemic has occurred. The death-rate has fallen considerably, and during the last 10 years or so it has become relatively stabilized at about 12 per 1,000 (Table XI).

TABLE XI

(*a*) DIARRHŒA AND ENTERITIS MORTALITY RATE FOR CHILDREN UNDER 2 YEARS OF AGE PER 1,000 LIVE BIRTHS IN THE THIRD QUARTER OF THE YEAR IN THE ADMINISTRATIVE COUNTY OF LONDON, AND (*b*) INFANTILE MORTALITY RATE FOR CHILDREN UNDER 1 YEAR OF AGE PER 1,000 LIVE BIRTHS IN THE SAME PERIOD AND DISTRICT

Years.	Live births.	Mean Diarrhœa and Enteritis death-rate.	Mean Infant Mortality rate.
1911–1915 . . .	136,879	72·0	128·5
1916–1920 . . .	112,232	27·1	80·5
1921–1925 . . .	115,787	21·2	61·7
1926–1930 . . .	91,503	12·2	48·1
1931–1935 . . .	74,669	11·4	47·7
1936	14,103	12·7	46·0
1937	13,612	12·3	46·0

The exact ætiology of summer diarrhœa is still obscure, but there are strong reasons for believing that milk plays an important part in its causation. Milk may become infected, either through human agency or through flies, with known pathogenic organisms such as the *shigae*, *flexneri*, and *sonnei* species of the dysentery bacilli, or

with potentially pathogenic organisms such as *Proteus vulgaris* and *Pr. morgani*. Sometimes organisms of the Salmonella group are incriminated.

In many instances, however, no pathogenic organisms can be found, but instead there is a profuse growth in the milk of organisms that are usually regarded as non-pathogenic, such as coliform bacilli, streptococci, and micrococci. As long ago as 1903 Park and Holt brought evidence to show that milk containing large numbers of these organisms, i.e. bacteriologically dirty milk, was liable to give rise to gastro-intestinal disturbance in infants. Since then, particularly during recent years, there has been an increasing volume of evidence to show that milk, or indeed any food, in which undue bacterial proliferation has occurred, is liable to give rise to food poisoning or diarrhœa, even in the complete absence of any known pathogenic organisms (see Topley and Wilson 1936).

The great decline of epidemic summer diarrhœa that has occurred in this country during the past twenty years or so is largely attributable to improvements in the cleanliness of the milk supply, to the partial replacement of loose by bottled milk, to the substitution of pasteurized, boiled, or dried for raw milk in infant feeding, and to the virtual abolition of flies in our large towns as a result of the supplanting of horse by motor traffic. The bottling of milk has greatly diminished the risk of contamination from flies in the house, and the almost universal practice in infant welfare centres of giving milk that has been heated in some way or other ensures that the enormous bacterial contamination that used to be so common now no longer occurs.

The common milk-borne diseases of bacterial origin, together with the way in which the milk becomes infected, are summarized in Table XII (pages 42 and 43).

Other Types of Milk-borne Disease. Various other diseases have been traced to milk in which bacteria are neither directly nor indirectly involved.

Nausea and vomiting not infrequently occur, either in sporadic or in epidemic form, after drinking milk. Recent examples are the outbreaks among children receiving milk in three schools at Melksham in 1936 in which 87 children were affected, and in a school camp in South Wales in the same year (Pole 1936). It has hitherto been impossible in instances of this sort to exclude the occurrence of toxin formation in the milk by staphylococci or other organisms. It is known, however, that *milk sickness* may be due to toxic substances in the milk derived from certain plants eaten by the cow, such as the white snakeroot or richweed (*Eupatorium urticæfolium*) and the rayless golden-rod (*Aplopappus heterophyllus*). The disease was not

uncommon among the early settlers in the United States, and Abraham Lincoln's mother is said to have died of it (Jordan 1931).

Milk Allergy. Another disturbance to which milk may give rise is that of milk allergy. Here the fault lies, not in the milk, but in the patient, whose tissues appear to be hypersensitive to certain of the milk proteins. The condition is not uncommon, and on account of the gastric and hepatic disturbances to which it gives rise is sometimes responsible for the dislike that many persons express for milk. Williams (1936), who inquired into the histories of 150 elementary schoolchildren who had refused milk at school, obtained evidence that 124, or 83 per cent. of them were probably suffering from allergy.

(For a review of milk-borne diseases, see Scott 1935, 1937 ; Smith 1939).

SUMMARY (For Sub-sectional summary see p. 32)

1. Reasons are advanced for believing that our knowledge of milk-borne disease is very incomplete, and that the recorded cases of disease grossly underestimate the real number.

2. Two different methods have been used for calculating the number of tuberculosis deaths of bovine origin in England and Wales. They show that this figure was somewhere between 1,500 and 2,000 in the year 1937.

3. About 400 to 500 cases of undulant fever are estimated to occur annually in England and Wales, the great majority of which are due to milk-borne infection.

4. The available evidence suggests that neither butter nor cheese is of any considerable importance in the causation of milk-borne tuberculosis or undulant fever, nearly all of which is attributable to infected raw milk or cream.

5. Actual records are available of 113 outbreaks of epidemic milk-borne disease in Great Britain between the years 1912 and 1937, affecting about 14,000 persons. The diseases include scarlet fever and septic sore throat, diphtheria, typhoid fever, paratyphoid fever, dysentery, and acute gastro-enteritis. The various ways in which the milk may become infected with the causative organisms of these diseases are briefly indicated.

6. The condition of staphylococcal intoxication, due to milk or cream in which staphylococci have grown outside the body and produced a specific toxin, is described. Since no satisfactory method of demonstrating the toxin in the milk has been available up till quite recently, the extent of this disease is unknown.

7. Milk from cows suffering from mastitis, and probably milk from other sources in which undue bacterial proliferation has occurred,

TABLE XII

INDICATING THE MORE COMMON TYPES OF MILK-BORNE DISEASE OF BACTERIAL ORIGIN AND THE WAY IN WHICH THE MILK BECOMES INFECTED

Bacterium.	Infections.		Mode of infection of the Milk.
	Disease caused in Animal.	Disease caused in Man.	
Mycobacterium tuberculosis (bovine type)	Tuberculosis in cattle	Tuberculosis.	Mainly from infected udder of the cow ; occasional organisms may gain access from cow's fæces.
Brucella abortus	Contagious abortion in cattle.	Undulant fever (often atypical)	From infected udder of the cow.
Streptococcus pyogenes	Very occasionally mastitis in the cow.	Septic sore throat or scarlet fever.	(1) From cough-spray of the milker or other person handling the milk. (2) From udder of the cow infected from a human source. Evidence suggests that large epidemics are mainly due to milk infected by the second route.
Corynebacterium diphtheriæ	Very occasionally found on ulcerated teats of cows.	Diphtheria.	(1) From the cough-spray of the milker or other person handling the milk. (2) From ulcerated teat of a cow infected from a human source. Evidence suggests that large epidemics are mainly due to milk infected by the second route.
Bacterium enteritidis, Bact. typhi-murium and certain other members of Salmonella group	Enteritis and septicæmic diseases, especially in older cattle. Rodent typhoid in rats and mice.	Acute gastro-enteritis (food poisoning).	(1) From fæces of infected cattle. (2) Occasionally from diseased udder of a cow. (3) Possibly from urine and fæces of rats and mice. (4) Possibly from fingers of a human case or carrier.

Bact. enteritidis var. *dublin*	Enteritis, especially in calves (calf diarrhœa).	Acute gastro-enteritis or paratyphoid-like fever.	(1) From fæces of infected cattle. (2) Possibly from fingers of a human case or carrier.
Bact. typhosum	Probably non-infective to cows.	Typhoid fever.	(1) From imperfectly cleansed fingers of typhoid case or carrier. (2) From contaminated water used for cleaning milk utensils or for watering the cows or for watering the milk.
Bact. paratyphosum B	Probably non-infective to cows.	Paratyphoid fever.	As with the typhoid bacillus.
Bact. flexneri, *Bact. sonnei*, *Bact. shigæ*, and certain other members of dysentery group.	Probably non-infective to cows.	Dysentery, diarrhœa, or sometimes acute gastro-enteritis.	As with the typhoid bacillus.
Streptobacillus moniliformis	May be identical with *Actinobacillus actinoides*, which gives rise to broncho-pneumonia in calves. Common nasal parasite of rats.	Rat-bite fever.	Probably from lungs of cattle or nose of rats.
	Intoxications.		
Staphylococcus aureus and possibly certain other organisms	Mastitis in the cow.	Acute gastro-enteritis (rapid onset) due to specific toxin formed in the milk before ingestion.	(1) From infected udder of the cow. (2) From nose or fingers of human carrier.
Various non-pathogenic organisms	Usually none.	Gastro-enteritis or diarrhœa due to non-specific toxic substances formed in the milk before ingestion.	Any cause of bacteriologically dirty milk associated with a warm temperature favouring prodigious bacterial multiplication.

may likewise give rise to acute gastro-enteritis. The ætiology of this condition is still imperfectly understood.

8. There are strong reasons for believing that epidemic summer diarrhœa in infants and young children is attributable in large part to bacteriologically dirty or infected milk. Owing to a combination of factors such as improvements in the cleanliness of milk, the partial replacement of loose by bottled milk, the substitution of pasteurized, boiled, or dried for raw milk in infant feeding, and the virtual abolition of flies in our large towns as a result of the supplanting of horse by motor traffic, the death-rate from this disease has diminished greatly during the past twenty or thirty years. In the first two decades of this century the loss of life it caused was enormous. In the hot year of 1911, for instance, it was responsible for something like 20,000 deaths in England and Wales.

9. Other milk-borne diseases, such as certain types of epidemic nausea and vomiting and milk allergy, in which bacteria play no part, are briefly described.

10. Taking milk-borne disease as a whole, during the years 1912 to 1937 there were at least 113 outbreaks of epidemic disease in Great Britain affecting about 14,000 persons. During the same time about 65,000 persons in England and Wales died of tuberculosis of bovine origin (assuming that 6 per cent. of all tuberculosis deaths were due to this cause), while an unascertained number, probably several thousand, suffered from undulant fever contracted through milk. If epidemic summer diarrhœa in infants under two years of age is included, some of the deaths during this period, which amounted in England and Wales to about 190,000, must be added to this total.

11. The evidence adduced here is believed to justify the statement that raw milk is probably the most dangerous article in our dietary.

CHAPTER IV

METHODS OF RENDERING MILK SAFE

Before discussing the various possible methods of rendering milk safe for human consumption, it is necessary to define our terms, and in particular to point out the difference between cleanliness and safety in relation to milk.

Cleanliness and Safety in Relation to Milk. We cannot do better here than to quote from the League of Nations Report (1937*b*):

" By ' cleanliness ' is generally understood the freedom of the milk from extraneous matter, such as manure and dust, from blood, and from an undue number of leucocytes and bacteria. It is an unsatisfactory term and refers to contamination of different types. It fails to distinguish between an initially dirty milk, and a milk which, though produced under good conditions, nevertheless contains large numbers of bacteria as the result of imperfect cooling. If the term is used in special relation to bacterial contamination, rather than to contamination with gross dirt, pus or blood, it may be suitably qualified, and the term ' bacterial cleanliness ' employed.

" By ' safety ' is meant the freedom of the milk from bacteria capable of giving rise to disease in man or animals.

" ' Cleanliness ' and ' safety ' in milk are two entirely different properties, and it is well to make this clear from the outset. A clean milk may be dangerous, and a dirty milk may be safe. Several outbreaks of disease in Europe and the United States have been traced to milk produced under exceptionally clean conditions. Two of the most striking recent instances are the Brighton-Hove outbreak of septic sore throat in 1929, and the Chilgrove outbreak of scarlet fever in 1934. In both instances, the incriminated milk supply was derived from farms where the greatest attention was paid to cleanliness. The producer of the Chilgrove milk had actually won the cup in the county clean-milk competition.

" The reason for the dissociation of cleanliness from safety is not far to seek. The organisms responsible for dirty milk come mainly from unsterilized milk utensils, mud and manure on the cow's udder, and dirt on the hands of the milker. On the other hand, the organisms responsible for dangerous milk come sometimes from the diseased udder of the cow, sometimes from human cases or carriers of infectious disease, and sometimes from a contaminated water supply. Milk may be produced under the most cleanly conditions, the udders may be

groomed, the milker's hands may be washed, all utensils may be sterilized by steam, and the resulting milk may contain only a very small number of bacteria; yet if the udder of one of the cows is diseased, or the milker is a carrier of hæmolytic streptococci or of enteric bacilli, or the water used on the premises is infected with typhoid bacilli, then pathogenic organisms may gain access to the milk, and the most serious consequences may follow.

" Not only are the organisms responsible for dirty milk qualitatively different from those responsible for dangerous milk, but there seems to be in fact some degree of antagonism between the two groups. That is to say, the presence of saprophytic organisms in milk is antagonistic to the development of pathogenic organisms (see for example Kliewe and Eldracher, 1935). Put in another way, pathogenic organisms have a better chance of survival in clean than in dirty milk."

As Sir Arthur MacNalty (1934), the late Chief Medical Officer of the Ministry of Health, has pointed out, " cleanliness " and " safety " in milk are two entirely different attributes, which may be associated together, or may be entirely dissociated from each other. Both are desirable. Clean milk is æsthetically more pleasing, it has a better flavour, and it keeps longer. Moreover, it is not likely to contain any of those toxic substances resulting from undue bacterial proliferation, which have an irritating effect on the gastro-intestinal tract—particularly of infants (see pp. 38, 39). The fewer organisms there are in milk, and the more bacterial proliferation is checked, the less liable is the milk to give rise to digestive disturbances of this type.

Methods of increasing the Safety of the Milk Supply

This book is concerned primarily with the safety of the milk supply, and there is therefore no need to describe the methods of production of clean milk. (For these see Report 1937*b*).

The methods of producing safe milk fall into three categories: (1) those concerned with the elimination of infectious disease from the animals; (2) those concerned with the elimination of infectious disease from the human personnel; and (3) those concerned with the destruction of pathogenic organisms by heat treatment of the milk. These may be dealt with in turn.

Control of Animal Disease

As has already been pointed out in Chapter II, the three most important diseases of cattle that affect the milk supply deleteriously are tuberculosis, contagious abortion, and mastitis. All of them are widespread in this country and all of them present the greatest diffi-

culties in control. The various ways in which attempts are being made to reduce their incidence can be briefly described.

Tuberculosis. Hitherto in this country chief reliance in the control of tuberculosis among cattle has been placed on the Tuberculosis Order of the Ministry of Agriculture and Fisheries. This Order was first issued in 1913, but did not come into operation till 1925. Certain alterations have been made in the most recent issue of 1938. Its main object is to secure the destruction of every cow found to be suffering from udder tuberculosis or to be secreting tuberculous milk, and of all cattle suffering from chronic cough and showing definite clinical signs of tuberculosis. The detection of these animals and their notification to the local authority rest on the owner and on private veterinary practitioners. On receiving a notification, the local authority informs the Divisional Officer, Ministry of Agriculture, who thereupon sends a veterinary inspector to examine the affected animal and to take such specimens as are necessary to confirm the diagnosis. Animals condemned by the veterinary inspector are slaughtered, and compensation is paid to the owner.

While not denying that this Order contains some useful provisions, it must be admitted that in practice it has failed to bring about any significant reduction in the proportion of cattle infected with tuberculosis. The Order assumes that the elimination of open cases of tuberculosis should be sufficient to control the disease. This may be true, but the real weakness of the assumption lies in supposing that open cases of tuberculosis can be detected in a sufficiently early stage to prevent the spread of infection to other animals in the herd. The difficulties of diagnosing open tuberculosis in the cow are, in fact, very considerable, and many weeks or months may elapse before a definite decision can be reached. During this time, if the animal has really been suffering from open tuberculosis, it has been excreting virulent tubercle bacilli either by the cough-spray, the milk, the fæces, or by some other channel, and has endangered the health of any normal animals in its vicinity. An attempt to prevent open tuberculosis by the elimination of open cases is, in practice, like shutting the stable-door after the horse has bolted.

It may be argued that, if the Tuberculosis Order was thoroughly implemented and if a sufficient number of properly trained veterinary inspectors were appointed who made periodical examinations of all the cattle within their area, it should be possible to reduce very considerably the number of cows suffering from udder tuberculosis and secreting tuberculous milk. This may be so, but there is no reason to believe that any substantial check would be put on the actual spread of infection. Diseased animals might be eliminated earlier, but their

brethren whom they had already infected would soon replace them to develop disease in their turn. Even the establishment of a State Veterinary Service under Part IV of the Agriculture Act, 1937, which takes over from the local authorities the duties of inspecting animals, is not likely to make any substantial alteration in this position. This is not to decry the value of veterinary inspection as such. In its educative aspects and in the diagnosis of clinically manifest disease it serves a very useful function; but the detection and eradication of latent and sub-clinical infection is not within its compass.

The fact that the proportion of milk samples found to contain tubercle bacilli has shown no apparent reduction during the twelve years following the introduction of the Tuberculosis Order (see Table IV, p. 14) merely confirms in practice what might have been expected on theoretical grounds. Even when veterinary inspection is carried out four times a year, as in the herds producing Accredited Milk, it is doubtful whether any real improvement has occurred in the proportion either of infected animals or of cows suffering from udder tuberculosis. Table V (p. 15), shows that the proportion of samples of milk entering London in 1937 found to obtain tubercle bacilli was actually higher in Accredited than in ungraded milk.

No method depending on the clinical detection of infected animals with their subsequent removal from the herd has so far been successful anywhere in preventing the spread of infection. Most countries that are interested in the control of tuberculosis in cattle have realized this, and have already adopted, or are in process of adopting, an eradication programme based on the use of the tuberculin test. This method involves the testing of every animal in the herd with tuberculin, the elimination of reactors, and the breeding of young stock from the non-reacting animals. Various modifications of this method are available, suited to different countries and different economic conditions, but they all agree in relying on (i) the tuberculin test for the detection of infected animals, and (ii) the segregation or removal of all infected animals, whether clinically diseased or not. That is to say, not only open cases, which are actually discharging tubercle bacilli into the outside world, but all infected animals which at some later stage may become open cases, are removed. The potentially dangerous, as well as the actually dangerous animal is got rid of, and further infection completely prevented from occurring.

The use of this method on a purely voluntary basis received its first official sanction in this country with the introduction by the Ministry of Health in 1922 of the Milk (Special Designations) Order laying down two grades of milk—Certified and Grade A Tuberculin Tested—which could be supplied under licence by producers who

undertook to test their animals with tuberculin at intervals of not less than two and not more than six months, to eliminate positive reactors, and to introduce into the herd only such animals as had already passed the tuberculin test. Since the cost of establishing tuberculin tested herds was very considerable, estimated at £13 per animal (Dixey 1937); since the owners of such herds had to pay a licensing fee for the privilege of producing milk free from tubercle bacilli; since they were subjected to a degree of official control from which the producers of ungraded milk were exempt; and since the members of the public who were prepared to pay extra for the milk so produced were numerically small, this scheme achieved only a very limited success. It is doubtful, whether by 1935, as much as 1 per cent. of the total liquid milk supply of the country was derived from tuberculin tested herds.

More recently, the Ministry of Agriculture and Fisheries have introduced their Tuberculosis (Attested Herds) Scheme, (England and Wales) 1937, designed to encourage the increase in tuberculin tested herds. There is no need to discuss the provisions of this scheme in detail, which have since been extended under Section 20 of the Agriculture Act, 1937, and by the Tuberculosis (Attested Herds) Scheme, 1938, made under Section 31 of the same Act. Briefly, powers are now available for assisting the voluntary attestation of herds on the basis of the tuberculin test; for the establishment of eradication areas when a substantial majority of the cattle in any given area are free from infection; and for the compulsory testing of non-attested herds in such an area with the destruction of any reactors found. Any area in which these measures have been taken may be declared an attested area, and the movement of cattle may be controlled so as to prevent the introduction of infection from outside. With the financial aid of the Ministry in the establishment of the herds and financial remuneration from the Milk Marketing Board at the rate of 1*d.* per gallon for the milk produced in addition to the 1*d.* per gallon that could be earned for milk reaching a certain degree of cleanliness, no fewer than 4,633 Attested Herds had been established in England and Wales by the end of March 1939. This figure represents about 3 per cent. of the dairy farmers in the country.

This is very promising, and everyone interested in the eradication of tuberculosis will wish success to this scheme. Nevertheless, with 40 per cent. of our cattle population infected with tuberculosis, it would be foolish to close one's eyes to the magnitude of the problem. The establishment of tuberculin tested herds is a costly proceeding, and the risk of failure, even after the apparently successful eradication of infected animals, is considerable. If entrance into the scheme was made compulsory, and if the Government were prepared to spend

several million pounds in direct and indirect financial assistance to the owners, progress would obviously be more rapid; but even the United States, in which an eradication programme under joint Federal and State supervision was inaugurated in 1917, has taken about twenty years to produce sufficient Tuberculin Tested milk to supply the needs of its population (Fuchs and Frank 1938). In this country the difficulties of prosecuting a successful eradication scheme are far greater than in the United States. The proportion of tuberculin positive animals is much higher than it was in the U.S.A. at the start of their campaign, the cattle population is much denser, the demands of the milk market favour the maintenance of "flying" herds, and any attempt to interfere with the long established custom of holding cattle markets in our country towns, which greatly favour cross-infection, would be resented. Moreover, few local authorities would dare to introduce the radical measures of milk control that have proved so successful on the other side of the Atlantic.

Unfortunately, owing to the war, the operation of the Attested Herds Scheme has had to be severely curtailed, and practically no new herds are now being accepted for official attestation. On the other hand, the exigencies of the food situation have led to considerable modifications in the normal movements of cattle. There is little trading in the markets, and most cattle leave the farms for slaughter only, and are purchased by the Ministry of Food at their collecting centres. In addition, 5 per cent of the cattle, comprising mainly the diseased and unthrifty, have been culled.

Reviewing the situation, however, we shall not be unduly pessimistic if we conclude that there is no prospect of supplying the whole population of this country with Tuberculin Tested milk for many years to come. Even if there were, the milk problem, as presented to the public health authorities, would be only partly solved. Though milk from tuberculin tested herds is usually free from the risk of carrying tuberculosis, it is just as likely as ordinary milk to be infected with other types of pathogenic micro-organisms. As Pullinger (1934, 1935) has shown (see pp. 16, 17), Tuberculin Tested milk is frequently infected with *Br. abortus* and with the streptococci of mastitis. So far as danger of contamination from human sources is concerned, there is no reason to suppose that the incidence of sore throats, scarlet fever, enteric fever, or dysentery is any lower among the personnel on farms producing Tuberculin Tested milk than on farms producing ordinary milk. Several outbreaks of scarlet fever, typhoid fever, and other diseases have been traced to Tuberculin Tested milk (Brooks 1930, Leeder 1932, Defries 1938, Harding 1939).

However satisfactory, therefore, the eradication of tuberculosis may

be to the agriculturist, it alone cannot solve the problem of providing the public with a safe milk supply.

Contagious Abortion. The methods of controlling contagious abortion are similar in principle to those for controlling tuberculosis. An attempt may be made by bacteriological methods to detect animals excreting *Br. abortus* in their milk, but this method is very slow and can hold out no hope of preventing the spread of infection. The only satisfactory method is complete eradication. All animals in an infected herd, whether they have aborted or not, are tested at frequent intervals for the presence of specific agglutinins in the blood, reacting animals are segregated or preferably eliminated, and young stock are raised exclusively from the non-reactors. Experience in Great Britain and in the United States shows that this method is usually successful provided the herd is small and self-contained, the proportion of reactors is not too high, re-infection from outside sources can be adequately guarded against, and blood tests are made at frequent intervals, such as two months, in the early stages of the process, so as to detect and remove freshly infected animals as rapidly as possible. Complete eradication from large herds, especially if the disease is very active, presents considerable difficulties, though there is reason to believe that success can be achieved in the long run if the process of eradication is conscientiously persisted in.

In Great Britain comparatively few farmers have adopted this method. Most of them allow the disease to run its course, or attempt to control its more flagrant clinical manifestations by the use of vaccines. No vaccine, however, has yet been devised that is capable of affording protection against *infection*. The wisdom of injecting living vaccines of uncontrolled virulence into herds supplying raw milk for human consumption is open to question, and the Ministry of Health has so far forbidden their use to producers of Tuberculin Tested milk.[1] There are, however, reasons for believing that the inoculation of calves or non-pregnant heifers with a living vaccine of suitably modified virulence may lead to a considerable reduction in the incidence of abortion and therefore of the total amount of infection. The use of such a vaccine, prepared from a specified and carefully controlled strain of *Br. abortus*, might well lead to a diminution in the degree of infection of the milk, and might justifiably be recommended for herds that are already infected, whether tuberculin tested or not.[1]

Mastitis. There is a considerable volume of evidence to show that in mastitis, as in tuberculosis and contagious abortion, the eradication method affords the only satisfactory means of eliminating the disease.

[1] Under the Milk (Special Designations) Regulations, 1942, herds producing Tuberculin Tested milk may now be inoculated with a living *Br. abortus* vaccine, provided the vaccine is approved by the Minister of Agriculture and Fisheries.

Frequent examinations of the milk from individual quarters of the udder for streptococci or other organisms capable of causing mastitis are required, followed by the elimination of badly infected animals. Injuries to the teats and udder must be avoided, hygienic methods must be employed in the milking shed, and healthy animals must be protected against infection by milking them first in the line. In practice, however, great difficulties are experienced, and many failures occur. For this reason increasing attention is being paid to the use of chemotherapeutic measures such as the administration of sulphonamide, and the irrigation of the udder with highly bactericidal dye solutions (Stableforth and Scorgie 1938), or with bacterial products such as gramicidin (Little, Dubos and Hotchkiss 1941).

Control of Human Personnel

Since the greater part of epidemic milk-borne disease is directly or indirectly of human origin, it is only logical to consider whether it is possible by medical and bacteriological control of the personnel on the farm and in the dairy to prevent infection of the milk at the source. The type of person who is most likely to prove dangerous is (1) the patient suffering from scarlet fever or septic sore throat, often in the incubation period, (2) the diphtheria case or carrier, (3) the patient suffering from typhoid fever, paratyphoid fever, dysentery, or gastro-enteritis, usually in an ambulant form, or the person who is a temporary or permanent carrier of the organisms of these diseases. What steps can be taken in practice to detect these persons and prevent them from handling milk during the time they are likely to be dangerous ? This is a question which very few workers have attempted to answer. What information is available comes mainly from the United States.

For some years the State of Connecticut insisted on a modicum of physical and bacteriological examination of handlers of certain grades of milk. Over a period of 6 years and 5 months, 91,257 laboratory examinations were made at an estimated cost of 48,000 dollars. Seventy-one carriers of pathogenic or potentially pathogenic organisms were detected, the average cost per carrier being 677 dollars. (Borman *et al.* 1935). This cost was sufficiently imposing to render it doubtful whether its continued incurrence was justifiable, and whether any extension of the scheme to the handlers of all grades of milk was advisable.

Accordingly a special investigation was undertaken (Foote *et al.* 1936) to find out what sort of results might be expected if regular and frequent examinations of the same milk handlers were made over a given period. Eighty-five employees of five dairies in Connecticut were selected, and nose and throat swabs were taken at weekly

intervals for the three months January to March inclusive. The swabs were examined for β-hæmolytic streptococci. Owing to absence of one sort or another not every employee was swabbed every week, but a total of 756 nasal and 756 throat swabs were taken. Hæmolytic streptococci, all of which belonged to Group A, were isolated from 8·7 per cent. of the swabs taken from individual employees (the nose and throat swabs from the same individual being considered together). Moreover, of the 85 employees, 23·5 per cent. yielded Group A streptococci at one time or another during the three-month period. Most of the employees carried these organisms for a time only, but 5 were persistent carriers. Medical examination at the time of swabbing revealed the occurrence of acute pharyngitis or tonsillitis on 22 occasions. In spite of the fact that some of the milk was consumed raw, no outbreak of milk-borne disease was reported as a result of these infections. The authors conclude that the percentage of persons harbouring β-hæmolytic streptococci is too great to permit of adequate control of milk-borne streptococci infections by employing any practical measures to eliminate carriers, and express the opinion that other methods must be used to protect against infections of this type.

Since this investigation was restricted to the examination of nasopharyngeal swabs, and included no attempt to detect enteric and dysentery carriers, it will be realized how extensive the machinery would have to be if a thorough control of all milk handlers was to be undertaken. The more the problem is considered, the more formidable it appears. The administrative and economic difficulties in collecting and examining specimens at frequent intervals from every milk handler on every farm and in every dairy in the country would be almost insuperable. These, however, might be faced if there was strong scientific evidence to show that by this means all carriers of pathogenic organisms would be detected, and that an effective guard would be placed against infection of the milk supply. But of this there is grave doubt. The carrier, and even diseased, state is often so transient in nasopharyngeal and intestinal infections that without almost daily examinations no guarantee to detect every infected person could be given. In addition, there is no justification for assuming that every carrier of pathogenic organisms will necessarily infect the milk, at least in sufficient degree to render it dangerous to other human beings. This conclusion is clearly exemplified by the results of the Connecticut workers. Whether the milk becomes dangerously infected, either directly from the cough-spray or the contaminated fingers of the milkers, or indirectly through udder infection of the cow, must be determined by a number of factors other than the mere presence of

the pathogenic organisms themselves in the nasopharynx or intestinal tract. The element of pure chance is bound to be considerable, and no amount of routine bacteriological control could do more than indicate the existence of potential danger. It may be concluded that any endeavour to supervise on an adequate bacteriological scale the human personnel on farms is bound to be frustrated by the immense administrative and economic difficulties involved.

On the other hand, there is a reasonably strong case to be made out for control of the employees in dairies and plants where large volumes of milk are being handled daily. More than one instance could be quoted of the infection of a bulked milk supply by a typhoid carrier. Even though pasteurization destroys typhoid bacilli, there is always a risk that through some breakdown in the machinery or carelessness on the part of the operator, the milk may not be effectively pasteurized, and typhoid bacilli may be distributed in the milk with disastrous results. Moreover, the possibility of infection of the milk in the bottling machinery after pasteurization is completed must always be borne in mind. For these reasons we believe that it would be sound practice to introduce a measure of bacteriological and medical control over the health of operatives in all dairies and plants where more than a given volume of milk—for example 100 gallons—is being dealt with daily. The supervision need not be very strict nor involve much expenditure. It should be directed mainly to avoiding the danger of including typhoid, paratyphoid, and dysentery carriers among the personnel, and should be of the same type as that recommended by the Ministry of Health (Report 1939*b*) for the control of men employed in water undertakings. The danger of bulked milk becoming infected from nasopharyngeal carriers of hæmolytic streptococci and diphtheria bacilli is probably so slight, considering that these organisms do not multiply in milk at ordinary temperatures, that their detection by bacteriological methods seems scarcely warranted. It should suffice to prevent any operative who is actually suffering from a sore throat from handling the milk. The fact that large dairies and pasteurizing plants are mainly situated in towns would render the administrative control of such a scheme very much easier than if specimens had to be taken on individual farms in the country.

Heat Treatment of the Milk

From what has been said hitherto it will be clear that no immediate practical method of abolishing milk-borne disease by controlling the health either of the animal population or of the human personnel handling the milk is available. It is true that by the expenditure of very large sums of money and extensive administrative reforms

tuberculosis or contagious abortion or mastitis in cows might be eliminated to a sufficient extent within the next twenty-five years or so to meet the demands of the liquid milk market, but the prospect of eliminating all three diseases within any reasonable time is remote. Many herds at present are free from one or other of these diseases, but the number of herds of any size in this country that are free from all three diseases is so small as to be negligible.

It will, moreover, be clear that the number of different types of pathogenic organisms of human origin that may infect milk and the number of different ways in which they may gain access to the milk are so great that any attempt to prevent the initial contamination of the milk is bound to break down sooner or later.

However much the eradication of animal disease may be desired by agriculturalists and veterinarians, it must be remembered that the problem presented to the public health administrator is urgent and presses for immediate solution. A great deal of milk-borne disease is occurring every year in this country. The only immediate and practicable method of putting a stop to it is not by protecting the milk from initial contamination—though this is desirable as far as possible—but by destroying all pathogenic organisms that may have gained access to the milk from various sources and by various channels on its way from the cow's udder to the distributing depot.

This can be accomplished by adequate heat treatment. On a large scale the milk can be pasteurized. Where this is impracticable, as in the home, the milk can be brought rapidly to the boil in a water-jacketed saucepan fitted with a lid to prevent scum formation, and, unless it is to be drunk immediately, cooled at once to as low a temperature as possible so as to interfere with the growth of any non-pathogenic organisms that may have survived.

The remainder of this volume will be devoted to the subject of pasteurization. It is this process alone which appears capable of providing the public immediately with a safe milk at a reasonable cost, and thus solving the double problem of abolishing milk-borne disease and improving the general nutritional state of the population.

Summary

1. The terms " clean " milk and " safe " milk are defined. It is shown that no necessary association exists between the two types of milk, and that a clean milk may be very dangerous. For human consumption milk that is both clean and safe is highly desirable.

2. The various ways in which the three most important diseases of milch cows, namely tuberculosis, contagious abortion, and mastitis, can be controlled are discussed. The only satisfactory method, it is

concluded, consists in the eradication of all infected animals, whether they are suffering from manifest disease or not.

3. The complete eradication of any one of these diseases presents a major problem that could not be solved within the next generation or two without the expenditure of very large sums of money and the introduction of far-reaching administrative measures.

4. The complete eradication of all three diseases, or even their eradication to an extent sufficient to meet the demands of the liquid milk market, presents in this country an almost insuperable task which there is little or no hope of accomplishing within any reasonable time.

5. Even if the eradication of these diseases from cattle could be successfully undertaken, the resulting milk would still be exposed to infection from the human personnel, from water, and from rodents.

6. The possibility of controlling milk-borne infection of human origin by medical and bacteriological supervision of all milk handlers on the farm is explored, but the administrative and economic difficulties are regarded as being far too great to justify the relatively meagre results that would probably be obtained.

7. On the other hand a strong case can be made out for a general measure of supervision of the personnel employed in dairies and plants where large volumes of milk are being handled. The control exercised should be similar to that already recommended by the Ministry of Health for the operatives of water undertakings, and should be designed mainly to exclude the employment of typhoid, paratyphoid, and dysentery carriers.

8. Since milk-borne disease is an urgent problem that cannot be immediately solved by controlling the health either of the cattle population or of the human personnel engaged in handling the milk, it is clear that other methods are required.

9. The only known method of satisfactorily solving this problem is by the heat treatment of milk, designed to destroy any pathogenic organisms that may have gained access to the milk on its way from the cow's udder to the distributing depot.

10. It is known that pasteurization, if carried out under proper conditions, destroys all pathogenic organisms in milk, and it is believed that this process alone affords a means of supplying the public with safe milk at a reasonable cost.

11. Where pasteurization is impracticable, the milk should be brought to the boil and rapidly cooled.

12. The remainder of this report will be devoted to considering pasteurization in its various health aspects.

CHAPTER V

THE OBJECT, DEFINITION, TYPES, AND CONTROL OF PASTEURIZATION

Object of Pasteurization

The observations of Louis Pasteur in France between the years 1860 and 1864 showed that abnormal fermentations and souring of wine could be prevented by exposing it for a short time to temperatures of 122–140° F. A little later Pasteur found that a similar treatment was effective in protecting beer against souring. The practical application of the process gave rise to the term "Pasteurization."

It was soon discovered that the souring of milk also could be greatly retarded by heat treatment. A process, which varied from one dairy to another but which was generally referred to as pasteurization, was gradually introduced into the larger towns of Europe and America with the purely commercial object of prolonging the life of the milk. Later, it was shown that heat treatment, if carried out under satisfactory conditions, destroyed not only most of the souring organisms but all the various pathogenic or disease-producing organisms in the milk. With increase in our knowledge of milk-borne disease this second, or public health aspect, of pasteurization has assumed more and more prominence, and now greatly outweighs its original commercial object.

It is only fair to say that the large dairy companies have been among the first to appreciate the public health importance of pasteurization. They realize the commercial value of a safe milk supply, and their efforts during the past two decades have been increasingly directed towards the conversion of an empirical and relatively slipshod method into a carefully regulated process of heat treatment, the efficiency of which is based on sound bacteriological and engineering principles. That this conversion is not yet completed, and that the efficiency of pasteurization in some of the smaller dairies still leaves much to be desired, must no more be allowed to interfere with the development of pasteurization than the imperfect chlorination of water by many of the smaller local authorities must be held to condemn a process which, in the hands of the larger water companies, has done more than any other single measure to protect the community against enteric disease.

So far as public health is concerned the object of pasteurization is to destroy the various pathogenic organisms that may be present

in milk and thus render it safe for human consumption, and to bring about this destruction with the least possible alteration to the physical, chemical, and nutritional properties of the milk. This latter purpose is more readily achieved by pasteurization than by boiling.

It must not be thought that pasteurization is demanded solely by the medical profession. The dairy industry itself demands it. The peculiar flavour of butter and cheese is due largely to the type of bacterial " starters " used in their preparation. These work much better when the majority of the contaminating micro-organisms in the cream or milk have been destroyed by heat treatment than in the original raw products with their diverse and more or less uncontrollable flora. For this reason nearly all factory butter in Great Britain and abroad is now made from pasteurized cream ; and the use of pasteurized milk in the preparation of cheese is becoming more and more prevalent (see Davis 1939).

Definition and Types of Pasteurization

Much of the current confusion about pasteurization is due to the fact that there is no universally accepted definition of the process. Pasteurization was introduced into practice before the scientific principles on which it is based were properly understood. One result of this was that attention became concentrated on the temperature

TABLE XIII

Time-temperature Equivalents necessary to bring about an equal degree of bacterial Destruction in Milk. $0^{10°\,C.} = 100$ (Report 1937*b*)

Temperature.		Time.
° C.	° F.	
60	140·0	63 minutes
62	143·6	25 ,,
63	145·4	16 ,,
64	147·2	10 ,,
65	149·0	6 ,,
66	150·8	4 ,,
70	158·0	38 seconds
72	161·6	15 ,,
74	165·2	6 ,,
76	168·8	2·4 ,,
80	176·0	0·4 ,,
85	185·0	0·04 ,,

to which the milk was exposed, while the time factor was largely neglected. It is now known that the coagulation of proteins and the destruction of bacteria are a function of both time and temperature, and that no definition of pasteurization which ignores this fact can be regarded as satisfactory.

Perusal of Table XIII will show that the same degree of bacterial destruction can be brought about by exposing the milk to a temperature of 140° F. (60° C.) for one hour, or to a temperature of 149° F. (65° C.) for 6 minutes, or to a temperature of 161·6° F. (72° C.) for 15 seconds. The time values given in this table for the upper ranges of temperature are only approximate, and are of greater academic than practical significance. Even though in the laboratory bacteria may be killed almost instantly at temperatures of 180° F. (82·2° C.) and over, under practical conditions the milk first entering the plant may fail to reach the desired temperature and a considerable margin of safety must therefore be allowed in calculating the required time exposure.

Since the same result can be achieved by variation of time and temperature, it follows that several combinations are possible. To the more usual of these special terms have been applied. Thus we have :

(1) Flash Pasteurization. In this the milk is momentarily exposed to a temperature of 140–176° F. (60–80° C.) or even higher. Neither the time nor the temperature is accurately controlled, and though the process may be of value for certain commercial purposes, it is unsuitable for the treatment of liquid milk destined for human consumption.

(2) High Temperature Pasteurization, or simply High Pasteurization. In this the milk is heated for a few seconds to 176–185° F. (80–85° C.). This method is frequently employed in Scandinavia. Though the temperature is controlled rather more than in flash pasteurization, there is still a considerable variation in the total amount of heating that the milk receives.

(3) High Temperature Short Time (H.T.S.T.) Pasteurization. In this the milk is heated for 15–20 seconds to 159·8–162° F. (71–72·2° C.). This method, which is sometimes referred to in Scandinavia as *low pasteurization* in contrast to the *high pasteurization* described under (2), and which must not be confused with the low temperature pasteurization to be described under (5) and (6), has been largely evolved in the United States. In the best types of apparatus the time and temperature of exposure are under automatic control, and the results can be predicted with a considerable degree of certainty.

(4) Stassanization. This is essentially the same process as high temperature short time pasteurization. It is carried out in a special type of apparatus devised by Stassano (1931), in which the milk is forced in a thin film through the annular space between two concentrically arranged pipes heated by hot water. The time-temperature combination employed varies somewhat, but in the most recent model the milk is heated for about 7 seconds to a temperature of 163·4–167·0° F. (73–75° C.).

(5) The Retarder Type of Low Temperature Pasteurization. This is sometimes spoken of as the continuous flow method. The same temperature is employed as in the holder method described below, but the heated milk, instead of being held in a closed vessel, flows continuously through an apparatus requiring an average of 30 minutes for its passage.

(6) Holder or Holding Type of Pasteurization, sometimes called Low Temperature Pasteurization. In this the milk is heated to 138–150·8° F. (58·9–66° C.) and kept at this temperature in a closed vessel for thirty minutes. The temperature varies in different countries, being higher in Great Britain than in most of the North American States. If, as is usual in the smaller plants, the milk is held in the same vessel as that in which it is heated, the term *vat* or *batch* pasteurization is generally applied. If, as is usual in the larger plants, the milk is held in a separate vessel from that in which it is heated, the term *pocket* type of apparatus is sometimes employed. When the milk is bottled before being heated, the process is referred to as the *in-bottle* method of pasteurization. In the holder process both the time and the temperature of exposure can be controlled with considerable accuracy.

In the first four of the methods just described the margin of safety is small, and slight fluctuations of temperature or time of exposure may affect the efficiency of the processing. Methods 1 to 5 are of the continuous flow type. In such a process there can be no objective certainty that every particle of the milk is exposed to the required temperature for the required time. In this respect objective certainty can be ensured only by the holder method of pasteurization, described under (6), where the milk, after being heated to the required temperature, is held at that temperature for the required time.

Whatever method of pasteurization is employed, it is important that the milk, after being heated, should be rapidly cooled to a low temperature, partly to avoid further changes in the milk caused by the heat, and partly to prevent multiplication of the surviving bacteria in the warm milk during the otherwise slow process of cooling to the temperature of the atmosphere. Though the cooling temperature

prescribed by official bodies is generally about 50–55° F. (10–12·8° C.), in practice the milk is frequently taken down to 38–40° F. (3·3–4·4° C.). The advantage of this greater degree of cooling is that bacterial multiplication is practically stopped, and the milk remains unchanged for a long time.

Official Requirements of Pasteurization in Great Britain. In England and Wales, up till 1941, only one official method of pasteurization was allowed—the holder method. As defined in the Milk (Special Designations) Orders, 1936 and 1938, made under Section 3 of the Milk and Dairies (Amendment) Act, 1922, pasteurization consists in retaining the milk at a temperature of not less than 145° F. (62·8° C.) and not more than 150° F. (65·6° C.) for at least thirty minutes, followed by its immediate cooling to a temperature of not more than 55° F. (12·8° C.). The milk must not be heated more than once. Indicating and recording thermometers must be used in connexion with the processing plant, and temperature records must be preserved for a period of not less than one month. The regulations in Scotland are very much the same, except that after processing the milk must be cooled to a temperature not exceeding 50° F. (10·0° C.).

In 1941, owing to shortage of skilled labour and of materials during war time, new regulations entitled " Milk (Special Designations) Regulations, 1941," were issued by the Ministry of Health permitting the use of the High Temperature Short Time (H.T.S.T.) method of pasteurization. The regulations provide that the milk treated by this process shall be retained at a temperature of not less than 162° F. (72·2° C.) for at least 15 seconds, and that the apparatus used shall be thermostatically controlled and be provided with an automatic device to divert the flow of any milk which has not been so treated. Separate licences are required for the Holder and the H.T.S.T. plants.

No milk that is not treated by one or other of these methods *in a plant licensed by the local authority* is allowed to bear the term " Pasteurized " on the bottle. This point must be made quite clear. There is nothing to prevent a dairyman heating milk to any temperature for any length of time and selling it as milk that has been treated in a pasteurizing plant, provided he does not use the actual printed word " Pasteurized " on the label of the receptacle in which he offers it for sale or describes it as officially pasteurized. Many of the faults ascribed to so-called " pasteurized " milk refer not to milk processed in licensed plants but to milk treated by heat in unlicensed plants over which no official control is exercised.

The time-temperature requirements laid down by the Ministry of Health for milk pasteurized by the holder method in this country are more stringent than those in most of the United States, where

a temperature of 142° F. (61·1° C.) for half an hour is widely used. As pointed out in Chapter XI, and as referred to later in this chapter under the phosphatase test (p. 63), the requirements in Great Britain are well above those required to destroy pathogenic organisms in milk, so that a considerable margin of safety is provided in the heat treatment. Slight faults in the technique of pasteurization, however regrettable, do not necessarily therefore impair the efficacy of the process in so far as the safety of the milk is concerned.

This is not the place to describe the mode of construction and operation of the various types of plant that conform to the licensing requirements laid down by the Ministry of Health. Those who are interested in this technical side of pasteurization may be referred to reports and publications by Hamill (1923), Frank, Moss, and Le Fevre (1927), Savage (1933*b*), Berry (1934), Scott and Wright (1935), Dalrymple-Champneys (1935), and Fuchs (1938). Suffice it to say that plants of the holder and H.T.S.T. type must be properly designed and operated if the processing of the milk is to be satisfactory. Many plants at present in use, particularly those that are not licensed, are imperfect in design and construction or are improperly and inefficiently operated and controlled. Under any system of compulsory pasteurization such plants would not receive their official licence and would therefore be rapidly eliminated.

Laboratory Control of Pasteurization

The control of the actual process of pasteurization can be carried out only by adequately trained inspectors who are present during the whole operation. The defects to be looked for and the type of control to be exercised are dealt with in some of the publications just mentioned, particularly in those by Berry (1934), Dalrymple-Champneys (1935), and Fuchs (1938).

Laboratory control is limited almost entirely to examination of the pasteurized product. The examination can be carried out either immediately after the processing, in order to test the efficacy of the heat treatment, or on the final bottled milk just before its delivery to the consumer in order to ascertain the amount of bacterial contamination resulting from the operations of cooling, bottling, storage, and delivery. Since this is not a laboratory manual, reference will be made to the various tests available in only the most general terms.

Chemical Methods

(*a*) **The Storch Colour Reaction.** This is extensively used on the Continent in the control of high temperature pasteurization, but is unsuitable for holder pasteurization, since the destruction of

the peroxydase on which it depends does not occur till the milk has been heated to 168·8° F. (76° C.) for 1·2 minutes or to 176° F. (80° C.) for 2·5 seconds.

The destruction of the enzyme *amylase*, on the other hand, occurs at too low a time-temperature combination to be of value for holder pasteurization; it is inactivated completely at 127·4° F. (53° C.) in 30–60 minutes.

Fortunately there is another enzyme, *phosphatase*, which is destroyed by a time-temperature combination corresponding almost exactly to that laid down in this country for holder and H.T.S.T. pasteurization. To Kay and Graham (1935) belongs the credit of having devised a test based on the destruction of this enzyme by heat, which can be used for the control of pasteurization.

(*b*) **Phosphatase Test: Interpretation.** This test depends on the fact that the enzyme phosphatase is destroyed by a temperature of 145° F. (62·8° C.) in 30 minutes. Milk exposed to 143·5° F. (61·9° C.) for 30 minutes or 145° F. (62·8° C.) for only 20 minutes will still contain detectable quantities of this enzyme. Though numerous modifications of the test have been described, the method commonly used in this country consists in incubating the milk for a given time with disodium phenyl-phosphate. If phosphatase is present, phenol will be liberated. Its amount can be measured colorimetrically, and used to estimate in arbitrary units the quantity of phosphatase present. In practice a milk containing less than 2·3 units may be regarded as having been satisfactorily pasteurized; with a milk having between 2·3 and 6 units there has been some small error in the pasteurization technique; with a milk having 6–10 units there has been some serious error in the technique; while with milks having above 10 units the error in the processing technique has been so gross as to amount virtually to non-pasteurization (Kay and Neave 1935).

This interpretation may need some slight alteration in the light of more extensive experience. Some milks are at present being condemned as imperfectly pasteurized because a reading of more than 2·3 units is obtained. There is reason to believe that this value may sometimes be exceeded even when the test is carried out by skilled technicians on milk that has been pasteurized under the best possible conditions. In practice a result slightly exceeding 2·3 units, though it may indicate the desirability of immediate inspection of the plant, should not be held to prove that pasteurization of the milk has been at fault. Experience in Canada and the United States also seems to point to allowing a greater range of phenol values than has been customary in the past (Smith 1938, Gilcreas 1939).

The phosphatase test is proving of the greatest value in this

country and abroad in the control both of holder and of high temperature short time pasteurization, and is serving to reveal multiple small errors in processing, many of them unsuspected and unintentional, which would otherwise escape detection (see Geiger and Davis 1937, Tiedeman 1938, Smith 1938, Krueger 1939). Its limitations, however, must not be overlooked; otherwise it will be subjected to the same type of abuse that so many previous laboratory tests have suffered at the hands of those who have failed to appreciate the fundamental principles on which they were based.

In the first place the test is one that demands exact standardization of all reagents and a high degree of technical skill in its performance. This is not an objection to the test *per se*, but it does mean that reliance can be placed on the result only if the test is carried out by adequately trained workers provided with all the necessary apparatus and facilities for doing accurate quantitative analysis. Even with skilled workers difficulties have arisen in practice over the standardization of the reagents (see Smith 1938), so that one lot of buffer solution has given a positive and another a negative result on samples of the same milk. Doubtless these difficulties will soon be overcome, but it must be borne in mind that a weakly positive reaction on pasteurized milk may sometimes be due not to imperfect heat treatment of the milk but to faulty technique in carrying out the phosphatase test. The greatest care should therefore be exercised in the performance of the test, and attention paid to the various precautions described in their monograph by Kay, Aschaffenburg, and Neave (1939).

Secondly, it has already been pointed out (p. 59) that the coagulation of proteins and the destruction of bacteria in milk are a function of both time and temperature. The inactivation of enzymes is no exception. Thus, phosphatase can be destroyed equally well by exposure to a temperature of 160° F. (71·1° C.) for 15–20 seconds, as to a temperature of 145° F. (62·8° C.) for half an hour. The fact, therefore, that in a given milk the phosphatase has been destroyed does not necessarily mean that the milk has been submitted to the pasteurizing requirements of the Ministry of Health. It may have been heated to a time-temperature combination giving results equivalent to those of holder pasteurization, such as 160° F. (71·1° C.) for 15–20 seconds, or it may have been frankly overheated. All the phosphatase test does is to show that the amount of heat applied to the milk has been above a certain minimal value; it gives no information about the excess of heat used nor about the manner in which it was applied.

Thirdly, the milk may have been adequately pasteurized, but owing to some defect in the plant or on the part of the operator, it

may have become admixed with a certain amount of raw or imperfectly heated milk. The phosphatase test, delicate though it is, is unable to detect less than about 0·2 per cent. of raw milk in otherwise adequately pasteurized milk. From the public health point of view this is of some importance. Consider, for example, a batch of 100 gallons of pasteurized milk that becomes contaminated with half a pint of raw milk containing tubercle bacilli. It is known that raw milk may at times contain tens, or even hundreds, of tubercle bacilli per millilitre, so that half a pint might well contain several thousand of these organisms. Such a milk, though infective for human beings, would nevertheless give a negative phosphatase reaction, and be erroneously regarded by many persons as satisfactory. The truth is that, while a positive phosphatase reaction shows that the milk has not been adequately pasteurized, a negative reaction cannot be regarded as proving the reverse. Inoculation into a guinea-pig of a pasteurized milk that has been contaminated with tuberculous raw milk in a volume too small to render the phosphatase reaction positive will almost certainly reveal the presence of living tubercle bacilli. Such slight undetectable contamination with raw milk would be even more serious if the milk contained typhoid, paratyphoid, or dysentery bacilli which are capable of multiplying in it.

How far this particular limitation is of importance in practice it is difficult to judge, because extensive series of comparative tests on pasteurized milk for phosphatase and for tubercle bacilli have not yet been recorded. What little has been done in this country suggests that a negative phosphatase test almost invariably means the absence of tubercle bacilli. On the other hand Sutherland (1937–1938), in Yorkshire, found that 3·5 per cent. of 334 pasteurized milks giving a negative phosphatase reaction nevertheless produced tuberculosis on inoculation into guinea-pigs. Moreover, experience in the United States has shown that leaky valves, permitting the contamination of pasteurized milk with raw, or the escape of imperfectly pasteurized milk, are one of the most frequent causes of the failure of pasteurized milk to pass the phosphatase test (Krueger 1939). It seems highly desirable that local authorities shall not rely exclusively on the phosphatase test in the control of pasteurized milk, but shall examine the milk from time to time by the guinea-pig inoculation method.

Fourthly, even when the raw supply is known to be infected with pathogenic organisms, it is a mistake to assume that a weakly positive phosphatase reaction on the pasteurized product necessarily indicates that the milk is dangerous. The time-temperature combination required to destroy the phosphatase in the milk is more severe than that required to destroy pathogenic organisms such as the tubercle

bacillus (see pp. 139-144). In other words, so far as the destruction of these organisms is concerned, the pasteurization requirements in this country provide a considerable margin of safety, while corresponding almost exactly with those needed for the inactivation of phosphatase. A degree of heat sufficient to destroy all the pathogenic organisms in the milk may be insufficient to inactivate the whole of the phosphatase, so that a weakly positive reaction may be recorded. Such a reaction must not, of course, be regarded with equanimity. It shows clearly that the official pasteurization requirements have not been complied with; nevertheless it does not prove that the milk is infective or necessarily dangerous for human consumption.

These last two corollaries to the interpretation of the phosphatase test may be summarized by saying that a negative phosphatase reaction does not *prove* that the milk is safe, nor a positive reaction *prove* that it is dangerous or even potentially dangerous. Though it is probable that these are the correct interpretations, the probability is not of such a high order as to be equivalent to certainty. From the legal point of view, therefore, these limitations are of importance, but from the practical point of view of plant control they may usually be ignored.

(*c*) **Cremometric Test.** This test, which was introduced by Orla-Jensen (1929, 1932), makes use of the fact that in milk submitted to low temperature pasteurization the cream rises more slowly than in the milk before heating. The effect of pasteurization is believed to be due to the destruction of an agglutinin in raw milk which causes the fat globules to adhere to one another. The aggregated globules rise more rapidly to the surface than when uniformly dispersed. In the actual test, which need not be described here (see Report 1935), the milk is diluted with an equal volume of water in a special tube, incubated for 5 minutes at 50° C., and then at 12–15° C. The cream line is measured in millimetres after 1 and 2 hours. Holland and Dahlberg (1940) have shown that at temperatures between 140 and 170° F. (60–76·1° C.) the curves denoting the commencement of reduction in creaming capacity and the inactivation of phosphatase agree with each other very closely. Thus at 170° F. (76·1° C.) exposure of the milk for 2½ seconds almost completely destroyed the phosphatase without impairing the creaming capacity. At 160° F. (71·1° C.) the corresponding time was about 15 seconds.

Unlike the phosphatase test, however, which can be used for skim milk, homogenized milk, or even cream, the cremometric test is unsuited for any of these types of milk, as well as for milk which is very rich in fat, like Jersey milk. The result is influenced by numerous factors, and the test is not to be compared in accuracy with the phosphatase

test. It is, however, simple and inexpensive to carry out, and may be of value for indicative or corroborative purposes (Report 1935*a*).

(*d*) **Suspension Tests.** It has been known for a long time that when blood is present in raw milk the corpuscles rise with the cream to the surface, but that when the milk is heated they fall to the bottom and form a pink sediment. It is probable that the mechanism involved is similar to that responsible for the formation of the cream line.

Various indicator suspensions have been recommended by different workers. Schern and Gorli (1932) added guinea-pig red blood corpuscles to the milk. Kohn and Klemm (1932) preferred charcoal, Tapernoux (1933) suggested carmine, and Pien and Baisse (1934) used indigo. The sedimentation of red blood corpuscles and of charcoal is brought about by heating the milk to about 142–145° F. (61·1–62·8° C.) for half an hour; carmine requires a temperature of 145–150° F. (62·8–65.6° C.), and indigo of 150·5–152·4° F. (65·8–66·9° C.).

The suspension test with these different substances has been tried out by Walker Hall and his colleagues (Report 1935*a*), who regard it as of limited value. For satisfactory results a high-grade technique and continuous practice are required. Even when carried out under optimal conditions, the test cannot be relied upon to distinguish definitely between perfectly and imperfectly pasteurized milk. Its main use appears to be in affording confirmation of other tests.

Bacteriological Methods

It may be said directly that there is no bacteriological method equal in efficiency to the phosphatase test for controlling the time-temperature exposure of the milk in holder pasteurization. On the other hand bacteriology can help considerably in confirming the results of the phosphatase test, in the search for undesirable organisms in the pasteurized milk, and in the detection of bacterial contamination of the milk after pasteurization. For a technical description of the various methods that will be briefly enumerated reference may be made to the report by Wilson and his colleagues (1935).

(*a*) **Breed Smear Method.** This method consists in a direct microscopical examination of the milk. It is mainly of value in detecting the presence of thermophilic organisms which may multiply in the plant during the period of holding and which do not give rise to colonies when the milk is examined by the official plate count method at 37° C.

(*b*) **Plate Count Method.** At 37° C. this method is not well suited to the control of the processing itself, since the number of

organisms that develop depends on several other factors than the time-temperature exposure of the milk. Its use is limited chiefly to bacterial control of the final quality of the milk as delivered to the consumer. If carried out at 55° C. immediately after processing it may be used to afford an estimate of the thermophilic organisms present.

(*c*) **Modified Methylene Blue Reduction Test.** This test has much the same limitations as the plate count. Carried out at 37° C. on the freshly pasteurized milk it can give no reliable indication of the efficiency of the heat treatment, but if performed on the milk as delivered to the consumer it is useful for gauging the degree of contamination or the adequacy of cooling after processing. According to Ciani (1939) it is of very much greater value for judging the keeping quality of pasteurized milk than the plate count. If performed at 55° C. on the milk immediately after processing, it can be used to detect the presence of active thermophilic organisms that might lead to deterioration of the milk.

(*d*) **Presumptive Coliform Test.** Several workers (for references see Wilson *et al.* 1935, Mattick, Hiscox, and Davis 1937) have found that coliform bacilli in raw milk are almost completely destroyed by holder pasteurization at 145–150° F. (62·8–65·6° C.) for half an hour. If the milk is examined immediately after processing these organisms should as a rule be absent from 10 ml. quantities of milk. Their presence in more than occasional samples should cast suspicion on the efficiency of the heat treatment. It should be made clear that the presence of coliform bacilli in pasteurized milk as delivered to the consumer can bear no such interpretation, since these organisms may gain access to the milk after pasteurization, or the few bacilli that survive processing may multiply if the milk is not maintained at a low temperature.

(*e*) **Tests for Pathogenic Organisms in the Milk.** If holder pasteurization is properly carried out all pathogenic organisms are destroyed. Search may be made in the finished product for the tubercle bacillus or *Br. abortus*, both of which are very common in raw bulked milk used for pasteurizing. Their presence in the pasteurized product indicates clearly that the time-temperature exposure has been insufficient or that the pasteurized milk has become contaminated with raw milk. The latter eventuality may be due to faults in the pasteurizing plant or occasionally to malpractice. As already pointed out (p. 65), it is unusual to find the tubercle bacillus in pasteurized milk which gives a negative phosphatase reaction, because the time-temperature conditions required for destruction of this organism are less severe than those for inactivation of the phos-

phatase. If, however, the milk has become contaminated after pasteurization with a small proportion of infected raw milk—too small to render the phosphatase test positive—tubercle bacilli may sometimes be demonstrated in the mixture owing to the greater sensitivity of the guinea-pig test, which will enable even minimal numbers of tubercle bacilli to be detected. Since the phosphatase test cannot reveal an admixture of less than about 0·2 per cent. of raw milk, while the guinea-pig test for tubercle bacilli may, if the raw milk is heavily infected, reveal much smaller proportions, it is wise, while relying for routine purposes mainly on the phosphatase test, to examine from time to time milk from every pasteurizing plant for evidence of tubercle bacilli in case contamination is occurring with infected raw milk that is not revealed by the phosphatase test.

Summary

(1) A brief description is given of the various types of pasteurization. It is pointed out that in this country official pasteurization is restricted to (*a*) the holder method in which the milk is exposed to a temperature of 145–150° F. (62·8–65·6° C.) for at least 30 minutes and cooled immediately to a temperature of not more than 55° F. (12·8° C.) or in Scotland 50° F. (10·0° C.), and (*b*) the high temperature short time method in which the milk is exposed to a temperature of not less than 162° F. (72·2° C.) for at least 15 seconds, and then cooled as in (*a*). No milk that is not treated in a plant licensed by the local authority is allowed to bear the label "Pasteurized" on the bottle in which it is offered for sale. Many of the objections that have been raised to pasteurized milk refer not to milk pasteurized under licence, but to milk treated in unlicensed plants over which no official control is exercised.

(2) The laboratory control of pasteurization is discussed, particular attention being paid to the importance and the limitations of the phosphatase test. While this test is a most valuable guide to inefficient heat treatment of the milk, it is not capable of detecting the admixture of the pasteurized product with very small quantities (less than about 0·2 per cent.) of raw or imperfectly heated milk. For this reason it is recommended that, while the phosphatase test should be used in the routine control of processing, it should be supplemented from time to time by the guinea-pig test for tubercle bacilli, which affords a more delicate means of revealing the presence of small proportions of infected raw milk in the final product.

(3) Recent work on the phosphatase test suggests that the range of phenol values given by properly pasteurized milk may be rather

greater than that which has hitherto been accepted. Caution should therefore be exercised in condemning pasteurized milk with values just over the 2·3 unit level.

(4) It is pointed out that the amount of heat necessary to destroy the phosphatase in milk is greater than that necessary to destroy pathogenic organisms, and therefore a pasteurized milk which gives a weak phosphatase reaction is not necessarily dangerous for human consumption, even though the raw milk was known to be infected.

CHAPTER VI

THE PRESENT EXTENT OF PASTEURIZATION

Apart from Canada and the United States little exact published information is available on the extent to which milk for human consumption is pasteurized in different countries. There is no question that pasteurization is on the increase, especially in the larger towns. The producer retailers are gradually diminishing in numbers, milk is being more and more bulked, and bulked milk is being increasingly subjected to some form of heat treatment.

Extent of Pasteurization in Other Countries. According to the League of Nations report (Report 1937*b*) pasteurization is quite common in some of the larger towns in Norway ; in Oslo, for example, about 50 per cent. is pasteurized. Similarly in the Netherlands, there are a number of towns, such as The Hague, Amsterdam, Rotterdam, Zutphen, and Deventer, where 40–75 per cent. of the liquid milk supply is pasteurized. In Denmark about 50 per cent. of the town supplies are heat-treated. There are, however, very great differences between different countries. In Switzerland, on the one hand, pasteurization is almost non-existent. In Sweden, on the other hand, as the result of a Royal Ordinance dated July 21st, 1937, which came into operation on July 1st, 1939, pasteurization of all milk and cream, with the exception of adequately supervised raw milk, is now universal and compulsory. In Germany pasteurization is extensively employed in the large towns. In France a law imposing compulsory pasteurization of all milk for human consumption was passed by the Chamber of Deputies on July 2nd, 1935. Exceptions were made for milk sold directly by the producer to the consumer, for high-quality raw milk, and for bulked milk not exceeding 600 litres a day in quantity. Two decrees controlling the quality of pasteurized and of raw milk came into operation on April 26th and April 28th, 1939, respectively. According to information, kindly supplied by Professor A. M. Leroy of the Institut National Agronomique, nearly the whole of the milk supply of Paris, and a considerable proportion of the milk supply of many of the large provincial towns in France is now pasteurized. Strasbourg has its own municipal pasteurized supply.

Detailed information is available about the extent of pasteurization in Canada and the United States of America. According to Berry (1938), who records the data for towns of 2,000 inhabitants and over, there are in Canada at least 38 municipalities, mostly in Ontario, in

which pasteurization is compulsory. Analysis of these data shows that in 154 municipalities for which actual figures are available the average proportion of milk pasteurized is 63 per cent. This figure is obtained by simply averaging the proportion of milk pasteurized in all the municipalities irrespective of their size. When a weighted mean is taken based on the total number of inhabitants it is found that an average of 91 per cent. of the population in these towns is supplied with pasteurized milk. Since Berry's report, pasteurization has been rendered compulsory for the whole Province of Ontario. A more recent (1941) report states that, two years after the introduction of compulsory pasteurization in 1938, 92 per cent. of all milk in Ontario sold for fluid consumption is believed to be pasteurized. All centres of population of over 500 inhabitants now come within the scope of legislation.

In the United States Fuchs and Frank (1938) collected data on the extent of pasteurization in urban communities of over 1,000 inhabitants in 1936. The average proportion of milk pasteurized in a total of 2,277 municipalities was 41 per cent. Taking the sizes of the different municipalities into account, it was found that an average of 73 per cent. of the population in these towns was supplied with pasteurized milk. In 176 municipalities all market milk, or all market milk except Certified, was pasteurized, and in 117 of these pasteurization was compulsory. It may be noted that in cities of 500,000 inhabitants and over 95 per cent. of the milk supply was pasteurized.

Extent of Pasteurization in Great Britain. Since no exact figures were available on the extent of heat treatment of the milk supply, the Ministry of Health in the spring of 1939 sent out the following questionnaire to the London County Council, to the 83 county boroughs of England and Wales, and to the Department of Health for Scotland for distribution to the four Scottish cities.

1. What proportion of the milk retailed in the district is milk which has been pasteurized under a licence granted by virtue of the Milk (Special Designations) Orders ?
2. What proportion of the milk retailed in the district is sterilized milk ? [1]
3. What proportion of the milk retailed in the district is heat-treated, other than (1) or (2) ?
4. What proportion of the milk retailed in the district is Tuberculin Tested milk [including Tuberculin Tested milk (Pasteurized) and Tuberculin Tested milk (Certified)] ?
5. Has pasteurization and/or sterilization of milk increased in the district during the last ten years, and, if so, to what extent ?

[1] See note on p. 78.

Answers to most of these questions were supplied with the exception of the last, information on which was too vague to be treated statistically.

Table XIV gives the percentage of various types of milk for the administrative county of London.

TABLE XIV

PERCENTAGE OF VARIOUS TYPES OF MILK IN THE ADMINISTRATIVE COUNTY OF LONDON IN 1939 (ESTIMATED POPULATION 4,100,000)

Pasteurized under licence.	Sterilized.	Heat-treated in other ways.	Tuberculin Tested, Raw and Pasteurized.	All heat-treated, excluding Tuberculin Tested.
92·7	2·3	3·6	0·4	98·6

It will be seen that nearly 93 per cent. of the milk is pasteurized under licence, and that between 98 and 99 per cent. is heat-treated in some way or other.

Table XV records the frequency distribution of the various types of milk in the county boroughs, including the four cities of Scotland.

TABLE XV

FREQUENCY DISTRIBUTION OF VARIOUS TYPES OF MILK IN THE COUNTY BOROUGHS OF ENGLAND AND WALES, INCLUDING THE FOUR CITIES OF SCOTLAND, IN 1939.

Percentage.	Pasteurized under licence (86).	Sterilized (85).	Heat-treated in other ways (84).	Tuberculin Tested Raw and Pasteurized (85).	All heat-treated, excluding Tuberculin Tested (85).
0–	2	69	63	79	1
10–	6	6	12	4	4
20–	12	4	4	1	6
30–	15	2	4	1	9
40–	22	1	1	0	9
50–	8	3	0	0	16
60–	6	0	0	0	10
70–	11	0	0	0	11
80–	4	0	1	0	12
90–	0	0	0	0	7
Weighted Mean	44·0%	7·1%	6·8%	3·6%	58·1%

Figures in parenthesis indicate number of county boroughs furnishing satisfactory returns. The weighted means have been worked out from the detailed, not the grouped, figures.

The average proportion of milk pasteurized under licence is less than half that in London, but the average proportion of milk sterilized is over three times as much.

In Table XVI the figures have been grouped according to the size of the populations in the different towns.

TABLE XVI

FREQUENCY DISTRIBUTION OF (1) MILK PASTEURIZED UNDER LICENCE AND (2) ALL TYPES OF HEAT-TREATED MILK IN THE COUNTY BOROUGHS OF ENGLAND AND WALES, INCLUDING THE FOUR CITIES OF SCOTLAND, ACCORDING TO SIZE OF POPULATION, IN 1939

	No. of C.B.	Average percentage.	
		Pasteurized under licence.	All heat-treated.
20,000–	38	43·4	53·3
100,000–	24	44·1	58·9
200,000–	12	49·2	71·6
300,000–	1	40·0	53·0
400,000–	3	57·3	62·3
500,000–	1	48·5	52·3
600,000–	0	—	—
700,000–	1	73·1	87·2
800,000–	1	30·3	55·2
900,000–	0	—	—
1,000,000 . .	2	39·0	88·6
Total	83		
Weighted mean for county boroughs		45·0	59·1
Weighted mean for population		47·0	70·0

The weighted means were worked out from the detailed figures when the number of observations in a group was less than 12. This partly explains the slight discrepancy between the figures for milks Pasteurized under licence and all heat-treated milks in this Table and Table XV. The other cause of the discrepancy is the fact that in Table XVI two or three county boroughs had to be excluded, because they did not furnish exact figures on which an average could be based, though they could be included in the grouped distribution of Table XV.

The weighted means have been worked out (1) to show the average percentage of milk pasteurized or heat-treated in the county boroughs, and (2) the average percentage of the population to which this milk is distributed. It will be noted that while the means for milk pasteurized under licence are much the same, those for all heat-treated milk differ by about 11 per cent. This is mainly due to the fact that the

two largest towns, namely Glasgow and Birmingham, have a large amount of milk heat-treated other than by pasteurization under licence.

In Table XVII an attempt has been made to estimate the average proportion of the population, living in the large towns of England and Wales and the four cities of Scotland, that is supplied with pasteurized or heat-treated milk.

The figures for Greater London are merely approximate estimates, since no detailed figures are available. While bearing this in mind, we shall not be far wrong if we conclude that over 60 per cent. of

TABLE XVII

APPROXIMATE ESTIMATE OF PERCENTAGE OF (1) MILK PASTEURIZED UNDER LICENCE, AND (2) ALL HEAT-TREATED MILK IN GREATER LONDON, THE COUNTY BOROUGHS OF ENGLAND AND WALES, AND THE FOUR CITIES OF SCOTLAND, IN 1939

Area.	Population.	Percentage pasteurized under licence.	Percentage all heat-treated.
Administrative county of London	4,100,000	92·7	98·6
Greater London, outside administrative county [1] . .	4,000,000	90·0	95·0
Aggregate county boroughs outside administrative county	13,300,000	44·7	70·2
Total	21,400,000		
Weighted Mean		62·0	80·0
Four cities of Scotland . .	1,800,000	51·7	77·8

[1] Excludes the three county boroughs of Croydon, East Ham, and West Ham, which are included in the aggregate county boroughs below.

the population in London and the county boroughs of England and Wales is supplied with milk pasteurized under licence and about 80 per cent. with milk heat-treated in some way or other. The population living in these areas constitutes rather more than half that of England and Wales as a whole. Of the remainder, about 12,000,000 live in urban areas and 7,000,000 in rural areas. What proportion of the population living in these areas receives heat-treated milk, there is no means of telling; but it is probable that a fair

proportion is supplied in the urban districts and a very small proportion in the rural districts. If, as a pure guess, we estimated the former proportion as 25 per cent. and the latter as 5 per cent., it would follow that about 50 per cent. of the population of England and Wales as a whole is at present being supplied with heat-treated milk.

Table XVIII is an attempt to estimate the increase that has occurred in pasteurization during the last nine years. In 1930 a questionnaire, more or less similar to that already reproduced, was sent out by the London School of Hygiene and Tropical Medicine to about half the county boroughs of England and Wales, and to Glasgow and Edinburgh. The returns have been compared with those furnished by the same towns in 1939.

TABLE XVIII

FREQUENCY DISTRIBUTION OF (1) MILK PASTEURIZED UNDER LICENCE, AND (2) STERILIZED MILK, IN THE SAME COUNTY BOROUGHS IN 1930 AND 1939

Percentage.	Pasteurized under licence (32).		Sterilized (28).	
	1930.	1939.	1930.	1939.
0–	9	1	24	22
10–	5	0	2	3
20–	4	3	0	1
30–	7	5	0	0
40–	2	14	2	0
50–	3	4	0	2
60–	0	2	0	0
70–	2	2	0	0
80–	0	1	0	0
90–	0	0	0	0
Weighted Mean	24·4	45·9	8·6	10·4
Percentage Increase	88		12	

Figures in parenthesis indicate number of county boroughs furnishing satisfactory returns.

The amount of sterilized sold appears to have increased only slightly, but the amount of pasteurized milk has increased by about 88 per cent. This figure probably underestimates the increase to some extent, since in the 1930 questionnaire the term " Pasteurized " was not qualified by the words " under licence," and it is probable that the 1930 returns included a certain amount of flash-pasteurized

milk. It would probably be fair to conclude that the amount of milk pasteurized under licence nearly doubled in the nine years between 1930 and 1939.

Summary

1. Compulsory pasteurization came into force for the whole of Sweden in July 1939. In France a law insisting on compulsory pasteurization of milk was passed in 1935, but has so far been only partly enforced.

2. In other European countries the proportion of milk pasteurized varies greatly. It is fairly common in the large towns of Western Europe, and appears to be increasing in extent.

3. In Canada pasteurization became compulsory for the whole Province of Ontario in June 1939. In addition, at least six municipalities have introduced compulsory pasteurization. Taking towns of 2,000 inhabitants and over, about 90 per cent. of the population in Canada in 1938 was supplied with pasteurized milk.

4. In the United States in 1936 data were available for 2,277 municipalities of 1,000 inhabitants or over. In 176 of these pasteurization was compulsory for all market milk, or for all except Certified. An average of about 73 per cent. of the population in the 2,277 municipalities was supplied with pasteurized milk. In cities of 500,000 inhabitants and over this figure was 95 per cent.

5. For Great Britain the following figures are available, based on a questionnaire sent out in the spring of 1939 to the London County Council, the county boroughs of England and Wales, and the four cities of Scotland:

(*a*) In the administrative county of London 92·7 per cent. of the milk is pasteurized under licence, and over 98 per cent. is heat-treated in some way or other.

(*b*) In the aggregate county boroughs the corresponding figures are 44 per cent. and 58·1 per cent. The latter figure includes 7·1 per cent. of sterilized milk.

(*c*) In the county boroughs 47 per cent. of the population is supplied with milk pasteurized under licence, and 70 per cent. with heat-treated milk.

(*d*) An approximate estimate shows that of the population of Greater London and the county boroughs about 62 per cent. is supplied with milk pasteurized under licence and about 80 per cent. with heat-treated milk.

(*e*) A very approximate estimate indicates that about 50 per cent. of the population of England and Wales is at present being supplied with heat-treated milk.

(*f*) In Edinburgh, Glasgow, Aberdeen, and Dundee the average amount of milk pasteurized under licence is 52 per cent. and of all heat-treated milk 66 per cent. The proportions of the population supplied with these two types of milk are 52 per cent. and 78 per cent. respectively.

6. A comparison of the returns furnished by about half the county boroughs of England and Wales in 1930 with those of the same county boroughs in 1939 shows that while the average proportion of sterilized milk rose by only about 12 per cent., the average proportion of milk pasteurized under licence nearly doubled during this period.

NOTE.—*Sterilized Milk.* There is no legal definition of sterilized milk, but the term is commonly applied to milk that has been heated to at least 212° F. (100° C.), and usually higher, for varying lengths of time. All, or nearly all, the micro-organisms are killed by this process, and the resulting milk is often bacteriologically sterile.

CHAPTER VII

COMPOSITION OF COWS' AND HUMAN MILK: FUNCTION OF THE VARIOUS CONSTITUENTS

General Composition

The composition of both cows' and human milk is subject to considerable variation, and the table presented (pp. 80, 81) is intended to give an approximate idea only of the quantities of the more important constituents found in Northern Europe. In preparing this table care has been taken to avoid as far as possible the inclusion of values at the beginning or the end of lactation, which often differ considerably from those when milk secretion is at its height. Numerous publications have been consulted, in particular those by Allen (1931), Davies (1936), Widdows, Lowenfeld, Bond, and Taylor (1930), Widdows and Lowenfeld (1933), Fixsen and Roscoe (1937–1938), and Dornbush, Peterson, and Olson (1940).

Milk is known to contain altogether about seventy different substances, including twenty amino-acids (Isaachsen 1932), and twenty different elements among the mineral salts (Allen 1931). Many of these are present in only minute quantities.

It will be observed that cows' milk contains two to three times as much protein as human milk. About six-sevenths of this protein is in the form of caseinogen and only about one-seventh in the form of lactalbumin; in human milk, on the other hand, lactalbumin constitutes about one-third of the protein. Lactalbumin, it may be remarked, forms a finer clot in the stomach than caseinogen and is therefore more digestible, and it contains larger quantities of the two amino-acids, cystine and lysine, which are of special importance for growth. The fat content of cows' milk tends to be rather higher and the sugar content rather lower than that of human milk. On account of these various differences it is common, when feeding cows' milk to young infants, to dilute it with water so as to diminish the quantity of protein, and to add extra sugar to compensate for the relative and absolute deficiency of the lactose.

In addition to these differences, cows' milk is over three times as rich as human milk in mineral salts, and contains a particularly abundant supply of calcium and phosphorus. Iron and copper, on the other hand, are relatively deficient in cows' milk, and may have to be supplemented in the diets of artificially fed infants to prevent the development of anæmia.

TABLE XIX

Average Chemical Composition of Cows' and Human Milk

(Figures refer mainly to the middle period of lactation)

Constituent.		Cow.	Human.	Remarks.
Lactose		4·75 gm. per 100 ml.	6·5 gm. per 100 ml.	Rises in human milk for a fortnight after birth and then remains fairly constant throughout lactation.
Fat		3·75 ,, ,,	3·6 ,, ,,	Subject to wide variations in human milk and in milk of different breeds of cow.
Protein	Caseinogen	3·0 ,, ,,	0·8 ,, ,,	Total protein in human milk falls during the first month after birth from 1·7 per cent. to 1·2 per cent., after which it remains fairly constant till towards the end of lactation when it falls gradually to 0·9 per cent.
	Lactalbumin	0·4 ,, ,,	0·4 ,, ,,	
	Lactoglobulin	Trace ,, ,,	? ,, ,,	
	Total	3·4 ,, ,,	1·2 ,, ,,	
Salts	Calcium: Soluble	0·03 ,, ,,	0·016 ,, ,,	Rises in human milk to reach a maximum at end of 4 months and then slowly declines.
	Calcium: Insoluble	0·09 ,, ,,	0·020 ,, ,,	
	Calcium: Total	0·12 ,, ,,	0·036 ,, ,,	
	Phosphorus: Soluble	0·038 ,, ,,	0·014 ,, ,,	Shows in human milk a similar rise and fall to that of calcium, but less marked.
	Phosphorus: Insoluble	0·057 ,, ,,	0·004 ,, ,,	
	Phosphorus: Total	0·095 ,, ,,	0·018 ,, ,,	
	Iron	0·027–0·14 mgm. per 100 ml.	0·05–0·19 mgm. per 100 ml.	
	Copper	0·012–0·02 mgm. per 100 ml.	0·04–0·08 mgm. per 100 ml.	
	Manganese	4–5γ per 100 ml.	?	
	Iodine	4–7γ per 100 ml.[1]	4·3–4·6γ [1] per 100 ml.	Depends on the iodine content of the diet. Figures for human milk based on 2 cases.
	Total	0·7 gm. per 100 ml.	0·2 gm. per 100 ml.	

Vitamins	Carotene	5–75γ per 100 ml.	5–60γ per 100 ml.	Higher in cows' milk in summer than in winter, depending on diet.
	A	20–130 I.U. ,, ,,	160–270 I.U. ,, ,,	
	Total A potency	28–280 I.U. ,, ,,	200–500 I.U. ,, ,,	
	B_1	15–25 I.U. ,, ,,	<1·6–4·4 I.U. ,, ,,[2]	Fairly constant in cows' milk.
	Riboflavin	0·1–0·3 mgm. ,, ,,		If present in human milk, not usually in free state.
	Nicotinic acid	0·5–1·4 ,, ,, ,,[3]	?	
	C	about 2 mgm. per 100 ml.	about 3–5 mgm. per 100 ml.	Fairly constant in cows' milk.
	D	Winter 0·3–1·7 I.U. per 100 ml. Summer 2·4–4·9 I.U. per 100 ml.	Usually less than in cows' milk.	In cows' milk depends on amount of sunshine to which cows are exposed.
	E	Very small	?	Even 25 per cent. of cows' milk fat in the diet is insufficient for rats.

[1] Magee and Glennie (1928), Matthews, Curtis, and Meyer (1939), Report (1939c).
[2] Neuweiler (1938).
[3] Irwin (1939).
γ = 1/1000 mgm. I.U. = international unit.

The lactose and the fat in milk constitute excellent sources of energy, while the protein, which has a high biological value, is of great importance in the building up of healthy tissue.

Vitamin A. Vitamin A is fairly abundant in cows' milk and considerably more so in human milk (Svensson 1936, Haas and Meulemans 1938). This vitamin is chemically related to the vegetable pigments known as the carotenes. Its structure is now known, and when pure it consists of a yellow oil. Carotene may be regarded as its natural precursor, for when ingested it is converted into vitamin A in the liver. The vitamin A content of cows' milk can be increased by the inclusion in the diet of substances like silage and dried grass which are rich in carotene. Vitamin A plays an important part in the growth of young animals. Its deficiency or absence from the diet for any length of time may be followed by degenerative changes in the epithelial cells lining the exposed surfaces of the body, by the eye condition known as xerophthalmia, and by night blindness or hemeralopia, which is due to deficiency of visual purple in the retina. In addition it seems to be concerned in the formation of the teeth, the nutrition of the skin, sexual fertility, and possibly other processes (Hopkins 1935). The observations of Green and Mellanby (1928, 1930) and numerous other workers indicate that it is of importance in the natural defence mechanism of the tissues against infection.

Vitamin B_1. This vitamin, which is sometimes referred to as aneurin or thiamin, is present in small quantity in cows' milk. Its output is fairly constant, and can be influenced to only a slight degree by the nature of the feed. It is a very important constituent of the diet, being responsible for the proper maintenance of the nervous tissues. Its absence from certain dietaries is the prime, though probably not the sole cause of Beri Beri, a disease in which nerve degenerations are a prominent feature. The adult requires 200–300 international units of vitamin B_1 per day; to satisfy this need, 2 to 3 pints of cows' milk would have to be consumed.

Vitamin B_2. This vitamin is a complex consisting of four or more factors. One, the growth factor, is a yellow fluorescent pigment known as riboflavin or lactoflavin. Another, the so-called antipellagra factor, consists of nicotinic acid. Other factors are of importance in the prevention of certain types of anæmia. Recent work has rendered it possible to measure the growth factor separately by fluorometric methods (see Kon 1938). Cows' milk is known to be a relatively abundant source of the vitamin B_2 complex as a whole. The quantity in which it is present is

determined to some extent by the diet of the cow (Bartlett *et al.* 1938).

Vitamin C. Vitamin C is of importance chiefly in the prevention of scurvy. It has now been obtained pure in the form of a white crystalline substance to which the name ascorbic acid has been given. The work of Kon and Watson (1936) has shown that, when secreted by the mammary gland, it exists in the reduced form, but that after exposure to light and air it undergoes oxidation, first of all to a biologically active and reducible substance known as dehydroascorbic acid, but later to substances which are biologically inactive and non-reducible. The total ascorbic content of cows' milk immediately after withdrawal from the udder is estimated at about 22 mgm. per litre; but owing to oxidation processes occurring as the result of exposure to light the ascorbic acid content of the raw milk of commerce may be reduced to one-half or even one-quarter of this figure. The adult requirements for vitamin C are about 22 mgm. a day. This quantity could be obtained from about $1\frac{3}{4}$ pints of freshly drawn milk, but in practice a considerably larger quantity would be required. Jacobsen (1935) at Copenhagen, for example, estimated the requisite daily quantity at $5\frac{1}{2}$ pints. Owing to the comparative poverty of active ascorbic acid in commercial raw milk, it is usual to add some more abundant source of this substance, such as orange juice, to the diet of infants and young children who are being fed mainly or exclusively on cows' milk. The vitamin C content of cows' milk varies but little throughout the year, and can be affected to only a minor degree by diet.

Vitamin D. This vitamin, which plays such an important part in the calcium metabolism of the body, and hence in the prevention of rickets and dental caries, belongs to a class of substances known as sterols. Its structure is now known, and the name calciferol has been applied to it. The quantity of this vitamin in cows' milk is subject to striking seasonal variation, being relatively abundant in the summer and almost lacking in winter. Though many workers have related this variation to the nature of the diet, the observations of Campion and his colleagues (1937) indicate that direct exposure of the cow to sunshine and skyshine, and not the pasture on which the animals are fed, is responsible for the higher content of vitamin D in the summer months. Vitamin D in the milk can, however, be increased by adding vitamin D concentrates or irradiated yeast to the cows' diet, or by actual irradiation of the milk itself (see Friedman 1934).

The amount of vitamin D required by the infant to protect it against the development of rickets is about 140 international units

a day. The quantity of milk an infant can drink contains far less than this amount. Fortunately, vitamin D is formed as the result of exposure of the skin to sunshine, so that in the summer rickets is unlikely to develop. In winter, however, when the ultra-violet rays of the sun are largely filtered off by impurities in the atmosphere, it is highly desirable to supplement the diet of the infant and growing child with some rich source of vitamin D, such as cod-liver oil.

Vitamin E. This substance appears to be necessary for the normal development and functioning of the testis in the male and the placenta in the female. The name " anti-sterility " vitamin, which has been applied to it, is misleading, since deficiency of vitamin A is also capable of leading to a failure in reproductive powers (Hopkins 1935). The chemical nature of vitamin E has recently been determined. Its presence in cows' milk has been a subject for dispute, Farine (1931), for instance, stating that no appreciable amount occurs, while certain other workers claim to have found it (see Mattill and Clayton 1926). More recent work indicates that it is present in very small quantity, but that the amount varies with the nature of the cows' diet.

Other Accessory Food Factors. Whether other vitamins occur in milk is not yet known with certainty. Elvehjem, Hart, Jackson and Weckel (1934), Stirn, Elvehjem and Hart (1935), and Kohler, Elvehjem and Hart (1937), working with rats in the United States, have brought some evidence to show the existence of a growth factor in summer milk which is absent from winter milk; but observations in this country by Henry, Ikin, and Kon (1937) on rats and by Drummond and his colleagues (1938) on children, have so far failed to confirm the findings of the American workers.

Summary

1. A table giving the approximate average composition of cows' and human milk is presented.

2. Cows' milk differs considerably from human milk. It contains two to three times as much total protein, of which more is constituted by caseinogen and less by lactalbumin. It contains over three times as much mineral matter, and is a particularly rich source of calcium and phosphorus. On the other hand it contains less sugar, and less iron and copper than human milk.

3. As regards vitamin content, cows' milk is richer than human milk in vitamin B_1, and probably in vitamins B_2 and D, but is slightly poorer in vitamin C and much poorer in Vitamin A.

4. To satisfy the requirements of the human infant, cows' milk, whether raw or pasteurized, if given as the sole source of nourishment, should be supplemented with iron, and possibly copper, with vitamin C, and in the winter months with vitamin D.

5. A brief account is given of the more important functions of the vitamins.

CHAPTER VIII

EFFECT OF PASTEURIZATION ON THE NATURAL CONSTITUENTS AND GENERAL PROPERTIES OF MILK

As a preface to this chapter it may be pointed out once again that we are concerned here with the effects of pasteurization as carried out under licence by the official holder process, in which the milk is exposed to a temperature of 145–150° F. (62·8–65·6° C.) for half an hour, and immediately cooled to 55° F. (12·8° C.) or below. If these conditions are not complied with and the heat treatment given is more severe, then changes may be apparent, particularly in the flavour and in the cream line, that are not observed in properly pasteurized milk. Some of the work to which reference must be made has been carried out with milk heated to a higher temperature or for a longer time than those laid down under the official regulations, or even submitted to boiling; and care must therefore be exercised in distinguishing between the effect of pasteurization as carried out by the official holder process and that of other methods of heat treatment. So few observations have yet been recorded on the changes produced in the milk by the recently authorized high temperature short time method of pasteurization (see p. 59) that we shall make no direct reference to them. Such information as we have, however, does not suggest that they are greater than those produced by the holder method.

As previously explained (p. 59) changes in bacteria and in the chemical composition of milk are a function of both time and temperature. Within a given range the same effects may be produced by exposure to a low temperature for a long time as to a high temperature for a short time. On this account both time and temperature will, when possible, be stated in describing the effects of exposure to heat, except when pasteurization under licence, or the corresponding conditions in the laboratory, are referred to.

Sugar, Fat, and Protein

Lactose. No change in this sugar is detectable as the result of pasteurization.

Fats. Chemically the fat is not altered by pasteurization, but the size of the fat globules is affected in such a way as to lead to a diminution in the proportion that rise to the surface on standing. According to Whittaker and his colleagues (1925) the average reduction

in the cream line of milk heated to 145–146° F. (62·8–63·3° C.) for half an hour is 8 per cent., and of milk heated to 148° F. (64·4° C.) is 31 per cent., but considerable variations are experienced with different milks. The same workers also found that the cream line could be partly preserved by cooling the milk to a low temperature after pasteurization. Similarly Weigmann (1932) showed that the more rapidly the milk was cooled after heating and the lower the temperature to which it was taken, the greater was the volume of cream that rose to the surface. In modern plants, in which the milk is heated to a temperature of about 145–146° F. (62·8–63·3° C.) and cooled rapidly to 40° F. (4·4° C.), the diminution in the cream line is very small indeed. It is hardly necessary to point out that the amount of fat in the milk and its nutritive value are not affected by the change in the physical aggregation of the fat globules resulting from heat treatment; a milk with a poor cream line after pasteurization contains just as much fat as the same milk with a good cream line before pasteurization.

Proteins. Caseinogen, which constitutes at least 85 per cent. of the protein of cows' milk, though readily precipitated by acids and coagulated by rennet, is very resistant to heat. Apart from the slight increase in coagulation time under the influence of rennet, which is due to its existence in milk in the form of calcium caseinogenate, it undergoes no detectable change as the result of pasteurization.

Lactalbumin and lactoglobulin, on the other hand, which are not coagulated by rennet, are susceptible to heat, and start to coagulate with a time-temperature exposure similar to that used in pasteurization. Numerous observations have been made by different workers on the amount of lactalbumin thrown out of solution on heating. Stirling and Blackwood (1933), who review the literature, conclude that about 5 per cent. of this substance is coagulated under the conditions of holder pasteurization. A figure of about the same order is reached in the analysis of various other papers by Rahn (1925) in Germany. Rowland (1933) found that 10·4 per cent. of the total soluble protein was denatured on heating to 63° C. for 30 minutes. It may be pointed out, however, that the total protein includes lactoglobulin which, though often present in the milk of cows suffering from mastitis, is practically absent from the milk of healthy cows. On the whole, it may be concluded, that the effect of holder pasteurization is to lead to the coagulation of about 5 per cent. of the lactalbumin fraction of the protein.

Whether this partial coagulation diminishes the physiological availability of protein has been questioned. According to Lane-Claypon (1916) coagulation of the albumin probably favours its

digestibility. This doubt has now been resolved by the experimental observations on calves of Blackwood, Morris, and Wright (1936) and on rats of Henry, Kon, and Watson (1937). Blackwood and her colleagues carried out three series of experiments on calves, the first and third lasting 72 days, the second 24 days. Altogether 16 calves were used. Raw and pasteurized milk were fed alternately, half the animals commencing with raw and half with pasteurized milk, so as to avoid any errors due to individual behaviour. The nitrogen intake in the diet and the output in the fæces and urine were measured. No significant differences were found in the assimilation and retention of nitrogen on raw and pasteurized milk.

In Henry, Kon, and Watson's experiments twelve young female rats belonging to two litters were divided when 25 days old into 6 pairs of litter-mates. The paired feeding method was used. After a preliminary period on a basal low-nitrogen diet the rats were fed for two periods on a basal nitrogen-free diet plus milk, the milk being given raw in one period and pasteurized in the other; a final period followed on the basal low-nitrogen diet. The nitrogen in the diet, the fæces, and the urine was measured, and the biological value of the proteins of the milk estimated by Mitchell's method. For raw milk a biological value of 80·73 and for pasteurized milk of 81·45 was obtained; this difference was not statistically important. The authors conclude that pasteurization has no significant effect on the biological value of the proteins of milk. This conclusion is supported by the findings of Fairbanks and Mitchell (1935) who, after more drastic heat treatment, detected practically no difference between the proteins of raw liquid milk and of milk dried by the low-temperature roller process.

It may be concluded that the slight degree of coagulation of the lactalbumin occurring during the pasteurization of cows' milk is without effect on its nutritive value.

Mineral Constituents. In studying the effect of pasteurization on the mineral constituents chief interest has centred on calcium and phosphorus, both of which are unusually abundant in cows' milk. Part of the calcium, estimated at about 25–30 per cent., is in the form of inorganic salts and is often referred to as the soluble or diffusible calcium; the remainder is combined chiefly with caseinogen and is non-diffusible. The phosphorus is in an even more complex state, some being present in the form of inorganic salts, some as protein, and some as lipoid. The soluble or diffusible fraction of the phosphorus constitutes about 30–45 per cent. of the total (see Allen 1931).

The effect of pasteurization on calcium and phosphorus has been studied by several workers, whose results have been reviewed by

Stirling and Blackwood (1933) and by Savage (1933*a*). The general findings indicate that about 6 per cent. of the calcium is rendered insoluble, though the total amount of calcium remains unaltered. The results with phosphorus are a little conflicting, some of the earlier workers reporting a slight diminution in the soluble fraction, and some of the later workers, like Mattick and Hallett (1929), failing to find any definite change.

More recently the problem has been submitted to careful study on both calves and rats. Blackwood, Morris, and Wright (1936) at the Hannah Dairy Research Institute, carried out three experiments on a total of 16 calves to compare the assimilation and retention of nitrogen, phosphorus, and calcium on raw and pasteurized milk. The results with nitrogen have already been described (p. 88). With regard to calcium and phosphorus the intake and output of these two substances were measured. No evidence was obtained to show that their retention was deleteriously affected by pasteurization of the milk.

Krauss, Erb, and Washburn (1933) in the United States compared the total ash, the calcium, and the phosphorus content of the bodies of 32 rats, 16 of which had been fed on raw and 16 on pasteurized milk for several weeks. No significant differences were found, and the authors concluded that the rats had utilized the minerals of pasteurized milk just as well as those of raw milk.

Henry and Kon (1937*a*) at Reading, using the paired-feeding method, endeavoured to find out whether commercial pasteurization [1] affects the availability of calcium and phosphorus for the growing rat. Twelve pairs of freshly weaned litter-mate rats of the same sex, comprising six pairs of bucks and six pairs of does, were fed on a basal diet of which more than 80 per cent. of the calcium and phosphorus were supplied by milk. The experiment lasted for five weeks, and the intake of these two elements in the diet and their excretion in the urine and fæces were measured. An additional estimate of the assimilation of calcium and phosphorus was made by killing the animals at the end of the experiment and comparing the calcium and phosphorus content of their tissues with that of litter-mates killed at the beginning of the experiment. The results of the two methods were in close agreement. It was found that the rats retained about 80 per cent. of the ingested calcium and phosphorus irrespective of whether they were given raw or pasteurized milk. The only statistic-

[1] For the sake of clarity it may be stated that the term "commercial pasteurization" used here and in the following pages refers to pasteurization in accordance with the Milk (Special Designations) Orders on a commercial scale in commercial pasteurizing plants.

ally significant difference was shown by the does receiving pasteurized milk, which retained 3 per cent. more calcium than their litter-mates on raw milk. The authors conclude that under the conditions of the experiment commercial pasteurization had no deleterious effects on the availability of the calcium or phosphorus of the milk.

A similar experiment, also lasting five weeks, was carried out on freshly weaned rats by Auchinachie (1937) at Aberdeen to control the results of Henry and Kon. Six sets of four litter-mates were used, three sets being male and three female. About 85 per cent. of the calcium and 75 per cent. of the phosphorus was retained independent of the nature of the milk. The author reached the conclusion that the retention of the calcium and phosphorus of the milk was unaffected by pasteurization.

From the practical point of view it is doubtful whether, if a minor change in the availability of these two substances did occur as the result of pasteurization, it would be of any importance in infant feeding. The content of calcium is three times and of phosphorus five times as high in cows' milk as in human milk, and there is therefore a large surplus available to meet the needs of the growing child.

With regard to *iodine*, Magee and Glennie (1928) compared the content of eight samples of milk before and after " pasteurization." The average figures obtained were 5·69 γ per 100 ml. for the raw and 4·51 γ per 100 ml. for the " pasteurized " milk (γ is 1/1000 milligramme). This was regarded as indicating a loss of 20 per cent. as the result of heating, apparently due to volatilization. The method of " pasteurization " employed consisted in heating the milk in a flask at a temperature of 70° C. (158° F.) for 30 minutes in a water bath, followed by rapid cooling (see Magee and Harvey 1926*a*). This treatment is far more severe than any method of pasteurization used in practice. Since the effect of heat appeared to be progressive—boiling brought about a 26 per cent. reduction—it is probable that the loss of iodine due to holder pasteurization (145–150° F. or 62·8–65·6° C. for half an hour) is less than 20 per cent. Observations on milk pasteurized under licence will have to be made before the exact effect of pasteurization on the iodine content of the milk can be stated.

Vitamins.

Vitamin A. Both vitamin A and its precursor carotene appear to be fairly resistant to heat (see Stirling and Blackwood 1933, Kon 1936). Even boiling the milk produces no significant diminution in the amount of these substances (Haas and Meulemans 1938). Quite recently Gillam, Henry, and Kon (1937) have made a special investi-

gation on the effect of commercial pasteurization on carotene and vitamin A in milk. Twenty samples of butter churned from raw and from pasteurized milk were examined over a period of five weeks. The vitamin A and carotene contents were estimated by Lovibond tintometer and spectrophotometric tests. Neither of these substances was found to be affected by pasteurization.

These results confirm those reported by Krauss, Erb, and Washburn (1933), who fed rats on a vitamin A deficient diet supplemented by various quantities of raw or pasteurized milk, and failed to detect any significant difference between the two groups of animals in their growth response or in their incidence of ophthalmia or respiratory disease.

Vitamin B. Stirling and Blackwood (1933), who review the literature, reach the conclusion that, in general, vitamin B_1 may be regarded as relatively thermolabile and vitamin B_2 as thermostable, and that the activity of the vitamin B complex in milk is probably unimpaired by pasteurization. On the other hand Dutcher, Guerrant, and McKelvey (1934), who made observations on rats, obtained evidence that pasteurization of milk at 62–63° C. (143·6–145·4° F.) for 30 minutes led to some destruction of both vitamin B_1 and B_2. Considerable variations, however, were observed, and the recorded figures in their paper are limited to " those animals which reacted most typically in their respective groups."

Krauss, Erb, and Washburn (1933), who carried out experiments on small numbers of rats fed on a diet deficient in vitamin B_1 or in the vitamin B_2 complex, obtained evidence that about 25 per cent. of the vitamin B_1 activity was destroyed by pasteurization, but that the vitamin B_2 complex was unaffected. According to Fixsen (1938) and Bigwood (1939) vitamin B_2 is unaffected by heating.

The subject has been investigated recently by Kon (1937) using rats. Seventeen pairs of litter-mate bucks and thirteen pairs of litter-mate does were placed after weaning on a diet deficient in the vitamin B complex. After the animals had begun to lose weight their diet was supplemented by 8 ml. daily of milk, one member of each pair receiving the milk raw, the other pasteurized. The experiment was continued for eight weeks, and the weight of the animals recorded. No significant difference was found at the end of this time between the does, but among the more rapidly growing bucks those receiving raw milk weighed on an average 10·2 gm. more than those on pasteurized milk. One buck on pasteurized milk developed symptoms of beri-beri on the 47th day of the experiment, while another at the end of the experiment showed some spasticity, but without any typical symptoms of beri-beri. The author concludes

that some loss of the vitamin B complex occurs as the result of commercial pasteurization. The amount could not be measured, but considering that it was detectable only with the members of one sex, it was probably quite small. In other experiments carried out by the same author (Henry and Kon 1937*b*) in which larger quantities of milk were given, no difference was found in the growth rate of rats fed on raw and pasteurized milk. Kay (1939), who reviews the evidence, concludes that 20–25 per cent. of vitamin B_1 is destroyed by pasteurization. Houston, Kon and Thompson (1940), however, using the fluorometric method, found that in commercially pasteurized milk the vitamin B_1 content was reduced by only about 10 per cent. Riboflavin was unaltered. Kon (1941) likewise puts the decrease in vitamin B_1 in pasteurized milk at 10 per cent.

It may be concluded that pasteurization leads to some destruction of vitamin B_1, amounting usually to 10 per cent. and at most to 25 per cent., but leaves the vitamin B_2 complex unaffected.

Vitamin C. Most workers have found that the vitamin C content of pasteurized milk is lower than that of raw milk. Stirling and Blackwood (1933) who review the literature, conclude that even under the most favourable conditions about 50 per cent. of the anti-scorbutic potency of the milk is lost during commercial pasteurization. The destruction is greater in the presence of copper, which catalyses oxidation, than in its absence. Some observers, such as King and Waugh (1934), have noticed very little destruction when the milk has been pasteurized in aluminium vessels, while others, such as Schwartze and his colleagues (1931), have found that, though copper leads to a far greater destruction than aluminium, nevertheless even in aluminium vessels the loss of vitamin C potency may reach 20–40 per cent. There is general agreement that less destruction occurs when milk is submitted to the short time high temperature process, i.e. 159·8–162° F. (71–72·2° C.) for 15–20 seconds, than when it is pasteurized by the usual holder method, i.e. 145–150° F. (62·8–65·6° C.) for 30 minutes (Rahn 1925, Sharp 1936, Whitnah *et al.* 1936). This may be due to the lessened exposure to oxygen in the former method.

On the whole the work of the last few years has indicated that the destruction of vitamin C that occurs during holder pasteurization is rather less than the 50 per cent. figure at which Stirling and Blackwood arrived. Reedman (1937), for example, states that the greatest destruction in his experience amounted to not more than 26·5 per cent., while Kon and Watson (1937*b*), who made observations over a period of four months on milk before and after processing, found that the average destruction of vitamin C during pasteurization was 20·8 per cent. Trout and Gjessing (1939), who compared the ascorbic

acid content of Grade A raw and of ordinary pasteurized milk after one day's storage at different times of the year, found that the average content was actually higher in the pasteurized than in the raw milk. Thus in the raw milk there were 6·0, 10·5, 8·8, and 7·7 mgm. per litre during winter, spring, summer, and autumn respectively, while the corresponding values for the pasteurized milk were 9·9, 12·9, 13·0, and 13·0 mgm. per litre. Holmes, Tripp, Woelffer and Satterfield (1939) in the United States of America measured the ascorbic acid content of pooled raw and pasteurized milk over a period of 18 months. They found that the maximum destruction of 53 per cent. occurred in August and the minimum of 4·5 per cent. in March. The average loss over the whole period due to pasteurization was 18·7 per cent.

Fresh light has been thrown on the problem by the recent studies of Kon and his colleagues at Reading. Kon's (1933) first observations showed that the vitamin C content of raw milk was apparently subject to marked and irregular fluctuations, and that even in raw milk of the highest quality produced under excellent conditions of feeding and management the vitamin C content suffered considerable loss within two to three hours of milking. Later, Kon and Watson (1936, 1937*a*) were able to show that this diminution was the result of exposure of the milk to light. As secreted by the cow the vitamin C, or ascorbic acid as it is now known to be, exists in the reduced form. Under the influence of the blue and violet rays of visible light, acting in the presence of oxygen, the ascorbic acid is oxidized to dehydroascorbic acid. In the early stages this oxidized product can be reduced by treatment with H_2S, but later dehydroascorbic acid undergoes decomposition and can no longer be converted back to the original reduced product. Guinea-pig tests show that the reversibly oxidized fraction is still biologically active, but that the irreversibly oxidized fraction has lost all its activity.

It was found that a pint bottle of milk exposed on the doorstep of a house for half an hour and then kept in darkness for an hour had lost fully half its original antiscorbutic potency. The vitamin C content of milk taken directly from the cow and containing only the reduced form of ascorbic acid was unaffected by pasteurization, provided copper utensils were avoided; but in milk that had been exposed to light for some time pasteurization destroyed the reversibly oxidized fraction of the ascorbic acid, while leaving the reduced form almost intact.

This work explains the divergent results obtained by previous observers. It shows that the destruction of vitamin C during pasteurization depends partly on the amount of previous exposure of the milk to light and partly on the presence of copper in the pasteurizing plant. If the milk after withdrawal from the udder is protected from light,

and if aluminium, stainless steel, or other copper-free materials are used for the processing equipment, the loss of vitamin C during pasteurization may be minimal. If, on the other hand, the raw milk is exposed to light and the processing equipment is made of copper, then very considerable destruction may occur.

Why vitamin C in milk should undergo photochemical oxidation when both ascorbic acid and oxygen are colourless has been the subject of inquiry by Hand, Guthrie, and Sharp (1938) in the United States. These workers bring evidence to suggest that lactoflavin—one of the vitamin B_2 components—which absorbs the blue rays of daylight, plays some part in the reaction. They found that when lactoflavin was removed from milk by adsorption, ascorbic acid was no longer oxidized by exposure to light. Lactoflavin, however, proved to be incapable of facilitating the oxidation of ascorbic acid unless oxygen was also present. If the dissolved oxygen was removed from the milk, practically no destruction of ascorbic acid occurred on exposure either to light or to pasteurization.

It will be realized that the extensive destruction of vitamin C which has hitherto been regarded as an inevitable accompaniment of pasteurization can be largely avoided. Provided the milk is protected from light after withdrawal from the udder, provided it is kept out of contact with copper during processing and cooling, and provided it is protected from light after bottling or is filled into amber bottles (Houston, Kon, and Thompson 1939), only a minimal loss of vitamin C need occur. Whether it will be possible in practice to remove the dissolved oxygen from the milk before processing is still in doubt, but the information already at our disposal makes it clear that the destruction of vitamin C during pasteurization should be much less in the future than it has been in the past.

Vitamin D. Vitamin D is known to play a large part in regulating the calcium metabolism of the body and to be concerned in the prevention of rickets and of dental caries (see Mellanby 1929, 1930, 1934). Since the observations of Blackwood, Morris, and Wright (1936) on calves, and of Henry and Kon (1937*a*) and Auchinachie (1937) on rats, which have already been described (pp. 89, 90), failed to show any diminution in the availability of calcium as the result of pasteurization, it seems very unlikely that the vitamin D is affected.

These metabolic experiments are supported by the observations of Kitchin and McFarland (1933), who, in a study of the development of rats' teeth, were unable to find any difference in the crystal structure of the enamel of animals fed on raw and those fed on pasteurized milk.

The only specific observations that appear to have been made on the effect of pasteurization on the vitamin D content of milk were

reported by Krauss, Erb, and Washburn (1933) in the United States. Two sets of experiments were carried out. In the first set weanling rats were placed for 3 weeks on a rickets-producing diet and were then given measured amounts of cream and butter-fat prepared from milk before and after pasteurization. After 10 days the animals were killed and the amount of healing in the bones determined by the line-test. In the second set of experiments rats were fed for 5 weeks on a rickets-producing diet supplemented from the start by known amounts of butter-fat made from raw or pasteurized milk. At the end of this time the animals were killed and the ash content of their femurs estimated. In neither the curative nor the prophylactic set of experiments was any difference apparent between the vitamin D content of raw and pasteurized milk. These results are in agreement with those of Daniels and Jordan (1928–1929), who found that even boiling had no detectable effect on the vitamin D. General experience, moreover, has shown that vitamin D is resistant to cooking (Fixsen 1938).

Vitamin D has now been obtained in a crystalline form and is known as calciferol. Observations in the laboratory show that it is even more resistant to heat and oxidation than vitamin A, which has been proved experimentally to be unaffected by pasteurization (see p. 90). It is therefore improbable that pasteurization causes any diminution in the vitamin D content of milk.

Vitamin E. Whether vitamin E, which appears to be present in very small quantity in cows' milk, is affected by pasteurization is unknown, but considering that it is very resistant to heat treatment when present in wheat-germ oil, this seems unlikely.

Enzymes. Though enzymes of different types are present in colostrum, in milk at the end of lactation, and in milk from diseased udders, they are found only in traces in milk secreted by perfectly healthy animals. Indeed their presence in healthy milk appears to be more or less accidental. Davies (1936) regards them as rudimentary, non-functioning constituents, on the ground that they bring about practically no changes in the milk for a considerable length of time. Most of the enzymes like the lipases, proteases, and peroxidases are not destroyed till they are heated to a temperature of 70–80° C. (158–176° F.) or higher. A few, like amylase and phosphatase, are inactivated by holder pasteurization, while others, like catalase and the Schardinger enzyme are weakened in their activity (Zeller *et al.* 1930). Phosphatase is destroyed almost completely by exposure to a temperature of 62·8° C. (145° F.) for 30 minutes, or of 72·2° C. (162° F.) for 15 to 20 seconds, and is now being used as a valuable index of the efficiency of the processing treatment (see p. 63). There is no evidence to suggest that any of these enzymes are of importance

in animal or human nutrition, and indeed most of them are probably destroyed during the natural processes of digestion.

Antibodies. Though often abundant in colostrum, antibodies are not found in milk secreted by the healthy udder. Their presence in milk shows that the mammary tissue is diseased and is unable to filter off the globulin in the blood serum with which the antibodies are associated. Since they are not normal constituents of milk, their behaviour under the influence of pasteurization need not be considered.

Bacteriostatic Property. In milk produced under cleanly conditions growth of bacteria, as estimated by the plate count, does not occur for some hours. This has generally been ascribed to the presence of a growth-inhibiting or actively bactericidal factor in the milk. How far this is true is rather doubtful. By use of the methylene blue reduction test it was shown by Wilson and his colleagues (1935), and later confirmed by Hobbs (1939), that, though the organisms may not divide for several hours, they are nevertheless growing in size and bringing about changes in the milk. In other words the lag phase affects multiplication rather than growth. It is possible that a real growth-inhibiting factor may exist in fresh clean milk, but its activity seems to be very much less than has been supposed by workers who relied exclusively on the plate count for its measurement. The effect of pasteurization on the multiplication-inhibiting factor seems to be negligible. Destruction does not occur till it has been exposed to a temperature of 70–75° C. (158·0–167·0° F.) for one hour.

The statement frequently made that bacteria grow more rapidly in pasteurized than in raw milk is without adequate foundation, provided the comparison is made between samples of the same milk before and after pasteurization and not between different milks of different ages and different degrees of cleanliness. It is admitted that, if milk is withdrawn from the udder under sterile conditions, one half of it left raw and one-half pasteurized, and both halves are inoculated with the same number of organisms, the plate count after a given time may be higher in the pasteurized than in the raw sample (Allen 1916). This, however, may be explained by the presence in raw milk of a non-specific agglutinating property which is responsible for the growth of many of the organisms in clumps. This property is destroyed by pasteurization, so that the organisms tend to separate more freely after division and therefore give rise to a larger number of colonies in the plate count (Hobbs 1939).

The only satisfactory way of comparing the growth rate in raw and pasteurized milk is to make use of some test that does not depend upon the nature of the spatial distribution of the organisms. Allen (1917), for example, compared the rate of gas formation from lactose

by coliform organisms growing in milk. He found that gas was formed rather more rapidly and more abundantly in pasteurized than in raw milk, suggesting that pasteurization favoured bacterial multiplication. Interesting as his results are, it would be dangerous to generalize from them, partly because they were carried out with organisms belonging to only one group, partly because the number of strains examined were few, and partly because no information is given as to whether the amount of dissolved oxygen in the two milks was equalized before inoculation of the organisms.

Wilson and his colleagues (1935), working with milk drawn under aseptic conditions, and inoculated with dirty raw milk, found that pasteurization led to some decrease in the reduction time of methylene blue. On the other hand they found that the average keeping quality of commercially pasteurized milk was longer than that of Certified milk, even though the average bacterial content at the time of examination was higher in the pasteurized samples.

The observations of Hobbs (1939), in which the metabolic activity of several different types of organisms was estimated by the methylene blue reduction test, showed that the growth rate varied from milk to milk, and from one type of organism to another. As a rule the growth rates in raw and pasteurized milk were very similar or actually identical; sometimes the growth rate was slightly higher in raw than in pasteurized milk, sometimes the reverse. Taking all the observations together there was little to suggest that pasteurization favoured the growth of organisms in milk.

There are clearly a number of factors that influence the growth rate of micro-organisms in raw and pasteurized milk, and until further observations have been made it would be unjustifiable to conclude that pasteurization favours bacterial growth.

Flavour. It is often stated that pasteurization imparts a cooked flavour to the milk. That so-called pasteurized milk may have an unpleasant flavour is not denied, but this is generally attributable to the fact either that the heat treatment of the milk has been more severe than that laid down under the official regulations, or that the milk contains aromatic substances derived from the diet of the cow or formed as the result of bacterial growth. Clean milk from cows on a suitable diet, pasteurized at a temperature of 145–150° F. (62·8–65·6° C.) for half an hour, cooled immediately to a temperature of 55° F. (12·8° C.) or below, and consumed fresh or after refrigeration has practically no detectable cooked flavour. By some persons it can be distinguished from fresh raw milk, not so much on account of its positive cooked flavour, as on account of its absence of that peculiar "cow" flavour which is present in milk recently drawn

from the udder or contaminated with manure, and which is largely removed by filtration and heating. Both the " cow " flavour of raw milk and the very slightly cooked flavour of pasteurized milk can be considerably reduced by aeration.

According to Gould and Sommer (1939) the development of a cooked flavour in milk is, like so many other changes, the resultant of both time and temperature. Under the conditions of their experiments, in which approximately 15 minutes were required to raise the temperature of the milk, the cooked flavour appeared after momentary heating at 76–78° C. (168·8–172·4° F.), after 3 minutes' heating at 74–76° C. (165·2–168·8° F.), or after 30 minutes' heating at 70–72° C. (158–161·6° F.). Reducing the acidity of the milk, increasing its butter-fat content, or adding sodium sulphite lowered the temperature at which the cooked flavour appeared. On the other hand increasing the acidity, lowering the butter-fat content, or adding copper raised the temperature at which it appeared. Evidence is brought to show that coincident with the development of the cooked flavour volatile sulphides were liberated and the oxidation-reduction potential of the milk began to fall. It is therefore concluded that the cooked flavour in milk is largely due to the production of volatile sulphides. It will be noted that the minimum temperature at which the cooked flavour developed in 30 minutes, namely 70–72° C. (158–161·6° F.), is considerably above the maximum temperature permitted in holder pasteurization in this country, namely 65·6° C. (150° F.).

Experiment to find out whether Pasteurized Milk can be distinguished by flavour from Raw Milk. Since the objection to pasteurized milk on account of its flavour is of some practical importance, it was determined to carry out an experiment to see what substance of truth there is in it. Though several observations of this type have been previously reported, particularly in the United States, we know of none in which such care was taken to avoid all possible sources of error. The experiment was carried out with the help of Dr. Tangye, County Medical Officer of Health, Wiltshire, on May 31, 1939, at Trowbridge.

Thirty milks were collected in pint or half-pint bottles, 12 in Wiltshire and 18 in London. They were of the following types :—

Raw Certified, 1 Raw Tuberculin Tested (T.T.), 4 Raw Accredited, 7 Raw Bulked, 2	. . .	14
Pasteurized Tuberculin Tested, 1 Pasteurized Homogenized, 1 Pasteurized, 14	. . .	16

All the raw milks were of different origin. Of the pasteurized milks other than the two Tuberculin Tested and homogenized samples, six came from different plants in Wiltshire, and eight from two firms in London. With these eight samples care was taken that the milk was derived either from different plants, or at different times from the same plant. No two samples were alike.

The milks were numbered more or less at random by persons not taking part in the actual tasting experiment. The order in which they were given out was determined by a second series of random numbers contained in Tippett's "Tracts for Computers, No. 15." Thus there was a double randomization, which would have effectively prevented any person who could have observed and memorized the labels, i.e., raw or pasteurized, on the bottles from making use of this knowledge. Any such observation, however, was rendered impossible by covering over the labels with sticky tape. With the exception of two samples, all milks had been collected on the previous day. The bulked raw and pasteurized milks were probably a day older than the single-herd milks, but exact information on this point was not obtainable. The average age of the pasteurized milks, determined from the time at which the milk was withdrawn from the cow, was probably several hours greater than that of the raw milks. All milks were kept cool either by storage in a refrigerator or by being packed around with dry ice; but for some time before distribution they were allowed to warm up, so as to reach a suitable drinking temperature.

Nine observers took part in the experiment. They were as follows:

Observer No. 1. County Sanitary Inspector. Male.
,, ,, 2. Clerk. Athlete. Big milk drinker. Male.
,, ,, 3. Wife of Agricultural County Organizer. Mother of a family.
,, ,, 4. School girl. Volunteered. Age 12.
,, ,, 5. School girl. Age 11. Said to be able to distinguish raw from pasteurized milk.
,, ,, 6. School boy. Age 12. Volunteered.
,, ,, 7. ,, ,, ,, 12. ,,
,, ,, 8. Veterinarian. Drinks raw milk only, and is opposed to Pasteurization. Male.
,, ,, 9. Doctor. Interested in milk and supports Pasteurization. Male.

The observers were seated around three sides of a square in the middle of which was a clerk who dispensed the milks. Each observer

had five glasses bearing his own number. Four of these were used for milk; the other contained water for rinsing out the mouth in between each milk. Dirty glasses were collected, washed out, and returned by assistants. After tasting each milk, the observer stated on a form whether he considered it raw or pasteurized, and made any relevant remarks about it. Once they were put down these decisions were not subsequently altered. Some persons swallowed the milk, others spat it out. Only one observer, No. 9, knew exactly how many milks were raw and how many were pasteurized; but

TABLE XX

MILK TASTING EXPERIMENT

Description of different milks by different observers on basis of flavour

Real No.	Order given out.	Observers. 1	2	3	4	5	6	7	8	9	No. Raw.	No. Past.	Type of Milk.
5	1	P	*R*	*R*	P	*R*	*R*	*R*	*R*	P	6	3	Past.
29	2	*R*	*R*	*R*	*R*	*R*	P	*R*	*R*	*R*	8	1	Certified *Raw.*
26	3	*R*	P	P	*R*	P	P	P	P	*R*	3	6	T.T. *Raw.*
17	4	*R*	*R*	*R*	P	P	P	P	P	*R*	4	5	Past.
2	5	*R*	*R*	*R*	*R*	*R*	P	P	*R*	*R*	7	2	*Raw* Accred.
27	6	P	P	P	P	P	*R*	P	P	P	1	8	Bulk *Raw.*
30	7	*R*	P	P	*R*	*R*	*R*	*R*	*R*	P	6	3	Past.
11	8	P	*R*	*R*	*R*	*R*	*R*	P	*R*	P	6	3	*Raw* Accred.
7	9	P	P	P	*R*	P	P	*R*	P	P	2	7	Past. homogenized
4	10	*R*	*R*	*R*	P	*R*	*R*	*R*	*R*	P	7	2	Past.
12	11	P	P	*R*	P	*R*	*R*	P	P	*R*	4	5	*Raw* bulk
25	12	*R*	P	P	*R*	P	P	*R*	P	P	3	6	*Raw* Accred.
20	13	P	P	P	*R*	P	P	P	*R*	*R*	3	6	Past.
28	14	*R*	*R*	*R*	*R*	P	P	*R*	P	*R*	6	3	Past.
18	15	P	P	P	*R*	P	*R*	*R*	*R*	*R*	5	4	Past.
21	16	*R*	P	*R*	P	*R*	P	P	P	*R*	4	5	*Raw* Accred.
24	18	P	P	P	P	P	P	*R*	P	P	1	8	Past.
14	19	P	*R*	P	P	P	*R*	P	*R*	P	3	6	Past.
10	20	P	P	*R*	*R*	*R*	*R*	*R*	*R*	P	6	3	Past.
1	21	*R*	P	*R*	*R*	*R*	P	*R*	*R*	*R*	7	2	T.T. *Raw.*
22	22	P	*R*	P	P	P	P	*R*	*R*	*R*	4	5	Past.
23	23	P	*R*	*R*	P	*R*	*R*	*R*	*R*	*R*	7	2	T.T. *Raw.*
6	24	*R*	*R*	P	*R*	P	*R*	P	P	P	4	5	Past. single herd
9	25	P	P	P	*R*	P	*R*	P	P	*R*	3	6	*Raw* Accred.
13	26	*R*	P	*R*	P	*R*	P	*R*	P	*R*	5	4	*Raw* T.T.
16	27	P	*R*	P	*R*	P	*R*	P	*R*	*R*	5	4	Past.
19	28	*R*	*R*	*R*	*R*	P	P	*R*	*R*	*R*	7	2	Past. T.T.
3	29	P	*R*	*R*	P	P	P	P	P	*R*	3	6	Past.
15	30	*R*	*R*	*R*	P	*R*	*R*	*R*	*R*	*R*	8	1	*Raw* Accred.
31	31	P	P	*R*	P	*R*	P	*R*	*R*	P	4	5	*Raw* Accred.
		14*R*	15*R*	17*R*	16*R*	14*R*	14*R*	17*R*	17*R*	18*R*	142	128	14*R* 16P
		16P	15P	13P	14P	16P	16P	13P	13P	12P	270		

Milk No. 8, given out as No. 17, has been omitted, since it was not pasteurized under licence.

probably two other observers, Nos. 1 and 2, suspected that they were more or less equal in number. Each milk was dispensed in turn. After the first twenty milks, a five-minute interval was allowed. The experiment was commenced about 2 to 3 hours after the midday meal and took about 50 minutes to complete.

The detailed results are recorded in Table XX.

This table is analysed in Table XXI.

TABLE XXI

MILK TASTING EXPERIMENT. ANALYSIS OF TABLE XX

Raw Milks 14.
Pasteurized Milks 16.
No. of observers 9.
Total no. of observations 270.
No. of observations describing milk as Raw 142.
" " " " Pasteurized 128.

Observer No.	Described Raw as Pasteurized.	Described Pasteurized as Raw.	Total No. of Mistakes.
1	6	6	12
2	9	10	19
3	4	7	11
4	7	9	16
5	4	4	8
6	8	8	16
7	7	10	17
8	7	10	17
9	4	8	12
Total	56	72	128
Expected No.	63	72	135

On the assumption that the distinction by flavour of raw and pasteurized milk is a pure matter of chance, there should have been 135 mistakes; in fact there were 128 mistakes. This difference is less than the standard error and is not significant. It will be observed that the actual number of mistakes in describing pasteurized milks was identical with the expected number, but that the number of raw milks wrongly identified was rather less than what would have been expected on chance alone. This difference is certainly not significant. If it means anything, it suggests that raw milk may have a slightly more distinctive flavour than pasteurized milk.

The expected number of mistakes made by each individual observer was of course 15; in fact, the number varied from 8 to 19, with an arithmetic mean of 14·2. One observer, No. 5, made only 8 mistakes

out of 30. The odds against this occurring by chance alone are over 100 to 1, and it would appear as if this particular observer, a schoolgirl aged 11, was in fact able quite frequently to identify correctly raw and pasteurized milks. None of the other observers was able to do this.

A further analysis of the results is given in Table XXII.

TABLE XXII

MILK TASTING EXPERIMENT

Analysis of sub-groups, based partly on information not included in Table XX

Sub-Group.	No. of Persons.	Nos. of Observers.	No. of Mistakes.	No. of Mistakes per Person.
Males	6	1, 2, 6, 7, 8, 9.	93	15·5
Females	3	3, 4, 5.	35	11·7
Adults	5	1, 2, 3, 8, 9.	71	14·2
Children	4	4, 5, 6, 7.	57	14·3
Milk drinkers	7	1, 2, 3, 4, 5, 7, 8.	100	14·3
Non-milk drinkers	2	6, 9.	28	14·0
Swallowed Milk	5	1, 2, 3, 4, 8.	75	15·0
Did not swallow Milk	4	5, 6, 7, 9.	53	13·3
Smokers	4	1, 3, 8, 9.	52	13·0
Non-smokers	5	2, 4, 5, 6, 7.	76	15·2

Sub-division of the observers into groups seems to show that the only difference of any possible importance is that between males and females. The number of observers, however, was very small, and the results were influenced to a considerable extent by the inclusion in the small group of females of Observer No. 5, who made only 8 mistakes.

Further analysis did not suggest that fatigue played any part in the results. On the contrary, the number of mistakes made diminished progressively throughout the experiment. Thus, in the first ten milks there were 45 out of 90 possible mistakes, in the second ten milks 42, and in the third ten milks only 41 mistakes.

Unfortunately very few remarks were made about the flavours of the milks. Only 39 observations of this type were recorded, and all but 6 of them were made by two observers, Nos. 8 and 9. For what they are worth, however, they are reproduced in Table XXIII.

It will be noted that the most frequent epithets applied to raw milk were salty, sweetish, sickly, and flavourless, and to pasteurized

TABLE XXIII

MILK TASTING EXPERIMENT. REMARKS ON FLAVOUR

Description of Flavour.	No. of Observations.	Type of Milks so described and No. of Observations.	
Cooked, boiled, or burnt	8	Pasteurized	5
		Pasteurized homogenized	2
		Raw Bulked	1
Sweet or sweetish	8	Pasteurized	4
		Raw bulked	1
		Raw Accredited	3
Salty	5	Raw Accredited	3
		Raw bulked	1
		Raw T.T.	1
Rich and creamy	3	Pasteurized	2
		Pasteurized T.T.	1
Rich, or very rich, and sweetish	3	Pasteurized	2
		Pasteurized homogenized	1
Rich and salty	1	Pasteurized	
Flat and salty	1	Pasteurized	
Sickly	3	Raw Accredited	1
		Raw T.T.	1
		Raw bulked	1
Rather flavourless	3	Raw T.T.	2
		Raw Accredited	1
Distinctive smell	2	Raw Accredited	
Unpleasant	1	Pasteurized	
Horrible metallic	1	Raw Accredited	
Total	39		

milk cooked, boiled or burnt, sweetish, rich and sweetish, and rich and creamy. The fact that pasteurized milk was described seven times as cooked, boiled, or burnt, and raw milk only once, does lend some support to the statement that pasteurized milk may have a slightly cooked flavour. It is quite clear, however, from Tables XXI and XXII that the majority of people are unable to distinguish raw and pasteurized milk by this means.

SUMMARY

1. In an experiment in which nine different observers tasted 14 raw and 16 pasteurized milks distributed at random the number of mistakes made was almost the same as would have been expected by chance alone on the assumption that there is no characteristic difference between them.

2. Only one observer, a schoolgirl aged 11, succeeded more often than would have been expected by chance alone in correctly identifying

the nature of the milks; and even she made 8 mistakes out of 30. None of the other observers was able to approach this record.

3. The number of observations on flavour was very small, and little attention can be paid to them. The epithets cooked, boiled, or burnt were applied to pasteurized milk seven times and to raw milk only once. There is therefore some ground for the assertion that pasteurized milk may have a slightly cooked flavour. That the majority of people, however, are unable to distinguish raw from pasteurized milk by this means is clear from the fact that of 144 observations on the 16 pasteurized milks, 72 were entered as raw and 72 as pasteurized.

4. The difference in taste between different raw milks appears to be as great as, or greater than, the difference between raw and pasteurized milk. Though many persons can detect a difference in flavour between different milks, it is quite exceptional for any one to be able with a reasonable degree of certainty to identify a given milk as raw or pasteurized.

Coagulability by Rennet. Mattick and Hallett (1929) compared the coagulability by rennet of raw milk and of milk heated for half an hour to temperatures ranging between 105° F. and 209° F. (40·6–98·3° C.). When the comparison was made immediately after heating, coagulation occurred rather more rapidly in milk heated to between 105° and 140° F. (40·6–60·0° C.) than in raw milk, but if the comparison was delayed for a few hours, the reverse occurred. Milk heated to between 145° and 151° F. (62·8–66·1° C.), that is under approximately the same time-temperature conditions as those laid down officially for pasteurization, coagulated at about the same rate as raw milk when the examination was made immediately after heating; but when the heated milk was left for 5 hours before being examined, its coagulation time was increased by about 25 per cent. At higher temperatures rennet coagulation took longer and longer, till in milk heated to near the boiling point it failed to occur within any reasonable time. General confirmation of these results has been brought by Moir (1930, 1931), who obtained evidence that the increased delay in rennet coagulation of heated milk was related to the decrease in the amount of soluble nitrogen found in the whey.

The mechanism of rennet coagulation is still obscure. It is known to depend on the presence of calcium salts, and it has been suggested that the decreased susceptibility of heated milk to coagulation with this enzyme is due to the partial precipitation of calcium salts that occurs. This suggestion is supported by the observation that the coagulability of heated milk can be restored by the addition of calcium salts. Other observations, however, render it doubtful whether

this explanation is wholly satisfactory (see Stirling and Blackwood 1933).

Digestibility. Numerous comparative observations have been made on the digestibility of raw and heated milk. Many of them have been conducted under highly artificial conditions, and their results afford little indication of what happens under natural conditions in the human intestinal tract. The subject is complicated by the differences in the gastric acidity existing in the infant and the adult. Reviewing the literature up to 1932, Stirling and Blackwood (1933) were unable to come to any definite conclusion on the comparative digestibility of raw and pasteurized milk.

Since that time Andross (1933), using the *in vitro* method, has made observations on the digestibility of raw, pasteurized, and boiled milk. She found that peptic digestion occurred best with raw milk, but that when pepsin was followed by trypsin, as occurs naturally in the human alimentary tract, the weight of the curd residue was practically the same for all three milks.

Ogilvie and Peden (1934), using the *in vivo* method, studied the gastric digestion of seventeen infants fed on raw and on boiled milk. Their observations included the free hydrochloric acid, total chlorine, total acidity, peptic activity, soluble non-protein nitrogen, and soluble calcium in the test-meal fluid. No significant differences were found, and the authors concluded that the digestion of milk was not adversely affected by boiling.

Graham and Morris (1937) fed two children alternately on raw and boiled milk, and measured the retention of calcium, phosphorus, and nitrogen, and the percentage absorption of fat. No evidence was obtained that during the five weeks of the experiment either the absorption or the utilization of the milk was impaired by boiling.

The last two experiments, it will be noted, were carried out with boiled milk. Since boiling is known to produce greater changes in the protein and the fat than pasteurization, it seems very unlikely that, when no effect can be demonstrated as the result of boiling, pasteurization can decrease the digestibility of the milk.

The most recent observations, which were made by Henry, Kon, and Watson (1937) on rats, and which have already been described (p. 88), afford no evidence that pasteurization lowers the digestibility of milk protein. For raw milk a true digestibility figure was obtained of 95·05, and for pasteurized milk of 95·63. The difference was insignificant.

Many years ago Lane-Claypon (1916) drew attention to the fact that heated milk was more readily digested by the human infant than raw milk on account of the greater softness and more open texture

of the clot. Lane-Claypon was working mainly with boiled milk, but there is reason to believe that the effects of pasteurization in rendering the clot more digestible are similar in kind though less in degree to those produced by boiling. Krauss, Erb, and Washburn (1933), for example, who compared the curd tension of milk before and after pasteurization, found that the curd was usually rather softer, and therefore presumably more digestible, after heat treatment. McCollum (1934), in a summary of the effects of pasteurization, likewise concludes that pasteurized milk is more easily digested than raw milk.

The available evidence, therefore, lends no support to the view that the digestibility of milk is in any way lowered by pasteurization. Indeed it points rather in the opposite direction.

Keeping Quality. Owing to the destruction of a high proportion of the bacteria in raw milk (see p. 57), pasteurization leads to an improvement in its keeping quality. The amount of increase will vary with the nature of the milk and the type of bacterial flora present. Generally speaking, the life of commercial milk is prolonged at ordinary atmospheric temperature by about a day.

Summary

Pasteurization of cows' milk by the holder method at 145–150° F. (62·8–65·6° C.) for half an hour followed by immediate cooling to 55° F. (12·8° C.) or below has the following effects:

(1) The cream line is reduced by about 10–30 per cent.

(2) About 5 per cent. of the lactalbumin is coagulated.

(3) There is a diminution in the soluble calcium and phosphorus, estimated variously by different workers, but approximating to a figure of about 5 per cent.

(4) Some of the iodine—almost certainly less than 20 per cent.—may be driven off as the result of volatilization. Since, however, no observations appear to have been made yet on milk pasteurized under licence, it is impossible to say what the effect on the iodine content really is.

(5) There is some destruction, amounting usually to 10 per cent. and at the most to 25 per cent., of vitamin B_1.

(6) There is a diminution in the vitamin C content, which is dependent on the degree of previous exposure of the milk to light, the presence of copper in the pasteurizing plant, and the amount of dissolved oxygen. At present this destruction is of the order of 5–50 per cent., with an average of about 20 per cent., but there is no reason why in future the greater part of it should not be avoided.

(7) The flavour of the milk is somewhat altered, owing to the

removal by filtration and heating of volatile substances derived from the cow itself or from manure. The acquirement of a cooked flavour is practically negligible in properly pasteurized milk, and very few persons are able to distinguish it. This was shown by a special experiment summarized on p. 103.

(8) In milk that has been pasteurized some hours previously, but not in freshly pasteurized milk, the coagulation time with rennet is increased by about 25 per cent. On the other hand, the resulting clot tends to consist of finer particles and to be of a more open texture than that formed from raw milk.

(9) Owing to the change in the rennet clot the digestibility of the milk for infants is perhaps slightly improved by pasteurization.

(10) Certain enzymes, including phosphatase, are destroyed by pasteurization. Since, however, these enzymes are probably destroyed in any case during the natural processes of digestion, there is no reason to believe that their presence in milk is of any value to the animal or human subject.

(11) A non-specific agglutinating factor, which favours the growth of micro-organisms in clumps rather than in smaller units, is destroyed by pasteurization. This factor is not to be confused with the so-called growth-inhibitory or bacteriostatic factor, which is resistant to pasteurization.

(12) The keeping quality of the milk is increased considerably.

Against these positive changes may be set a number of negative findings. Thus it is found from feeding experiments on rats and calves that the biological value and digestibility of the proteins is not decreased, that there is no detectable change in the availability of calcium and phosphorus, that there is no loss of vitamin A or D, and that the total energy value of the milk remains unaltered (see Chapter IX).

The differences between raw and pasteurized milk are far less than those between cows' milk and human milk, or even between summer and winter samples of raw milk (see Kay 1939).

CHAPTER IX

EFFECTS OF PASTEURIZATION ON THE TOTAL NUTRITIVE VALUE OF MILK

Numerous experiments have been carried out on animals and children in an endeavour to determine whether pasteurization lowers the total nutritive value of cows' milk. Many of these have been reviewed by Stirling and Blackwood (1933), Savage (1933*a*), Brown (1931), and Krauss, Erb, and Washburn (1933), and need be referred to only very briefly here.

Before describing the more recent experiments it may be pointed out that much of the earlier work was vitiated by faults of one type or another. Sometimes the raw and pasteurized milks that were compared were not derived from the same source, and differed in their content of fat, minerals, and other constituents ; at other times the nutritive comparison was obscured by the development of infection conveyed by the milk or occurring naturally ; and in many experiments the number of animals observed was so small that the conclusions drawn by the authors could not be supported by statistical analysis. In the following account it will be convenient to deal with the observations on different animals separately.

Rats. Early Experiments. Of the early work on rats reference must be made briefly to the experiments carried out by Scott and Erf (1931) in the United States, and to those of Mattick and Golding (1931) at Reading, since they have been widely quoted in support of the view that pasteurization lowers the nutritive value of the milk. Scott and Erf's experiments have been reviewed and severely criticized by Brown (1931) and by Krauss, Erb, and Washburn (1933). Suffice it to say that in three out of four of their experiments the raw milk was drawn from cows to whose regular feed had been added a complex mineral mixture, consisting of bone meal and other ingredients, and of dried fish-meal. The pasteurized milk was procured from a corner grocery store, came from cows presumably receiving no such dietetic supplement, and had a rather lower butter-fat content than the raw milk. In the one experiment in which the two milks were derived from the same source, only eight animals were included in the " pasteurized " group.

Mattick and Golding's experiments (1931) have been reviewed and criticized by Brown (1931) and by Stirling and Blackwood (1933) and need not be described in detail here. The number of animals used

was very small, and the main differences observed were between raw and sterilized milk. Only one experiment is described in which the effect of raw and pasteurized milk, supplemented by a white flour biscuit, was compared. After four months on this diet the average weight of 6 bucks in the raw milk group was 256 gm., and of 6 bucks in the pasteurized milk group 235 gm.; the corresponding weights of the 6 does in each group were 184 gm. and 183 gm. The fertility of the two groups was very similar (see p. 129), but whereas 18 young animals from 4 litters were reared in the raw milk group, only 7 young animals from 1 litter were reared in the pasteurized milk group. Mattick and Golding also found that generation after generation of healthy young rats could be reared on a diet of raw milk and white flour biscuit. A comparison in this respect with pasteurized milk was not made till later (see p. 111).

Nutritional Anæmia of Rats. What is particularly striking in the work of these two sets of observers is that the rats are stated to have developed satisfactorily, either on an exclusive milk diet as in Scott and Erf's experiments, or on a milk diet supplemented only by a white flour biscuit as in Mattick and Golding's work.

Repeated observations by numerous workers have rendered it abundantly clear that young rats cannot thrive on an exclusive diet of cows' milk; instead they develop a progressive anæmia, cease to grow, lose weight, and sooner or later die. By the addition of iron, copper, and manganese to the milk this nutritional anæmia can be prevented (see, for example, Waddell, Steenbock *et al.* 1928; Waddell, Elvehjem *et al.* 1928; Hart *et al.* 1928; McHargue *et al.* 1928; Titus *et al.* 1928; Waddell *et al.* 1929; Krauss 1929*a*, *b*, 1930; Nevens and Shaw 1930; Stein and Lewis 1932; Elvehjem and Sherman 1932; Kemmerer *et al.* 1932; Josephs 1932; Orten *et al.* 1932; Mitchell 1932; Drummond 1933; Rominger *et al.* 1933; Keil *et al.* 1933; Schultze and Elvehjem 1933; Underhill *et al.* 1933; Geraghty *et al.* 1933; Bing *et al.* 1934; Stein *et al.* 1936). Cows' milk is too poor in those minerals required for blood formation to enable the rat to develop satisfactorily. For this reason all the later workers, with the exception of Mattick and Golding (1936), have added a mineral mixture to the diet when comparing raw and pasteurized milk.

Rats. Later Experiments. In an endeavour to confirm Mattick and Golding's observations Drummond (1933) carried out three experiments. In the first experiment litter mates were divided into two groups, each containing 5 bucks and 7 does. Milk was given *ad libitum*, and supplemented by biscuit made from white flour and water. The milk was pasteurized, not in the laboratory, but in a commercial plant of the holding type. Both the raw and the pas-

teurized milk were derived daily from the same bulk supply. The experiment was continued for one year. No detectable difference occurred in the rate of growth of the animals in the two groups during the first 6 months, but at the end of 50 weeks the average weight of the animals on raw milk was rather greater than of those on pasteurized milk. The actual figures were 354 gm. and 250 gm. respectively for the bucks and does in the raw milk group, and 312 gm. and 214 gm. for those in the pasteurized milk group. Post-mortem examination suggested that this difference was attributable to a greater storage of fat rather than to an increase in the body-weight, but no definite conclusions on this point were reached. None of the animals became definitely anæmic, because of the presence of appreciable quantities of iron and copper in the white flour biscuit, which had been baked in metal trays. Attempts at breeding proved unsatisfactory, and in neither group could any litters be raised (see p. 132).

Thinking that there might be some difference between the quality of the milk or the biscuit in London and those used by Mattick and Golding at Reading, Drummond carried out a second experiment in which a comparison was made between London raw milk and London biscuit and Reading raw milk and Reading biscuit. Four groups of animals were observed over a period of 26 weeks. No difference in their rate of growth was detectable and reproduction was again unsatisfactory (see p. 132).

In his third experiment Drummond put up eight groups of animals, each containing 5 bucks and 5 does from divided litters. London milk and London biscuit were used, and the diet of different groups was supplemented by yeastrel as a source of Vitamin B, or by a mineral mixture containing iron and copper, or by both. The experiment lasted 24 weeks. No significant difference was observed in the rate of growth of the animals in the various groups (Table XXIV).

Breeding was again unsatisfactory on the milk and biscuit diet alone, but was improved when supplements of yeastrel or iron and copper were added (see p. 132).

Drummond was thus unable to confirm Mattick and Golding's conclusion that pasteurization lowered the nutritive value of the milk. He further differed from these workers in finding that a simple diet of milk and white flour biscuit was insufficient to enable young female rats to reproduce and rear their litters satisfactorily.

Krauss, Erb, and Washburn (1933) in the United States, using the paired feeding method, made observations on twelve pairs of rats. The diet consisted exclusively of raw or pasteurized milk supplemented in each instance by a mixture of ferric chloride and copper sulphate. The experiment was continued for 12 weeks. No significant difference

TABLE XXIV

DRUMMOND'S THIRD EXPERIMENT. AVERAGE WEIGHT OF RATS FED ON RAW OR PASTEURIZED MILK AND WHITE FLOUR BISCUIT WITH SUPPLEMENTS OF YEASTREL AND MINERAL MIXTURE

Group.	Type of Milk.	Supplements.		Average weight in Grams.			
				12 weeks.		24 weeks.	
		Yeastrel.	Mineral Mixture.	Bucks.	Does.	Bucks.	Does.
1	Raw	−	−	179	151	266	183
2	Past.	−	−	178	140	261	179
3	Raw	+	−	180	152	248	192
4	Past.	+	−	183	140	237	162
5	Raw	−	+	173	148	252	189
6	Past.	−	+	170	146	239	180
7	Raw	+	+	180	153	249	186
8	Past.	+	+	172	141	240	180

was found in the rate of growth between the animals on raw and those on pasteurized milk.

Channon and Channon (1936) at Liverpool likewise endeavoured to repeat Mattick and Golding's observations, restricting themselves, however, to a comparison of raw and sterilized milk. This comparison has no bearing on our particular problem, but it may be noted that the animals on raw milk showed evidence of marked anæmia within a month. Even when their diet was supplemented by a mineral mixture containing iron, copper and manganese, their reproductive behaviour proved unsatisfactory, since the does were unable to rear their litters.

Mattick and Golding (1936) reported further observations on the growth and reproduction of rats fed on a diet of raw or pasteurized milk supplemented by a white flour biscuit. The reproductive behaviour of these animals will be described later (see p. 130). The effect on growth was estimated by weighing the animals in successive generations when they were four months old. Breeding failed in the sixth generation in the raw milk group and in the fifth generation in the pasteurized milk group so that only four generations are available for comparison (Table XXV).

It will be noted that the average weight of the animals fed on raw milk was rather greater than of those fed on pasteurized milk, but the differences were not large and the number of animals available for comparison was sometimes very small. Some of the second and

TABLE XXV

MATTICK AND GOLDING'S (1936) SECOND EXPERIMENT. AVERAGE WEIGHT OF YOUNG RATS AT 120 DAYS IN SUCCESSIVE GENERATIONS REARED ON RAW OR PASTEURIZED MILK SUPPLEMENTED BY WHITE FLOUR BISCUIT

Generation.	Milk.	Bucks		Does.	
		No. of Rats.	Av. wt. in gm.	No. of Rats.	Av. wt. in gm.
I	Raw	6	225	6	168
	Past.	6	200	6	166
II	Raw	11	212	13	163
	Past.	12	211	13	150
III	Raw	14	225	19	176
	Past.	23	162	25	135
IV	Raw	1	232	1	185
	Past.	10	192	11	143

third generation animals in the pasteurized milk group showed loss of hair, which was regarded as evidence of vitamin B deficiency.

In a further experiment, starting with 9 bucks and 9 does in each group, breeding failed in the raw milk group in the third generation, while still continuing in the pasteurized milk group, so that only two generations are available for comparison (Table XXVI).

TABLE XXVI

MATTICK AND GOLDING'S (1936) THIRD EXPERIMENT. AVERAGE WEIGHT OF YOUNG RATS AT 120 DAYS IN SUCCESSIVE GENERATIONS REARED ON RAW OR PASTEURIZED MILK SUPPLEMENTED BY WHITE FLOUR BISCUIT.

Generation.	Milk.	Bucks.		Does.	
		No. of Rats.	Av. wt. in gm.	No. of Rats.	Av. wt. in gm.
I	Raw	8	262	9	194
	Past.	9	257	9	190
II	Raw	17	155	15	128
	Past.	13	168	21	132

These figures are as close as could possibly be expected, and there is nothing in them to suggest that the growth of the rats fed on pasteurized milk was in any way inferior to that of rats fed on raw milk. It is stated, however, that in the third generation on pasteurized

milk the young animals in one litter were abnormally small, and that loss of hair was observed among some of the does in the second and third generations on pasteurized milk.

The latest observations to be recorded are those of Henry and Kon (1937*b*) at Reading. Two experiments were carried out, the first lasting 6 months and comprising twelve litter-mate pairs, the second lasting 8 weeks and comprising ten litter-mate pairs of male weanling rats. For each experiment the paired feeding method was used, in which the consumption of milk is equalized for each pair of animals. A milk diet was used exclusively, but was supplemented by iron, copper, and later by manganese. Growth was measured by gain in weight and in body-length, and by the composition of the carcases on chemical examination. Both raw and pasteurized milks appeared to be equally palatable; no signs of vitamin B deficiency were noted, and no significant differences were found in either experiment in the rate of growth between the two groups of animals (Table XXVII).

TABLE XXVII

HENRY AND KON'S EXPERIMENTS. AVERAGE GAIN IN WEIGHT OF RATS FED ON RAW OR PASTEURIZED MILK SUPPLEMENTED BY A MINERAL MIXTURE

No. of Experiment.	Milk.	No. of Rats.	Average gain in weight in gm.	
			8 Weeks.	6 Months.
1	Raw	12	88·6	207·9
	Past.	12	90·4	198·8
2	Raw	10	136·7	—
	Past.	10	133·3	—

Taking these various experiments together it seems very doubtful whether pasteurization has any effect on the total nutritive value of cows' milk for the rat. The experiments of Mattick and Golding at Reading do point rather in this direction, but their results were not altogether consistent, and in only two or three groups did the difference in weight between the animals on raw and pasteurized milk approach statistical significance. In none of the experiments carried out by Drummond in London, by Krauss, Erb, and Washburn in the United States, or by Henry and Kon at Reading itself were statistically significant differences observed. It is noticeable, however, that in Drummond's first experiment and to a less extent in his third, and in Henry and Kon's experiments there was a tendency for the animals

on raw milk to put on slightly more weight than those on pasteurized milk. Whether this was determined purely by chance, whether it was related to the difference in the distribution of the fat globules (see p. 115), or whether it was due to a real difference in the nutritive value of the two types of milk, it is impossible to say. The only justifiable conclusions are (1) that no definite diminution in the total nutritive value of cows' milk for rats as the result of pasteurization has yet been established, and (2) that if such a diminution does occur it must be of a very low order. It must be remarked, however, that rats do not require vitamin C, so that any destruction of this substance occurring through exposure to light or heat would not be reflected in the growth of these animals.

Mice. Very few experiments have been made on mice, in spite of the advantages that these animals possess. Wilson and Cowell (1933) carried out a series of preliminary experiments in which they attempted to confirm on mice the findings of Mattick and Golding (1931) on rats. Altogether 151 mice on a basal diet of raw milk and white flour biscuit were compared with the same number of animals on a basal diet of pasteurized milk and white flour biscuit. Both types of milk were found inadequate to promote normal growth, and young mice on this dietary generally died within a few weeks in an emaciated and often severely anæmic condition. The addition of an iron and copper mixture improved growth and development and prevented the occurrence of anæmia, but did not suffice as a rule to enable the does to rear their litters. The addition of yeastrel, however, to the basal diet overcame this difficulty, and when both mineral mixture and yeastrel were added, growth, reproduction, and rearing of the litters appeared to be almost as satisfactory as on a normal stock diet.

Comparing the behaviour on the different dietaries of the two groups of animals, all of which had been reared by does on stock diet and were, at the commencement of the experiment, about half grown, it was found that there was little difference between the nutritive value of raw and pasteurized milk in influencing general development, survival time, production of litters, or successful rearing of the young. With mice, however, that had been brought up from birth by does on a milk-and-biscuit diet, the average weight of the animals at weaning time was significantly heavier in the raw than in the pasteurized milk group.

From these preliminary experiments it was tentatively concluded (1) that cows' milk is deficient in iron and copper, and possibly in some vitamin which is required for successful lactation, and (2) that though raw and pasteurized milks appear to be of approximately equal

value for the growth and reproduction of mice subsequent to weaning, for the development of mice during their first four weeks of life raw milk appears to possess certain advantages over pasteurized milk.

Further experiments along these lines were carried out by Wilson and Maier (1937). The comparison was again made between raw and pasteurized milk, supplemented by a white flour biscuit. A mineral mixture containing iron, copper and manganese was, however, always added to the milk, and the breeding bucks at the time of mating, and the breeding does from the time of mating to the time of weaning, were given yeastrel. Growth was followed in the different experiments over a period of 7–19 weeks. The total number of animals observed in each group was about 1,000.

In the first series of experiments, as in those recorded by Wilson and Cowell, the milk was given in *inverted tubes*. The general results confirmed those previously obtained, and owing to the greater number of animals under observation it was possible to establish a definite superiority in the rate of growth of the young mice fed on raw milk.

In the second series of experiments the milk was given in *open vessels* in order to try and compensate for the difference in fat intake between the animals on raw and pasteurized milk when the inverted tube method was used. The results were different from those previously obtained, the animals on pasteurized milk being heavier, though not significantly so, at the end of the experiment than those on raw milk.

The explanation of the difference between the results in the two series of experiments was not clear, but may have been related to the difference in the amount of milk ingested (Kon 1938). The authors were unable to draw any definite conclusions from their experiments, since the majority of their observations had been made by the inverted tube method of feeding. They pointed out, however, that no progressive deterioration of the animals occurred on either type of milk, and that the growth of the young mice in the second and third generations was equal to that of mice brought up on a simple stock diet. They suggested that, if any difference did exist in the nutritive value of raw and pasteurized milk, it must be very small, and could probably not be determined satisfactorily without using very large numbers of animals.

Pigs. The few experiments made on pigs by Orr and his colleagues (1926) and Magee and Harvey (1926*b*) have been reviewed by Stirling and Blackwood (1933) and by Savage (1933*a*). Their main result was to show the unsuitability of cows' milk for the young pig. Sows' milk is very much richer in both calcium and phosphorus than cows' milk, and young pigs brought up on cows' milk are liable to develop rickets.

Calves. The early experiments on calves of Orr and his colleagues (1926) and of M'Candlish and Black (1932, 1933) have been reviewed and criticized by Stirling and Blackwood (1933) who are of the opinion that no definite conclusions can be drawn from the recorded results. The most recent experiments will be described here.

M'Candlish and Black (1935) at the West of Scotland Agricultural College made observations on a total of 35 Ayrshire and British Friesian calves. They were divided into three groups of winter heifers and three of winter bulls in a trial lasting 150 days, and into two groups of spring heifers and two of spring bulls in a trial lasting 120 days. The calves came from herds of cows infected to a considerable extent with tuberculosis. They were fed for 5 to 10 days on colostrum. Thirteen of the animals were then given raw milk and twenty-two pasteurized milk. The raw milk was apparently taken from the separate morning and evening milkings of the College herd and was fed fresh twice daily. The pasteurized milk came from the same herd, but consisted of the mixed morning and evening milk of the previous day, so that when fed it was between 12 and 36 hours old. The processing of the milk was carried out locally in a vat pasteurizer for part of the time, and in a " starter can " or cheese-vat for the rest. The heating was for 30 minutes at 145–150° F., but was not automatically controlled, and the recording thermometer was broken during the experiment. The temperature to which the milk was cooled is not stated. Small quantities of milk were given at first, but the allowance was increased rapidly till each animal was receiving 1 lb. per 10 lb. body-weight. When the animals reached 150 lb. in weight, their allowance of milk was restricted to 15 lb. daily. From 21 days onwards they were given hay *ad libitum* and a grain mixture twice daily.

In their description of the results the authors draw attention to the fact that the bull-calves grew better on raw than on pasteurized milk ; but it is obvious from their tables that the heifer-calves behaved in exactly the opposite way. When all the animals are taken into consideration the weighted mean increase in body-weight is found to have been 308·4 per cent. for the calves on raw milk and 311·3 per cent. for those on pasteurized milk. There is nothing in these figures to suggest that the growth of the animals was prejudiced in any way by pasteurization of the milk.

It is stated, however, that the calves on raw milk had better coats than those on pasteurized, and suffered less from scours and navel ill. If true, this latter statement would not be altogether surprising, considering that (1) the average length of time that the raw milk calves received colostrum was 8·8 days, while the corresponding

time for the pasteurized milk calves was only 7·5 days, and (2) the raw milk was fed fresh and the pasteurized milk was fed stale. Examination of the figures, however, shows that the proportion of calves developing white scours and navel ill was 30·8 per cent. on raw and 45·5 per cent. on pasteurized milk. The standard error of the difference of these figures is greater than the observed difference, so that no significance can be attached to them.

When subjected to the tuberculin test, one calf on raw milk and three calves on pasteurized milk are stated to have reacted positively. One of the calves on pasteurized milk gave a positive reaction on the fifth day of the experiment. Considering that the animals were derived from herds infected with tuberculosis, that they were fed on colostrum for 5 to 10 days, and that the calves on pasteurized milk were kept in the same pens as those receiving raw and potentially infected milk, it is clear that no conclusions can be drawn except that the experimental conditions were unsatisfactory. There is abundant evidence to show that proper pasteurization destroys all tubercle bacilli in milk (see Chapter XI). Consequently if calves fed on pasteurized milk are found to be infected with tuberculosis the conclusion must be drawn that either (1) pasteurization was inadequately performed, or (2) the calves were infected congenitally or by drinking colostrum before being put on to pasteurized milk, or (3) the calves became infected during the experiment, because they were given or allowed access to raw milk instead of pasteurized milk by mistake, or because, being in the same pens as the animals on raw milk, they were subjected to aerial or contact infection.

In spite of the wide publicity which has been accorded to this experiment, as showing that raw milk is superior to pasteurized milk in the feeding of calves, it is extremely doubtful whether there is any justification whatever for such a conclusion. Apart from the observation—largely subjective and not capable of exact measurement—that the calves on raw milk had better coats than those on pasteurized milk, there is nothing in the recorded data to prove that pasteurization had any deleterious effect on the milk. The actual weight increase was greater in the pasteurized milk group than in the raw milk group; the difference in incidence of white scours and navel ill might easily have occurred by chance alone; and the difference in the proportion of reactors to the tuberculin test merely shows that the experiment was badly designed and executed.

Wilson, Minett and Carling (1937) carried out an experiment at Peppard in which an attempt was made to avoid most of the errors of their predecessors, and to obtain a straightforward answer to the question whether a given quantity of pasteurized milk is of equal

nutritive value for calves as the same quantity of raw milk. For this reason they selected a small self-contained herd of healthy Shorthorn cows that were free from tuberculosis, contagious abortion, and clinical evidence of mastitis. Alternate calves, as they were born into the herd throughout the year, were fed for three and a half days on colostrum, and were then transferred to a group which was fed twice daily on either raw or pasteurized milk. The milk was derived from the mixed morning's milk of the herd. It was divided into two parts. One was left raw; the other was pasteurized at 145° F. (62·8° C.) for 30 minutes in a specially constructed batch pasteurizer fitted with a motor-driven agitator and an auto-thermostatic control, and then cooled to 90° F., if it was to be fed immediately, or, like the raw milk, to the temperature of tap water if it was to be kept till the evening feed. The quantity of milk fed was in proportion to the body-weight. Apart from milk, the animals were given hay *ad libitum*. For the first month they merely " played " with it, but after that each animal consumed about 1 lb. a day, rising to 3 lb. by the eighth week. The experimental period of observation on each animal lasted 8 weeks.

With the exception of two weaklings which died after 15 and 23 days respectively from causes apparently unconnected with the nature of their diet, all the animals—25 in the " raw " and 23 in the " pasteurized " group—throve well and showed no signs of rickets or anæmia. The average increase in weight over the eight-week period for the animals on raw milk was 61·18 per cent. and for those on pasteurized milk 62·94 per cent. The highest individual gain among the bull-calves—one of 80 lb.—and the highest individual gain among the heifer-calves—one of 63 lb.—both occurred in animals fed on pasteurized milk. Observers, both lay and professional, were called in at different times during the experiment, but none of them was able to distinguish between the two groups of animals. No evidence was therefore obtained in this experiment to suggest that the nutritive value of pasteurized milk was in any way inferior to that of raw milk.

Wilkie, Edwards, Fowler, and Wright (1937) at the Hannah Dairy Research Institute, Ayr, carried out an experiment designed to answer a rather different question, namely whether calves fed on commercially pasteurized milk behaved as satisfactorily as those fed on raw milk from the same source. Bull-calves from tuberculin tested Ayrshire herds, after receiving two to five feeds of colostrum, were allocated alternately to two groups, one of which was fed on raw, the other on commercially pasteurized milk. The two groups were housed separately. The milk, which came from untested herds, was given

twice daily in an amount strictly proportional to the body-weight. It was supplemented from the eighth week onwards by $\frac{2}{3}$ lb. of hay per head per day. The experimental period of observation on each animal lasted 12 weeks. Of the 92 calves in the experiment, 42 received pasteurized milk and 50 raw milk. Nineteen calves died during observation from gastritis, pneumonia, and other causes, 5 in the pasteurized group and 14 in the raw milk group.

The 36 animals receiving raw milk and completing the twelve weeks of observation showed an average increase in weight of 74·5 $\pm$ 2·7 per cent.; the 37 corresponding animals receiving pasteurized milk showed an average increase in weight of 80·2 $\pm$ 3·1 per cent. This difference is not significant. Marks awarded by experienced stock judges called in to examine the animals showed consistent differences in favour of the animals fed on pasteurized milk.

Seventy per cent. of the samples of raw milk proved on guinea-pig inoculation to contain tubercle bacilli; the pasteurized milk samples were uniformly negative. Corresponding to these differences 23 of the 36 calves on raw milk were found, when killed at the end of the experiment, to contain macroscopic lesions of tuberculosis, while not one of the 37 calves on pasteurized milk proved infected.

The results of this experiment show that calves fed on commercially pasteurized milk grow just as well as those fed on raw milk, and that they are protected by reason of their diet from becoming infected with tubercle bacilli present in the raw milk.

Bartlett, Cotton, and Mackintosh (1938) at the National Institute for Research in Dairying at Reading made observations on 16 pairs of bull-calves from tubercle-free Shorthorn herds. On arrival at the Institute the calves were given raw and pasteurized milk mixed in equal quantities for a few days, and were then distributed in pairs of approximately similar age and weight. One of each pair was henceforward given raw milk, the other pasteurized milk, the choice being made by toss of coin. The milk was derived from non-tuberculin-tested herds; part of it was left raw, and part of it was pasteurized by a commercial firm. The amount of milk fed to the animals was in proportion to their weight. A hay supplement was included after 8 weeks, and a meal supplement after 17 weeks. All animals were castrated when they reached a weight of 130 lb., that is at about 4 to 5 weeks of age. The experiment lasted for 26 weeks.

In assessing the results 5 pairs of calves were omitted on the ground that three animals on raw milk died of pneumonia, one animal on raw milk was ill, and one animal on pasteurized milk had intestinal obstruction. The average increase in weight of the 11 animals fed on raw milk was 358·00 $\pm$ 20·37 lb., and of the 11 animals fed on

pasteurized milk 358·72 ± 20·50 lb. There was a very slight and statistically insignificant difference, in favour of the raw milk group, in the depth of chest, the height at the withers, and the heart girth.

Examination of the raw milk showed that 30 per cent. of samples contained tubercle bacilli, while none of the pasteurized milk samples did so. Tuberculin tests made towards the end of the experiment revealed 9 reactors out of 13 calves in the raw milk group and one reactor out of 14 calves in the pasteurized milk group. Of the 11 animals on the raw milk that were used in the final analysis 8 reacted to tuberculin, and of the 11 animals on pasteurized milk 1 reacted. As in the experiments of M'Candlish and Black the fact that tuberculous infection was introduced into the group of animals fed on pasteurized milk shows that the experimental conditions were imperfect. The defects in the Reading experiment consisted in feeding all the calves on a mixture of raw and pasteurized milk before they were divided into two groups, and in housing both groups together during the whole course of the observations.

The general results, however, of this experiment are very clear. They afford no evidence that the nutritive value of the milk was detrimentally affected by pasteurization, while they do show that pasteurization serves as a valuable safeguard against the danger of contracting tuberculous infection from milk.

Crichton and Biggar (1938) carried out two experiments at the Rowett Research Institute, Aberdeen. In the first experiment 32 calves were purchased locally from non-tuberculin-tested herds. They were reasonably uniform in type, having been bred by Aberdeen Angus bulls from Irish Shorthorn cows. After receiving colostrum for 3 days, they were divided into two roughly similar groups, each of which contained 6 heifer and 10 bull-calves. One group was given raw milk, the other pasteurized milk from the same mixed source. Pasteurization was apparently carried out by a commercial firm. Milk was fed three times a day according to appetite till the animals were 122 days old, when it was reduced to 20 lb. per head daily, and supplemented by a meal mixture. Oat straw was used as litter, and fed *ad libitum* during the whole course of the observations. The animals in the two groups were housed together. The bull-calves were castrated when they reached a weight of 120 lb., that is at about 5 weeks of age. The experiment was terminated after 183 days.

One calf in the pasteurized milk group and three calves in the raw milk group had to be discarded for reasons not associated with the diet. The average increase in weight of the 13 calves fed on raw milk was 406 per cent., and of the 15 calves fed on pasteurized milk 380 per cent. This difference is not statistically significant.

The average height and girth were very slightly greater in the pasteurized than in the raw milk group.

Examination of the milk supply showed that 74 per cent. of samples of raw milk contained tubercle bacilli and 74 per cent. *Br. abortus*; of the pasteurized milk 7 per cent. of samples contained tubercle bacilli and 5 per cent. *Br. abortus*. Four of the calves on raw milk and 3 on pasteurized milk reacted to tuberculin; and 6 calves on raw milk and 5 on pasteurized developed serum agglutinins for *Br. abortus*. It is a little difficult to understand why the pasteurized milk should have been infected, considering that pasteurization was carried out under daily medical supervision and that the phosphatase test was uniformly negative. Whatever the explanation, it is clear that the processing was inefficiently carried out. Since the calves were not purchased from tuberculin tested herds, since they were all housed together, and since pasteurization was unsatisfactorily performed, there is no difficulty in understanding why some of the calves fed on pasteurized milk became infected with the organisms of tuberculosis and contagious abortion.

Crichton and Biggar (1938) carried out their second experiment in much the same way as their first. The following were the more important differences. The calves were all males. They were given colostrum for 7 days instead of 3, and were then divided at random into two groups of 10 each. The maximum daily allowance of milk was limited to 15 lb. per head daily, and meal was fed as soon as the calves would take it. The animals were not castrated till the end of the experiment, which lasted 187 days.

The average increase in weight of the 10 calves fed on raw milk was 320 per cent., and of the 10 calves fed on pasteurized milk 288 per cent. This difference is not significant. The height and girth of the animals were not measured, but no difference in their general health was observed.

The same infected milk was used as in the first experiment, with the result that 4 animals on raw milk and 1 on pasteurized reacted to the tuberculin test.

In discussing these two experiments, the authors point out that, though the difference in weight increase between the animals on raw and pasteurized milk is not statistically significant in either experiment separately, it becomes just significant if the two experiments are taken together. The significance of this difference, however, is somewhat diminished by the fact that in the first experiment the growth of the animals was by no means uniform, and that after 122 days the weight of the calves on pasteurized milk was equal to that of those on raw milk.

As regards the general conduct of the two experiments much the same criticism applies as to the experiments of M'Candlish and Black (1935). The animals were not derived from tuberculin tested herds, they were fed on potentially infected colostrum, the processing of the milk was unsatisfactory, and the calves on pasteurized milk were housed together with those on raw milk. Under these conditions many of the animals on pasteurized milk became infected with tuberculosis, thus obscuring one of the main benefits of pasteurization.

It can hardly be emphasized too strongly that in all nutritional work bacterial and other parasitic infections liable to obscure the effect of the diet itself should be avoided. No doubt it is of interest to learn, as the authors of the Hannah Dairy Research Institute experiment were anxious to do, that healthy calves fed on infected raw milk frequently become tuberculous, while healthy calves fed on pasteurized milk from the same source and housed separately do not become tuberculous; but how is it possible to tell from such an experiment whether, if the milk had been free from tubercle bacilli, the animals fed on raw milk might not have thrived better than those fed on pasteurized? It may be answered that tuberculous infection is not likely to influence the growth of animals during the relatively short experimental periods of observation, but such an answer is not wholly satisfactory.

The only experiment so far carried out on calves in which infection was completely avoided was that at Peppard; and the results of this experiment failed to show any difference in the growth rate or general condition of the animals in the raw and the pasteurized milk groups. Even to this experiment it may be objected that hay was given in addition to milk. Such an objection applies also to the other experiments, in all but one of which a hay, straw, or meal supplement was provided. The difficulty of carrying out a really satisfactory experiment is due to the fact that, as Knoop, Krauss, and Washburn (1935) and others have shown, calves do not thrive for long on milk alone, but develop anæmia and other disturbances which may prove fatal. Cows' milk is deficient in some of the minerals and vitamins required by the growing calf, and unless these are supplied in some other article of diet, normal growth for long is impossible.

Bartlett (1938) has performed a very useful service in compiling a table in which the main conditions and results of the various experiments performed on calves in Great Britain since 1926 are conveniently summarized. Taking 250 calves in all, divided into two approximately equal groups, it is found that the weighted mean live weight increase of the animals on raw milk was 1·232 lb. per day, and of those on pasteurized milk 1·273 lb. per day (see Table XXVIII). The general

TABLE XXVIII

FEEDING EXPERIMENTS WITH RAW AND PASTEURIZED MILK ON CALVES SINCE 1926 (MODIFIED FROM BARTLETT 1938)

Experimenters.	Duration of Experiment in Days.	Nature of Supplement.	No. of Calves completing Experiment.		Average daily gain in Weight in lb.	
			Raw.	Past.	Raw.	Past.
Orr *et al.* (1926)	125	Concentrates. ? nature of roughage	4	4	1·54	1·41
Orr *et al.* (1926)	45	None	2	3	1·22	1·07
M'Candlish and Black (1932, 1933)	90	Hay and concentrates	6	5	1·20	1·10
M'Candlish and Black (1935)	120 and 150	Hay and concentrates	13	22	1·88	1·89
Wilson, Minett and Carling (1937)	56	Hay	25	23	0·96	0·96
Wilkie, Edwards, Fowler and Wright (1937)	84	Hay	36	37	0·60	0·67
Bartlett, Cotton and Mackintosh (1938)	182	Hay and concentrates	11	11	1·97	1·97
Crichton and Biggar (1938)	180	Oat straw and concentrates	13	15	1·86	1·77
Crichton and Biggar (1938)	180	Oat straw and concentrates	10	10	1·61	1·45
	Total		120	130		
	Weighted mean. . .				**1·232**	**1·273**

results, therefore, fail to reveal any significant difference between the nutritive value of raw and pasteurized milk under the experimental conditions employed, but do show that, when adequately carried out, pasteurization of tuberculous milk is able to protect the animals against the danger of contracting infection.

Lest it should be thought that these results are of purely academic significance, reference may be made to the report of Roadhouse and Perry (1930), who state that in practice the Hansen and Orloff Dairy in California has been very successful in raising calves on pasteurized milk. Of the first 300 animals brought up in this way only 9 reacted to tuberculin at 5 to 6 months of age. These results were obtained when the calves were left with their mothers for one day and then fed with pasteurized milk from a non-tested herd.

It is also apposite to point out that the compulsory pasteurization of milk to be used in the feeding of animals in Attested herds is

explicitly enjoined by the Ministry of Agriculture and Fisheries in the following terms : " No milk or dairy by-product shall be brought on to the premises of an Attested Herd for feeding to animals except direct from the premises of another Attested Herd unless such milk or dairy by-product is pasteurized or sterilized by heat " (Report 1937*f*).

Children. In spite of the numerous experiments carried out on the feeding of milk to infants and children, not one has yet been performed under really satisfactory conditions in which a straightforward comparison has been made between the effect on growth of raw and pasteurized milk given in the same quantity from the same source.

The well-known observations of Corry Mann (1926) on the improvement in physique that followed the daily administration of one pint of milk to a group of institutional boys fed on an otherwise satisfactory diet are not strictly relevant to this chapter, since all the milk was pasteurized and no comparison was therefore available with boys fed on an equivalent amount of raw milk.

The observations in the United States of Daniels and Stearns (1924), Ladd, Evarts, and Franks (1926), Lewis (1927, 1929), and Daniels, Hutton, Stearns, and Hejinian (1929) have been critically reviewed at some length by Stirling and Blackwood (1933) and Savage (1933*a*). In no instance was a satisfactory comparison made between raw and pasteurized milk from the same source. Daniels and Stearns (1924), for example, compared the effect of boiling and of pasteurization on diluted milk fortified with orange juice and dextri-maltose or lactose. Ladd, Evarts, and Franks (1926) compared two modified milks of different origin, namely raw Certified and Grade A pasteurized, and tried the effect of supplements of cod-liver oil and orange juice. Lewis (1927, 1929) carried out his observations on boiled Certified and boiled Pasteurized milk of different origin and greatly different fat content. Daniels, Hutton, Stearns, and Hejinian (1929) compared modified pasteurized with modified boiled milk, supplemented or not with cod-liver oil and sometimes with wheat embryo extract. In only one of these series of observations, therefore, was a comparison made between raw and pasteurized milk, and in this experiment the milks were of different origin and were modified to suit the dietetic requirements of the infants.

The field survey of Frank and his colleagues (1932) on over 3,700 children in the United States to determine whether the average height and weight of children up to 6 years of age differed among those who had been brought up on raw or on heated milk has again been critically reviewed by Stirling and Blackwood (1933). No significant differences

were found, and the slight differences that did occur were in favour of the group fed on heated milk. So many factors, however, affect the outcome of a survey of this kind that the conclusions drawn must necessarily be accepted with reserve.

The large scale *ad hoc* feeding experiment of Leighton and McKinlay (1930) in Lanarkshire, in which the growth of 5,000 children receiving daily an additional three-quarters of a pint of raw Grade A Tuberculin Tested milk was compared with that of 5,000 children receiving a similar quantity of pasteurized milk from the same source, might be expected to have settled the question of the relative value of a supplement of raw or pasteurized milk to the ordinary diet of school children. In fact, however, the experiment was carried out in such a way that the comparison between the two groups of children was not wholly satisfactory (see Stirling and Blackwood 1933, Savage 1933*a*). Raw and pasteurized milk, for example, were given to separate schools, never to children in the same school, and the two groups of children were not strictly similar. The authors' conclusions that the effects of raw and pasteurized milk on growth in height and weight appear to be equal have been challenged by Fisher and Bartlett (1931), Bartlett (1931), and " Student " (1931). The final analysis of the results by Elderton (1933) and Pearson (1933), however, in which the comparison was restricted to pairs of children of approximately the same initial height and weight, confirmed Leighton and McKinlay's original conclusions, and failed to reveal any difference between the effect of raw and of pasteurized milk in their effect on growth.

The recent experiment (Report 1938*b*, 1939*c*) carried out under the direction of the Milk Nutrition Committee to ascertain the effect of dietary supplements of raw and pasteurized milk on the growth and health of schoolchildren must be described more fully. Observations were made on a total of 8,000 children in five different areas —Luton, Wolverhampton, Burton-on-Trent, Huddersfield, and Renfrewshire. In each area the children, who were 5 to 14 years of age, were divided into four equal groups. Group I received biscuit with a calorie value of 52 ; Group II received one-third of a pint of pasteurized milk ; Group III received two-thirds of a pint of pasteurized milk ; and Group IV received two-thirds of a pint of raw milk. In any one area the raw and pasteurized milks came from the same bulk source. The allocation of the children to their respective groups was made in strict rotation without reference to their physical condition or to the economic status of their parents. The children were examined medically every three months, their nutritional state being gauged by physical measurements, clinical examination, and certain functional tests. The period of observation extended over one school year.

The more important results are summarized in Table XXIX.

TABLE XXIX

PERCENTAGE INCREASES IN HEIGHT AND WEIGHT OVER INITIAL VALUES OF 6,099 SCHOOLCHILDREN GIVEN A DAILY SUPPLEMENT OF BISCUIT OR MILK FOR ONE YEAR

Group and Dietary Supplement.	Boys.			Girls.		
	Number.	Height.	Weight.	Number.	Height.	Weight.
Group I. Biscuit	802	4·16	10·87	681	4·66	12·75
Group II. ⅓ pint pasteurized . .	821	4·32	11·41	719	4·77	13·51
Group III. ⅔ pint pasteurized . .	838	4·46	12·02	715	4·87	14·00
Group IV. ⅔ pint raw	834	4·37	12·07	689	4·83	14·53

Of the 8,000 children at the start of the experiment, 6,099 attended school regularly and were present at all four medical examinations. The general results showed that children on a milk supplement increased in height, weight, and chest circumference to a greater extent than children in the control group on a biscuit supplement, and that the children given two-thirds of a pint of milk grew more than those given only one-third of a pint. The differences, however, between the milk-fed and the control groups were very small. Even between the groups receiving two-thirds of a pint of milk and the control group receiving biscuit they did not amount to more than about 0·1 inch in height, ½ to 1 lb. in weight, and 0·1 to 0·2 inch in chest circumference during the entire year. Somewhat unexpectedly the growth-promoting power of the milk appeared to be greater in children who were assessed clinically as above the average than in those who were poorly nourished at the start.

So far as the comparison between children receiving equal quantities of raw and pasteurized milk is concerned, that is between Groups III and IV, no constant differences could be detected in either height, weight, or chest circumference. The growth varied in different age, sex, and place groups. The only consistent differences related to increase in muscular strength as estimated by the dynamometer test, and these were in favour of the group receiving pasteurized milk. The differences, however, though uniformly in the same direction, were small, and could not be considered statistically significant.

The general conclusions to be drawn from this feeding test on children are that milk has a stimulating effect on growth, and that

there is no detectable difference in this respect between raw and pasteurized milk when fed to school children as part of their ordinary diet.

Once more it may be pointed out that no satisfactory comparison of the effect of raw and pasteurized milk on the human subject has yet been made. Such a comparison should preferably be conducted on infants who are being fed on an exclusive milk diet, supplemented only by a mineral mixture containing iron, copper, and possibly manganese to prevent the development of anæmia (see Mackay 1931, Sheldon 1932, Parsons 1933, v. Haam and Beard 1935, Hutchison 1938), by orange juice to prevent scurvy (see Hess and Fish 1914, Hess 1915, 1916), and possibly by some source of vitamin D to protect against rickets and dental caries (see Report 1923, Hess 1932, Mellanby 1929, 1930, 1934). The most that can be said for the experiments so far conducted on infants and children is that no satisfactory evidence has yet been adduced to prove the occurrence of any deleterious effect on the nutritive value of cows' milk resulting from pasteurization.

Summary

(1) Numerous experiments during the past few years have been carried out on rats, mice, pigs, calves, and children, in which a comparison has been attempted between the effect on growth of raw and pasteurized milk. Many of these experiments, particularly those on infants and children, have been faulty in design and execution, and their conclusions cannot be accepted without reserve.

(2) Difficulties exist in making a really satisfactory comparison, because cows' milk is not a perfect food for any of these animals, even the calf, and maintenance on an exclusive diet of milk for any length of time is likely to be followed by anæmia, rickets, scurvy, or other kind of disturbance.

(3) Several careful experiments, however, have been conducted between groups of animals fed on raw or pasteurized milk with just a sufficient supplement to prevent the development of nutritional disease.

(4) The most recent experiments on rats at Reading carried out by the paired feeding method failed to show any significant difference in the rate of growth of the animals or in the assimilation of the various food substances contained in the milk.

(5) The observations on mice are less numerous than those on rats, and are a little difficult to interpret, since the behaviour of the animals appeared to depend on the way in which the milk was fed. They suggest that if any difference does exist in the nutritive value

of raw and pasteurized milk, it must be very small and could be determined only by the use of very large numbers of animals.

(6) The experiments on calves are of particular interest, since they were conducted by six different groups of workers on a total of 250 animals. Though in some experiments the animals on raw milk grew better than those on pasteurized milk, in other experiments the reverse occurred. Taking all the observations together the mean increase in weight of the animals on raw milk was 1·232 lb. per day, and of the animals on pasteurized milk 1·273 lb. per day, the small advantage therefore being in favour of the latter group. Incidentally it was found that adequate pasteurization of tuberculous milk protected the calves from contracting infection.

(7) In the most recent comparison on children very little difference was noticed between those fed on raw and those fed on pasteurized milk. On an average the children gained slightly more in weight on raw milk and slightly more in height on pasteurized milk, but the differences were insignificant.

(8) The results of the various experiments on different animals, including children, are in general accordance with each other, and fail to show that pasteurization of milk by the holder method has any significant effect in lowering its total nutritive value for the growing animal.

CHAPTER X

EFFECT OF PASTEURIZATION ON THE FERTILITY OF ANIMALS FED ON A MILK DIET

It has been suggested that the fertility of animals and human beings may be less on pasteurized than on raw milk (Picton 1938, Sutherland 1938).

Considering that milk is essentially a food for the very young animal, and considering that sexual maturity is not reached in man for several years after weaning, it seems on *a priori* grounds to be improbable that this statement holds any substance of truth. Nevertheless it is of such importance that the evidence bearing on it must be examined in some detail.

Observations on Rats. Mattick and Golding (1931, 1936) at Reading carried out a number of experiments which have already been referred to in Chapter IX. Litter mates were divided into two more or less equal groups. These were fed on raw or pasteurized milk from the same bulk source with the addition of a biscuit made from white flour and water. Pasteurization was carried out by the holder method in the laboratory, the milk being kept at 145–150° F. for 30 minutes and then cooled to between 56° and 69° F. The results, so far as fertility is concerned, may be summarized as follows :

Mattick and Golding's First Experiment. (1931). Six bucks and 6 does were included in the raw milk group, and the same number in the pasteurized milk group. After the experiment had lasted 4 months the animals within each group were cross-mated, with the following results (Table XXX).

TABLE XXX

FERTILITY OF RATS FED ON RAW OR PASTEURIZED MILK. MATTICK AND GOLDING'S FIRST EXPERIMENT (1931).

Group.	Does available for Breeding.	Litters produced.	No. of Young Born.
Raw Milk	6	6	47
Pasteurized Milk	6	6	42

Five of the young in the raw milk group and 20 in the pasteurized milk group appear to have been born dead. So far as fertility is

concerned there is nothing in these figures to suggest that the behaviour of the animals in the two groups was significantly different.

Mattick and Golding's Second Experiment (1936). In this experiment, which likewise started with 6 bucks and 6 does in each group, breeding was continued for several successive generations. The results are a little difficult to summarize and interpret, because it would appear that not every doe was used for breeding. The actual figures given, however, are as follows (Table XXXI):

TABLE XXXI

FERTILITY OF RATS FED ON RAW OR PASTEURIZED MILK. MATTICK AND GOLDING'S SECOND EXPERIMENT (1936)

Generation.	Raw Milk Group.			Pasteurized Milk Group.		
	Possible Matings.	Litters produced.	No. of Young born.	Possible Matings.	Litters produced.	No. of Young born.
1st (original) . .	17	13	91	16	13	91
2nd	31	21	159	27	25	186
3rd	39	23	120	34	25	163
4th	4	4	16	12	6	37
5th	3	1	7	0	0	0
6th	0	0	0	0	0	0
Total	94	62	393	89	69	477
Arithmetic mean per Mating . .	—	0·66	—	—	0·78	—
Arithmetic mean per Litter . .	—	—	6·34	—	—	6·91

It will be observed that breeding failed in the raw milk group in the sixth generation and in the pasteurized milk group in the fifth generation. The average number of litters produced per mating and of young born per litter was rather higher in the pasteurized than in the raw milk group.

Mattick and Golding's Third Experiment (1936). This experiment resembled the second, except that the initial number of rats was 9 bucks and 9 does in each group. All does were mated except those that were in obviously bad condition. The results are shown in Table XXXII.

Breeding failed in the raw milk group in the third generation, while it was still continuing in the pasteurized milk group. Again it

TABLE XXXII

FERTILITY OF RATS FED ON RAW OR PASTEURIZED MILK. MATTICK AND GOLDING'S THIRD EXPERIMENT (1936)

Generation.	Raw Milk Group.			Pasteurized Milk Group.		
	Attempted Matings.	Litters produced.	No. of Young born.	Attempted Matings.	Litters produced.	No. of Young born.
1st (original) . .	17	15	99	17	13	103
2nd	30	16	122	40	27	202
3rd	0	0	0	11	7	44
Total	47	31	221	68	47	349
Arithmetic mean per Mating . .	—	**0·66**	—	—	**0·69**	—
Arithmetic mean per Litter . .	—	—	**7·13**	—	—	**7·43**

will be noted that the average number of litters per mating and of young born per litter was rather higher in the pasteurized than in the raw milk group.

The results of the second and third experiments may be summarized as follows (Table XXXIII):

TABLE XXXIII

FERTILITY OF RATS FED ON RAW OR PASTEURIZED MILK. MATTICK AND GOLDING'S SECOND AND THIRD EXPERIMENTS COMBINED

	Raw Milk Group.	Pasteurized Milk Group.
Possible or attempted Matings	141	157
Litters produced	93	116
No. of Young born	614	826
Mean no. of Litters per Mating . . .	**0·66**	**0·74**
Mean. no. of Young per Litter . . .	**6·60**	**7·12**

From these experiments it seems perfectly clear that the breeding performance of rats fed on pasteurized milk was just as good as that of rats fed on raw milk, and it is difficult to understand how any other conclusion could be drawn from them.

Picton (1938), who quotes them in support of his thesis that pasteurization of milk might accelerate the present fall in the birth-

rate of the country, confuses pasteurized milk with sterilized milk; while Sutherland (1938), who likewise quotes them to show that pasteurization has an adverse effect on fertility, refers merely to the preliminary experiment of 1931. It is interesting to note that Mattick and Golding themselves carefully refrained in their papers from drawing any such conclusions. The figures suggest that the fertility of rats fed on pasteurized milk was rather greater than that of rats fed on raw milk, and this may have been true. In view, however, of the apparent absence of perfect comparability of mating in Mattick and Golding's second experiment, it would perhaps be wiser to refrain from drawing this conclusion.

After the publication of Mattick and Golding's first paper, Drummond (1933) endeavoured to carry out a similar series of experiments at University College, London. The only important difference was that the milk was pasteurized, not in the laboratory, but in a commercial plant. Both the holder-pasteurized and the raw milk used were derived daily from the same bulk supply. The diet was supplemented with biscuit made from white flour and water.

Drummond's First Experiment. Five bucks and 7 does were included in each group. Observations were made on the animals for a whole year, during which numerous matings were attempted. The results were unsatisfactory, and only 7 litters were produced in the raw milk and 12 in the pasteurized milk group. Since the young in each group died soon after birth, second generation matings were not possible.

Drummond's Second Experiment. Thinking that there might be some difference between the quality of the milk or the biscuit at Reading and those in London, arrangements were made for a daily supply of raw milk from Reading as well as of a batch of the same flour as that used by the Reading workers. Observations were then made on four groups of rats in which the effect of London raw and Reading raw milk, and of London flour and Reading flour made into biscuit, were compared. In none of the groups was breeding satisfactory, and only one litter of 8 animals, none of which survived for more than 2 days, was produced in the group of rats fed on Reading raw milk and Reading biscuit. The conclusion was drawn that rats do not breed satisfactorily on a diet consisting of milk, whether raw or pasteurized, and a simple white flour biscuit.

Drummond's Third Experiment. Eight groups of animals, each containing 5 bucks and 5 does, taken from divided litters, were set up. The first two groups were fed on raw and pasteurized milk respectively supplemented by biscuit. Groups 3 and 4 received in addition 0·5 gm. of yeastrel. Groups 5 and 6 received a mineral

supplement of iron and copper. Groups 7 and 8 received both yeastrel and a mineral mixture. There were thus four groups of animals on raw milk and four on pasteurized milk, given the same type of supplementary feeding. The experiment lasted 24 weeks. Matings within the groups were carried out when vaginal smears, which were made daily, appeared favourable, and only bucks of proved fertility were used. The results are summarized in Table XXXIV.

TABLE XXXIV

FERTILITY OF RATS FED ON RAW OR PASTEURIZED MILK WITH CERTAIN SUPPLEMENTS. DRUMMOND'S THIRD EXPERIMENT (1933)

Nature of Supplement.	Raw Milk Group.			Pasteurized Milk Group.		
	Matings.	Litters produced.	No. of Young born.	Matings.	Litters produced.	No. of Young born.
Biscuit . . .	3	2	12	3	2	16
Biscuit + Yeastrel	8	6	40	4	4	31
Biscuit + Mineral Mixture . . .	7	5	33	4	4	29
Biscuit + Mineral Mixture + Yeastrel . . .	8	3	25	6	4	30
Total	26	16	110	17	14	106
Arithmetic Mean per Mating . .	—	0·62	—	—	0·82	—
Arithmetic Mean per Litter . .	—	—	6·88	—	—	7·57

It will be noted that in all groups receiving a supplement of yeastrel or mineral mixture, or both, breeding was more satisfactory than in those receiving milk and biscuit alone.

So far as fertility is concerned, there is nothing in these results of Drummond's to suggest that rats on pasteurized milk are less productive than those on raw milk.

The observations of Channon and Channon (1936) at Liverpool, who endeavoured to repeat Mattick and Golding's work on raw and sterilized milk, have already been referred to. The reproductive behaviour, however, of their animals on raw milk may be dealt with here a little more fully. In their first experiment the young rats received raw milk *ad libitum* together with a white flour biscuit.

Within a month they showed evidence of marked anæmia, and their diet was therefore supplemented with iron, copper and manganese. In subsequent experiments this mineral mixture was given from the start. The fertility of the bucks and does was tested separately, by crossing them with healthy animals on a stock diet. The results are summarized in Table XXXV.

TABLE XXXV

FERTILITY OF RATS FED ON RAW MILK AND CROSSED WITH ANIMALS FED ON A STOCK DIET. CHANNON AND CHANNON'S EXPERIMENT (1936)

	No. of Animals tested.	No. of Matings.	No. of Matings proving positive.	No. of Matings proving infertile or followed by resorption.	No. of Litters.	No. of Young.	No. of Young reared up to 4 days.
Bucks.	8	34	6	—	—	—	—
Does .	18	86	18	2	16	128	8

With the bucks 6 out of 34 matings proved positive: with the does 18 out of 86 matings proved positive, but only 16 litters were produced. Although the ability to reproduce might be considered as satisfactory, the does were unable to rear their litters. In only one litter did the animals survive for more than 4 days, and these all died eventually. These results are in striking contrast to those of Mattick and Golding, who reported that a diet of raw milk and white flour biscuit without any mineral supplement was adequate for the rearing of rats for several generations.

Waddell (1931) in the United States observed that male rats fed on a diet of whole cows' milk supplemented by iron and copper sometimes became completely sterile owing to testicular degeneration; while Waddell, Steenbock, and Hart (1931) observed that on a similar diet reproduction was unsatisfactory, and that the females had difficulty in rearing their young owing to defective mammary secretion. No evidence, however, of resorption of the young pointing to a vitamin E deficiency was observed.

Taken as a whole, the experiments on rats show (1) that cows' milk, even when supplemented by a mineral mixture to prevent the development of anæmia, is not conducive to satisfactory breeding, and (2) that pasteurization of the milk does not appear to diminish the fertility of the animals fed on it.

Observations on Mice. Wilson and Cowell (1933), like Drummond (1933) and Channon and Channon (1936), also carried out experiments to test the correctness of Mattick and Golding's results,

but used mice instead of rats. Their work followed the same general lines as those of Drummond. It was found that a simple diet of raw or pasteurized milk, supplemented by a white flour biscuit, was unable to support normal growth or breeding, but that the addition of a mineral mixture and of yeastrel enabled both growth and breeding to occur.

Fifteen experiments, lasting 7 to 52 weeks, were carried out, a total of 151 mice being fed on raw milk and an equal number on pasteurized milk. The pasteurized milk and the biscuit were from the same source of supply as in Drummond's experiments. The results may be summarized in a composite table (Table XXXVI).

TABLE XXXVI

FERTILITY OF MICE FED ON RAW OR PASTEURIZED MILK WITH A MINERAL MIXTURE AND YEASTREL SUPPLEMENT. WILSON AND COWELL'S EXPERIMENTS (1933)

Milk.	Does available for breeding.	Litters produced.	No. of Young reared for 28 days.	Average no. of Litters per Doe.	Average no. of young Mice reared per Litter.
Raw . . .	42	46	105	**1·10**	**2·40**
Pasteurized .	38	42	90	**1·11**	**2·14**

The number of young were not counted at birth, but those reared for 28 days were available for comparison.

Though the average number of young mice reared per litter was slightly higher in the raw milk than in the pasteurized milk group, the fertility of the animals as judged by the number of litters produced was practically identical in the two groups.

Following on these preliminary observations, Wilson and Maier (1937) carried out nine experiments comprising a total of 428 mice fed on raw milk and 417 fed on pasteurized milk. The diet was supplemented with a white flour biscuit and mineral mixture. In addition the breeding bucks at the time of mating, and the breeding does from the time of mating to the time of weaning, were given 3 per cent. yeastrel. The pasteurized milk was derived from the same commercial source as in Wilson and Cowell's experiments, and was always taken from the same bulk supply as that used for the provision of the raw milk.

After the growth of the animals had been observed for a time varying in different experiments from 7 to 19 weeks, mating was begun. Five does were run in a cage with 1 buck. As soon as a doe showed signs of pregnancy it was transferred to a separate cage. After

parturition the young were kept with the mother for 4 weeks. They were then removed and placed on a diet free from yeastrel. In five experiments, when the growth of the young mice had been observed for a sufficient length of time, they were mated as before. In this way three successive generations of mice fed on raw and pasteurized milk with the supplements described above were raised. It may be noted that there was no apparent falling off in growth rate or in breeding performance in either of the groups. The breeding results are summarized in Table XXXVII.

TABLE XXXVII

FERTILITY OF MICE FED ON RAW OR PASTEURIZED MILK WITH A MINERAL MIXTURE AND YEASTREL SUPPLEMENT. WILSON AND MAIER'S EXPERIMENTS (1937)

	Raw Milk Group.	Pasteurized Milk Group.
No. of Does available for breeding . .	183	167
Total no. of Litters produced	110	104
Total no. of Young Mice reared to 28 days	434	364
Average no. of Litters per Doe . . .	0·60	0·62
Average no. of young Mice reared per Litter produced	3·95	3·50
Average no. of days from placing with Buck to parturition	32·53	30·14

Judging from the average number of litters per doe and the average rapidity of conception and parturition after mating, the two groups behaved almost identically. As in Wilson and Cowell's experiments, the average number of young mice reared per litter was slightly higher in the raw milk than in the pasteurized milk group. This, however, is not a perfectly fair test of fertility, since the number of young mice reared depends on other factors. It would have been better to count the young mice at birth, but this was impracticable.

There is no evidence in these experiments to show that the fertility of mice kept for three successive generations on a diet of pasteurized milk with suitable supplements is inferior to that of similar mice kept on raw milk.

Observations on Calves. Wilson, Minett, and Carling (1937) carried out an experiment in which alternate calves born into a herd of healthy cattle at Peppard were fed on raw or holder-pasteurized milk from the bulked supply of the herd. Pasteurization was carried out on the farm itself, the milk being held in the usual way at 145–150° F. for 30 minutes. Observations were made for 8 weeks on 25 calves fed on raw milk and on 23 calves fed on pasteurized milk. Hay was

ERRATA

Page 136, *three lines from the bottom.*
" 145–150° F." should be " 145° F."

Page 149, *three lines from the bottom.*
" 140–145° F." should be " 145–150° F."

allowed *ad lib*, though little was actually consumed during the first four weeks. The intention was to find out whether any difference in growth rate occurred between the two groups of animals. As has been already described (pp. 117, 118), no significant difference was in fact observed. The average increase in weight for the animals in the raw milk group was 61·18 per cent., and in the pasteurized milk group 62·94 per cent.

At the conclusion of the experiment many animals were sold, but 6 heifers in the raw-milk group and 11 in the pasteurized milk group were retained for adding to the main herd. No experimental observations were made on the breeding capacity of these animals, but in May 1938, nearly 4 years after the beginning and 20 months after the conclusion of the original experiment, an inquiry was made into the after-history of these animals. These results may be summarized as follows (Table XXXVIII).

TABLE XXXVIII

FERTILITY OF COWS THAT HAD BEEN BROUGHT UP ON RAW OR PASTEURIZED MILK. WILSON, MINETT, AND CARLING'S EXPERIMENT (1937)

	Heifers reared on Raw Milk.	Heifers reared on Pasteurized Milk.
No. used for breeding	6	9 [1]
No. calved satisfactorily	3	6
No. in Calf	3	2

[1] One animal in the pasteurized milk group, after being taken to the bull, had to be destroyed because of a uterine growth. Two animals which were retained in the herd were still too young at the time of collection of the data to be taken to the bull.

It may be noted that by May 1938, 1 heifer in the raw milk group had calved twice and 1 was in calf for the second time. In the pasteurized milk group 4 animals were in calf for the second time. The two groups cannot be strictly compared, because some of the animals in each group were sold with their first calves, so that no opportunity was available for studying them for their further breeding behaviour. It is quite clear, however, that not a single animal in either group proved sterile.

One further point may be mentioned. One of the bulls in the pasteurized milk group was registered for breeding purposes. This animal proved most satisfactory while it was kept in the Peppard herd, being used successfully to serve several heifers and cows. Later it was sold locally to the owner of a large herd, who likewise reported that it was proving most satisfactory.

This experiment on calves was not primarily designed for breeding

purposes. The data, however, collected on the breeding performance of the animals after the conclusion of the observations on growth failed to reveal any difference in fertility between animals reared on raw and those reared on pasteurized milk.

Summary

The recorded observations dealing with the fertility of rats, mice and calves fed on raw or pasteurized milk have been analysed. In none of the experiments is there any evidence to show that the fertility of animals fed on raw milk was greater than that of animals fed on holder-pasteurized milk. Indeed the suggestion is rather the opposite. In rats, for example, both in Mattick and Golding's and in Drummond's experiments, the average number of litters produced per mating and the average number of young born per litter were almost invariably higher in the pasteurized than in the raw milk group. There appears at present to be no evidence to justify the statement or the surmise that the use of pasteurized milk in the diet is in any way likely to impair the fertility of animals consuming it.

CHAPTER XI

EFFECT OF PASTEURIZATION ON THE DESTRUCTION OF PATHOGENIC ORGANISMS IN THE MILK

Of the various pathogenic micro-organisms that may be present in cows' milk the tubercle bacillus is regarded by general consent as the one which is most resistant to heat. If the process of pasteurization is sufficient to destroy this organism, then it may be safely concluded that all other pathogenic organisms will also be destroyed. It is therefore of considerable importance to ascertain what exactly are the time-temperature conditions requisite for destroying tubercle bacilli in milk. Fortunately there is a wealth of experimental data on this subject.

Before considering this it may be well to point out once more (see p. 58) that disinfection, like the coagulation of proteins, is a function of both time and temperature, and that strictly speaking the expression " thermal death point " is unjustifiable. It may also be pointed out that, since disinfection proceeds logarithmically, the time taken to reach complete sterility is dependent on the initial number of organisms to be destroyed. The heavier the degree of infection of the milk, the longer is the time taken at any given temperature to render the milk non-infective. This fact has not always been generally appreciated, and is responsible for some of the smaller discrepancies between the reports of different observers.

The present chapter will be devoted to a review of the *experimental* evidence gained in the laboratory and in large scale plants. The evidence furnished by the routine examination of pasteurized milks for pathogenic organisms will be more conveniently dealt with at a later stage (pp. 174–176).

Tubercle Bacillus. It is not proposed to abstract in detail the records of the numerous experiments that have been carried out in the laboratory and in processing plants during the past 50 years or so to determine the heat resistance of the tubercle bacillus. Most of the early records have been reviewed by Smith (1899), Rosenau (1908), North and Park (1927), and Zeller and his colleagues (1928). North and Park, reviewing the literature in 1927, were able to collect 89 reports since 1879. Much of the early work was vitiated by failure to realize the correct conditions for making a proper experiment and by imperfect technique. Such factors as scum or foam formation on the surface of the milk, curdling of the milk due to too high an acidity, imperfect distribution of the organisms in the milk through the use

of semi-solid inocula such as crude caseous material or sputum, and confusion of the lesions caused in guinea-pigs by dead tubercle bacilli with those caused by living, along with various other technical imperfections, all led to erroneous results of one type or another. Except for the more recent work, which has not yet been reviewed, it will be wiser to neglect these experiments and concentrate on those which were carried out under satisfactory conditions. Even so it will be impossible to deal here with any but the more important series of observations.

Theobald Smith (1899), one of the most prominent of all American bacteriologists, made a considerable number of observations on the heat resistance of tubercle bacilli, and found that these organisms, when suspended in distilled water, normal saline, nutrient broth, or milk, were destroyed completely in 15 to 20 minutes at 60° C. (140° F.). He observed, however, that if a pellicle was allowed to form on the surface of the milk, living tubercle bacilli might still be present after an hour.

Rosenau (1908) at Washington carried out an extensive series of experiments on milk that had been very heavily infected with pure cultures of bovine tubercle bacilli. Nine experiments were made using five different strains, and 121 guinea-pigs were inoculated directly from the milk before and after heating. With the exception of one animal in the first experiment the results were consistent in showing that all tubercle bacilli were killed by exposure to a temperature of 60° C. (140° F.), for 20 minutes.

Barthel (1918) and Barthel and Stenström (1918) in Sweden conducted a series of field experiments under conditions normally prevailing in practice, and found that tubercle bacilli in milk were destroyed by exposure to 60° C. (140° F.) for 10 minutes.

The most extensive and thorough observations ever carried out in any country were those recorded in 1925 by North, Park, Moore, Rosenau, Armstrong, Wadsworth, and Phelps in the United States. Experienced bacteriologists from six different laboratories, together with sanitary engineers, co-operated in this investigation. Pasteurizing plants of several different types were collected at a milk depot at Endicott on the Erie railroad and studied with the greatest care. The milk was infected with tubercle bacilli of human and bovine type, derived from cultures, from ground-up bovine tissues, and from human sputum. The degree of infection was sometimes very heavy, amounting to 10,000,000 tubercle bacilli per millilitre of milk. Altogether 1,958 guinea-pigs were inoculated from heated and unheated milk samples. The results showed that in properly designed and operated plants of the vat and pocket types tubercle bacilli were completely

destroyed by a temperature of 138° F. (58·9° C.) maintained for 30 minutes.

A corollary to this work was reported by North and Park (1927) in New York, who studied the heat resistance of the tubercle bacillus in the laboratory. Known quantities of bovine tubercle bacilli from cultures were added to the milk, which was then run into a lead coil submerged in water of the desired temperature. Their results may be recorded in tabular form (Table XXXIX).

TABLE XXXIX

TIME-TEMPERATURE CONDITIONS NECESSARY TO DESTROY BOVINE TUBERCLE BACILLI IN MILK (NORTH AND PARK 1927)

Temperature.	Time.
150° F. (65·6° C.)	2 minutes
145° F. (62·8° C.)	6 ,,
142° F. (61·1° C.)	10 ,,
140° F. (60·0° C.)	10 ,,
138° F. (58·9° C.)	20 ,,
136° F. (57·8° C.)	30 ,,
134° F. (56·7° C.)	40 ,,
132° F. (55·6° C.)	60 ,,
130° F. (54·4° C.)	60 ,,

It will be noted that, whereas most previous workers found that tubercle bacilli were destroyed at 60° C. (140° F.) in 20 minutes, North and Park found that 10 minutes were sufficient. This may have been due to a less heavy infection of the milk than that used by other observers, or to more uniform and rapid heating of the milk. It may be pointed out, however, that the performance of disinfection experiments in metal apparatus is not desirable, since traces of dissolved metal frequently have a toxic effect on bacteria.

No such objection applied to the more recent experiments in the United States of Corper and Cohn (1937, 1938), who used a glass tube for containing the material to be heated. Tubercle bacilli of human type suspended in buffer solution of *p*H 7·0, not in milk, were studied, and their survival was tested for by cultivation on suitable media instead of by the more usual method of guinea-pig inoculation. Though on general grounds it is less convincing, this method does answer the objection that heat merely lowers the virulence of the bacilli without actually destroying them. It was found that under these conditions mammalian tubercle bacilli were destroyed by exposure to a temperature of 55° C. (131° F.) for 15 to 30 minutes, or 60° C. (140° F.) for less than 5 minutes. Further observation, however, showed that, when

grown on media containing broth, their resistance was increased, so that they survived for about an hour at 55° C. and about 15 minutes at 60° C. At 65° C. (149° F.) they were killed almost immediately.

Incidentally North and Park, who reviewed much of the previous literature, reached the conclusion that bovine tubercle bacilli added to milk from artificial cultures were of the same degree of heat resistance as the bacilli present in naturally infected milk. This conclusion is of importance in interpreting the results of different workers who used artificially or naturally infected milk for their experimental inquiries.

In Great Britain experiments in recent years have been undertaken by White (1926), Jenkins (1926), and Meanwell (1927). White worked with naturally infected milk and with milk artificially infected with cultures or with caseous material. The degree of infection was sometimes very heavy, amounting to 1,600,000,000 tubercle bacilli per millilitre. The milk was heated in 1-litre quantities in an earthenware pot fitted with a perforated tin lid and standing in a pan of hot water. The temperature of the milk was 62·5° C. (144·5° F.) and was maintained for 30 minutes. Of 47 guinea-pigs inoculated with milk heated in this way, only 1 developed tuberculosis. Since another animal inoculated with the same milk at the same time showed no evidence of infection, wrong labelling of the first animal was suspected. In another series of experiments milk containing a minimum of 10,000 infective doses per millilitre before heating was found to be completely non-infective after exposure to 62·5° C. (144·5° F.) for only 5 minutes.

Jenkins (1926) made a few laboratory observations on naturally infected milk and on milk infected with cultures of human and bovine types of tubercle bacilli. She found that all tubercle bacilli were destroyed within 30 minutes by temperatures of 60° C. (140° F.) and 62·8° C. (145° F.), but not by one of 57·5° C. (135·5° F.).

Meanwell (1927) used naturally infected milk and carried out his observations on a pasteurizing plant of the holder type.

Of 156 guinea-pigs inoculated with milk that had been heated to 62·8° C. (145° F.) for 30 minutes, 2 developed tuberculosis.

Of 100 guinea-pigs inoculated with milk heated to 60° C. (140° F.) for 30 minutes, none became tuberculous.

Of 72 guinea-pigs inoculated with milk that had been heated to 60° C. (140° F.) for 20 minutes, 2 developed tuberculosis.

Of 18 guinea-pigs inoculated with milk that had been heated to 59·3° C. (138·8° F.) for 20 minutes, 10 became tuberculous.

It will be noted that four positive results were obtained with milk heated to 140° F. or over for 20 to 30 minutes. In three of these, flocculi developed in the milk as the result of heating. As previous workers have shown, this may lead to survival of the bacilli owing to

their protection by coagulated protein material. In the fourth the milk appeared to be satisfactory, but the guinea-pig is stated to have shown enlarged and caseating mesenteric glands. Most workers have found that these glands do not become caseous after subcutaneous or intramuscular inoculation with tubercle bacilli. The occurrence of enlarged caseating mesenteric glands is strongly suggestive either of natural infection by the mouth or of pseudo-tuberculosis. It is therefore very doubtful whether the exceptional development of tuberculosis in the guinea-pigs in Meanwell's experiments can be held to invalidate the general finding that tubercle bacilli in milk are destroyed by a temperature of 140° F. (60° C.) in 20 minutes.

In Germany recent observations on actual pasteurizing plants include those of Machens (1925), Proescholdt (1925, 1926*a*, *b*, 1927), Seelemann (1926), and Zeller and his colleagues (1928). Seelemann relied on guinea-pig inoculation for testing the infectivity of the milk, while Machens and Proescholdt in addition fed the milk to calves or young pigs. All of these workers came to the conclusion that milk heated at 63° C. (145·5° F.) for 30 minutes in pasteurizing plants of the holder type was rendered completely non-infective to animals.

Zeller, Wedemann, Lange, and Gildemeister (1928) carried out an extensive series of experiments on two different holder pasteurizing plants. Naturally infected tuberculous milk was added to ordinary raw milk in a proportion of 0·15–1·9 per cent. and heated to 60–63° C. (140–145·4° F.) for 30 minutes. Tests for tubercle bacilli were made by feeding the milk to calves and young pigs, killing them after suitable intervals, examining the organs for evidence of tuberculosis, and inoculating any doubtful lesions into guinea-pigs. Omitting animals dying of intercurrent disease, the results may be summarized by saying that 29 out of 32 pigs and 15 out of 16 calves fed on raw milk showed evidence of tuberculosis, while none out of 30 pigs and only 4 out of 22 calves fed on pasteurized milk did so. The fact that a few calves fed on tuberculous milk after pasteurization became infected is disquieting, but careful examination of the records shows that this experiment, like so many before it, was vitiated by technical defects. In order to have sufficient tuberculous milk available for any given test, it had to be collected from tuberculous cows for some days beforehand, with the result that much of it was old and contained gross floccules which served to protect the tubercle bacilli from the full effects of the heat treatment.

Finally, for the sake of interest, it may be worth while referring to a paper by Dahlberg (1932), in which he plotted the results obtained by Marquardt and Dahlberg (1931) on the effect of heat on the creaming capacity of milk (see pp. 66, 86) against the results obtained by North

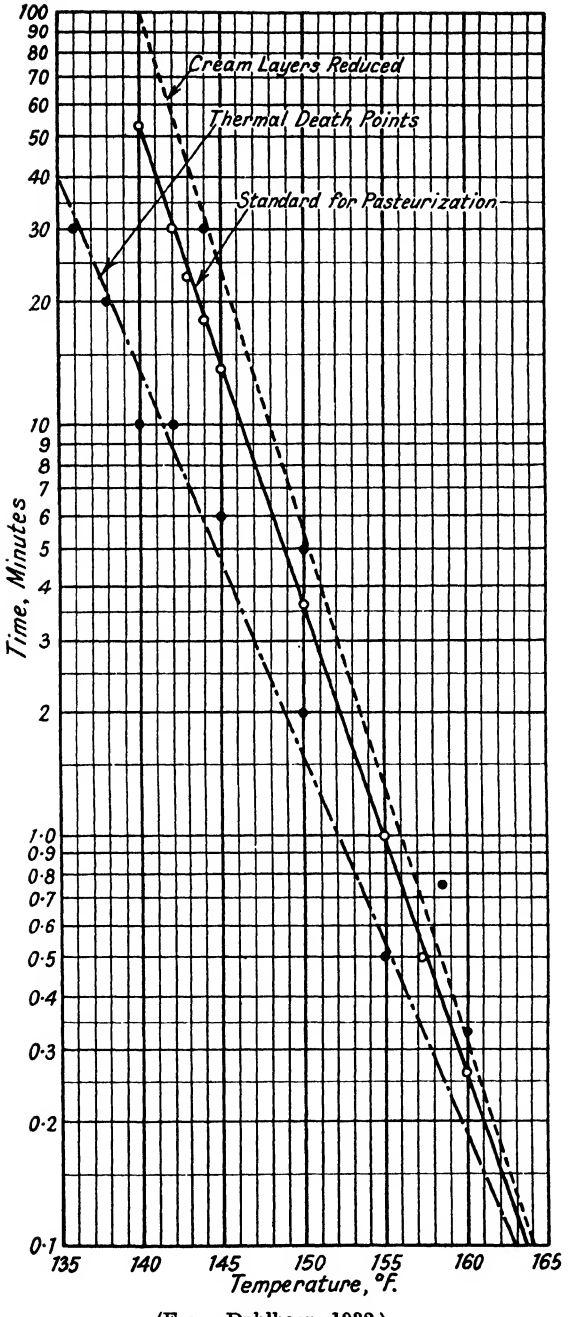
and Park (1927) on the destruction of tubercle bacilli by heat (see p. 141). Both curves appeared to be similar to that for a monomolecular reaction. Between them Dahlberg inserted a third curve to act as a guide to the time-temperature combinations that would afford an adequate safety margin for the destruction of tubercle bacilli without interfering with the creaming properties of the milk (see Figure, p. 145).

It will be noted that at a temperature of 145° F. (62·8° C.) tubercle bacilli are recorded as being killed in 5 minutes, and at 150° F. (65·6° C.) in about 1½ minutes. Since the regulations in Great Britain for holder pasteurization demand that the milk shall be held for at least 30 minutes at a temperature of 145–150° F., it is clear that a wide safety margin is provided by the current method of holder pasteurization.

Published observations on the destruction of tubercle bacilli by a time-temperature combination similar to that used in *High Temperature Short Time pasteurization* are so far few in number. Mattick and Hiscox (1939) carried out investigations on 37 samples of raw milk, 28 of which contained living tubercle bacilli. The milk was heated in an old type of A.P.V. cast-plate heat exchanger. A temperature of 158·5–164° F. (70·3–73·3° C.) was reached in about 16 seconds, and the milk was then kept for a further 11·5 seconds at a temperature of 158–163·5° F. (70–73° C.). The deposit from 100 ml. of the heated milk was inoculated into 2 guinea-pigs, which were killed not less than 9 weeks later. None of the pasteurized samples proved to be infected with tubercle bacilli, though 4 of them, which had been kept at a temperature below 160° C., yielded a positive phosphatase reaction. The authors conclude that under the conditions of their experiments tubercle bacilli in naturally infected milk are killed within 11·5 seconds at a temperature of 158° F. It should be pointed out, however, that in fact only 1 sample was heated to as low a temperature as this, and that all but 5 of the samples were kept at 160° F. or over.

Taking this work in conjunction with the results of Dahlberg (1932) and of previous workers quoted by Mattick and Hiscox, it may be tentatively concluded that tubercle bacilli in naturally infected milk are destroyed within 12 to 15 seconds at 160° F. (71·1° C.). Since the regulations in Great Britain for the High Temperature Short Time process demand that the milk shall be retained at a temperature of not less than 162° F. (72·2° C.) for at least 15 seconds, it will be seen that there is some margin of safety afforded.

Typhoid Bacillus. Since typhoid bacilli are less resistant to heat than tubercle bacilli, there is no need to review the literature on their destruction in milk in any detail.



(From Dahlberg, 1932.)

Rosenau (1908), working in the laboratory, found that typhoid bacilli in milk were killed by exposure to a temperature of 60° C. (140° F.) in 2 minutes, whereas tubercle bacilli required 20 minutes.

Krumwiede and Noble (1921), also working in the laboratory, infected milk heavily with typhoid bacilli, exposed it to a temperature of 60° C. (140° F.), and made cultures after 10 to 20 minutes. Under these conditions the bacilli were invariably destroyed, but the minimal degree of heat required was not determined.

North and his colleagues (1925), working with pasteurizing plants of the holding type, found that typhoid bacilli were destroyed at 132° F. (55·6° C.) in 30 minutes whereas tubercle bacilli required a temperature of 138° F. (58·9° C.) for the same length of time.

Zeller and his colleagues (1928) in Germany, who carried out a large number of experiments in pasteurizing plants of the holder type, found that typhoid bacilli and other members of the Salmonella group, were invariably destroyed by pasteurization of the milk at 60–63° C. (140–145° F.) for 30 minutes. The minimum degree of heat exposure required was not determined.

Beamer and Tanner (1939), who made particularly careful observations in the laboratory, found that, when added to broth in the enormous concentration of 5 to 10 million organisms per millilitre, typhoid bacilli were destroyed at 60° C. (140° F.) within 10 minutes and at 65° C. (149° F.) within 5 minutes.

Brucella abortus. This organism is likewise readily destroyed by such time-temperature combinations as are commonly used in holder pasteurization.

Rosenau (1908) made a few experiments with the closely related organism, *Br. melitensis*, and found that exposure to a temperature of 60° C. (140° F.) for 20 minutes was more than sufficient to destroy this organism in milk.

Zeller and his colleagues (1928), on the basis of experiments carried out in the Imperial Health Office in Berlin, state that under laboratory conditions suspensions of *Br. abortus* are sterilized by exposure to a temperature of 55° C. (131° F.) for 25 to 30 minutes, 60° C. (140° F.) for 10 to 15 minutes, and 65° C. (149·0° F.) for 5 to 10 minutes. In pasteurizing plants of the holder type numerous experiments showed that *Br. abortus* was invariably destroyed by a temperature of 60–63° C. (140–145·4° F.) maintained for 30 minutes.

Park, Graham, Prucha, and Brannon (1932), working with porcine strains of *Brucella*, found that when added to milk in enormous quantities, such as 500,000,000 per millilitre, and exposed in sealed tubes to prevent scum formation, the organisms were all killed by a tem-

perature of 144° F. (62·2° C.) in 7 minutes, and 142° F. (61·1° C.) in 15 minutes.

Streptococci. Numerous experiments have been carried out on the heat resistance of pathogenic hæmolytic streptococci in milk.

Rosenow (1912) isolated strains of streptococci from milk and from epidemic sore throats, suspended them in broth and in milk, and found that all virulent strains were killed at 60° C. (140° F.) within 20 minutes.

Ayers, Johnson, and Davis (1918) made observations on 27 strains derived from pathological sources. Suspended in milk, 22 strains were killed by temperatures between 51·7° C. (125° F.) and 57·2° C. (135° F.) maintained for 30 minutes, while 5 strains required a temperature of 60° C. (140° F.) for 30 minutes.

Davis (1918) studied 24 strains of pathogenic hæmolytic streptococci of human origin, and found that all were destroyed by exposure to a temperature of 60° C. (140° F.) for 30 minutes. Of 74 strains of hæmolytic streptococci isolated from milk, all of which were practically avirulent, 20 resisted heating to 68·3° C. (155° F.) for 30 minutes. These experiments show that, though strains of hæmolytic streptococci of human type, which may occasionally gain access to milk, are readily destroyed by the usual pasteurizing conditions, not all strains of hæmolytic streptococci isolated from milk are equally susceptible to heat. So far as is known, however, only strains of human type are responsible for milk-borne disease in man.

Zeller and his colleagues (1928), who made observations on holder pasteurizing plants, likewise showed that mastitis streptococci were not completely destroyed by exposure to 60–63° C. (140–145·4° F.) for 30 minutes, though their numbers were very greatly diminished.

On the other hand Pullinger (1935), who examined 58 samples of raw bulked milk containing β-hæmolytic streptococci, found that in all of them the organisms were destroyed by holder pasteurization in a commercial plant at 62·8° C. (145° F.) for 30 minutes.

It seems clear that *Str. pyogenes*, which is responsible for scarlet fever and septic sore throat in man, is readily destroyed in milk by pasteurization. With regard to β-hæmolytic streptococci of bovine types, the data are somewhat discrepant, some workers finding that they are all destroyed by pasteurization at 63° C. (145·4° F.) for 30 minutes, others finding that they are not. Further observations are called for.

Staphylococcus aureus. This organism, though responsible for a small proportion of cases of mastitis in cattle, is not known to be pathogenic to man when ingested by the mouth. Certain strains, however, may grow in the milk after it is withdrawn from the udder and produce a toxin which gives rise to food poisoning in man. This

condition has only recently been recognized, and very few experiments seem to have been made on the effect of pasteurization on the destruction of staphylococci. Laboratory experiments, however, show that this organism is unusually resistant to heat, and is not destroyed with certainty by a temperature of 60° C. (140° F.) maintained for 30 minutes, though a temperature of 65° C. (149° F.) almost invariably proves fatal within this time.

Diphtheria and dysentery bacilli, cholera vibrios, and foot-and-mouth virus. The observations of Rosenau (1908), of North and his colleagues (1925), and of Zeller and his colleagues (1928) all show that these organisms are readily destroyed by holder pasteurization carried out at 60–63° C. (140–145·4° F.) for 30 minutes. In Rosenau's experience diphtheria bacilli and cholera vibrios proved very susceptible, being nearly always killed by the time the milk had reached 55° C. (131° F.), and none of them surviving 60° C. (140° F.) for 1 minute. Dysentery bacilli were a little more resistant than typhoid bacilli, but were invariably killed within 10 minutes at 60° C. (140° F.). Foot-and-mouth virus, in the experience of Zeller and his colleagues, was destroyed by holder pasteurization at 60–63° C. (140–145·4° F.) within 10 minutes.

Summary

1. Experimental observations in the laboratory and in actual pasteurizing plants, dealing with the destruction of pathogenic organisms that may occur in milk, have been reviewed.

2. It is pointed out that the destruction of bacteria by heat is a function of both time and temperature, and is influenced, among other things, by the initial number of organisms present. The greater the degree of infection of the milk, the longer is the time necessary at any given temperature to render it non-infective. The experimental data recorded in this chapter refer to milks that have been heavily infected, though not always to the same degree.

3. From the vast number of experiments that have been carried out during the past 50 years by different workers in different countries, it may be concluded that mammalian tubercle bacilli, whether naturally present in the milk or added artificially, are completely destroyed by exposure to a temperature of 138° F. (58·9° C.) for 30 minutes, 140° F. (60° C.) for 20 minutes, 145° F. (62·8° C.) for 5 to 10 minutes, 150° F. (65·6° C.) for 2 to 5 minutes, or 160° F. (71·1° C.) for 12 seconds.

4. Other pathogenic organisms, with the exception of *Staphylococcus aureus* (see below), are all destroyed by a time-temperature exposure equal to or less than that necessary to destroy the tubercle bacillus.

5. Thus at 60° C. (140° F.) the following organisms are destroyed in the following times :

Diphtheria bacilli and cholera vibrios in about 1 minute.
Typhoid bacilli in about 2 minutes.
Dysentery bacilli in about 10 minutes.
Br. abortus in 10–15 minutes.
Str. pyogenes within 30 minutes.

6. With regard to bovine types of β-hæmolytic streptococci, which are probably non-pathogenic to man, the available evidence is somewhat conflicting. On the whole, it looks as if strains of some types are not completely destroyed by holder pasteurization at 63° C. (145·4° F.) in half an hour.

7. *Staph. aureus*, which though it may form a toxin in the milk is not pathogenic for human beings when ingested by the mouth, is not destroyed with certainty by exposure to a temperature of 60° C. (140° F.) for 30 minutes, but is destroyed by a temperature of 65° C. (149° F.) within this time.

8. The available experimental evidence, therefore, leaves no doubt that, with the occasional exception of staphylococci, all pathogenic non-sporing bacilli which are liable to contaminate milk are destroyed by exposure to a temperature of 60° C. (140° F.) within 30 minutes.

11. Since the official regulations for holder pasteurization in this country require the exposure of the milk to a temperature of 140–145° F. (62·8–65·6° C.) for not less than 30 minutes, it will be seen that there is a considerable margin of safety afforded to the consumer.

CHAPTER XII

THE EFFECT OF PASTEURIZATION ON THE EXTENT OF MILK-BORNE DISEASE

Difficulties in obtaining satisfactory Data. If milk containing pathogenic organisms not infrequently gives rise to disease, and if adequate pasteurization destroys these organisms, then the introduction of compulsory pasteurization should lead to a disappearance of milk-borne disease. There is good reason to believe in the general truth of this proposition, but the difficulties of supplying wholly satisfactory evidence to support it are considerable. This is due to several causes.

In the first place, epidemic milk-borne disease is very capricious in its incidence. A given town may be free for several years, and then suffer a disastrous outbreak. Unless the incidence is studied over a long period, the results cannot be wholly convincing. Pasteurization on a large scale is of such comparatively recent introduction that there are few areas in which satisfactory data on the incidence of milk-borne disease are yet available.

Secondly, even when data are available on the incidence of milk-borne disease, they are not always reliable. In general, pasteurization is practised most extensively in large municipalities with a competent health service, and raw milk is drunk most extensively in smaller municipalities and rural areas where the health services are less efficient. If an outbreak of milk-borne disease does occur, it is far more likely to be detected in a large than in a small town. Indeed, as is pointed out elsewhere (pp. 20–23), there is strong reason to believe that a great deal of milk-borne disease is never recognized as such, particularly in the less densely populated areas. It follows, therefore, that the available figures for the incidence of milk-borne disease are liable to underestimate its extent in the very areas where most raw milk is consumed.

Thirdly, comparison of the incidence of a given disease before and after the introduction of compulsory pasteurization into a particular town is not an ideal method of determining the effect of pasteurization, since pasteurization may not be the only factor affecting the incidence of the disease that has changed during the period under examination. Summer diarrhœa of infants is a case in point. The mortality from this disease has fallen enormously in our large towns in the past 15 or 20 years. During this period pasteurized, boiled, or dried milk has

been generally substituted for raw milk in infant feeding, and the conclusion may be drawn that this change has been responsible for the fall in the mortality. But other factors affecting the incidence, such as the prevalence of flies, the cleanliness of the milk, the number and activity of infant welfare centres, and so on have also changed, and it is impossible to know how much of the decrease in the mortality should be attributed to the heat treatment of the milk and how much to these other factors. If half the infant population was fed on raw and half on heated milk, and the two groups were of similar composition and were treated in every other way alike, then an answer might be obtained; but under present conditions this is impossible.

Fourthly, with a disease such as tuberculosis only a small proportion of which is caused by the bovine type of bacillus, statistical comparisons between towns in which the greater part of the milk is pasteurized and those in which the greater part is consumed raw are liable to be vitiated by differences in the case incidence of disease due to the human type. Imagine, for example, a large industrial town in which the whole of the milk supply is pasteurized, but in which, owing to unfavourable evironmental conditions, the incidence of tuberculosis of human origin is very high, and a small country town in which the whole of the milk is drunk raw, but in which the incidence of tuberculosis of human origin is very low. Comparison of the non-pulmonary tuberculosis death-rate of these two towns might be very misleading, for even if not a single death of milk-borne origin occurred in the first town, and quite considerable numbers occurred in the second town, the non-pulmonary tuberculosis death-rate, which is determined mainly by infections due to the human type, would be considerably higher in the first town than in the second. Nothing short of a bacteriological examination to determine the type of infecting bacillus in every patient dying of non-pulmonary tuberculosis would reveal the true state of affairs in regard to milk-borne tuberculosis in these two towns. So far, an investigation of this type has been carried out in only two towns, namely Toronto and Paris. The results in both these instances were most striking (see p. 156).

Sufficient has been said to show that our information on the effect of pasteurization on the diminution of milk-borne disease is less extensive and satisfactory than it might be. Nevertheless we may review briefly the available evidence in relation to the incidence or the mortality of different diseases.

Tuberculosis. Under this heading two sources of evidence may be considered, (1) The fall in the non-pulmonary tuberculosis death-rate, and (2) The diminution or disappearance of tuberculosis due to the bovine type of bacillus.

(1) *Fall in the Non-pulmonary Tuberculosis Death-rate.* Winslow and Gray (1924) selected 22 cities in the United States and divided them into four groups according to the extent of pasteurization between 1915 and 1920. Group I had less than 50 per cent. of its milk supply pasteurized in 1920 and Group IV had more than 90 per cent. of its milk supply pasteurized before 1915. A comparison was then made in these groups between the pulmonary and the non-pulmonary tuberculosis death-rates in children under 4 years of age. It was found that the fall in the *pulmonary* tuberculosis death-rate was much the same in all four groups, namely about 30 per cent. Similarly, the fall in the non-pulmonary tuberculosis death-rate in infants under 1 year of age was much the same in all four groups, namely about 38 per cent. But in the age group 1–4 years the fall in the *non-pulmonary* tuberculosis death-rate corresponded more or less with the extent of pasteurization. Thus the figures for the four groups were + 13, − 16, − 2, and − 34 per cent. In Group I the absolute rate was about 100 per 100,000 and in Group IV only 75 per 100,000. The decrease of 25 per 100,000 is ascribed to the beneficial effects of pasteurization.

These figures are suggestive, but for reasons pointed out above (p. 150) they cannot be regarded as wholly conclusive. Other factors besides pasteurization may have influenced the fall in the non-pulmonary tuberculosis death-rates in the various groups, such as differences in the amount of Tuberculin Tested milk consumed, or the activity of child welfare centres. Nevertheless the fact that the pulmonary tuberculosis death-rate fell to the same extent in the four groups, while the non-pulmonary tuberculosis death-rate altered differentially, does suggest that changes were occurring in the amount of bovine type infection.

The fall in the non-pulmonary tuberculosis death-rate in the United States is not confined to the 1–4 year age group. Chadwick (1935), for example, showed that in Massachusetts, where pasteurized milk began to be used about 1910, the non-pulmonary tuberculosis death-rate at all ages had fallen by 92 per cent., while the pulmonary tuberculosis death-rate had fallen by only 64 per cent. Here again the great increase that has occurred in the proportion of Tuberculin Tested milk consumed may claim with pasteurization some share in the enormous diminution in the amount of non-pulmonary tuberculosis.

In Great Britain various observers have pointed out that the death-rate from non-pulmonary tuberculosis has fallen more rapidly than the corresponding rate for pulmonary tuberculosis. If this is due wholly or in part to a diminution in the amount of infection of bovine origin, it must be attributed mainly to an increase in the heat treatment of milk, since it has already been shown that tuberculous infection

TABLE XL

FALL IN THE PULMONARY AND NON-PULMONARY STANDARDIZED TUBERCULOSIS DEATH-RATES PER 100,000 IN DIFFERENT AREAS IN ENGLAND AND WALES 1911–1937. (ALL AGGREGATES REFER TO OUTSIDE ADMINISTRATIVE COUNTY OF LONDON)

Area.	Death-rates.								Percentage fall in death-rate between 1911 and 1937.	
	1911.		1921.		1931.		1937.			
	P.T.	N.P.T.	P.T.	N.P.T.	P.T.	N.P.T.	P.T.	N.P.T.	P.T.	N.P.T.
London administrative county .	130	46	100	25	80	15	61	11	53	76
Aggregate county boroughs . .	126	48	99	31	84	21	65	16	48	67
Aggregate urban districts . .	92	42	78	28	62	18	48	14	48	67
Aggregate rural districts . . .	82	34	70	23	53	17	40	14	51	59

P.T. = pulmonary tuberculosis. N.P.T. = non-pulmonary tuberculosis.

of the raw milk supply is just as frequent now as it was twenty or thirty years ago (pp. 13, 14). To test the truth of this conclusion it is worth while studying the rates of fall in the tuberculosis death-rates in different areas according to the extent of pasteurization of the milk supply. For this purpose we may choose London, and the aggregate county boroughs, the aggregate urban districts, and the aggregate rural districts outside London, and compare the fall between the years 1911 and 1937. In London pasteurization began to be employed in the early years of the century, and has been increasing both in amount and efficiency ever since, till in 1937 probably 98 per cent, of the milk supply was pasteurized or heat-treated in some other way. In rural districts, on the other hand, there has been very little pasteurization, and it is only within recent years that pasteurized milk has been available in a few areas. In the towns, the extent of pasteurization has, on the average, been more or less intermediate between that of London and that of the rural districts. If, therefore, pasteurization has played an important part in the fall of the non-pulmonary tuberculosis death-rate, it should follow that this rate should have decreased most in London and least in the rural districts. Table XL (p. 153) shows that this is in fact what has occurred.

Though the *pulmonary* tuberculosis death-rate has fallen to almost the same extent in the rural districts as in London between 1911 and 1937, the fall in the *non-pulmonary* tuberculosis death-rate in the rural districts has been about only three quarters of that in London.

TABLE XLI

FALL IN THE NON-PULMONARY TUBERCULOSIS DEATH-RATE PER 100,000 AT AGES IN DIFFERENT AREAS IN ENGLAND AND WALES BETWEEN 1911 AND 1937. (ALL AGGREGATES REFER TO OUTSIDE ADMINISTRATIVE COUNTY OF LONDON)

Area.	Death-rates.								
	0–5 years.			5–15 years.			15–25 years.		
	1911.	1937.	% fall.	1911.	1937.	% fall.	1911.	1937.	% fall.
London administrative county	231	33	86	46	10	78	18	9	50
Aggregate county boroughs	237	52	78	45	15	67	23	14	39
Aggregate urban districts	198	49	75	38	12	68	22	12	45
Aggregate rural districts	136	44	68	29	10	66	22	12	45

Analysis of the death-rates shows that the greatest saving of life from non-pulmonary tuberculosis has occurred in children under 15 years of age (Table XLI).

It will be noticed that while the fall in London and the rural districts in the 15–25 age group has been almost the same, in the 0–5 and the 5–15 age groups the fall in London has been far greater than in the rural districts. This sequence is what would be expected on the assumption that a proportion of the non-pulmonary tuberculosis deaths in infancy and childhood are the result of milk-borne infection, and that the danger of this infection is removed by adequate pasteurization or other form of heat treatment of the milk.

(2) *Diminution in the Amount of Tuberculosis due to the Bovine Type of Bacillus.* One of the most striking observations made by clinicians in the United States, Canada, Great Britain, and certain other countries is that during the past 20 or 30 years primary abdominal tuberculosis in infants and children, which bacteriological study has shown to be almost exclusively due to the bovine type of bacillus, has changed from being one of the commonest clinical conditions met with in the out-patient departments of our large hospitals to being one of comparative rarity. In 1911 in England and Wales 2,700 deaths occurred from this cause in children under 5 years of age; in 1937 only 143 deaths occurred. The death-rate from abdominal tuberculosis fell in fact from 70 per 100,000 to 5 per 100,000, that is by about 93 per cent. Unfortunately the Registrar-General does not provide figures to show the distribution of deaths from this cause in different areas, but judging from clinical experience it is probable that the fall in London, where practically all the milk is pasteurized or otherwise heat-treated, has been nearer 100 than 93 per cent. Similarly in Glasgow, Macgregor (1930) has pointed out that the diminution in the abdominal form of tuberculosis has been outstandingly rapid. Not the whole of this fall can, of course, be ascribed to pasteurization. The increased boiling of milk and the provision of dried milk for infants, and the greater amount of Tuberculin Tested milk now available, can probably claim a large share. Nevertheless it appears to be true that the extensive replacement of raw by heated milk in the feeding of infants and children during the last 20 or 30 years is largely responsible for the diminution in the death-rate from primary abdominal tuberculosis.

This statistical experience is supported by pathological observations. Oesterreich (1937), for example, in Germany during the course of 1,605 post-mortem examinations at Greifswald, met with primary tuberculosis lesions 503 times; of these, 102, or 20·1 per cent. were in the mesentery. In other German cities the proportion of primary

abdominal tuberculosis found at autopsy was only 1–5 per cent. The author ascribes this difference to the fact that most of the cadavers examined in the Greifswald pathological institute came from the country districts, where milk was drunk raw, while in the large cities the cadavers were mainly derived from the hospital population of the cities themselves where most of the milk was pasteurized or derived from tuberculosis-free herds.

We may now refer to the most striking piece of evidence that has so far been obtained to prove that adequate pasteurization of the whole milk supply leads to a complete disappearance of all milk-borne tuberculous infection. Price (1934) in Toronto recorded the results of a painstaking investigation that she had carried out during the previous eight years. Working at the Hospital for Sick Children she studied the type of infecting bacillus in a series of 300 cases of non-pulmonary tuberculosis in children under 14 years of age. Of these, 15 per cent. were found to be infected with the bovine tubercle bacillus. Inquiry revealed that every one of these children came from parts of Ontario where the milk was drunk raw. In Toronto itself, where compulsory pasteurization had been in force since 1915, not a single case of infection with the bovine type was discovered, in spite of the fact that 4 per cent. of the churn milk and 26 per cent. of the bulked milk entering the city contained tubercle bacilli. That pasteurization was carried out effectively was shown by an examination of 100 samples of milk after processing, none of which proved infective for guinea-pigs. This investigation, based as it was on a bacteriological study of the infecting types of tubercle bacilli, affords far and away the most satisfactory and convincing evidence that adequate pasteurization of a tuberculous milk supply affords complete protection of the population against milk-borne tuberculosis. It is small wonder that compulsory pasteurization for the whole province of Ontario has now been introduced and carried effectively into operation.

Since Price's work in Toronto, observations have been carried on along similar lines in Paris. Lesné and his colleagues (1936), working at the Trousseau Children's Hospital, isolated tubercle bacilli from 130 infants and children suffering from meningitis. All of these strains except 9 proved to be of the human type. The 9 patients infected with the bovine type were not native Parisians, but were brought up in the country and fed on raw milk.

More recently Armand-Delille (1939) in Paris has studied material from 50 young children suffering from various forms of tuberculosis. Not one strain of bovine type was isolated, showing that in Paris, where children are brought up on pasteurized or some other type of heated milk, the danger of milk-borne tuberculosis appears to have

been eliminated. Careful inquiry disclosed a source of human infection for each of the tuberculous patients studied.

Undulant Fever. Very much less attention has been paid to undulant fever than to tuberculosis, and our knowledge of its incidence is grossly deficient. Nevertheless there is reason for believing that adequate pasteurization of milk and cream protects against this disease.

Dolman and Hudson (1938) in Vancouver, for example, found that 53 out of 55 different dairies were supplying milk infected with *Br. abortus*. An investigation of 5,068 sera from febrile patients showed that 101, or 2 per cent., agglutinated *Br. abortus* to a titre of 1/80 or over, indicating that the patients were probably suffering from undulant fever. Fifteen cases of the disease were actually diagnosed by city physicians. Inquiry revealed that every one of these patients consumed raw milk prior to the onset of illness. Considering that 78 per cent. of the milk in Vancouver is pasteurized and only 22 per cent. is consumed raw, the odds due to chance alone against getting fifteen cases in the raw milk group and none in the pasteurized are several thousand to one.

London has had a very similar experience. In spite of its large population, comparatively few cases of undulant fever have been recorded. Some of these have occurred in patients who have spent their holiday in the country and appear to have been infected by drinking raw milk on farms or elsewhere. Of the few indigenous cases, practically all give a history of drinking raw milk, such as Tuberculin Tested. This is the more remarkable in view of the fact that raw milk constitutes less than 2 per cent. of the total supply in London. Not a single case has so far been brought to light in which pasteurized milk and cream were alone concerned.

Epidemic Milk-borne Disease. The effect of pasteurization on epidemic milk-borne disease may be considered from various aspects.

(1) Both in Great Britain and America epidemic milk-borne disease can almost invariably be traced to the consumption of raw milk or cream. Thus in New York State, excluding New York City, during the years 1917 to 1940 no fewer than 160 out of 163 outbreaks were traced to raw milk (Ward 1941). Similarly, 57 out of 58 outbreaks in New Jersey during the years 1910 to 1937 were traced to raw milk (Blanchard 1937). Again, of 18 outbreaks in Massachusetts during the years 1927 to 1932, 16 were due to raw milk. Of the two outbreaks due to pasteurized milk, both resulted from infection of the milk after processing (Bigelow and Forsbeck 1930, Bigelow and Feemster 1933). In practically all of the 113 outbreaks recorded in Great Britain between 1912 and 1937 raw milk or cream appears to have been involved.

(2) Milk-borne epidemics occur mainly in areas where raw milk is extensively drunk. It is significant that in the United States milk-borne disease has been practically banished from the large cities where the great majority of the milk is pasteurized, and is now almost entirely confined to the rural districts and smaller towns where most of the milk is consumed raw (Report 1931*b*).

Since Toronto introduced compulsory pasteurization in 1915 not a single case of infectious disease carried by milk has been recognized. In this connection it must be remembered that Toronto has an excellent public health and bacteriological service, fully alive to the danger of milk-borne infection.

(3) The introduction of pasteurization has often been followed by a marked diminution in the amount of epidemic summer diarrhœa of infants. The enormous fall in the death-rate from this cause that has occurred in London during the past twenty years or so has already been commented on (p. 39); but it was pointed out that, since other factors had almost certainly been operative as well, it was impossible to say how much of the fall could legitimately be ascribed to the increasing heat treatment of the milk supply. A classic illustration, however, of the effect of pasteurization is afforded by the experience of a children's institution on Randall's Island, New York, where a mortality of 44·36 was promptly reduced to one of 19·8 after all the milk was pasteurized, no other hygienic measures having been put into operation (Report 1931*b*).

(4) Numerous instances are on record where the same supply of milk has been drunk both raw and pasteurized, and where disease has been confined to consumers of the raw milk portion (Report 1932*b*, 1936*e*, Camps and Wood 1936, Blanchard 1937). A typical example is afforded by the recent outbreak of gastro-enteritis at Wilton, where the raw portion of the infected milk gave rise to at least 132 cases of acute food poisoning, while the pasteurized portion was consumed with impunity.

(5) So effective is pasteurization in destroying pathogenic organisms in milk that it is now the routine public health procedure adopted in outbreaks of milk-borne disease. For example, in the large typhoid outbreak at Bournemouth, Poole, and Christchurch in 1936 the Medical Officer of the Ministry of Health, who was called in to advise on the procedure to be adopted, recommended immediate pasteurization, confidently anticipating that no further primary cases of the disease would occur after the expiry of the necessary incubation period. This anticipation was fulfilled. Once the milk supply was rendered safe, an inquiry was set on foot into the method by which it became infected. This sequence of events, it may be noted, namely pasteurization first

and inquiry after, is the same as was followed in the Croydon water-borne typhoid outbreak of 1937, pasteurization being replaced by its equivalent of chlorination.

Summary

1. Reasons are given for believing that the evidence we have on the effect of pasteurization in diminishing the extent of milk-borne disease is less complete than it might be.

2. In the United States the fall in the non-pulmonary tuberculosis death-rate has been considerably greater in towns where the majority of the milk is pasteurized than in towns where a smaller proportion is pasteurized, even though the fall in the pulmonary tuberculosis death-rate has been much the same in the two groups.

3. In this country the fall in the non-pulmonary tuberculosis death-rate between 1911 and 1937 has been greatest in London, where pasteurization has been most extensively practised, and least in the rural areas where little pasteurized milk has been available. The county boroughs and urban districts occupy an intermediate position in both respects. Since the decrease in the *pulmonary* tuberculosis death-rate has been much the same in all districts, it is clear that some special factor has been operating in London and the larger towns to accelerate the rate of fall in the *non-pulmonary* tuberculosis death-rate. It is believed that this factor is heat treatment of the milk.

4. Analysis of the non-pulmonary tuberculosis death-rate shows that the greatest difference in the rate of fall between London and the rural districts has occurred in the 0–5 and 5–15 year age groups. This is what would be expected if the increased rate of fall in London was attributable to pasteurization.

5. The most convincing evidence of the effect of compulsory pasteurization on eliminating the risk of milk-borne tuberculosis is provided by the careful bacteriological observations of Price in Toronto. Price found that 15 per cent. of 300 children suffering from non-pulmonary tuberculosis were infected with the bovine type of tubercle bacillus. All of these children came from parts of Ontario where milk was drunk raw. In Toronto itself, where compulsory pasteurization has been in force since 1915, not a single case of infection with the bovine type was found, in spite of the fact that 26 per cent. of the bulked milk entering the city contained tubercle bacilli.

6. Similar observations in Paris have likewise shown that bovine infections in children are invariably contracted in the country where raw milk is drunk. Not one case of infection with this type has been encountered of recent years in native Parisian children brought up on pasteurized or other type of heated milk.

7. As regards undulant fever, experience in Toronto, Vancouver and London has shown that, when milk-borne, this disease is met with only in those who have consumed raw milk or cream.

8. Examination of the available figures for epidemic milk-borne disease shows that (i) milk-borne outbreaks are almost invariably due to raw milk or cream ; (ii) milk-borne outbreaks in the United States have practically ceased to occur in areas where compulsory pasteurization has been introduced, and are now confined mainly to small towns and rural districts where most of the milk is consumed raw ; (iii) pasteurization of the milk supply seems to have played a part in the diminution of epidemic summer diarrhœa in infants ; (iv) several recent instances are on record where the same supply of milk has been drunk both raw and pasteurized, and where disease has been confined to consumers of the raw milk portion ; (v) pasteurization is so effective that it has now become the routine public health procedure to prevent further infection in milk-borne epidemics.

CHAPTER XIII

OBJECTIONS RAISED AGAINST PASTEURIZATION

In the following pages we shall attempt to answer only those objections to pasteurization that have some bearing on health. Economic and political objections are outside the scope of this book. A few objections of insufficient weight to be mentioned here are dealt with in an appendix.

"*Pasteurization diminishes the nutritive value of the milk*"

The effect of pasteurization on the nutritive value of the milk has been dealt with at length in Chapters VIII and IX, and there is no need here to present the evidence afresh. As a whole the numerous observations and experiments that have been made can be summarized by saying that the amount of change brought about by holder pasteurization in the chemical constitution of the milk is very slight, and that the effect of holder pasteurization on the total nutritive value of the milk is almost negligible.

This interpretation is now generally agreed upon by scientific investigators both in this country and in America. That some persons prefer to put a different interpretation upon them is not surprising.

Heap (1939), for instance, maintains that pasteurization does affect the nutritional value of the milk, because (1) it affects the taste and palatability; (2) it affects the cream line; (3) it affects the acidity; (4) it reduces the percentage of calcium and phosphates; (5) proteins and enzymes start coagulating at five degrees higher and continue as the temperature rises; and (6) vitamin C is destroyed, whilst the effect on vitamins B, D, and E is uncertain.

Of these objections it may be remarked that (1), (2), and (3) have nothing to do with the nutritive value of the milk, that (4) is untrue, that (5) is irrelevant, and that (6) is largely untrue. The statement frequently made that pasteurization reduces the calcium and phosphorus in the milk is not in accordance with fact. There is a slight diminution, about 5 per cent., in the amount of *soluble* calcium and phosphorus, but the total amount of these elements remains practically unchanged. Even if the statement were true, it is very doubtful whether it would be of any practical importance, since the amount of calcium and phosphorus in cow's milk is over three times that in human milk.

With regard to vitamin C, it has been pointed out that milk taken straight from the cow undergoes no diminution in its vitamin C content

on pasteurization, provided the interior of the processing plant is free from copper. It is only when the milk has been exposed to light that the vitamin C becomes susceptible to heat. The average amount of destruction of vitamin C that occurs in modern plants is probably of the order of 20 per cent. A diminution as great as, or greater than this, may readily occur in raw milk if it is exposed to sunlight. In any case, cows' milk is a poor source of vitamin C, and when given to infants in either the raw or the pasteurized state it should always be supplemented with orange juice or other suitable source of vitamin C.

Of the other three vitamins mentioned, there is some destruction of the B_1 fraction, too small to be detected by rat feeding experiments unless the milk is given in minimal quantities. Vitamin D appears to be heat resistant. Vitamin E is present in such a minute amount that whether it is affected or not by heat is unlikely to be of any importance to civilized human beings, who do not usually attempt to reproduce their species till they have been living on a mixed diet for at least fifteen or twenty years.

Objection has been raised to the animal feeding experiments on the ground that the young animals to which the pasteurized milk was fed were brought up on their mother's milk (Tate 1939). This was true only of the rat experiments. In nearly all the calf-feeding experiments the young animals were put on to the raw or pasteurized milk within a few days of birth.

Exception has also been taken to the calf-feeding experiments in the following terms : " Certain experiments have been carried out on calves, but no experiment which is carried out on any animal as to the value of pasteurized or unpasteurized milk is scientific if the pasteurized milk was bought from a commercial plant, because no one can prove whether such milk was or was not genuinely pasteurized. No experiment is of any value unless the milk is pasteurized where experiment is made " (Tate 1939). In answer to this objection it may be pointed out that in all the recent experiments in which the milk was obtained from a commercial firm the efficiency of pasteurization was carefully controlled by the phosphatase test and by guinea-pig inoculation. In the Peppard experiment, which probably constituted the purest test of the nutritive value of pasteurized milk, and which failed to reveal any significant difference between the animals fed on raw and those fed on pasteurized milk, the milk was pasteurized on the premises in a plant specially designed for this purpose.

" *Pasteurized milk may diminish resistance to disease* "

This objection is frequently expressed, though, so far as we are aware, no natural or experimental evidence has ever yet been brought

to support it. Indeed it would be very surprising if it were true. Our knowledge of the effect of diet on resistance to disease is admittedly small, but the work of the last twenty years seems to show that in animals fed on an otherwise well balanced diet only gross deficiencies in vitamin A are able to bring about a lowering in their resistance to spontaneous and artificial infections. Vitamins B and D appear to have little, if any, influence on resistance, while the observations on vitamin C are confusing and difficult to interpret. Vitamin A, it will be remembered, is heat resistant and is unaffected by pasteurization, while vitamin C is susceptible to heat only if the milk has been previously exposed to light.

If there was any truth in the statement that pasteurized milk led to a diminution in resistance to disease, it might be possible to obtain evidence for it by comparison of the death-rates for various diseases in London, where practically the whole milk supply is heat-treated, with those in the rural districts, where there is very little heat treatment of the milk. A direct comparison of this sort, however, would be unsatisfactory, since the density of the population in the two areas is very different, and the chances of infection in London are considerably greater than those in the country. A better method would be to compare the death-rates from various diseases in London before and after pasteurization became general, and to find out whether the fall was greater or less than that in the rural districts during the same period. This has been done for (1) pulmonary and non-pulmonary tuberculosis at all ages, (2) non-pulmonary tuberculosis under 5 and 15 years of age, (3) the infant mortality rate, and (4) the combined rate for scarlet fever, whooping cough, measles, and diphtheria under 15 years of age. Since the death-rates in the last categories fluctuate to a greater or less extent from year to year, the mean values for periods of 5 or 10 years have been taken to exclude the effect of gross irregularities.

Pasteurization in a crude form began to be practised in London about 1903, and by 1911 probably about 20 per cent. of the milk was heat-treated in some way or other. It would have been better, therefore, in compiling our tables, if we could have taken the death-rates for the last 10 years of the last century, or even for the first 10 years of the present century. There are various objections to this, chief among which is the fact that the method of allocation of deaths by the Registrar General was altered in 1911, and figures previous to this could not justifiably be compared with those of subsequent returns. For this reason it has been considered wise to make the comparisons from 1911 onwards, even at the risk of weighting the figures against pasteurization. Table XLII summarizes the data that we have collected.

TABLE XLII

THE DEATH-RATE PER 100,000 FROM VARIOUS CAUSES IN LONDON AND THE AGGREGATE RURAL DISTRICTS BEFORE AND AFTER PASTEURIZATION BECAME GENERAL IN LONDON

Cause of Death.	Periods compared.	London.			Aggregate Rural Districts.		
		1st Period.	2nd Period.	Percentage change.	1st Period.	2nd Period.	Percentage change.
Pulmonary Tuberculosis	1911 and 1937	130	61	— 53	82	40	— 51
Non-pulmonary Tuberculosis	1911 and 1937	46	11	— 76	34	14	— 59
Non-pulmonary Tuberculosis under 5 years	1911 and 1937	231	33	— 86	136	44	— 68
Non-pulmonary Tuberculosis under 15 years	1911 and 1937	46	10	— 78	29	10	— 66
Infant Mortality	1911–1915 and 1931–1935	108·6	63·4	— 42	89·6	54·8	— 39
Common infectious [1] diseases under 15 years	1911–1920 and 1928–1937	257·5	134·9	— 48	131·6	59·4	— 55

[1] Includes measles, whooping cough, scarlet fever, and diphtheria.

It will be seen that in every instance but one London compares favourably with the rural districts. Most attention should probably be paid to the pulmonary tuberculosis death-rates, since the relation of under-nutrition to this disease is well known. There is nothing in any of these figures to suggest that pasteurized milk leads to a decrease in resistance to disease.

On the other hand, the death-rate from all these different diseases is determined by so many factors other than nutrition that, with the exception of non-pulmonary tuberculosis, for which milk is often directly responsible, it would be quite unjustifiable to regard the greater percentage fall in the death-rates in London as being determined by the quality of the milk supply. Nothing but a direct experimental comparison of raw and pasteurized milk on infants or children, with other factors controlled, could be trusted to detect a difference.

With regard to animals, there is still very little direct evidence available on the comparative resistance of those fed on raw and those fed on pasteurized milk. Watson (1937) and Watson and her colleagues (1938) made a series of very careful observations on mice reared on different diets and tested for resistance to infection with *Bact. typhi-murium*—a natural parasite giving rise to rodent typhoid. Her most important finding was that the resistance of these animals both to artificial infection by the mouth and to contact infection was considerably higher when dried skimmed milk was included in the dietary. Dried milk is not the same as pasteurized milk, but the degree of heating it receives is rather greater, so that it may justifiably be concluded that the resistance-increasing factor present in dried milk is almost certainly present also in pasteurized milk.

The only direct comparison available between raw and pasteurized milk appears to be that made by G. S. Wilson (1937) on the resistance of mice to infection with *Bact. typhi-murium*. Groups of mice were fed on raw or pasteurized milk—both supplemented by a mineral mixture and a white-flour biscuit—and were tested for resistance either by intraperitoneal inoculation or by feeding. All the mice were fed on their respective diets for several weeks before infection, and in some of the groups they constituted the second or third generation of mice on these diets. Every mouse dying, and every mouse killed at the end of the experiment, was submitted to a bacteriological examination, so as to determine whether death was due to specific infection or whether the animal was still harbouring the specific organism. The results on a total of 551 mice fed on raw and 495 on pasteurized are summarized in Table XLIII.

It will be noted that, with one exception, none of the observed

TABLE XLIII

RESISTANCE TO INTRAPERITONEAL OR ORAL INFECTION WITH BACT. TYPHI-MURIUM OF MICE FED ON RAW OR PASTEURIZED MILK

Mode of Infection.		Raw.	Pasteurized.	O.D.	S.E. diff.	$\frac{\text{O.D.}}{\text{S.E. diff.}}$
Intraperitoneal	No. of animals	191	166			
	Specific deaths	99	85	− 0·63%	5·30	− 0·12
	Total infected	182	152	− 3·72%	2·60	− 1·43
	Average survival time in days	8·06	6·93	+ 1·13 days	0·49	+ 2·31
Feeding	No. of animals	360	329			
	Specific deaths	137	146	+ 6·32%	3·75	+ 1·69
	Total infected	243	217	− 1·54%	3·59	− 0·43
	Average survival time in days	13·95	14·09	− 0·14 days	0·61	− 0·23

O.D. = observed difference.
S.E. diff. = standard error of this difference.
+ means in favour of raw milk mice.
− means in favour of pasteurized milk mice.

differences between the " raw " and " pasteurized " groups in respect of any of the three factors analysed approached even twice its standard error. Some of the differences were in favour of the mice on raw, others in favour of those on pasteurized milk. Differences as great as, or even greater than, those observed are often met with between two groups of mice of the same batch kept under identical conditions.

These experiments afford no evidence to show that mice fed on pasteurized milk differ from those fed on raw milk in their resistance to infection with a natural parasite such as *Bact. typhimurium.*

It will thus be seen that neither an analysis of the death-rates from various causes in London and rural districts, nor the results of experimental observations in the laboratory, lend any support to the contention that the resistance to disease of man or animals is any less on a diet of pasteurized than on a diet of raw milk.

" *Pasteurized milk interferes with the proper development of the teeth and predisposes to dental caries* "

These allegations are based very largely on the observations of Sprawson (1932, 1933, 1934), who found that in a certain institution, in which the boys were given raw milk, the incidence of dental caries appeared to be unusually low. Whatever the interpretation of these findings may be, it is impossible to draw any definite conclusions from them, since no comparable group of children receiving pasteurized milk was examined.

The factors responsible for the development of the teeth and for their preservation in a healthy condition are still imperfectly understood, but the painstaking observations of Mellanby (1929, 1930, 1934) seem to show that among the most important factors are the calcium and phosphorus, the vitamin A, and the vitamin D content of the dietary. Pasteurization has apparently no effect on vitamin A or D, and apart from throwing about 5 per cent. of the calcium and phosphorus out of solution, it brings about no change in these mineral elements. As has already been pointed out, the total amount of calcium and phosphorus remains unchanged. Even if the 5 per cent. fraction rendered insoluble was no longer available for metabolism—and for this there is no evidence—little importance could be attached to it, since the calcium and phosphorus content of cows' milk is at least three times that of human milk.

So far as we are aware, there is no scientific evidence to show that pasteurized milk is inferior to raw milk in favouring the development of the teeth or in protecting them from dental caries.

"*Pasteurization, by eliminating tuberculosis of bovine origin in early life, would lead to an increase in pulmonary tuberculosis in adult life*"

This objection is fully dealt with in the League of Nations Report (1937*b*), from which we may quote freely.

"This argument," which applies with equal force to milk from Tuberculin Tested Herds, "assumes that infection of bovine origin in early life may produce an active immunization sufficient to protect against a subsequent infection of human origin. Its value would be easier to assess if we had any adequate statistical data on the degree of immunity to pulmonary tuberculosis possessed by populations exposed in infancy and childhood to bovine infection. Unfortunately, we have none that can be regarded as satisfactory. No high negative correlation exists in different countries between the death-rate from non-pulmonary and that from pulmonary tuberculosis, as might be expected if this thesis held generally true. For example, taking the figures for the years 1928 to 1930 (Report 1930), we find that the mean non-pulmonary tuberculosis death-rate for Finland, Germany, England and Wales, Denmark and Czechoslovakia was 0·143 per 1,000, while for the Netherlands, France, Scotland, the Irish Free State, Switzerland and Norway it was 0·273. The corresponding death-rates from pulmonary tuberculosis were 0·814 and 0·999 per 1000 respectively. In spite of the fact, therefore, that in the first group of countries the mean non-pulmonary tuberculosis death-rate was only about half that in the second group, the mean pulmonary tuberculosis death-rates were almost equal.

"Supporters of the argument sometimes quote the experience of Denmark (see Englebreth 1935). In Copenhagen, where a certain proportion of the raw milk contained tubercle bacilli, the pulmonary tuberculosis death-rate fell between 1923 and 1931, while in Bornholm, where tuberculosis in cattle has recently been eradicated, the pulmonary tuberculosis death-rate remained stationary. Deductions, however, from observations of this type made on populations differing in so many other important respects are peculiarly dangerous. In Sweden, for example, as Ostenfeld, Heitmann and Neander (1931) have shown, the geographical incidence of pulmonary tuberculosis has changed considerably during the past century, decreasing in the more industrial districts of the south and increasing in the more rural districts of the north. The truth is, of course, that the incidence of the disease is determined by so many different factors that it is very difficult to assess the part played by any one factor.

"Though we are unable to measure the possible advantage conferred on a population supplied with tuberculous milk, we have got some

data regarding the disadvantage from which such a population suffers. In Great Britain, for example, we know that, in 1931, about 2,600 persons died from tuberculosis of bovine origin, and that there were over 6,000 fresh cases of the same disease. In Denmark, approximately 250 deaths occur annually from this cause. In the face of these figures, it is not easy to justify a method of indiscriminate vaccination that results in such a serious incidence of disease and actual loss of life. What other method of vaccination, we may ask, would be tolerated that exacted such a sacrifice ? If we do want to vaccinate against pulmonary tuberculosis, let us at least do so by some more scientific and less dangerous procedure than that of distributing raw milk containing an unknown number of virulent tubercle bacilli.

" The principle on which public health has relied in the past is to prevent disease whenever it is possible. There are the strongest grounds for believing that compulsory pasteurization would virtually abolish milk-borne disease. We can see no more reason for refusing to adopt this measure because of some future hypothetical ill effect than for refusing to purify our water supplies on the ground that the adult population might thereby become more susceptible to enteric fever. The purification of water supplies to our large towns has in fact resulted in an enormous decrease in the incidence of enteric fever and dysentery, and we may confidently anticipate a similar reduction in the incidence of the various milk-borne diseases as the result of compulsory pasteurization."

Fortunately this objection to pasteurization and to Tuberculin Tested milk is belied by practical experience. In the United States over 99 per cent. of milk distributed in towns of 1,000 inhabitants and over comes from tuberculin tested herds and 73 per cent. is pasteurized. Pulmonary tuberculosis should therefore be on the increase, but the figures show the exact opposite. Thus the pulmonary tuberculosis death-rate fell from 124 per 100,000 in 1916, to 76 in 1926, and to 51 in 1936.

The argument that pasteurization, or the use of Tuberculin Tested milk, in early life will lead to an increase of pulmonary tuberculosis in adult life, comes from the arm-chair, is of very doubtful validity, is not supported by practical experience, and certainly cannot be allowed to outweigh the numerous positive advantages that are known to follow the provision of a safe milk supply.

" *Pasteurized milk has a cooked flavour* "

It has already been pointed out on p. 97 that clean milk from cows on a suitable diet, pasteurized at a temperature of 145–150° F. (62·8–65·6° C.) for half an hour, cooled immediately to a temperature

of 55° F. (12·8° C.) or below, and consumed fresh or after refrigeration has practically no detectable cooked flavour. It is true that the flavour of pasteurized milk may differ from that of raw milk owing to the removal during the heating process of certain volatile products which are often present in raw milk, but very few persons appear capable of differentiating between the two types of milk with any degree of certainty.

Point was given to the objection that pasteurized milk is said to have a distinctive cooked flavour when the County Medical Officer of Health for Wiltshire stated in a private communication that on more than one occasion the consumption of milk in schools had fallen off when raw milk was replaced by pasteurized. To learn what truth there was in this objection, it was decided to make actual observations on the ability of different persons to distinguish between raw and pasteurized milk. With Dr. Tangye's help an experiment was carried out at Trowbridge on May 31st, 1939. This experiment has already been described on p. 98, and reference may be made to a summary of the results on p. 103. The evidence obtained showed clearly that the majority of persons are unable to distinguish the flavour of raw and of pasteurized milk.

The observation that milk consumption in schools occasionally falls off when raw milk is replaced by pasteurized is unlikely to be due to any difference in the flavour of the milks. A more probable explanation, supported by actual experience, is that the children of farmers selling raw milk are told to object to pasteurized milk on grounds of flavour ; and as children are naturally imitative, the objection is liable to spread and the consumption of milk to decrease.

"*Pasteurization fails to destroy bacterial toxins in milk*"

In Chapter XI conclusive evidence has been brought to show that holder pasteurization destroys all pathogenic organisms in milk. There is at least one organism, however, namely *Staph. aureus*, which may under favourable conditions grow in milk outside the body and form a specific heat-resistant toxin which on ingestion gives rise to acute gastro-enteritis. This toxin is not destroyed by pasteurization, so that pasteurized milk containing this toxin might prove harmful to human beings. The raw milk, however, would be equally toxic. Pasteurization does not render the milk more harmful than it is already. Happily under the conditions of milk production and distribution prevailing in Great Britain, opportunities for this organism to render milk toxic do not seem to be numerous. Most of the recorded outbreaks of acute staphylococcal intoxication have followed the consumption of articles containing added carbohydrate, such as cream

puffs, layer cakes, and custards, in which the organisms have had favourable time and temperature conditions to multiply and produce their toxin.

The real fault lies not in the failure of pasteurization, but in the faulty production of the milk. If all milk was produced under cleanly conditions and kept cool before pasteurization, then toxic substances of this type would never be formed in sufficient quantity to be harmful.

It is probable that other types of toxins may be formed as the result of bacterial action in milk, but our knowledge of these and of their heat resistance is still very scanty. All the available information suggests that they result from the inordinate growth of bacteria in the milk. The answer is therefore the same, namely that no milk in which undue bacterial proliferation has occurred is really suitable for human consumption. Milk should be both clean and safe. The former can be achieved by careful conditions of production and transit, the second by pasteurization.

"*Compulsory pasteurization would diminish the incentive to clean milk production*"

This argument can be supported neither in theory nor in practice. Though it may make a dangerous milk safe, pasteurization can never make a dirty milk clean. The cleaner the milk to be pasteurized, the better is the finished product. No one knows better than the large firms who are at present pasteurizing milk how important it is to secure an initially clean raw supply. So far from there being an antagonism between pasteurization and clean milk production, there should be a very close association. Dirty milk may contain certain types of organisms which are either thermoduric and are not destroyed by the usual pasteurizing temperatures, or which are thermophilic and may actually multiply during the process of pasteurization. In either instance the pasteurized product may contain a high bacterial count, and thus fail to conform to the official standard laid down by the Ministry of Health, i.e. not more than 100,000 bacteria per millilitre.

Experience in the United States and Canada, where compulsory pasteurization has been in force for several years in certain cities, has lent no support to the argument that compulsory pasteurization leads to a deterioration in the cleanliness of the incoming raw supply.

So far as Great Britain is concerned, it may be remarked that the production of graded milk during the twelve years following the issue of the Milk (Special Designations) Orders, 1922, 1923, hung fire almost completely. But as soon as the Milk Marketing Board in 1935 introduced a bonus payment at the rate of 1*d.* per gallon for Grade A,

later Accredited, milk, the production leapt up from about 12 million gallons supplied by 778 licensed producers in 1933–1934 to 377 million gallons supplied by 22,711 licensed producers in 1937–1938 (Report 1939*a*). This enormous increase was achieved in spite of the fact that a large part of the Accredited milk produced was being taken to London and the large towns to be pasteurized.

To the ordinary farmer it makes little difference whether his milk is to be consumed raw or pasteurized. The real stimulus to cleanliness of production is financial, and the experience of many countries shows the value in this respect of a bonus system of payment. It may be argued that a farmer who has previously taken considerable trouble to produce clean milk will relax his precautions if he finds that in future his milk is to be mixed with that of a lower standard of cleanliness from other farmers. This may be true, but it is an argument, not against pasteurization, but against the bulking of milk.

To supply our large towns, the bulking of milk is inevitable. It is of the utmost importance that every milk composing the bulk should be of a reasonably high standard of cleanliness, since even a small proportion of dirty milk may affect detrimentally the quality of a large volume of clean milk. Measures are, in fact, required to control more stringently the bacterial quality of milk that is to be bulked. If this is done, then the milk for pasteurization will be clean and the finished product will be satisfactory. Compulsory pasteurization might, in fact, be used to raise the standard of cleanliness of milk production throughout the country, rather than to lower it, as its opponents maintain.

"*Compulsory pasteurization would remove the stimulus to the eradication of diseased animals from milking herds*"

This argument is false both in theory and in practice. It assumes that all types of infection gaining access to milk come exclusively from the cow. This is quite untrue. Even if tuberculosis, contagious abortion and mastitis were eliminated from our cattle population, the risks of contamination of milk with typhoid, paratyphoid, dysentery and food-poisoning bacilli from the human personnel or an infected water supply would still remain, and outbreaks of milk-borne typhoid, paratyphoid, dysentery, food poisoning, scarlet fever, septic sore throat and diphtheria would still occur if the milk was consumed in the raw condition.

It is highly desirable that udder disease should be eradicated from the milch cows of this country, but the difficulties to be faced are enormous. The problem is essentially a veterinary and agricultural one, and the programme of eradication should be undertaken with the

aim of breeding a healthy animal population rather than with any immediate reference to the public health. A healthy animal population is more economic and more profitable in every way, and this must be the main reason for combating animal disease. Even, however, if such a campaign was undertaken, with large Government subsidies, there would be little or no prospect of eliminating these three diseases in this country within our generation or the next. In other words, however desirable the eradication of cattle disease may be, it can have little effect on the quality of our milk supply in the immediate future.

In order to destroy pathogenic bacteria that may gain access to milk from all the various sources enumerated, and in order to supply the public at once with a safe milk, pasteurization affords the only practical solution. If the milk can be derived from disease-free animals, so much the better, but the provision of safe milk to the public must not be allowed to rest on an eradication programme that will certainly take many years to complete.

Just as with clean milk, so the stimulus to a farmer to eradicate disease from his cows must be primarily economic. If the farmer is paid at a higher rate for milk derived from cows free from tuberculosis and contagious abortion, there is a hope that many producers will undertake the programme of eradication. Whether the milk is to be subsequently pasteurized or not is of no real importance to the producer.

Summarizing, we may say that the eradication of disease from the herds is essentially an agricultural and veterinary problem, and should be undertaken on economic, and not on public health, grounds. The provision of a safe milk supply to the human population is essentially a public health problem, and can be solved satisfactorily only by compulsory pasteurization. The two problems should be kept distinct, and the eradication of animal disease should be made contingent not on the payment by the consumer of a higher price for milk from disease-free herds, but by suitable Government and other subsidies.

These theoretical considerations are strongly supported by practical experience in the United States and in this country. In the United States a drive for the eradication of tuberculosis from cattle has been carried out by the Department of Agriculture simultaneously with a drive for compulsory pasteurization by the Public Health Service. The result is that by 1936 no less than 99·8 per cent. of raw milk and 99·2 per cent. of pasteurized milk distributed in towns of 1,000 inhabitants and over was derived from Tuberculin Tested herds (Fuchs and Frank 1938). The two figures are so nearly identical as to lend no colour to the view that pasteurization retards the process of disease eradication.

In this country a scheme for the production of Tuberculin Tested

milk, which had to be sold raw if it was to bear the label " Certified," was introduced in 1923. No financial support was given to the producer, and the result was an almost complete failure. In 1937 a Tuberculosis (Attested Herds) Scheme was introduced, giving financial support to the producer who was anxious to eradicate tuberculosis from his herd. The result was an altogether unexpected success, and by the end of March 1939 the number of fully attested herds in England and Wales had reached 4,633. Tuberculin Tested milk, from being a luxury article, rapidly increased in amount, and according to returns received in the spring of 1939, constituted 5 per cent. or over of the total liquid milk supply in 16 of the 83 county boroughs of England and Wales. Unfortunately the war has had an unfavourable effect on the further progress of the Attested Herds Scheme.

The truth is that, as with clean milk production, the stimulus to eradicate disease is almost purely financial, and is unaffected by the subsequent history of the milk.

" *Pasteurization is often inefficient* "

It is alleged that pasteurization frequently fails to destroy pathogenic organisms in the milk, and therefore affords a sense of false security. This statement will be examined under three heads.

(*a*) *Frequency of tubercle bacilli in pasteurized milk*

It is admitted that so-called pasteurized milk does occasionally contain tubercle bacilli and possibly other pathogenic organisms. The exact frequency with which this occurs is difficult to estimate because usually no distinction is drawn between milk pasteurized and offered for sale under the conditions laid down by the Ministry of Health, and milk which is merely heat-treated, often by an inefficient method, and which is not sold as officially pasteurized milk.

In an inquiry undertaken in Scotland (Report 1933*c*) it was found that of samples of raw milk in churns from individual farms coming into Aberdeen, Dundee, Edinburgh and Glasgow, 10 per cent. contained the tubercle bacillus, and that 37·5 per cent. of samples of tank milk contained this organism. Of 1,243 samples of milk pasteurized by the holder process, 2·8 per cent. contained tubercle bacilli. It was found, however, that all the positive samples were derived from three out of the eight plants examined, one plant alone supplying 21 of the 33 positive samples. Inquiry showed that in some of the plants from which positive samples were derived, the temperature or the time of holding was below that officially laid down, while in others the design of the plant was unsatisfactory. The information afforded in the report does not make it clear whether, or how many of, the milks

examined were being pasteurized under licence, and it is impossible therefore to know with what frequency such milk contained the tubercle bacillus. A further point that may be noted is that Grade A pasteurized milk was excluded from examination.

The past few years have seen the development of a very live interest in the technique of pasteurization, and two reports, one from the Ministry of Health (Dalrymple-Champneys 1935) and one from the Hannah Dairy Research Institute (Scott and Wright 1935), have been published on this subject. It is not surprising therefore that more recent investigations than those referred to in Scotland have yielded very much more favourable results on the efficiency of pasteurization in destroying pathogenic organisms in milk. It appeared of interest to examine figures for the towns recorded in Table III (p. 13) from 1934 onwards. Unfortunately no data appear to be recorded for Brighton, Birmingham, Newcastle-on-Tyne, Edinburgh, or Aberdeen. As regards the remaining towns, the following information is available on the frequency of tubercle bacilli in pasteurized milk.

Pullinger (1934) examined 63 samples of rail tank milk coming into London, all of which were contaminated with tubercle bacilli. In the samples taken from the corresponding milks after holder pasteurization by a commercial firm, the tubercle bacillus was not found once.

In Manchester 206 samples of pasteurized milk were examined in 1936. All were free from tubercle bacilli, even though 12 per cent. of the samples of the raw unbulked milk supply contained these organisms.

In Sheffield 131 samples of pasteurized milk were examined during the years 1934 to 1936. Again not a single sample proved to be infected, even though 8·6 per cent. of samples of the raw unbulked supply contained tubercle bacilli.

In Liverpool, Humphriss, Peden, and Wright (1937) examined 440 samples of pasteurized milk distributed to schools in 1935 and 1936, and were unable to demonstrate tubercle bacilli in a single one, even though they were present in 9·7 per cent of ordinary samples taken from the city supply during the same period. Moreover an examination of 102 samples of pasteurized milk collected from a single plant during the course of a year failed to reveal tubercle bacilli in any of them, although these organisms were present in 61 of the corresponding raw milk samples.

In Glasgow during the years 1931 to 1933, 429 samples of pasteurized milk were examined and 1·6 per cent. were found to contain tubercle bacilli. Steps were then taken to eliminate various technical faults, with the result that during the following four years, 1934 to 1937, not one of 497 samples examined proved to be infected.

It thus appears that of 1,439 samples of pasteurized milk taken in some of our large towns during the years 1934 to 1936 not one was found to contain living tubercle bacilli.

(*b*) *Mode of infection of pasteurized milk*

That so-called pasteurized milk may occasionally contain pathogenic organisms of one sort or another is not denied. The most striking example of this is afforded by the Montreal outbreak of typhoid fever in 1927, when a total of over 5,000 cases followed the consumption of milk that was believed at the time to have been pasteurized. The real truth about this epidemic will probably never be known. The special board of officers sent by the United States Public Health Service (Report 1927) to investigate the causation of the outbreak reached the conclusion that the milk was infected on one or more of the farms in the country and that a considerable proportion of it was distributed from the Montreal dairy without being pasteurized at all. On the other hand Pease (1931), impressed by the fact that the supervisor of the pasteurizing plant was a typhoid carrier, and that one of the employees developed typhoid fever while still working on the plant, formed the opinion that the milk had been infected after pasteurization. All are agreed on the fact that the conditions in this particular plant were thoroughly unsatisfactory.

Other examples could be quoted of the infection of a pasteurized or so-called pasteurized milk supply. Thus McKay and his colleagues (1932) describe an outbreak of paratyphoid fever at St. Catharines, Ontario, in which inquiry revealed that the raw milk was infected on one of the farms supplying the city dairy, and that in the pasteurizing plant, owing to the provision of a common outlet for the raw and the pasteurized milk tanks, some of the raw milk had been inadvertently pumped to the cooler, thus short-circuiting the heating unit.

Again an outbreak of scarlet fever in Glasgow in 1931 (Seiler 1932) was traced to so-called pasteurized milk, not produced under licence. Investigations showed that, owing to a broken valve in the plant, the heating had been quite inadequate.

In the Montreal, St. Catharines, and Glasgow outbreaks there is strong reason for believing that the milk was either not pasteurized at all or was inefficiently pasteurized. Sometimes, however, the milk has been properly pasteurized, but has been contaminated from human sources after processing. Thus in a very small outbreak of typhoid fever in New York State in 1931, involving only four persons, the pasteurized milk was found to have been infected from a typhoid carrier employed in hand-capping the bottles (Report 1932*c*).

Similarly, in a recent outbreak of scarlet fever in Baltimore (Report

1938*a*), pasteurized milk was incriminated. The milk had been distributed in 5-gallon churns to a cafeteria attached to an institution. It was poured from these churns into pitchers, and from the pitchers into glasses; after each meal the unused milk was poured back into the pitchers. Prior to the outbreak one of the employees engaged in this work had been suffering from septic sore throat for three days and there was reason to believe that he had infected the milk. This interpretation was supported by the fact that pasteurized milk which had been distributed by the same plant in bottles was consumed in the institution with impunity.

Accidental contamination of pasteurized milk from human sources appears to be very exceptional in this country. It is likely to occur only as the result of some gross defect in the pasteurizing plant, or through the practice of filling or capping milk bottles by hand. With adequate control of the personnel employed (see p. 52) and with the installation of machinery to avoid any human contact with the milk after processing, the chances of contamination from this source must be negligible. That accidents are inevitable wherever machinery is controlled by human beings is the unfortunate experience of many branches of industry; but there is no more reason for condemning pasteurization on this account than there is for condemning the chlorination of water supplies because of the Croydon typhoid outbreak, or the use of railway transport because of the occasional failure of the signal system. The remedy is clearly to make each process as fool-proof as possible. That this can be done with pasteurization there is no reasonable doubt.

(*c*) *Alleged inefficiency of pasteurization: the phosphatase test and the plate count.*

Most of the statements on the inefficiency of pasteurization refer to faults in the heat treatment of the milk itself. Some are based on the experience of many years ago, when faults in the design and operation of pasteurizing plants were not uncommon. Since the publication of the Scottish report in 1933 and of the reports from the Ministry of Health and the Hannah Dairy Research Institute in 1935, notable progress has been made. Two other factors have contributed to improve the position still further in the last few years, namely the new regulations in regard to the control of pasteurization laid down in the Milk (Special Designations) Orders, 1936 (Report 1936*f*), 1938 (Report 1938*c*), and the introduction of the phosphatase test.

Under the Orders of 1936 and 1938 both indicating and recording thermometers must be used to check the temperature of the milk, and temperature records must be preserved for at least one month and be

open to inspection by the licensing authority. Moreover, the Ministry of Health (Report 1935*c*, 1936*f*) have drawn the attention of Sanitary Authorities to the importance of adequate supervision of both pasteurizing equipment and method.

The phosphatase test has already proved of immense service in detecting errors of one type or another and in checking the sanitary inspector's survey. There is reason to believe that by its use it should be possible to exercise an almost perfect control over the processing of pasteurization. It must be pointed out, however, that owing to its great delicacy it requires special care in its performance, and owing to occasional difficulties in the standardization of the reagents employed it should not be used to condemn off-hand milks that show slight deviations from the normal figures for properly pasteurized milk. In Chapter V it is shown that the degree of heat treatment required to destroy the phosphatase in milk is greater than that required to destroy pathogenic bacilli. Minor deviations, therefore, from the standard figure, even if proved to be significant, do not by any means necessarily indicate that the pasteurized milk is unsafe. While not wishing to reduce in any way the stringency of the precautions taken to guard against imperfect pasteurization, it is necessary to make it clear that until the technique and interpretation of the phosphatase test are perfected, too much attention should not be paid to minor failures in conformity to the standard.

Pasteurized milk is often condemned because the plate count happens to be rather above the legal limit. Such an occurrence may be due to uncleanliness of the raw supply, to insufficient heat treatment, to the presence of thermoduric organisms, to contamination of the milk after processing in the cooling and bottling sections of the plant, to the maintenance of the milk at too high a temperature before distribution to the consumer, or to the inherent error of the plate count itself. However undesirable a high plate count may be, it is often of very little importance from a hygienic point of view, and the organisms present may bear little relation either to the safety or to the keeping quality of the milk. It certainly cannot be regarded with the same stringency as the presence of tubercle bacilli, nor is it by any means necessarily an index of faulty pasteurization.

(*d*) *Summary.*

Though pasteurization has not infrequently been open to criticism in the past, the technique has improved very much of late years. Analysis of the recent figures published by some of the larger towns in Great Britain has failed to reveal infection of a single sample of pasteurized milk with tubercle bacilli. Some pasteurized milk at

present is perhaps being condemned unjustly on the basis of the phosphatase test, but it is believed that, when the technique and interpretation of this test have been perfected, it should prove one of the most valuable methods at our disposal for ensuring that pasteurization is efficiently carried out.

SUMMARY

Careful consideration has been given to the more important objections raised against pasteurization, such as that it diminishes the nutritive value of the milk, it removes the incentive to clean milk production and to the eradication of diseased animals from milking herds, that it is often inefficient, and that it imparts a cooked flavour to the milk. In addition, fears that pasteurized milk may diminish the resistance to disease, predispose to dental caries, and favour the development of pulmonary tuberculosis in adult life are dealt with. Evidence is brought to show that no serious objection has so far been raised which rests on a firm basis of fact, and which can be weighed in the scales against the immense demonstrable advantage of a safe milk supply conferred by pasteurization.

CHAPTER XIV

GENERAL SUMMARY AND CONCLUSIONS

For Chapter Summaries see pp. 4, 17, 41, 55, 69, 77, 84, 106, 127, 138, 148, 159, and 179.

1. In this volume an attempt has been made to furnish a conspectus of our knowledge on the medical and scientific aspects of pasteurization. The evidence presented consists of *facts*. The few opinions quoted in its favour (see Appendix, p. 193) are restricted to those expressed by authoritative bodies after reviewing the facts. Considerable attention has, however, been devoted to meeting the objections put forward by individual antagonists of pasteurization.

2. Cow's milk occupies a place of peculiar importance in the human dietary. The high biological value of its protein, and its content of vitamins and mineral elements, combine to render it specially favourable for the growth and maintenance of the body tissues. It is not, however, a perfect food. It differs both qualitatively and quantitatively from human milk; it is relatively deficient in iron and copper, both of which are required for the formation of hæmoglobin; it is relatively deficient in the important growth-promoting and anti-infective vitamin A, and in the anti-scorbutic vitamin C; while vitamin D, which in combination with calcium and phosphorous plays an important part in the development of bone and the prevention of rickets and dental caries, is practically absent during the winter months.

3. Though the sale of liquid milk in Great Britain has increased of late years, the average *per caput* consumption is still considerably less than that in several other countries. One reason for this is believed to be the distrust, felt both by the medical profession and by the more educated members of the laity, for our raw milk supply, which is more heavily infected with pathogenic micro-organisms and more likely to give rise to disease than any other article of common use in our dietary. Not until milk is regarded hygienically in the same light as water, and freed by suitable measures from the risk of causing disease, will doctors feel justified in recommending its unrestricted use as a food and a beverage without incurring, as they do at present, the moral responsibility for endangering the health of those under their care.

4. Raw milk may be infected with pathogenic bacteria derived from cattle, from human beings, from water, or from rodents. The more important organisms that may gain access to the milk come

from the diseased udder of the cow infected with the tubercle bacillus, *Br. abortus*, or occasionally *Str. pyogenes*; from the fæcal discharges of cattle suffering from enteritis; from the cough spray of human patients affected with scarlet fever or diphtheria; from the imperfectly cleansed fingers of human patients or carriers infected with typhoid, paratyphoid, dysentery, or food-poisoning bacilli; from water contaminated with the urinary or fæcal discharges of enteric patients; and from the excreta of rats and mice suffering from rodent typhoid. With such a number of different organisms and so many diverse ways of access, it is not surprising that raw milk in this country frequently contains pathogenic bacteria. Records of milk samples examined in our large cities show that about 5–10 per cent. of farmers are sending out milk infected with virulent tubercle bacilli, and about 20–40 per cent. with *Br. abortus*. Accredited milk appears to be as heavily infected with tubercle bacilli as ungraded milk, and Tuberculin Tested milk appears to be as heavily infected with *Br. abortus* as ungraded milk, if not more so.

5. Several reasons are advanced for believing that our knowledge of both sporadic and epidemic milk-borne disease is seriously deficient, and that a considerable amount of it is being at present overlooked. Careful estimates made in two different ways show that the probable number of deaths in England and Wales in 1937 from infection with the bovine type of tubercle bacillus was between 1,500 and 2,000. The number of cases of undulant fever due to infection with *Br. abortus* in England and Wales was probably of the order of 400–500. The evidence suggests that in the causation of both these diseases raw milk and cream play a major part, and that the rôle of butter and cheese is almost negligible. With regard to epidemic milk-borne disease, excluding that caused by milk products, there are records available in Great Britain between the years 1912 and 1937 of at least 113 outbreaks of scarlet fever, septic sore throat, diphtheria, typhoid fever, paratyphoid fever, dysentery, and acute gastro-enteritis affecting about 14,000 persons. To these must be added an unknown number of outbreaks of acute nausea and vomiting due to milk rendered toxic before ingestion by staphylococci or other organisms. Part also of the 190,000 deaths in England and Wales during the years 1912 to 1937 caused by summer diarrhœa of infants and young children must be attributed to raw milk. In view of these figures it is impossible to deny the gravity of milk-borne disease.

6. A distinction is drawn between "clean" milk, which is free from gross dirt, from pus, from blood, and from an excessive number of bacteria, and "safe" milk, which is free from organisms capable of giving rise to disease. There is no necessary association between

these two types of milk, and many outbreaks of human disease have resulted from the use of clean milk that was contaminated with *Str. pyogenes*, typhoid bacilli, or other pathogenic organisms. Both cleanliness and safety should be demanded of any milk intended for liquid consumption. Safe milk is desirable for obvious reasons. Clean milk is desirable because it is æsthetically more pleasing, it has a better flavour, it keeps longer, and it is less liable to contain toxic substances resulting from undue bacterial proliferation.

7. Three methods of rendering milk safe are discussed, namely eradication of cattle disease, control of the human personnel, and destruction of pathogenic organisms by heat treatment of the milk itself. (*a*) The complete eradication of tuberculosis, contagious abortion, and mastitis in this country presents a very difficult task, which, even with the assistance of generous Government subsidies, could not be successfully accomplished within any reasonable time. Even if it were possible, the resulting milk would still be exposed to infection from the human personnel, from water, and from rodents. (*b*) The economic and administrative difficulties of controlling milk-borne infection of human origin by medical and bacteriological supervision of all milk handlers on the farm are too great to be practicable. On the other hand, there is much to be said for a general measure of supervision of the personnel employed in dairies and plants where large volumes of milk are being handled. (*c*) Since milk-borne disease presents an urgent public health problem that cannot be solved immediately by controlling the health either of the cattle population or of the human personnel engaged in handling the milk, it is concluded that the only satisfactory method of providing the public with safe milk is some form of heat treatment designed to destroy any pathogenic organisms that may have gained access to the milk from the numerous and diverse sources available. Of the various ways of applying heat on a large scale, pasteurization under properly controlled conditions is undoubtedly the most satisfactory.

8. Pasteurization is a loose term covering a multiplicity of processes, not all of which are reliable. In this country pasteurization is restricted to (*a*) the holder method in which the milk is exposed to a temperature of 145–150° F. (62·8–65·6° C.) for at least thirty minutes and cooled immediately to a temperature of not more than 55° F. (12·8° C.), or in Scotland 50° F. (10·0° C.), and (*b*) the high temperature short time method in which the milk is exposed to a temperature of not less than 162° F. (72·2° C.) for at least 15 seconds and then cooled as in (*a*). No milk that is not treated in a plant licensed by the local authority is allowed to bear the label "Pasteurized" on the bottle in which it is exposed for sale.

9. The laboratory control of pasteurization is discussed, and it is pointed out that by means of the recently introduced phosphatase test the efficiency of the processing can now be controlled in a way which was impossible a few years ago.

10. Compulsory pasteurization, with exceptions for certain types of raw milk, has been introduced into Sweden, France, and the Province of Ontario. It is also common in many towns in the United States. In Canada about 91 per cent. of the population living in towns of over 2,000 inhabitants, and in the United States about 73 per cent. of the population living in towns of over 1,000 inhabitants, is supplied with pasteurized milk. In London 92·7 per cent. of the milk is pasteurized under licence and over 98 per cent. is treated by heat in some way or other. In the aggregate county boroughs of England and Wales the corresponding figures are 44 and 58·1 per cent. It is estimated that about 62 per cent. of the population of Greater London and the county boroughs is supplied with milk pasteurized under licence and about 80 per cent. with heat-treated milk. A very approximate estimate suggests that about 50 per cent. of the population of England and Wales is at present being supplied with heat-treated milk.

11. The effect of holder pasteurization on the natural constituents and the general properties of cows' milk has been carefully investigated. The changes brought about are summarized in full on pp. 106, 107, but briefly they are as follows: The cream line is reduced by 10–30 per cent.; about 5 per cent. of the lactalbumin is coagulated; about 5 per cent. of the calcium and phosphorus is rendered insoluble; a small proportion, almost certainly less than 20 per cent., of the iodine is probably driven off by volatilization, though this still requires confirmation; some destruction, usually 10 and at the most 25 per cent., occurs of vitamin B_1; and vitamin C undergoes a reduction of about 5–50 per cent. with an average of about 20 per cent., if the milk has been previously exposed to light. Feeding experiments, however, on animals show that the availability of calcium and phosphorus, the biological value of the protein, and the total energy value of the milk remain unaltered. The flavour of the milk is somewhat changed owing to the removal by filtration and heating of volatile substances derived from the cow or from manure, but the acquisition of a cooked taste is practically negligible, and as actual experiment shows cannot be detected by the great majority of ordinary persons. It will be realized that the changes produced by pasteurization are comparatively slight. In any event the differences between raw and pasteurized cows' milk are far less than those between cows' milk and human milk, or even between summer and winter samples of raw milk.

12. Particular attention has been devoted to the change in nutritive

value of the milk brought about by holder pasteurization. The difficulties of making a really satisfactory experimental comparison in this respect between raw and pasteurized milk are considerable, because cows' milk is not a perfect food for any animal—even the calf—and has to be supplemented in various ways to prevent the development of anæmia, rickets, scurvy, or other kind of disturbance. Many of the early experiments were badly designed, and their results have had to be largely discounted. Of recent years, however, several careful comparisons have been made under properly controlled conditions on rats, mice, calves, and children. The results of these are all in general agreement with each other and show that pasteurization has no significant effect in lowering the total nutritive value of the milk for the growing animal.

13. Only a comparatively small number of experiments have been undertaken to determine the effect of pasteurization on fertility. Analysis of the recorded observations on rats, mice, and cattle does not lend any support to the view that the fertility of animals fed on pasteurized milk is less than that of animals fed on raw milk. Indeed the suggestion is rather the opposite.

14. The destruction of pathogenic organisms in milk by heat has been the subject of numerous and important studies by workers in many countries. The results are in general agreement in showing that under laboratory conditions, or in properly controlled pasteurizing plants, all pathogenic organisms in milk likely to give rise to disease on ingestion are destroyed by exposure to a temperature of 138° F. (58·9° C.) within half an hour. Since each degree rise in temperature greatly accelerates the process of killing, and since a temperature of 145–150° F. (62·8–65·6° C.) for at least thirty minutes is enjoined officially for holder pasteurization in this country, it is clear that a considerable margin of safety is afforded to the consumer of pasteurized milk.

15. An attempt has been made to learn something of the effect of pasteurization on the prevalence of milk-borne disease. For several reasons our information is less complete than it might be. In London, where practically all milk is heat-treated, the fall in the non-pulmonary tuberculosis death-rate during the period 1911–1937, particularly in children under 15 years of age, was considerably greater than in the rural districts where much of the milk is drunk raw. The most convincing evidence, however, in favour of the beneficial effect of pasteurization in preventing tuberculosis of bovine origin is provided by the bacteriological observations of Price in Toronto. Examining 300 children suffering from non-pulmonary tuberculosis, she found that 15 per cent. were infected with the bovine type of bacillus. Every one of

these children came from parts of Ontario outside Toronto where the milk was drunk raw. In Toronto itself, where pasteurization has been compulsory since 1915, not a single case of infection of bovine origin was found, in spite of the fact that 26 per cent. of the bulked milk entering the city contained tubercle bacilli. Similar observations made in Paris point to the same conclusion. The beneficial effect of pasteurization is further shown by the practical disappearance of undulant fever and of epidemic milk-borne disease from towns where all milk and cream are heat-treated.

16. Answers are given in Chapter XIII and in the Appendix to twenty-five different objections of one type or another to pasteurization. Many of the statements made by the opponents of pasteurization are based on incomplete or inaccurate data, and many are simply untrue. Others consist of vague fears about certain hypothetical consequences of compulsory pasteurization, which find no justification either in the laboratory or in the field. No serious objection has so far been raised which rests on a firm basis of fact, and which can be weighed in the scales against the immense demonstrable advantage of a safe milk supply conferred by pasteurization.

17. The medical and scientific case in favour of pasteurization is now so well documented that to postpone the practical introduction of some measure of compulsory pasteurization on the ground that more time for inquiry is needed, or that the matter is of such importance as to deserve the appointment of a Royal Commission, would on the grounds of health be quite unjustifiable.

18. It is not the purpose of this report to make detailed practical suggestions for the solution of the milk problem. It is suggested, however, that the privilege enjoyed by calves in Attested herds of being supplied with pasteurized milk should be extended to human beings, and that the public conscience should be as much exercised over the problem of safe milk as it is over that of safe water. No Local Authority would knowingly allow infected water to be distributed to the population under its care. The necessary measures of filtration and chlorination would be undertaken to ensure its safety. On what ground can the distribution of infected milk be justified, when by the simple expedient of pasteurization it can be rendered entirely innocuous without producing more than minimal changes in its general properties or nutritive value ?

19. If some measure of compulsory pasteurization is introduced, it is important that the supply of milk and its heat treatment should be as satisfactory as possible. It is therefore suggested that (*a*) a pre-pasteurization standard of bacterial cleanliness should be laid down so as to ensure a satisfactory article for processing ; (*b*) the licensing

of pasteurizing plants should be transferred from Local Authorities to the County Councils; (*c*) the personnel of pasteurizing plants should be subject to the same type of medical and bacteriological control as that recommended by the Ministry of Health for the employees of water undertakings; (*d*) all pasteurizing plants should be subject to regular and frequent inspection; (*e*) since most sanitary inspectors have neither the time nor the detailed knowledge and experience to perform this duty satisfactorily, specially trained whole-time inspectors should be appointed, preferably on a regional basis, whose duty it would be to advise County Councils on the granting of licences, to inspect plants in operation, and to take samples of the milk for examination both before and after processing; (*f*) samples of the pasteurized milk should be examined as a routine by the phosphatase test, but this test should be checked at intervals by the method of guinea-pig inoculation.

20. In conclusion it is legitimate to point out that a great deal of unnecessary disease is at present being caused in this country by milk, and that practically all of this could be avoided by the introduction of compulsory pasteurization into our urban areas. There are many diseases of whose causation we are still uncertain, and whose mode of prevention is either unknown to us, or, if known, is impracticable; but it may be doubted whether, with the possible exception of diphtheria, we are so conversant with the causation of any other disease and so well equipped to prevent it by a sure and practicable means, as that of the group of milk-borne infections. To refuse to act on this knowledge, and to allow the continuation of a wholly unnecessary amount of suffering, invalidity, and death, may be justifiable on grave economic grounds, but can no longer be excused on the plea of ignorance except by those who, through natural or acquired defect, are too blind to see or too dense to understand.

APPENDIX

MINOR OBJECTIONS RAISED AGAINST PASTEURIZATION

The expression " minor " is used here chiefly in the sense of trivial or unimportant, but sometimes to indicate that, though the objection itself is of some moment, the evidence advanced in its support is too weak to justify serious consideration.

" *Pasteurization reduces the cream line* "

As pointed out on p. 87, the amount of fat that rises to the surface after heating is dependent on both the temperature and time of heating and on the degree of subsequent cooling. In modern plants in which the milk is heated to a temperature of about 145–146° F. (62·8–63·3° C.), and cooled rapidly to 40° F. (4·4° C.) the diminution in the cream line is very small indeed.

It is hardly necessary to point out that the total amount of fat in the milk and its nutritive value are unaffected by the state of physical aggregation of the fat globules.

" *Pasteurization of milk diminishes the fertility of animals fed on it, and might increase the present fall in the birth-rate* "

The effect of pasteurization on the fertility of rats, mice, and calves has been carefully examined in Chapter X, and no evidence whatever has been obtained to show that animals fed on pasteurized milk are less fertile than those fed on raw. Indeed, if anything, the suggestion is rather in the opposite direction.

Sutherland (1938), who fears that depopulation and national decline may follow the introduction of compulsory pasteurization, suggests that experiments should be carried out on rats and mice to see whether feeding on pasteurized milk has any effect on their fertility. Experiments to determine this point had already been made and published at the time of Sutherland's suggestion. They are reviewed in Chapter X, and, as mentioned above, lend no substance to his fear.

" *Pasteurization is unnecessary, because raw milk does not give rise to tuberculosis* "

According to the late Sir Arnold Wilson (1937*b*), " There is, however, no evidence, as far as I know, that the disease (tuberculosis) has been artificially induced in any healthy animal under laboratory conditions by a diet of tuberculous milk. It can, in fact, be induced only by highly concentrated intradermal injections."

The fact that tuberculosis is caused by the tubercle bacillus, and that the bovine type of tubercle bacillus in milk may give rise to tuberculosis in man and animals is as well attested as any other fact in the whole realm of bacteriology. If these facts are doubted then the whole germ theory of disease must be abandoned, together with the hygienic code that is based upon it.

It is hardly necessary to set out the evidence in detail relating the tubercle bacillus to the disease which it causes. Part of it is direct, part of it is circumstantial, and reference must be made to textbooks of bacteriology by those who desire to study it in full (see Griffith 1930, Topley and Wilson 1936). Suffice it to state here that naturally tuberculous milk was conclusively shown by the workers on the Royal Commission of Tuberculosis (Griffith 1907) to give rise to tuberculosis when fed to guinea-pigs, rabbits, cats, pigs, calves, monkeys, and chimpanzees, and by workers at the Hannah Dairy Research Institute (Wilkie, Edwards, Fowler, and Wright, 1937) to give rise to tuberculosis when fed to calves. Reference was made to this last observation on p. 118, but it may be recalled that no fewer than 23 out of 36 calves fed for 12 weeks on raw milk developed macroscopic lesions of tuberculosis, whereas not one of 37 calves fed for the same time on the same source of milk after holder pasteurization showed any sign of tuberculosis.

"*The death-rate from tuberculosis remains uniformly lower in rural areas where all milk is drunk raw than in cities where all milk is pasteurized*"

So far as non-pulmonary tuberculosis is concerned, this statement of the late Sir Arnold Wilson (1937*a*), does not appear to be supported by facts. Reference to Table XL (p. 153) shows that in 1937 the standardized death-rate from non-pulmonary tuberculosis in London, where over 98 per cent. of the milk is heat-treated, was 11 per 100,000, while in the aggregate rural districts of England and Wales, where nearly all milk is supplied raw, it was 14 per 100,000. Moreover, the death-rate from non-pulmonary tuberculosis fell by 76 per cent. in London between the years 1911 and 1937, but by only 59 per cent. in the country districts. In the 0–5 year age group (see Table XLI, p. 154), the percentage fall in London during the period was as high as 86 per cent., as compared with 68 per cent. in the aggregate rural districts.

"*Children and invalids thrive better on raw milk*"

No definite evidence is available to prove the truth of this statement. It is usually made on the basis of observations on occasional children and invalids, never on groups of sufficient size to give statistically significant results. Infants frequently seem to be capricious in their likes and dislikes, and it is easy to find examples of those that do well on one food after having done badly on another.

In support of this objection to pasteurized milk observations have sometimes been quoted from the work of the earlier American workers, such as Daniels and Stearns (1924), Ladd, Evarts, and Franks (1926), Lewis (1927, 1929), and Daniels, Hutton, Stearns, and Hejinian (1929). In no instance was a satisfactory comparison made between raw and pasteurized milk from the same source (see p. 124). The more recent experiments made in the United States and in Great Britain, which have already been described in Chapter IX, lend no support to the view that children thrive better on raw than on pasteurized milk.

"*Pasteurization would lead to an increase in infant mortality*"

When Manchester in 1933 was promoting a Bill containing a compulsory pasteurization clause, the opponents of the clause placarded the city with the slogan "Pasteurized milk will kill your babies."

If such an allegation was true, then our infant mortality rate ought to have risen considerably during the past ten or twenty years, when an increasing number of infants have been brought up on pasteurized, boiled, or dried milk. Moreover, the infant mortality rate should have risen sharply in London, where practically all milk is heat-treated, and presumably fallen in country districts where pasteurized milk is uncommon. That this has not happened is seen from Table XLIV, which shows that the infant mortality rate has fallen more rapidly in London, the county boroughs and the urban districts than in the rural districts.

TABLE XLIV

INFANT MORTALITY RATES IN DIFFERENT PARTS OF ENGLAND AND WALES IN 1911–1915 AND 1931–1935

Years.	Average Annual Infant Mortality Rate (per 1,000 live births).			
	Administrative county of London.	Aggregate county boroughs.	Aggregate urban districts.	Aggregate rural districts.
1911–1915	108·6	124·4	106·8	89·6
1916–1920	92·4	102·6	86·6	77·0
1921–1925	70·8	87·0	72·8	64·6
1926–1930	64·0	77·8	63·8	59·0
1931–1935	63·4	71·8	60·0	54·8
Percentage fall between 1911–1915 and 1931–1935 . .	41·6	42·3	43·8	38·8

Akin to this objection is the statement that " the infant mortality in Toronto, where all milk is pasteurized, is double that in Victoria and Vancouver, where all milk is drunk raw " (A. Wilson 1937*b*). The infant mortality rate is dependent on a large number of factors other than milk-borne infection, and the comparison is therefore invalid. But the facts themselves are incorrect. In Victoria, for example, 33 per cent., and in Vancouver 89 per cent. of the milk is pasteurized (Murray 1934). The difference between the infant mortality in these cities and in Toronto must therefore be due to other causes.

" Pasteurized milk will not clot with rennet," and related objections to its use in cooking

The coagulability of heated milk by rennet has already been discussed in Chapter VIII, to which reference may be made (p. 104). Briefly, milk treated by the holder method of pasteurization clots as readily as raw milk immediately after processing, but if left for some hours its coagulation time increases by about 25 per cent. The coagulability of pasteurized milk can be restored to normal by the addition of calcium salts.

The statement that pasteurized milk will not clot with rennet is untrue. If the milk is heated to higher temperatures than those allowed by pasteurization under licence, its coagulability by rennet falls off progressively to near

the boiling point, when rennet coagulability is completely destroyed. But milk pasteurized under licence still clots perfectly well, though its coagulation time is somewhat increased. Pasteurized milk is actually used by some factories in cheese-making—a process that would be impossible if it did not clot with rennet.

Numerous objections have been raised to the use of pasteurized milk in cooking, such as (1) Devonshire junket cannot be made with pasteurized milk, (2) pasteurized milk will not make curds and whey, and (3) pasteurized cream cannot be whipped. Provided the term " pasteurized " is applied to milk pasteurized under licence, these statements are simply untrue. Many housewives in London, where over 98 per cent. of the milk is heat-treated, know this quite well. Devonshire junket may take a minute or two longer to clot when made with pasteurized than with raw milk. Pasteurized cream, if kept cold for about 18 hours, as is the usual practice of the large firms, can be whipped as well as raw cream, and the drainage from it is no greater, i.e., it does not " flop."

" *Pasteurization favours the growth of bacteria in the milk* "

This objection has already been considered in Chapter VIII, to which reference may be made (p. 96). It is pointed out that the factor responsible for inhibiting bacterial multiplication is not destroyed in milk till it has been exposed to a temperature of 158–167° F. (70–75° C.) for one hour. The time-temperature combination laid down for holder pasteurization under licence is considerably lower than this, namely 145–150° F. (62·8–65·6° C.). Most of the results on which this objection is based have been obtained by methods that did not permit a real comparison of the growth rate of bacteria in raw and pasteurized milk to be made. If it were true it would be difficult to account for the observations of Wilson and his colleagues (1935) that the average keeping quality of commercially pasteurized milk was longer than that of Certified milk, even though the average bacterial content at the time of examination was higher in the pasteurized samples.

" *Pasteurization destroys the healthy lactic acid bacteria in milk, and pasteurized milk goes putrid instead of sour* "

Fortunately pasteurization does destroy many of the " souring " bacteria and thereby prolongs the life of the milk. Few normal persons are likely to be distressed by the fact that pasteurized milk remains sweet longer than raw milk. For those, however, who take a fortuitous interest in bacteria, it may be pointed out that lactic acid bacteria, like antibodies, are not present in pure milk. They are derived entirely from contamination of the milk after it has left the udder. Calves sucking directly from the udder therefore ingest no lactic acid bacteria. If the milk is collected under other than aseptic conditions, lactic acid bacteria frequently gain access from contaminated utensils and other sources, and if the milk is allowed to remain at a suitable temperature these organisms frequently multiply and become the predominant bacterial flora in the milk. If bacterial proliferation is allowed to continue unhindered sufficient acid may be produced to inhibit the growth, and perhaps even to bring about a partial destruction of certain pathogenic organisms that may be present in the milk. It may be pointed out, however, that no milk destined for human consumption in the raw state should ever be allowed to reach such a condition.

Many of the lactic acid bacteria are destroyed by pasteurization, and it is frequently stated that pasteurized milk does not sour on standing, but rather goes putrid. This is very largely a misrepresentation of fact. Commercially pasteurized milk usually does sour on standing, though it takes considerably longer to do so than the corresponding raw milk. If the raw milk before pasteurization is of a very high standard of cleanliness, containing therefore very few lactic acid bacteria, the pasteurized product may fail to sour. Instead it undergoes changes associated with destruction of the protein. The same sequence of events, however, is observed in raw milk produced under very clean conditions, and it is common to find that milk of Certified standard becomes putrid before becoming sour. It may be repeated that milk for human consumption in the liquid state, whether raw or pasteurized, should never be allowed to reach a state in which changes due to the proliferation of the lactic acid bacteria become obvious.

"*Pasteurization destroys beneficent enzymes, antibodies, and hormones, and takes the 'life' out of the milk*"

The effect of pasteurization on enzymes and on antibodies in the milk has already been described in Chapter VIII (pp. 95, 96).

Raw milk contains some enzymes that are inactivated by pasteurization, and others that are not. There is, however, no evidence to suggest that these enzymes, which in the milk of healthy cows are present in only minute amount and whose presence appears to be largely accidental, are of any importance to the human infant. It is highly probable that even in raw milk they are destroyed by the natural digestive processes.

Heat-labile antibodies in milk are supposed, by the opponents of pasteurization, to play an important part in building up the resistance of the child to disease. This argument hardly deserves serious consideration. Milk from the healthy udder of the cow contains no antibodies, or not more than a mere trace. Antibodies come from the globulin fraction of the circulating blood plasma. The passage of these globulins from the circulating blood into the *milk* occurs only when the udder is diseased. In other words, the very presence of antibodies in milk is evidence that the milk is abnormal. It is true that *colostrum*, which is a fluid secreted during the first few days of lactation and which is very different in chemical composition from milk, may contain antibodies, and it is true that these may be absorbed by the newly born calf and serve to protect it against certain types of disease to which it is peculiarly susceptible. But colostrum is rapidly replaced by milk which, if normal, contains no antibodies, and the intestinal canal of the calf soon ceases to be permeable to these substances. Since colostrum may not be sold as milk, it follows that a child receiving normal milk ingests no antibodies, and that a child receiving abnormal milk containing antibodies cannot absorb them except possibly during the first few days of life.

With regard to hormones, very little is known about their presence in milk. The only ones likely to be met with are the œstrogens, androgens, thyroid, and adrenal cortical hormones. These would probably be active to some extent if taken by the mouth. Our present knowledge renders it improbable that they are destroyed by pasteurization. Since these hormones are elaborated in the tissues of the growing animal itself and are distributed by the blood stream, there is no reason to suppose that their presence in the natural milk is other than fortuitous, or is likely to be of any special value to the offspring.

The objection that pasteurization takes the " life " out of the milk is merely one instance of a method of argument in which the fundamental issue is obscured by the tacit insertion of an unjustifiable premise. It assumes that milk has " life." What life ? The only living matter in milk of which we have any knowledge consists of bacteria, moulds, and other similar organized particles. Most of these are derived from the diseased udder of the cow, from contaminated milk ducts, or from external sources after the milk has been withdrawn. The whole tendency in clean milk production is to reduce these organisms to a minimum, so as to have as little " life " in the milk as possible. How then can objection be taken to the destruction of these organisms by heat, when one of the chief aims of milk production is to achieve virtually the same end ?

If by " life " is meant enzymes, then some evidence must be brought to show that these substances, which in milk from healthy cows are present more or less accidentally and in only minute quantities, are of any value to the young animal. As pointed out above, the probability is that they are destroyed during the natural processes of digestion.

" *Pasteurization kills the bacilli in milk and leaves their carcases to decompose directly it is exposed to the air* "

The answer to this ingenious objection, which was made by Sir Herbert Matthews (1939), has been partly given in Chapter XIII (p. 170). If milk is produced under cleanly conditions, then there will not be large numbers of organisms to be destroyed by pasteurization, and the milk will be free from substances liable to cause gastro-intestinal disturbance.

With regard to the decomposition of the killed bacteria on exposure to air, Sir Herbert seems to be a little confused about the real nature of fermentation. Before the time of Pasteur the decomposition of certain organic substances in the presence of air was well known, and was believed to be due to the presence of ferments. Pasteur showed that these ferments were living ferments, that is live micro-organisms, and that it was as the result of their metabolic activity that fermentation was brought about. Sir Herbert now apparently goes a step further and states that the ferment itself can ferment. How this process is accomplished is not explained.

That dead bacteria may undergo a certain amount of autolysis is not denied, but apart from a few species of organisms, which are not likely to be found in milk, bacterial autolysis appears to occur very slowly. Moreover the enzymes that are responsible for the autolysis appear to be destroyed by an exposure to heat similar to that used in pasteurization. Heat-killed typhoid and coliform bacilli, among others, remain apparently unaltered in contact with the air for several years.

The presence in milk of large numbers of bacteria, alive or dead, is very undesirable, because there is reason to believe that the body substance of certain types of bacteria is toxic when ingested in considerable quantity. Any decomposition that occurred in this body substance would have the effect of altering its chemical structure and probably destroying its toxicity. But to the best of our knowledge no such decomposition does occur.

" *Pasteurization legalizes the right to sell stale milk* "

If the term " stale " is meant to imply milk more than a certain number of hours old, then there is no existing legislation in Great Britain to prevent

its distribution in either the raw or the pasteurized state. But if, as is usually intended, the term " stale " is applied to milk which has been repeatedly pasteurized to prevent its going bad, then reference may be made to the Milk (Special Designations) Order, 1936, which lays it down that milk to which the designation " Pasteurized " is applied shall not be pasteurized more than once and shall not be otherwise treated by heat.

Unfortunately pasteurization is still voluntary, and it is open to any dealer to heat the same milk more than once, provided he does not sell it with the label " Pasteurized " on the container. If compulsory pasteurization was introduced, this irregular practice would be naturally eliminated.

" *Imperfectly pasteurized milk is worse than raw milk* "

This statement is frequently made, but without any evidence to support it. Indeed all the evidence that is available points in the opposite direction. There is a fairly big margin of safety in holder pasteurization as practised in Great Britain. The tubercle bacillus in milk is destroyed, as a rule, by a temperature of 138° F. (58·9° C.) in about 30 minutes, of 145° F. (62·8° C.) in 5–10 minutes, of 150° F. (65·6° C.) in less than 2–5 minutes, and of 162° F. (72·2° C.) in about 12 seconds. In holder pasteurization under licence the milk is heated at 145–150° F. for at least 30 minutes. In practice, the total heat exposure of the milk is increased by the time required for the preliminary warming up of the milk and for the emptying of the holding tanks. If, therefore, the temperature during the holding period is a few degrees lower than 145° F., or the holding tank is emptied after 20 instead of 30 minutes, the probability is that all the tubercle bacilli will be killed. It may be pointed out that the margin of safety for pasteurization is considerably higher in Great Britain than in many parts of the United States, where a temperature of 142° F. is frequently laid down.

Even if there is gross failure to fulfil the conditions imposed, and the heat treatment is inadequate to destroy all tubercle bacilli, the probability is that the great majority of them will be killed. This is due to the fact that the destruction of bacteria by heat conforms more or less to the formula for a monomolecular reaction, in which the rate of change in any given period of time is proportional to the amount of substance (or number of bacteria) present at the beginning of that period. Thus, supposing that (1) an exposure of 138° F. (58·9° C.) for 30 minutes is required to destroy all tubercle bacilli, (2) there are 10 million tubercle bacilli in a given volume of milk, and (3) the milk is heated to 138° F. (58·9° C.) for only 20 minutes, it would follow that 9,970,000 or 97 per cent. of these organisms would be dead by the end of 10 minutes, and 9,999,987 or 99·99 per cent. by the end of 20 minutes. To say that such grossly underheated milk was more dangerous than raw milk would be a mere travesty of the facts.

" *The medical profession is not unanimous in support of pasteurization* "

This objection, which is frequently urged, is a gross misrepresentation of the truth. Pasteurization is of such importance to the medical profession in its endeavour to prevent unnecessary disease that it has been discussed and approved of by nearly all the important representative bodies concerned with it. Thus resolutions in its favour have now been passed by the Royal College of Physicians, the Royal College of Surgeons, the British Medical Association, the Society of Medical Officers of Health, and the Joint Tuber-

culosis Council. With regard to the British Medical Association, which has a very large membership, a certain amount of opposition might have been expected. In fact, however, though the subject was brought up on three separate occasions before the annual representative meeting, at which the whole of the Association's 200 local divisions were represented, on only one occasion was there so much as a single dissentient.

It is not, of course, maintained that there is complete unanimity on this subject. Dietetic faddists are present everywhere, and it could hardly be expected that a profession containing 43,000 practising individualists would agree completely on any one point. But it is maintained that there is probably more unanimity in the medical profession on the desirability of pasteurization than on any other important problem with which it is at present concerned.

It is perhaps worth while to inquire briefly into the objections raised to pasteurization by members of the medical profession. Great play has been made by the National Federation of Milk Producer-Retailers with the views of Dr. Ralph Vincent. Inquiry shows that Dr. Vincent, whose contributions are entirely unknown to most scientific workers, died in 1922, and published his last scientific paper in 1914. Considering that the great mass of evidence on which the public health case for pasteurization rests has been built up since that date, it is hardly fair to Dr. Vincent himself still to ascribe to him views which he might quite well have altered in the light of subsequent work—as so many of his colleagues have actually done.

Referring to the objections of medical men who are still alive, the case against pasteurization was presented in a number of letters to the British Medical Journal in the first half of 1938. These may be summarized.

(*a*) Dr. Norman Macfadyen (1938) of Letchworth regards Tuberculin Tested (Certified) milk as superior to Pasteurized, and supports his opinion by quoting some bacteriological results on a small number of samples of milk, which revealed the presence of coliform organisms in some of the pasteurized but in none of the Certified samples. Since there is no coliform standard laid down for pasteurized milk, and since there is no reason to believe that these organisms, which are inhabitants of the normal intestine, are likely to prove harmful if ingested in small numbers, the objection seems to be trifling. But surely, if a comparison had to be made, it would have been fairer to make it between Tuberculin Tested (raw) and Tuberculin Tested (Pasteurized) milk, not between Tuberculin Tested (Certified) milk, which is produced under exceptionally clean conditions and is bottled on the farm, and ordinary pasteurized milk, which is usually the bulked milk from a number of farms on which the general standard of cleanliness is lower than that on farms producing the highest grade of raw milk.

(*b*) Dr. Halliday Sutherland (1938) fears that compulsory pasteurization would remove the stimulus to produce clean milk and Tuberculin Tested milk. These fears have already been fully dealt with (pp. 171–174) and shown to be groundless.

(*c*) Dr. Halliday Sutherland (1938), and Dr. Lionel Picton (1938) of Holmes Chapel, are afraid that the general consumption of pasteurized milk would lead to a decrease in human fertility and to a consequent fall in the birth-rate. Both of these refer to certain experiments carried out by Mattick and Golding on rats at Reading. These experiments have already been described and analysed (pp. 129–132), and have been shown, when considered as a whole, to lend no support to the belief that the fertility of rats

on pasteurized milk is less than that on raw. Nor do the results of other experiments on mice and calves, which have also been reviewed (pp. 134–138). The fear that the introducion of compulsory pasteurization would lead to a fall in the national birth-rate is devoid of any foundation.

(*d*) Dr. Chalmers Watson (1933) of Edinburgh, who, like many other objectors to pasteurization, is an advocate of Tuberculin Tested milk, is afraid that pasteurization may affect deleteriously some subtle nutritive quality of the milk at present unknown to chemists or nutritional experts. While such a possibility cannot be denied, none of the observations reviewed in Chapters VIII and IX has hitherto succeeded in demonstrating it.

It will be realized that the three main objections, namely that pasteurization decreases the nutritive value of the milk, that it is detrimental to fertility, and that it removes the stimulus to the production of clean milk and to the eradication of diseased animals from milking herds, are all hypothetical, are unsupported by evidence, and are largely disproved by observations both in the laboratory and in the field. These objections are put forward by advocates of Tuberculin Tested milk. There is no antagonism between pasteurization and the production of clean and Tuberculin Tested milk. The better the source of supply, the more satisfactory will be the pasteurized article. But what so many of the advocates of raw Tuberculin Tested milk forget is that, though reasonably safe from the risk of carrying tuberculosis, it is just as likely as any other type of milk to contain pathogenic organisms derived from the human personnel and other sources. Several outbreaks of scarlet fever, typhoid fever, and other diseases have in fact followed its use (see p. 50). The only way to ensure the safety of the final product is by some form of heat treatment.

Summarizing, we may say that there is an extraordinary degree of unanimity in the medical profession in favour of pasteurization, and that the objections of the relatively few individual members who oppose it are based on fears of possible unfortunate consequences. These fears appear to be entirely groundless, and should not be allowed to weigh against the great demonstrable advantages of pasteurization in supplying the human population with safe milk.

"*Pasteurization is not advocated by the Pasteur Institute*"

This statement of the late Sir Arnold Wilson (see Heap 1938) is surprising. In the first place it is difficult for anyone who knows that the Pasteur Institute was established primarily for the treatment of rabies to understand why it should wish to express an opinion on milk at all. And secondly, it is highly improbable that an institute which was erected in honour of the discoverer of pasteurization should directly repudiate the very principle he introduced.

To clear up any possible doubt, however, a personal letter was sent to Dr. Louis Martin, the present Director of the Pasteur Institute, asking him whether there was any substance in the statement made by Sir Arnold Wilson. There is no need to reproduce his reply in full. Suffice it to say that he expressed profound surprise at such a statement, completely disclaimed its truth, and remarked that it was all the more surprising in view of the fact that the Pasteur Institute had afforded facilities to M. Stassano for working out the method of pasteurization (Stassanization) which is extensively used on the Continent.

"*Raw milk is better than no milk*"

Though this statement cannot be strictly regarded as an objection to pasteurization, it is nevertheless relevant to it and can be conveniently considered here.

During the course of a debate on the anti-tuberculosis service in Wales and Monmouthshire in the House of Commons on March 22nd, 1939, Mr. Kenneth Lindsay (1939), Parliamentary Secretary to the Board of Education, is reported to have made the following authoritative statement: "Where there is neither T.T. nor pasteurized milk obtainable the value of milk is so great that the children will on balance be in more danger of tuberculous infection if they are debarred from taking milk than if they take the risk and drink ungraded milk."

It would be interesting to know on what evidence this statement was based. As an expression of personal opinion it may be justifiable, but as a record of scientific fact it is quite unjustifiable. We know of no published evidence to prove the truth of such a contention.

The real answer is that no such contingency as that envisaged by the Parliamentary Secretary should be allowed to arise. If neither Pasteurized nor Tuberculin Tested milk can be obtained, then the milk should be boiled, or dried milk made up with some suitable flavouring agent should be supplied.

If it is objected that the school-teachers have no time to do this, then voluntary social help should be called in.

To recommend the consumption by young children of raw potentially infected milk, when the milk can be rendered safe by a very little trouble, can hardly be described as a progressive step on the road towards national fitness.

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(With few exceptions the references given below are to the main text, not to the Summaries at the end of each chapter nor to the General Summary and Conclusions on pp. 180–186.