Milk consumption and tuberculosis in Britain, 1850-1950

Dr P.J. Atkins, Department of Geography, University of Durham

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Introduction

The `cultural turn' which has swept through the Anglo-American social sciences in the 1990s has as one of its major themes the consumption of food and the various discourses which are deployed to understand it. This theme of consumption is particularly apposite because in recent times we have also seen a surge in consumer concern about the ethics of food production and the quality of foodstuffs at various stages in the food system. The sight of middle class English protesters blocking the export of live calves to the Continent in 1995 was symbolic of a growing resistance throughout society to intensive farming and the declaration on 20th March 1996 that Mad Cow Disease (B.S.E.) can be transferred from cattle to humans has confirmed our worst fears about consequences for health. The green and consumer lobbies are keen to present these issues as new but I wish to argue that the concern, in a fully mature form, about food quality is at least one hundred years old. In this paper I will outline the history of bovine tuberculosis in Britain and argue that its study presents us with one possible way that food historians might approach the links between consumption and health.

The debate about bovine tuberculosis

Tuberculosis was the single greatest cause of death and disability in nineteenth century Britain. People in their 30s and 40s had a depressingly high chance of dying from it. It was a plague to match cancer or AIDS in our own time. Unfortunately the World Health organisation has just warned that respiratory tuberculosis is returning at present in a new form that is resistant to drug therapy and that deaths will exceed one hundred million world-wide in the next fifty years.[1]

The German microbiologist Robert Koch discovered the tubercle bacillus in 1882 and almost immediately it was realised that milk might be a powerful agent of its spread. Only four years later a survey by the Association of Municipal Corporations found that 85 per cent of English Medical Officers of Health believed that tuberculosis was communicable via raw milk and undercooked meat, a view endorsed subsequently by various parliamentary enquiries.[2] This was a remarkable sea-change in opinion, the medical and public health professions having previously been convinced that heredity and environment were the major factors. Scientific belief and public action are often lagged, however, and indeed very little was done to put into practical effect the new rhetoric that dominated the public health journals and local Medical Officer of Health Reports in the 1890s and 1900s.

There were two reasons for this. First, neither was there an administrative structure for inspecting cattle or collecting samples of milk, nor had technology for mass screening yet become readily available. These features appeared first in large cities such as Manchester at the turn of the century and gradually filtered down to smaller centres. Second, the belief in the transmissibility of the bovine strain of tuberculosis, *Mycobacterium bovis*, to humans was fiercely contested. Ironically it was Koch himself, at the International Congress on Tuberculosis held in London in 1901, who declared that he had found experimentally that the human version of the mycobacterium did not produce tuberculosis in cattle, the implication being that there was no danger of an infection in the other direction either. It is difficult to exaggerate the consternation this caused in the medical community but it did at least stimulate debate and there was a surge in tuberculosis-related laboratory research in Britain, Germany, France and the United States.[3]

In this country a Royal Commission was appointed with a brief to take evidence and conduct experiments. Several strains of bacilli were identified, with different pathogenicities.[4] Under laboratory conditions the most virulent was the *M. bovis*, which in humans was shown conclusively to be principally responsible for non-pulmonary forms of tuberculosis.

Tuberculosis in the cattle herd and livestock products

A major reason for the prevalence of bovine tuberculosis in humans was the endemicity of the disease among the cattle herds that supplied Britain's liquid milk and much of its beef, especially those cows confined to cramped urban sheds where the probability of infection by airborne droplets was maximized.[5] From Table 1 it is clear that the cattle population in general, kept on the whole on open pastures, were infected to a far lesser extent.

The problem for farmers was that the early stages of tuberculosis in cattle are invisible. By the time there are symptoms, such as emaciation, a chronic cough or a diseased udder, both

Table 1: Tuberculosis discovered at slaughter

	Cattle slaughtered	% TB
Belfast 1909-13, 1917, 1919, 1922-4	161,306*	15.00
Birkenhead 1892-8, 1915, 1920	225,228	0.53
Blackburn, 1920	*	9.69
Bradford 1918-21	56,659	2.74
Bury 1911-25	52,292*	1.33
Croydon 1920	757	1.72
Derby 1937	*	53.00
Edinburgh 1919-21, 1924-25	4932*	42.19
Halifax 1915	9,185	0.05
Liverpool 1913-16, 1918, 1920 Liverpool 1921, 1925	95,950 4,674*	1.02 11.90
London (Metropolitan Cattle Market) 1881 London (Metropolitan Cattle Market) 1929 City of London abattoirs 1918-27	* * *	90.00 51.00 33.33
Newcastle 1917, 1919-25 Newcastle 1943	158,700 *	0.68 47.5
Salford 1920	1,365	2.05

Note: * cows only
Sources: Medical Officer of Health Annual Reports; Creighton (1881); Savage (1929), 41-2; Francis (1947), 22-24.

the carcass and milk are potentially dangerous for human consumption.[6] In the decade or so before the First World War it was generally realised that tests were required to ensure detection at the earliest possible moment. There were several possibilities.

First, the veterinary inspection of cows was attempted. This was easiest among beasts tethered in urban settings but some local authorities also employed inspectors to tour the country areas supplying milk. The results were varied at first (Tables 2 and 3) but gradually farmers came to realise that diseased animals threatened their livelihoods and a `slink' trade in `wasters' developed as a hidden circuit in the livestock economy of some regions. In Cheshire, for instance, vets employed by the City of Manchester were well aware of the problem but were powerless to act:

`The number of cows suffering from tuberculosis of the udder found in this piece of inspection was comparatively small, but this is to a great extent accounted for by the fact that, as soon as it became known that the farms were being visited, large numbers of suspected animals were sent into the local auction market every week, and disposed of to individuals who evidently had no difficulty in disposing of this class of cattle'.[7]

Table 2: Evidence of udder tuberculosis in the early twentieth century

	Cows inspected	**% TB udder**
Birmingham: 1900-5, 1917-19, 1923, 1925	14,445	0.14
Blackburn 1902-12, 1920	unknown	0.6-2.9
Bradford 1910-11, 1915, 1918-19	16,212	2.10
Cumberland 1927-30	39,641	0.10
Gloucestershire 1927-31	327,434	0.06
Lanarkshire 1926-9	141,285	0.12
Leeds 1914	2,000	0.85
Liverpool 1901-25	47,185 city cows 29,325 country cows	0.83 0.79
Manchester 1900, 1903-11, 1921	92,073 city cows 24,851 country cows	0.02 0.81
Newcastle 1908-25	9,223	0.54
Salford 1921	91	3.30
Sheffield 1901-14, 1921-29	unknown	country cows 0.59-4.84 city cows 0.14-1.10
Yorkshire, West Riding 1928-30	256,988	0.13

Sources: Medical Officer of Health Annual Reports; Anon. (1932); Savage (1929).

Table 3: Udder tuberculosis detected by Liverpool inspectors

	City cowshed visits				**Country cowshed visits**			
	Cows examined	**TB udders**	**%**	**Convictions**	**Cows examined**	**TB udders**	**%**	**Convictions**

1901-5	1,156	184	15.9	21	2,345	76	3.2	12
1906-10	1,110	24	2.2	0	3,445	29	0.8	11
1911-15	12,526	73	0.6	0	5,837	20	0.3	4
1916-20	7,499	23	0.3	0	4,278	29	0.7	0
1921-25	24,894	89	0.4	0	13,420	77	0.6	0

Source: Medical Officer of Health Annual Reports

Second, the tuberculin test (TT) was available for detecting tuberculosis. A fold of the animal's skin was injected with dead mycobacteria and a reaction of swelling was taken as an indication that there was a problem of infection. This TT was adopted only slowly in Britain by comparison with countries like Denmark, one possible reason being the alarming results. Cows supplying Birmingham's milk were the first to be tested on a large scale, and from 1907 to 1927 no less than 40.4 per cent were found to be reactors.[8]

Third, the microscopic detection of *M. bovis* in milk allowed the sampling of a city's supply and the identification of infected herds. This was a cost-effective approach pioneered in the largest cities but it came to be used by many Medical Officers of Health around the country as a means of providing ammunition in their campaign about the dangers of the milk supply. Figure 1 is a compilation of data from Birmingham, Liverpool, London, Manchester, Newcastle, Salford and Sheffield. It shows that the proportion of samples found to be tuberculous was as high, if not higher, in 1930 as at the start of the series. This proved that government-sponsored remedial measures were necessary because the well-meaning efforts of local authorities had been ineffective.

<Figure 1 here.>

This pooling of data from a number of cities hides the puzzling spatial variations of disease incidence shown in Table 4. Note the high figure for Manchester by comparison with other authorities. There are possible explanations in the differences in sampling methodology adopted and in the technicalities of testing but these are insufficient reasons to account for the extremes of geographical variation.

Table 4: Tuberculosis in milk samples

	Dates	Samples	%		Dates	Samples	%
Aberdeen	1920-30	1,561	5.7	Leeds	1913, 20-30	1,080	
Birmingham	1907-37	20,047	8.1	Liverpool	1896-1937	26,978	6.2
Blackburn	1915, 20-30	1,908	2.2	London	1908-37	53,195	8.1
Brighton	1915, 28-37	737	9.2	Manchester	1901-37	18,995	11.3
Bristol	1920-30	450	6.7	Monmouth	1927	213	1.4
Burton on Trent	1904-5	117	10.3	Newcastle	1913-37	6,819	4.4
Cambridgeshire	1927	46	0.0	Northumberland	1927	58	5.2
Cardiff	1920-30	682	4.6	Reading	1920-30	282	11.0
Coventry	1909-10	53	15.1	Salford	1904-6, 13-16, 20-30	4,401	8.0
Croydon	1900-21	547	8.0	Sheffield	1902-14, 20-37	20,580	8.0
Derby	1907, 09	94	8.5	Somerset	1926-8	683	2.2
Dorset	1926-7	243	4.9	Southport	1902-20	724	8.2
Edinburgh	1904, 6-7, 11-12, 26-37	3,541	8.5	East Suffolk	1927	33	6.1
Glasgow	1921-5, 28-37	4,647	6.7	Warwickshire	1927	85	3.5
Huddersfield	1926-30	237	6.3	Yorkshire, West Riding	1923-33	6,951	5.8
Hull	1925-30	449	6.7	**Total**	1896-1937	178,189	7.8
Lancashire	1924-7	1,753	10.2				

Sources: Medical Officer of Health Annual Reports; Anon. (1932); Savage (1929).

The impact on humans

In some regions it seems likely that **all** of the milk drinking population would have been infected with bovine tuberculosis at one time or another. Thus 1,420 post mortems of children under 12 in the 1880s showed that 30 per cent had tuberculosis, no doubt mostly of bovine origin, and in 1930 58.3 per cent of a sample of London children reacted positively to the TT.[9] My estimates indicate that at least half a million (and possibly 800,000) human deaths in the period 1850-1950 are directly attributable to bovine TB, especially among young children who were the main milk drinkers.

One positive outcome of the Royal Commission on Tuberculosis was some detailed work on the typing of the mycobacteria found in human sputum, in surgical biopsies and in autopsy specimens. Stanley Griffith's work was the outstanding contribution, continuing through to the 1940s. As a result of his investigations we have a fair idea of the proportion of tuberculosis in

each organ caused by bovine infection. From the data in Table 5 it seems that respiratory tuberculosis was rarely a consequence of milk consumption, but the non pulmonary forms were.

Table 5: The estimated site-specific tuberculosis mortality of bovine origin

Tuberculosis type	**Savage (1929)**		**Griffith (1937, 1941) all ages**	
	0-4 years (%)	**>5 years (%)**	**England (%)**	**Scotland (%)**
Respiratory	0	1	2	6
Abdominal	80	33	45	45
Nervous	20	20	25	30
Bones & joints	30	15	20	30
General	25	10	-	-
Other	20	5	34	36
Mean	29.5	3.2	27.8	34.2

Sources: Savage (1929), 27; Griffith (1937), 530-1; Griffith and Munro (1943).

The overall number of deaths from tuberculosis fell markedly between 1840 and 1920, but the proportion of this mortality attributable to non-pulmonary forms rose, peaking in the 1890s (Figure 2).[10] It seems likely that the respiratory disease was radically reduced in response to the environmental improvements of the late nineteenth century, especially in housing conditions, for the probability of catching `phthisis' had been exacerbated by over-crowded and ill-ventilated accommodation. As the use of cows' milk increased in the second half of the century, *M. bovis* infections became more common, especially amongst small children who had been taken from the breast. Unfortunately, the British taste for raw milk made this problem worse than it might have been had boiling been as common as on the continent.[11] Other dairy products were also sources of infection: *M. bovis* can survive in butter for up to five months, and also for long periods in some types of cheese.[12]

<Figure 2 here>

In Scotland the non-respiratory tuberculosis death rate was consistently higher than in England, and there were also regional variations, the highest county figures lying in the northern half of the country. It seems likely that this distribution of human disease reflected that in cattle, but interestingly it was the inverse of the map of total tuberculosis mortality. It has been argued that this was due to an inoculation effect whereby regular low doses of *M. bovis* induced a resistance to infection of the lungs by the respiratory route.[13]

The local state and the regulation of production and distribution

It was generally recognised in the late nineteenth century that poor cattle housing was responsible for dirty and diseased milk.[14] The Dairies, Cowsheds and Milkshops Orders (1879, 1885) empowered local authorities to regulate the physical conditions in which animals were kept but improvements were gradual.[15] The crowding and lack of ventilation, which together created ideal circumstances for the spread of tuberculosis from animal to animal, remained common in rural areas into the 1920s and 1930s.[16]

Another hazard arose, ironically with technological progress in the 1920s. This was the mixing in 3,000 gallon tankers of the milk of 1,000 cows. If only one cow was diseased and excreting mycobacteria then the whole batch was contaminated because tuberculous milk can be diluted 10,000-1,000,000 times and still be infective.[17] Some forms of tuberculosis, such as that of the bones and joints, can be initiated by even small invasions of mycobacteria.

The dangers of tuberculous milk infected with tubercle bacilli may have been scientifically known from the 1880s, but policy reaction at the local level was slow. In 1885 the town council of Hull did enquire of the Privy Council about the possibility of using the Contagious Diseases (Animals) Acts to deal with the source of the disease, but nothing seems to have come of their initiative. Three years later a Departmental Committee even recommended compulsory slaughter of animals with tuberculosis, but this aspect of their report was ignored.[18]

Glasgow was the first city, under its Police (Amendment) Act of 1890, actually to take powers for its medical and sanitary officers to inspect any cowshed supplying the city and to prohibit the sale of any milk `dangerous or injurious to health'.[19] Weak and permissive English law, however, was an obstacle to progress south of the border. Promisingly, Article 15 of the

Dairies, Cowsheds and Milkshops Order of 1885, stated that `If at any time disease exists among the cattle in a dairy or cowshed, the milk of a diseased cow therein shall not be mixed with other milk; and shall not be sold or used for human food', but the definition of `disease' used was that of the 1878 Contagious Diseases (Animals) Act, which did not include tuberculosis.

Under the Infectious Diseases Prevention Act (1890) and The Public Health (London) Act (1891), sanitary authorities were given relevant powers.[20] A local authority's Medical Officer of Health had to obtain a magistrate's order to inspect a dairy and/or cows. Another order could then be made against the dairyman, forbidding the local sale of his milk. The procedure was cumbersome and there was nothing to prevent the dairy farmer or dairyman from switching milk to an area other than the one mentioned in the prohibition order. As a result few prosecutions were ever obtained.

In the absence of any real central government intervention it was left to a few pioneering local authorities to initiate measures against bovine tuberculosis. The so-called `milk clauses' of the private Manchester Corporation (General Powers) Act of 1899, as amended in 1904, became a bench mark.[21] Officials were enabled to:

- prosecute anyone who knowingly sold milk from cows with tuberculosis of the udder;[22]
- demand the isolation of infected beasts;
- demand the notification of any cow exhibiting signs of tuberculosis of the udder;
- inspect the cows in and take samples from herds which supplied milk to the city.

By 1910 67 boroughs and 24 urban districts had similar powers. Section 27 of the London County Council (General Powers) Act of 1904 allowed the compulsory slaughter, with compensation, of diseased animals, and Section 24 of the 1907 version of the same Act empowered the L.C.C. to take samples at railway stations and to prevent tuberculous milk being sent to London. It was not until 1925 that central government measures advanced beyond this stage.

The central state and the rights of the consumer

Faced with a mounting public outcry and with an overwhelming body of evidence, successive administrations felt the need to demonstrate, at the very least, a momentum in the direction of change. Concerted action was very slow in coming but there were experimental forays into enabling legislation.[23]

In 1909, after many years of discussion, it seemed likely that a Milk and Dairies Bill would receive the royal assent. This comprehensive legislation was meant, inter alia, to prohibit the supply and sale of milk likely to cause disease, including tuberculosis (Clauses 2 and 3). The Bill was not passed, however, and the Local Government Board was forced to withdraw its parallel Tuberculosis Order which would have given all local authorities powers of inspection and slaughter. A similar Order, made in 1913, was replaced in June 1914 by another, which in turn was revoked on the outbreak of hostilities.[24] The war also delayed the implementation of the Milk and Dairies Act (1914) and its replacement, the Milk and Dairies (Consolidation) Act of 1915. The Milk and Dairies (Amendment) Act (1922) further postponed any action until 1925, when at last the Tuberculosis Order (1925) and the Public Health (Prevention of Tuberculosis) Regulations (1925) were made.[25]

Such a messy legislative process significantly delayed progress. One problem was that, although the problem of cattle disease was relatively well understood in the farming community, milk producers were on the whole small farmers who did not have the resources to clear out their own herds and start again with tuberculosis-free stock. There needed to be a policy of carrot (compensation for slaughter) and stick (legally binding regulation) but the politics of pragmatism meant that successive governments were content with steady, incremental change. Thus, the Tuberculosis Order of 1925 did establish the principle of slaughter with compensation, but the scheme was low key and not uniformly applied, and in practice it proved difficult to prevent tuberculous animals being recycled in the slink trade.[26] All of this contrasted with the United States where a more forceful slaughter policy was adopted, and most continental countries which settled for versions of Professor Bang's method of separating healthy from infected animals.

It was clear, to the veterinary and medical professions at least, that little improvement had been achieved by the early 1930s. Some consumers in urban areas had access to pasteurised milk,

but not all of the equipment used was effective in killing the mycobacteria, and anyway it was merely treating the problem at an unacceptably late stage in the food chain. The People's League of Health in 1932 and the Gowland Hopkins Committee in 1934 both reported a major problem with the milk supply as a result of a minimum of 40 per cent of the dairy herd being infected with tuberculosis. This was difficult to ignore and the Milk Act of 1934 set aside a sum for upgrading milk quality. The notion of an `attested herd' was introduced where cattle had passed the TT successfully, and milk guaranteed as disease-free attracted a premium. In 1937 the Ministry of Agriculture began moving towards an area eradication policy, an important advance that was interrupted by the Second World War.[27]

It was not until 1950 that bovine tuberculosis again came to the top of the political agenda. After decades of half measures the government finally decided on a stringent policy of area eradication which involved much disruption to the farming industry, through restrictions on the movement of cattle, and a considerable expenditure in compensation. The whole country was declared attested in 1960, and `TT milk' as a category was abolished in 1964.[28]

Table 6 displays the geographical distribution of cattle taken for slaughter 1926-40 under the Tuberculosis Orders (1925, 1938). An alternative index would have been the proportion of positive reactors to the tuberculin test, but by 1938 only a small and unrepresentative sample of herds had been tested.[29] The map shows heavily infected areas in north west and south east England, and in central and south west Scotland. Many of the worst affected counties were also the chief milk producing areas. Cheshire, for instance, in the mid 1940s still had 60-80 per cent of its cows infected, when the estimated average for Britain was 30-35 per cent.[30] Large herds and older cows were especially susceptible.[31] Table 6 hints that people drinking milk produced in north west England were most at risk.[32] Much of that milk entered into the long distance supply of cities such as Manchester and may help to explain the high incidence of bovine tuberculosis among their citizens.

Table 6: The proportion of off-farm liquid milk sales in each Milk Marketing Board region in England and Wales, 1924-5-1938/9, compared with the percentage of cattle slaughtered under the Tuberculosis Order 1926-40

Region	Milk sales (%)	Cattle slaughtered (%)
North	6.63	6.60

North West	28.01	38.44
East	5.36	6.98
East Midlands	7.70	6.57
West Midlands	8.44	7.47
North Wales	2.49	4.59
South Wales	3.83	2.73
South	7.49	3.41
Mid West	15.23	7.98
Far West	4.18	2.68
South East	10.65	12.56
Total	100.01	100.01

Sources: Francis (1947), 28-31; Barnes (1958).

Conclusion

Recent public concern about Mad Cow Disease is by no means the first time that there has been a furore about cattle disease infecting humans. There have been many scares over the last one hundred years, and the reaction of politicians has been consistently reluctant and inadequate. On each occasion there appears to be a pattern of behaviour in the Ministry of Agriculture which suggests that public health is not, and never has been, at the top of its agenda.

By way of an overall conclusion we may assert that historians have a very important role to play in the analysis of food systems and food consumption. Examination of the temporal dimension can assist with a fuller understanding of structural and policy problems, many of which are not new. The ultimate aim is a theory of governance and food quality regulation that would act as context for analysing issues of food consumption and health.

Notes

1. *Guardian*, March 22nd, 1996, p 3.

2. *Royal Commission (*1898), q. 155. Subsequently it transpired that meat was far less likely than milk to have been a source of infection. Savage (1929), 1.

3. This was by no means the first time that Koch's work on tuberculosis had been found wanting. Cobbett (1917), 132; Rosenkrantz (1985).

4. There are 28 species of mycobacteria, nine of which can cause human disease. Youmans (1979), 3-4.

5. *Royal Commission* (1896), qq 1508-9.

6. About 4 per cent of TT reactors excrete bacilli in their milk, but only 25 per cent of these show any udder lesions. Kaplan (1962), 48.

7. Medical Officer of Health, Manchester, *Annual Report* (1909), 134.

8. Savage (1929), 38.

9. Cronje (1984), 81; Smith (1988), 12.

10. It should be noted that there are serious doubts about the accuracy of the mortality data on tuberculosis. The disease has complex manifestations and its diagnosis is very likely to have been confused with other causes of death. The term "phthisis", for instance, was a general term for wasting diseases and would therefore have covered pulmonary tuberculosis,
leukaemia and various internal cancers. Hardy (1988), 392. There are complex interactions between tuberculosis and other diseases which also need to be considered. Some deaths may, for instance, have been due to tubercular `breakdown disease' in former sufferers initiated through a weakening of resistance by an unrelated infection. Szreter (1988), 17; Rich (1944).

11. Thorne-Thorne (1899), 30; Ashby (1922), 187; Smith (1988), 190. Boiling reduces the nutritive value of milk and anyway was rejected as a practice by mothers in the 1880s as giving rise to constipation. Roberts (1973), 86.

12. Savage (1929), 2-6. The consumption of cream was also risky, but dried milk and condensed milk less so because of the heating process in their manufacture.

13. Savage (1929), 137, 152; Ministry of Agriculture (1965), 217; Cronje (1984), 82; Bryder (1989), 135.,

14. Atkins (1977); Atkins (1992).

15. The Dairies, Cowshed and Milkshops Order of 1899 amended Article 15 of the 1885 Order to include tuberculosis for the first time. The Order remained permissive, however, and even those local authorities which did make their own regulations were not necessarily conscientious in carrying them out.

16. Before the First World War few rural authorities seem to have taken the problem of cattle welfare seriously. In many areas there was no veterinary inspection whatsoever. Woodruff (1911), 87.

17. Individual cows may intermittently excrete five million bacilli per ml. Forrester (1927), 17; Francis (1947), 131; Kaplan (1962), 48.

18. *Departmental Committee* (1888) xxxii, p. xxiii.

19. *Royal* Commission (1898), q 155; Pennington (1982), 89-91.

20. Under these powers the London County Council kept a record of the number of instances of disease on milk retailing premises in their jurisdiction. The results were startling. In the short period 1895/6-1900/1, for instance, there were 680 cases of scarlet fever, 398 of diphtheria, 108 of enteric fever, and 23 of smallpox. London County Council, *Annual Reports*.

21. But one contemporary writer was sceptical that the Manchester Milk Clauses had any positive effect beyond their educational impact. Savage (1912), 338; Savage (1929), 104.

22. In practice it proved very difficult to track down the sources of tuberculous milk. Large dairy companies mixed their milk at rural depots before consignment. Savage (1929), 107.

23. The farming lobby was politically strong enough to mobilise opposition in parliament to any anti-tuberculosis measure which might threaten the prosperity of dairying. Smith (1988), 176-77.

24. Ministry of Agriculture (1965), 216.

25. Grades of milk were established by the Milk (Special Designations) Order (1923) which guaranteed that the source herd had been tuberculin tested. The 1925 Tuberculosis Order was amended in 1938.

26. Notification and slaughter, with compensation, were compulsory for animals suffering from tuberculosis of the udder, emaciation, or chronic cough with clinical signs of the disease.

27. By 1947 only 15 per cent of herds had qualified as `attested'. Francis (1947), 26.

28. Myers & Steele (1969), 269-70.

29. The Milk and Dairies (Scotland) Act of 1914, which came into effect in 1925, was responsible for a rather different system of regular veterinary inspection north of the border.

30. Francis (1947), 27, 32.

31. Ritchie (1964), 4.

32. In Cheshire, Derbyshire, Lancashire, Staffordshire and the West Riding of Yorkshire.

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