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The incidence of *Brucella* infections in producer-retailer herds in North Lancashire from 1965 to 1972

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SUMMARY

The results are presented of testing untreated producer–retailer herd milk samples for the presence of *Brucella abortus* during the period 1965–1972 in the North Lancashire region.

There was a steady decline in the incidence of infected herds from 22% in 1965 to 12% in 1971. A sharp fall to 5% in 1972 suggests that the introduction of the Brucellosis Incentives Scheme and the eradication programme has helped to reduce the practice of selling brucella-infected cattle in the open market which was prevalent in the period 1965 to 1970.

This practice of selling brucella-infected cattle may also be a prime factor in the changing pattern of distribution of the biotypes of *B. abortus* which was observed during the period 1965 to 1970.

A comparison of the two areas in the region show that the incidence of herd infection was always greater in the area containing flying herds than in the area in which self-contained herds predominated.

INTRODUCTION

In the United Kingdom brucellosis is primarily a disease of cattle and infections in other animals and man are invariably associated with infected cattle or their products, except for the occasional infection which is acquired either abroad or in the laboratory. An important vehicle in the transmission of the disease from cattle to man is untreated, brucella-infected milk. Whilst the pasteurization of milk is a reliable method for protecting the consumer, the eradication of brucella infection in cattle is the only measure which will eliminate human infections contracted by direct contact with infected animals.

In Lancashire there are approximately 1000 producer-retailer herds, the majority of which are in the north of the county supplying untreated milk directly to the public. In this report an account is given of the incidence of brucella infection in producer–retailer herds in the area served by this laboratory (North Lancashire) since 1965.

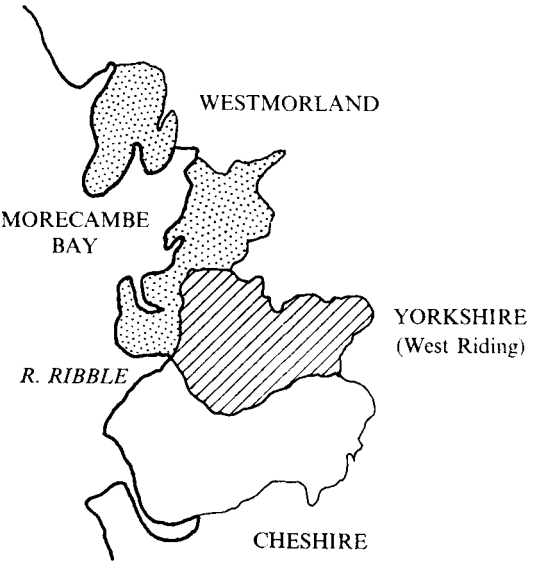

Fig. 1. Lancashire showing Area I ▦ mainly self-contained herds and Area II ▨ mainly flying-herds.

MATERIALS AND METHODS

The region under investigation can be divided into two areas designated I and II, as shown in Fig. 1. In Area I most of the producer–retailer herds are self contained units and replacements are reared from their own stock in contrast to Area II where the majority are 'flying herds', replacements being bought as required. Samples of bulk milk from these herds were screened with the milk ring test (MRT) (Report, 1956) and MRT-positive samples were cultured on selective media, and inoculated into guinea-pigs.

Direct culture

The milk samples were transferred to sterile, stoppered test tubes (7 × 1 in). Each sample was kept overnight in the refrigerator (approximately 4° C.) and a sample of gravity cream, withdrawn with a spiral wire (Mair, 1955), was spread over the selective agar with a bent, sterile, glass rod. During the period of the investigation a number of selective media were used and included media described by Mair, 1955; a modification of the medium described by Morris, 1956; Ryan, 1967; and Farrell, 1974. The plates were incubated at 37° C. in air containing 20 % CO_2 and were examined every 2 days for 10 days. Suspected brucella colonies were provisionally identified by slide agglutination.

Guinea-pig inoculation

Milk ring test positive samples were inoculated into guinea-pigs, but MRT-negative milk samples were inoculated only twice yearly, unless from herds which had previously been MRT-positive. The centrifuged deposit (1400 *g* for 30 min) from 100 ml. of milk was emulsified in the cream layer and 2 ml. inoculated intramuscularly into the thigh of a guinea-pig. When the guinea-pig was killed

Table 1. *The incidence of brucella-infected producer–retailer herds in North Lancashire in the period* 1965–1972

Year	Area I	Area II	Total
1965	36/210* (17)	114/484 (24)	150/694 (22)
1966	30/201 (15)	82/479 (17)	112/680 (16)
1967	29/255 (11)	107/565 (19)	136/820 (17)
1968	33/336 (10)	113/546 (21)	146/882 (17)
1969	19/220 (9)	95/510 (19)	114/730 (16)
1970	13/281 (5)	93/481 (19)	106/762 (14)
1971	16/353 (6)	67/435 (15)	83/688 (12)
1972	1/208 (0·5)	27/366 (7)	28/574 (5)

* Number of herds excreting/number tested.
Figures in parentheses are percentages.

Table 2. *The biotypes of* Brucella abortus *isolated from untreated milk*

Year	No. of herds excreting	No. typed	Biotype			More than one biotype excreted
			1	2	4, 5 or 9	
1965	150	133	110 (83)	11 (8)	19 (14)	7
1966	112	110	86 (78)	12 (11)	17 (15)	4*
1967	136	135	93 (69)	14 (10)	32 (24)	4
1968	146	137	101 (74)	18 (13)	25 (18)	7
1969	114	108	71 (66)	11 (10)	35 (32)	8*
1970	106	104	70 (67)	13 (13)	32 (31)	11
1971	83	80	56 (70)	9 (11)	21 (26)	5
1972	28	25	22 (88)	5 (20)	2 (8)	4

* Including one herd excreting three different biotypes.
Figures in parentheses are percentages.

6 weeks later the spleen and deep inguinal gland adjacent to the site of inoculation were removed and cultured for brucella organisms on 5 % blood agar containing 100 μg./ml. of cycloheximide.

Identification and biotyping

The biotype of each strain of *B. abortus* was determined by the criteria of Alton & Jones (1967): (1) CO_2 dependence; (2) H_2S production on serum-dextrose agar; (3) sensitivity to basic fuchsin and thionin; (4) agglutination with *B. abortus* and *B. melitensis* monospecific sera; (5) sensitivity to *B. abortus* bacteriophage, strain Tblisi.

RESULTS

The incidence of herds excreting brucella organisms in milk was higher in Area II (flying herds) than in Area I (self contained herds) in each of the 8 years of the investigation. *B. abortus* was isolated from the milk of 22 % of all producer–retailer herds in 1965, the incidence declining steadily to 12 % in 1971, then falling sharply to 5 % in 1972 (Table 1). This sudden decrease was more pronounced in Area I, which contributed only one of the 28 herds excreting brucellas in 1972.

In 1965, 83% of the infected herds were excreting *B. abortus*, biotype I, and 8% excreting biotype 2 (Table 2). Since 1965 the incidence of biotype 2 has not altered significantly whereas the incidence of biotype 1 fell steadily until 1969, when it was responsible for 66% of infections. This was accompanied by an increase in the incidence of infections with biotypes 4, 5 and 9, from 14% in 1965 to 32% in 1969; biotype 4 has never contributed more than 1% of the infections for any year and has been included with biotypes 5 and 9 mainly for convenience. Biotypes 5 and 9 would previously have been described as 'British Melitensis'. After 1969 the incidence of these biotypes fell to 8% whilst the incidence of biotype 1 increased from 66% to 88% (Table 2).

DISCUSSION

Cattle are usually infected with *Brucella* by ingestion or inhalation of contaminated material. As a source of contamination the infected pregnant cow is especially dangerous during parturition when large numbers of organisms are discharged in the amniotic fluids, placenta, and fetus, and post partum when vaginal discharges containing large numbers of organisms are present for several weeks. In many infected cows the organism becomes localized in the udder and brucella organisms are excreted in the milk.

Leech and his colleagues (1964) estimated that in 1960–61 25,000 to 30,000 (25 to 30%) of British dairy herds were infected with *Brucella abortus*, and that 2% of dairy cows were infected, approximately 50% (36,500) of which were excreting the organism in their milk.

Short-term control measures were evolved before the introduction of a programme for the eradication of brucellosis. The clinical manifestations of the disease in cattle were controlled with brucella vaccines, S-19 and 45/20, thus reducing the all too familiar 'abortion storms', with great economic advantage to the farmer, but the problems of udder infections and, to a lesser degree of infertility, have remained. The transmission of brucellosis from infected cattle to man has been contained by pasteurization of milk, and where this was not feasible, by the restriction of the sale of brucella-infected, untreated milk. It must be emphasized however, that the risk to those in occupational contact will remain until the disease in cattle is eradicated.

In 1966 the Ministry of Health indicated that existing legislation should be vigorously applied against the sale of brucella-infected, untreated milk (Ministry of Health Circular 17/66). It was suggested that untreated milk supplies should be examined monthly, whenever possible, for the presence of brucella organisms and the sale of infected milk restricted by compulsory pasteurization.

In the North of Lancashire during 1959, Robertson (1961) found that 158/842 (19%) producer–retailer herds were infected, a figure comparable with our findings for 1965. Despite the increased surveillance recommended by the Ministry and the announcement of the proposed eradication programme (Hansard, 1966) the decline in the incidence of brucella infection in these herds was slight, for in 1969 114/730 (16%) of herds were producing for sale brucella-infected, untreated

milk. During the time covered by this report there was intense activity by the producer-retailers to identify the infected animals within their herds and these animals were then sold in the open market and introduced infection into other herds, thereby increasing the dissemination of the disease both within the region under investigation and elsewhere in the United Kingdom. This problem was appreciated by Henderson (1969) who described the practice of selling brucella-infected cattle in the open market in Worcestershire.

In Lancashire one County Borough rigorously excluded all brucella-infected raw milk by allowing only ring test negative milk to be sold within its boundaries. The farmers had the ring test positive, and thus potentially infected, animals identified by laboratory tests and proceeded to sell ring test negative milk within the County Borough and ring test positive milk in the contiguous municipal boroughs and rural areas.

In April 1967, the voluntary Brucellosis (Accredited Herds) Scheme was launched with the aim of identifying and registering those herds which could be used as sources of brucella-free replacement stock for other herds.

In April 1970, the replacement Brucellosis Incentives Scheme was introduced. To qualify for entry on the national register, a herd has to have three consecutive negative blood tests at four-monthly intervals.

In November 1971, eradication of brucellosis was started in three main areas of Great Britain with a programme of compulsory blood testing of all herds not already in the voluntary schemes.

Once the Brucellosis Incentives Scheme was introduced and the eradication areas and extension zones were specified the attitude of stock owners appeared to alter. The region described in this communication is not an initial eradication area, but a large part of Area I, which contains mainly large self-contained herds, is included in the extension zone for the next phase to be started in November 1973. These factors have undoubtedly contributed to the large decline in infections already described.

The distribution of the biotypes of *B. abortus* shows that the frequency of biotype 1 infection fell during the period 1965 to 1969 but with a corresponding increase in infection with biotype 4, 5 and 9 (Table 2), until in 1969 these biotypes caused 32 % of the brucella infections in this region.

It is significant that this change in the epidemiological picture occurred at a time when infected animals were being identified and their removal from herds actively encouraged, but without adequate powers to control their movement or to demand their slaughter.

Since 1970, the reversal of this trend has coincided with the acceptance of the Brucellosis Incentives Scheme by the producer-retailers in North Lancashire, with the slaughter of infected animals and their replacement by brucella-free stock.

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