

Survey of the incidence and aetiology of mastitis on dairy farms in England and Wales

A. J. BRADLEY, K. A. LEACH, J. E. BREEN, L. E. GREEN, M. J. GREEN

A survey of clinical and subclinical mastitis was carried out on 97 dairy farms in England and Wales, selected at random from members of a national milk recording scheme. The farmers were asked to collect aseptic milk samples from five consecutive cases of clinical mastitis and from five quarters with high somatic cell counts using a defined protocol, and they completed a questionnaire that included information on the cows sampled, the herd and the history of mastitis in the herd. The samples were collected throughout the year. The mean incidence of clinical mastitis was 47 cases per 100 cows per year (estimated from historic farm records) and 71 cases per 100 cows per year (estimated from the samples collected). *Streptococcus uberis* and *Escherichia coli* were isolated in pure culture from 23.5 per cent and 19.8 per cent, respectively, of the clinical samples; 26.5 per cent of the clinical samples produced no growth. The most common isolates from the samples with high cell counts were coagulase-negative staphylococci (15 per cent), *S. uberis* (14 per cent) and *Corynebacterium* species (10 per cent). *Staphylococcus aureus* and coagulase-positive staphylococci together accounted for 10 per cent of the samples with high somatic cell counts; 39 per cent produced no bacterial growth.

AS a result of the widespread application of the five-point-plan (Kingwill and others 1970) significant progress was made in reducing the incidence of clinical mastitis in UK dairy herds between the 1960s and the 1980s (Booth 1997, Bradley 2002). Wilesmith and others (1986) reported an incidence of 41 cases per 100 cows per year in 1982, compared with a figure of over 150 cases per year in the 1960s (Wilson and Kingwill 1975). In the last decade, a mean incidence of 17 cases to 43 cases per 100 cows per year have been reported (Table 1). Of these, the only studies on a national scale were those by Kossaibati and others (1998) and Berry (1998), and other studies were based on one or a small number of veterinary practices, or on a specific subset of herds. Those that were retrospective are likely to have underestimated the incidence because of a failure to detect or report all clinical cases of mastitis.

The UK literature describing the pathogens associated with clinical mastitis is even less extensive than that relating to incidence. Wilesmith and others (1986) reported that 47 per cent of clinical mastitis was caused by *Escherichia coli* or *Streptococcus uberis*. Bradley and Green (2001) reported that environmental organisms dominated, causing 61 per cent of clinical mastitis cases, and of these *Escherichia coli* was the most common. Milne and others (2002), also reported that environmental organisms dominated, causing 60 per cent of clinical mastitis, but in this case *S. uberis* was isolated more frequently than *E. coli*. The last two studies were conducted with small numbers of farms and in restricted geographical regions and thus the results may not represent the national dairy population.

There are no reliable data available in recent peer-reviewed literature on the incidence and aetiology of subclinical mastitis in the UK. Over 25 years ago, a national survey was conducted to estimate the prevalence of subclinical mastitis (Wilson and Richards 1980) in which major mastitis pathogens were reported in 14 per cent of quarters. However, there has been a large reduction in bulk milk somatic cell counts (BMSCCs) since that time, which suggests that there may have been a reduction in the prevalence of subclinical intramammary infections or that farmers may be able to manipulate the BMSCC by withholding milk with a high somatic cell count (SCC). Because of this lack of reliable data, it is difficult to assess the current importance of mastitis to the UK dairy industry objectively. The aim of this study was to estimate the incidence of clinical mastitis in a random selection of dairy herds in England and Wales and to identify the patho-

gens associated with clinical and subclinical mastitis in these herds.

MATERIALS AND METHODS

Farms

Dairy farmers were randomly selected from the list of farmers recording the SCC of individual cows in the National Milk Records (NMR) recording scheme, Chippenham; NMR is the largest milk recording organisation in Europe, recording more than 50 per cent of UK dairy farmers (NMR 2006). Five hundred and fifty letters were sent to eligible NMR users and a response indicating their willingness to participate was received from 125 farmers. These farmers were all enrolled in the study, although five had stopped milk production before the study began and six more sold their herds before completing or commencing their sampling.

Sample collection

The participants were asked to collect an aseptic pretreatment milk sample from each of the first five clinical cases of mastitis that occurred after they had received the sampling kit, and to freeze the samples immediately; the sample tubes contained glycerol as a cryopreservative. Five aseptic milk samples were also collected from quarters of cows with a SCC of more than 200,000 cells/ml at the previous milk recording. These cows were selected from all the cows with a SCC of more than 200,000 cells/ml as follows: after discarding any cows that had had a case of clinical mastitis in the previous four weeks, the cows with a SCC of more than 200,000 cells/ml at the last milk recording were sorted by line number (NMR reference number); cows for sampling were then selected alternately from the top and bottom of this list. A California Mastitis Test (CMT) kit was supplied and used to identify the infected quarters of each cow selected, and the positive quarters (maximum two per cow) were individually sampled. This procedure was repeated with the next eligible cow until five milk samples had been obtained. The milk samples were frozen before being posted to the laboratory for bacteriological analysis. A written questionnaire was completed by each farmer to collect information about the herd and details of the cows sampled. To ensure that samples were collected throughout the year, sampling kits were dispatched to 10 participants each month, beginning in February 2004 and ending in January 2005. The kits

Veterinary Record (2007)
160, 253-258

A. J. Bradley, MA, VetMB, PhD, DCHP, DipECBHM, MRCVS,
K. A. Leach, BSc, MSc, PhD,
J. E. Breen, BVSc, CertCHP, MRCVS,
School of Clinical Veterinary Science,
University of Bristol, Langford, Bristol BS40 5DU
L. E. Green, BVSc, MSc, PhD, MRCVS,
Ecology and Epidemiology Group,
Department of Biological Sciences, University of Warwick, Coventry CV4 7AL
M. J. Green, BVSc, PhD, DCHP, DipECBHM, MRCVS,
School of Veterinary Medicine and Science, and School of Mathematical Sciences, University of Nottingham, Sutton Bonington Campus, Sutton Bonington LE12 5RD

Dr Bradley is also at Quality Milk Management Services, Unit 1, Lodge Hill Industrial Park, Station Road, Westbury-sub-Mendip BA5 1EY

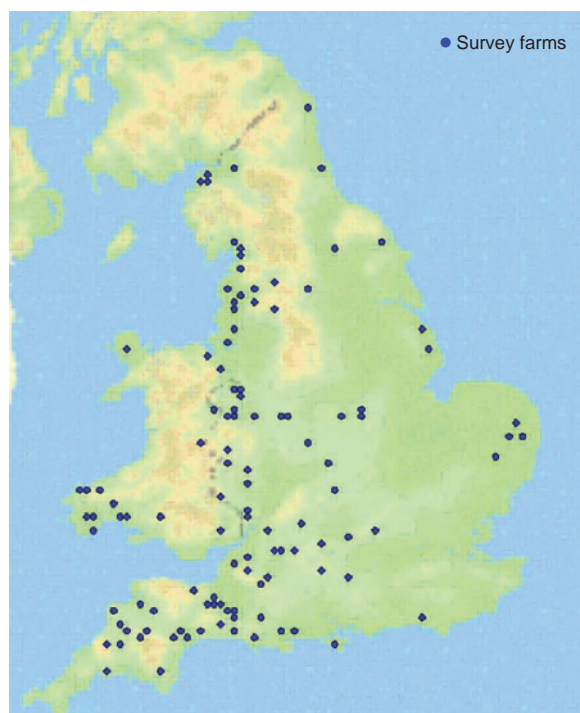


FIG 1: Location of the farms recruited for the survey of clinical and subclinical mastitis. Reproduced from Ordnance Survey map data by permission of the Ordnance Survey Crown copyright 2001

contained all the necessary equipment, full instructions and standard operating procedures for taking milk samples and performing the CMT.

Telephone calls were made during the month after the sampling kits were dispatched to ensure that they had been received and the instructions understood. Follow-up calls were made if samples had not been returned within three months of the kits being dispatched, and thereafter at three monthly intervals. Delays in sampling were recorded. A final round of telephone calls was made in September 2005.

Laboratory methods

The clinical and subclinical mastitis samples were analysed by standard laboratory methods for the microbiological analysis of milk (National Mastitis Council 1999). A volume of 10 µl secretion was inoculated on to blood agar and Edward's agar; 100 µl of secretion was inoculated on to MacConkey's agar to enhance the detection of Enterobacteriaceae. The plates were incubated at 37°C and read after 24, 48 and 72 hours. Organisms were identified and quantified using standard laboratory techniques (National Mastitis Council 1999). If a pathogen was isolated it was recorded as an infection regardless of the number of colony forming units identified. Samples were defined as contaminated when more than three colony types were identified.

Calculation of incidence of clinical mastitis

The incidence of clinical mastitis for each herd was calculated by two methods. First, the time in days between the first (t_1) and the fifth (t_5) clinical sample was used to derive the incidence, assuming that this was the time during which four cases had occurred. The incidence was calculated as follows:

$$\text{Cases per 100 cows per year} = (4 \times 365 \times 100) / ([t_1 - t_5] \times \text{herd size})$$

Secondly, the incidence was calculated from the farmers' questionnaires. The farmers were asked to report, from their

TABLE 1: Summary of UK literature reporting the incidence of clinical mastitis between 1980 and 2001

Date of study	Type of study	Sample population	Incidence (cases per 100 cows per year)	Reference
1980-1982	Prospective	Selected sample from 1000 volunteers: 273 herds (1980); 209 herds (1981); 159 herds (1982)	55 (1980); 50 (1981); 41 (1982)	Wilesmith and others (1986)
1994-1996	Retrospective	144 'DAISY' recording herds	43 (range 8-113)	Kossaibati and others (1998)
1995-1996	Retrospective	516 herds with 'on farm' consultants	Cubicles, 30 (range 0-163); Straw yards, 38 (range 0-231)	Berry (1998)
1997	Retrospective	1771 low BMSCC herds	23	Peeler and others (2000)
1997-1998	Prospective	6 herds in Somerset, BMSCC <250,000 cells/ml	42 (range 14-75)	Bradley and Green (2001)
1998	Prospective	482 herds, BMSCC <150,000 cells/ml	37	Peeler and others (2002)
1999-2001	Prospective	60 herds in Devon	17 (range 1-73)	Milne and others (2002)

DAISY Dairy Information System, BMSCC Bulk milk somatic cell count

TABLE 2: Levels of compliance with the protocol by farmers enrolled in the survey of mastitis in dairy cows

Level of compliance	Bacteriology (clinical)	Bacteriology (subclinical)	Used in incidence calculations
Complete set of samples	97	94	90
Spoilt/incomplete set of samples	4	7	0
No return	13	13	0
Sold herd before receiving kit	5	5	0
Sold herd after receiving kit	6	6	0
Total	125	125	90

own records, the number of cases of clinical mastitis and the mean herd size in the year before they had received the sampling kit. An estimate was then made of the number of clinical cases per 100 cows per year.

RESULTS

The compliance of the original 125 farmers and the data they provided are summarised in Table 2; the location of the farms is shown in Fig 1. Ninety-seven farmers provided clinical samples and 94 provided subclinical samples. Some of the herds were excluded from the calculations of incidence because sampling was delayed or interrupted, and 90 herds remained in the analysis. Among the 13 farms that failed to return any samples, the most common reason given was lack of regular milking staff. The mean herd size was 136 cows (median 132, range 28 to 400 cows) and the mean rolling annual BMSCC (weighted means from individual cow recordings) was 194,000 cells/ml (median 179,000, range 83,000 to 464,000 cells/ml).

Incidence of clinical mastitis

Samples were collected from cases of clinical mastitis between February 26, 2004 and October 6, 2005. The mean herd incidence of clinical mastitis, calculated from the time between the first and fifth cases, was 71 cases per 100 cows per year (median 41, range three to 849 cases). The herd with an incidence of 849 cases per 100 cows per year had two cows affected on successive days, the second with all four quarters affected. When this herd was excluded from the analysis, the mean was reduced to 65 cases per 100 cows per year (median 40 cases). The incidence of clinical mastitis for these herds are shown in Fig 2. There was no relationship

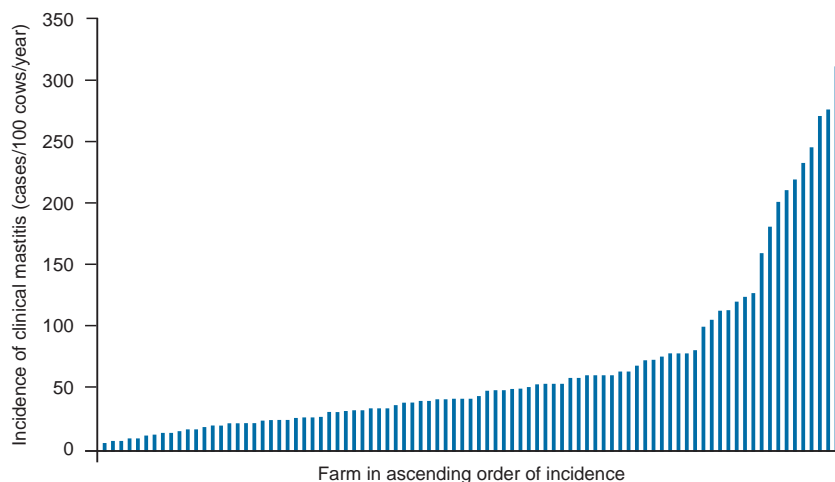


FIG 2: Incidence of clinical mastitis calculated from five milk samples in 89 herds, in order of increasing incidence; one farm (not shown) had an incidence of 849 cases per 100 cows per year

between the herd size and the incidence of clinical mastitis (Fig 3).

Historical data of clinical mastitis cases in the previous 12 months were provided by 84 farmers. From these data, the mean number of cases per 100 cows per year was calculated to be 47, (median 39, range nine to 162 cases). The mean was slightly higher for the 49 farmers who reported using prospectively collected records (mean 50, median 42, range 11 to 162 cases) than for the 35 farmers who provided an approximation by using other methods (mean 42, median 30, range nine to 146 cases).

Bacteriological results

Clinical samples A total of 480 clinical samples were cultured (Tables 3, 4), which represents less than five samples per farm, because one farm returned only one sample and one returned only four samples. Pure cultures of *S uberis* and *E coli* accounted for 23.5 per cent and 19.8 per cent of the samples, respectively, and 26.5 per cent produced no growth. *Staphylococcus aureus* was isolated in pure culture from 3.3 per cent of the samples and coagulase-positive staphylococci were grown in pure culture from 4.6 per cent of the samples. *S agalactiae* was not isolated.

A pure culture of coagulase-negative staphylococci was identified in 8.1 per cent of the samples and *Corynebacterium* species in 3.5 per cent of the samples. A total of 20 samples had a mixed culture of two organisms (Table 4). No sam-

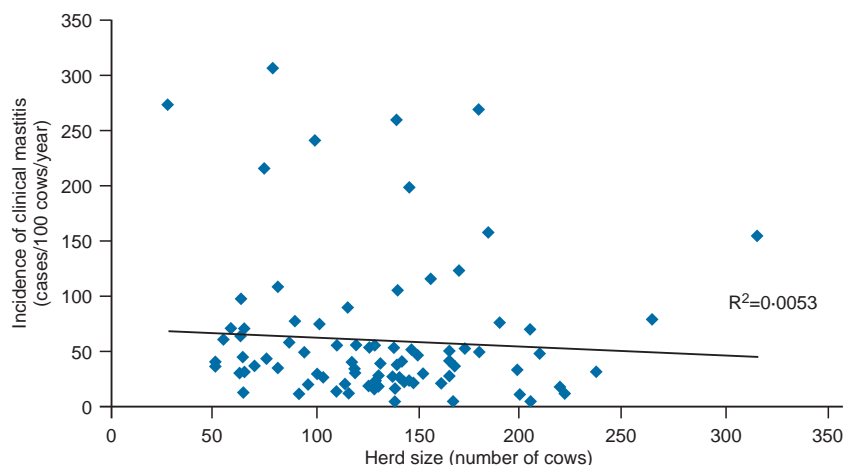


FIG 3: Relationship between the incidence of clinical mastitis and herd size

TABLE 3: Pathogens isolated by bacteriological culture from the cases of clinical mastitis, in order of frequency

Culture result	Number (%)
Major pathogens	
<i>Streptococcus uberis</i>	113 (23.5)
<i>Escherichia coli</i>	95 (19.8)
Coagulase-positive staphylococci	22 (4.6)
<i>Staphylococcus aureus</i>	16 (3.3)
<i>Streptococcus dysgalactiae</i>	7 (1.5)
<i>Bacillus</i> species	7 (1.5)
Yeast species	5 (1.0)
<i>Proteus</i> species	4 (0.8)
Streptococci (other)	2 (0.4)
<i>Enterobacter</i> species	1 (0.2)
<i>Klebsiella</i> species	1 (0.2)
<i>Lactococcus</i> species	1 (0.2)
<i>Pasteurella</i> species	1 (0.2)
<i>Serratia</i> species	1 (0.2)
<i>Arcanobacterium pyogenes</i>	1 (0.2)
All Enterobacteriaceae	102 (21.2)
Total staphylococci (not CNS)	38 (7.9)
Minor pathogens	
CNS	39 (8.1)
<i>Corynebacterium</i> species	17 (3.5)
Mixed aetiology	20 (4.2)
No growth	127 (26.5)
Total	480

CNS Coagulase-negative staphylococci

ples were contaminated. The distribution of pathogens isolated from samples with a positive culture is shown in Fig 4. The proportions of pathogens isolated between April and September 2004 and between October 2004 and March 2005 are shown in Fig 5. For all the pathogens, more cases were reported to be first cases during a lactation than repeat cases in the same quarter (Table 5).

Subclinical samples Ninety-four farmers returned subclinical mastitis samples, providing a total of 464 cases (Tables 6, 7). The most common isolates were coagulase-negative staphylococci (14.9 per cent) followed by *S uberis* (13.8 per cent) and *Corynebacterium* species (9.9 per cent). *S aureus* or coagulase-positive staphylococci accounted for 10 per cent of the samples and 38.6 per cent produced no growth. The distribution of pathogens from the samples with a positive culture is shown in Fig 6.

DISCUSSION

This is the first national survey of mastitis in dairy cows in England and Wales for over 20 years. It is difficult in sur-

TABLE 4: Cultures with more than one pathogen isolated from cases of clinical mastitis, in order of frequency

Mixed cultures	Number
<i>Escherichia coli</i> , <i>Streptococcus uberis</i>	4
<i>Aerococcus</i> species, <i>Bacillus</i> species	2
<i>Bacillus</i> species, <i>E coli</i>	2
<i>Bacillus</i> species, <i>S uberis</i>	2
Coagulase-positive staphylococci, <i>S uberis</i>	2
<i>Bacillus</i> species, <i>Pantoea</i> species	1
<i>Bacillus</i> species, streptococci (other)	1
<i>Corynebacterium</i> species, CNS	1
<i>E coli</i> , <i>Proteus</i> species	1
<i>E coli</i> , streptococci (other)	1
<i>Enterobacter</i> species, <i>S uberis</i>	1
<i>Staphylococcus aureus</i> , <i>S uberis</i>	1
<i>S aureus</i> , yeast species	1
Total	20

CNS Coagulase-negative staphylococci

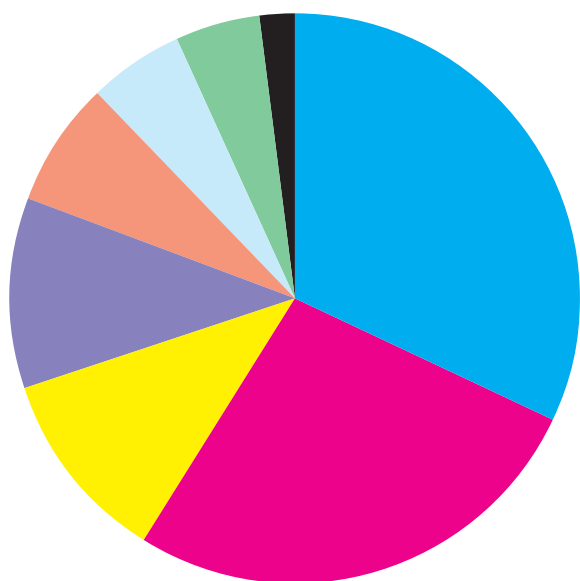


FIG 4: Percentages of bacterial species cultured from the cases of clinical mastitis in which a pathogen was identified

- *Streptococcus uberis* (32.0%)
- *Escherichia coli* (26.9%)
- Coagulase-negative staphylococci (11.0%)
- Coagulase-positive staphylococci, *Staphylococcus aureus* (10.8%)
- Other (7.1%)
- Mixed (5.4%)
- *Corynebacterium* species (4.8%)
- *Streptococcus dysgalactiae* (2.0%)

FIG 4: Percentages of bacterial species cultured from the cases of clinical mastitis in which a pathogen was identified

veys of this nature to be sure that the results truly represent the target population. The herds were selected from herds recording with NMR and the incidence of mastitis in these herds may not have been representative of that in the whole of England and Wales. However, because the estimation of the SCC of individual cows is a central activity of milk recording, it is unlikely that these herds were less motivated than the population 'average' in terms of mastitis control. The distribution (Fig 1) and size of the herds were consistent with the overall distribution of NMR herds suggesting that they were representative of NMR herds. Another problem is the potential selection bias resulting from farmers having to volunteer to participate in the survey. It is possible that volunteering was related to the farmers' perceptions of mastitis or its incidence in the herd, and that herds with either a high or low incidence were more likely to have participated. However, this possibility is difficult to assess because it was not possible to obtain accurate data from the farmers who declined to participate and a direct comparison with these

TABLE 5: Percentages of the main mastitis pathogens isolated from cases of clinical mastitis that were categorised as either first or repeat cases in a quarter in a lactation

Culture result	Number	First case	Repeat case
CPS, <i>Staphylococcus aureus</i>	38	76.3	23.7
<i>Escherichia coli</i>	95	83.2	16.8
<i>Streptococcus uberis</i>	113	69.0	31.0
<i>Streptococcus dysgalactiae</i>	7	57.1	42.9
CNS	39	74.4	25.6
<i>Corynebacterium</i> species	17	82.4	17.6
No growth	127	76.4	23.6
Total	436	75.7	24.3

CPS Coagulase-positive staphylococci, CNS Coagulase-negative staphylococci

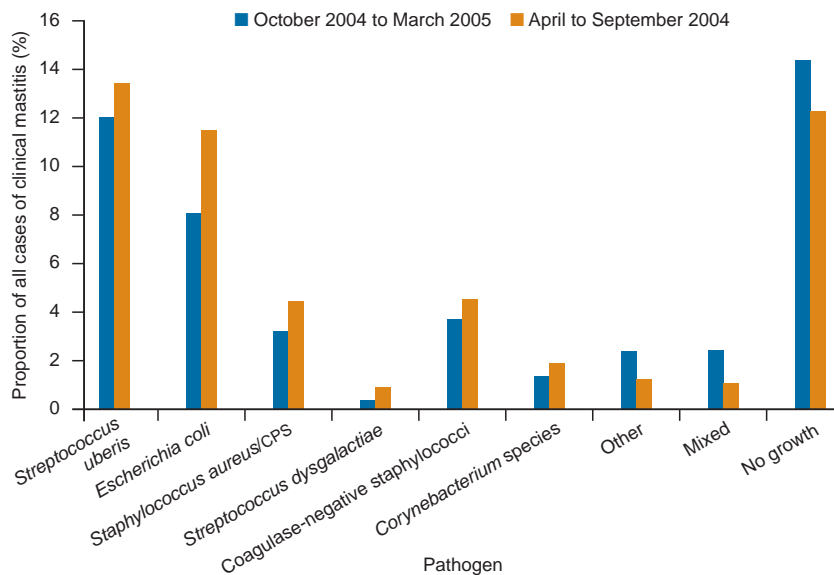


FIG 5: Main pathogens associated with 376 cases of clinical mastitis reported either between April and September 2004 or October 2004 and March 2005. CPS Coagulase-positive staphylococci

herds cannot be made. Previous studies of the incidence and prevalence of mastitis have all encountered the possibility of volunteer selection bias (Wilesmith and others 1986, Berry 1998, Kossaibati and others 1998, Bradley and Green 2001, Peeler and others 2002, Milne and others 2002) and so in this sense the studies are comparable. The method used to calculate the incidence of clinical mastitis from prospectively collected survey samples could have produced an over-estimation if the mastitis cases had been heavily clustered within herds. Alternatively, farmers could have omitted to sample or report some cases within the five samples, resulting in an underestimation of the incidence. The use of historic farm records is likely to have underestimated the incidence because under-recording is more likely than over-recording. As a result, a pragmatic estimate for the current mean incidence of clinical mastitis for the population appears to be between 47 and 65 cases per 100 cows per year,

TABLE 6: Pathogens isolated from the cases of subclinical mastitis, in order of frequency

Culture result	Number (%)
Major pathogens	
<i>Streptococcus uberis</i>	64 (13.8)
<i>Staphylococcus aureus</i>	24 (5.2)
Coagulase-positive staphylococci	22 (4.7)
<i>Escherichia coli</i>	14 (3.0)
<i>Bacillus</i> species	9 (1.9)
Enterococci	8 (1.7)
Yeast species	7 (1.5)
<i>Serratia</i> species	3 (0.7)
<i>Proteus</i> species	2 (0.4)
<i>Streptococcus dysgalactiae</i>	2 (0.4)
<i>Aspergillus</i> species	1 (0.2)
<i>Mucor</i> species	1 (0.2)
Streptococci (other)	1 (0.2)
All Enterobacteriaceae	19 (4.1)
Total staphylococci (not CNS)	46 (9.9)
Minor pathogens	
CNS	69 (14.9)
<i>Corynebacterium</i> species	46 (9.9)
Mixed aetiology	11 (2.2)
No growth	179 (38.6)
Contaminated	1 (0.2)
Total	464 (100)

CNS Coagulase-negative staphylococci

TABLE 7: Cultures with more than one pathogen isolated from cases of subclinical mastitis, in order of frequency

Mixed cultures	Number
<i>Corynebacterium</i> species, CNS	2
<i>Staphylococcus aureus</i> , <i>Streptococcus uberis</i>	2
<i>Serratia</i> species, <i>S. uberis</i>	2
<i>Bacillus</i> species, <i>Escherichia coli</i>	1
Coagulase-positive staphylococci, <i>S. uberis</i>	1
<i>E. coli</i> , <i>S. uberis</i>	1
<i>E. coli</i> , <i>S. aureus</i> , <i>S. uberis</i>	1
Total	11

CNS Coagulase-negative staphylococci

apparently higher than previously thought. This is certainly a cause for concern and suggests that recently reported estimates (Kossaibati and others 1998, Berry 1998, Bradley and Green 2001, Milne and others 2002) may not represent the national picture. There was no relationship between the size of the herds and the incidence of clinical mastitis, although mastitis is often considered to be more of a problem in larger herds.

S. uberis and *E. coli* were the predominant pathogens isolated from the clinical mastitis cases and *S. uberis* from the subclinical cases, confirming the importance of these 'environmental' pathogens, and suggesting that more research and education about their epidemiological features and control methods is required. These bacterial species remained important causes of clinical mastitis during the grazing period as well as during the winter housing period (Fig 5), suggesting that they may not be simply a result of poor environmental hygiene in winter. The contagious pathogens were generally of less importance, although for individual herds they were a potential threat. The 'minor' pathogens coagulase-negative staphylococci and *Corynebacterium* species were isolated in pure culture from a number of cases of clinical and subclinical mastitis. Their importance is difficult to assess – they

could have been a cause of mastitis or they could have been an incidental finding in a quarter from which no true causal pathogen was isolated. These species warrant further investigation.

The presence of a Gram-negative pathogen in 8.5 per cent (24 of 284) of the subclinical samples in which a diagnosis was made, emphasises the importance of either making a definitive diagnosis (of Gram-positive aetiology) or using broad-spectrum therapy for the intramammary treatment of cows with high SCC.

The prevalences of the bacterial species in the cases of clinical and subclinical mastitis in this survey were similar to those reported from laboratory diagnoses in the Veterinary Investigation Surveillance Report (VIDA) (VLA 1997). This suggests that despite some selection bias due to samples being more likely to be collected from 'outbreaks', the VIDA results are likely to provide a useful method of surveillance of the aetiology, but not the incidence of mastitis.

Approximately a quarter of the clinical cases were recorded as a recurrence, that is, the second or subsequent case, in a quarter in that lactation. This suggests that the success of treatments could be improved and, together with obtaining a better understanding of the mechanisms of persistence, this should be the subject of further research.

The results of this survey suggest that incidence of clinical mastitis in dairy herds in England and Wales is probably between 47 and 65 cases per 100 cows per year, higher than previously thought. The major mastitis pathogens most commonly isolated from the clinical cases were *S. uberis* and *E. coli* and those most commonly isolated from cows with high SCCs were *S. uberis*, coagulase-positive staphylococci and *S. aureus*.

ACKNOWLEDGEMENTS

The authors thank the Milk Development Council for funding this research and Mr James Booth for help and support throughout the project. The contribution of NMR to the recruitment and delivery of sampling kits is gratefully acknowledged. The authors also thank the farmers for their participation and cooperation.

References

- BERRY, E. J. (1998) Mastitis incidence in straw yards and cubicles. *Veterinary Record* **142**, 517-518
- BOOTH, J. M. (1997) Progress in Mastitis Control – an Evolving Problem. Proceedings of the British Mastitis Conference. Stoneleigh, UK, October 8, 1997. pp 3-9
- BRADLEY, A. J. (2002) Bovine mastitis: an evolving disease. *Veterinary Journal* **164**, 116-128
- BRADLEY, A. J. & GREEN, M. J. (2001) Aetiology of clinical mastitis in six Somerset dairy herds. *Veterinary Record* **148**, 683-686
- KINGWILL, R. G., NEAVE, F. K., DODD, F. H., GRIFFIN, T. K., WESTGARTH, D. R. & WILSON, C. D. (1970) The effect of a mastitis control system on levels of sub-clinical and clinical mastitis in two years. *Veterinary Record* **87**, 94-100
- KOSSAIBATI, M. A., HOVI, M. & ESSLEMONT, R. J. (1998) Incidence of clinical mastitis in dairy herds in England. *Veterinary Record* **143**, 649-653
- MILNE, M. H., BARRETT, D. C., FITZPATRICK, J. L. & BIGGS, A. M. (2002) Prevalence and aetiology of clinical mastitis on dairy farms in Devon. *Veterinary Record* **151**, 241-243
- NATIONAL MASTITIS COUNCIL (1999) Laboratory Handbook on Bovine Mastitis. Madison, WI, National Mastitis Council
- NMR (2006) NMR. www.nmr.co.uk. Accessed December 20, 2006
- PEELER, E. J., GREEN, M. J., FITZPATRICK, J. L. & GREEN, L. E. (2002) Study of clinical mastitis in British dairy herds with bulk milk somatic cell counts less than 150,000 cells/ml. *Veterinary Record* **151**, 170-176
- PEELER, E. J., GREEN, M. J., FITZPATRICK, J. L., MORGAN, K. L. & GREEN, L. E. (2000) Risk factors associated with clinical mastitis in low somatic cell count British dairy herds. *Journal of Dairy Science* **83**, 2464-2472
- VLA (1997) Veterinary Investigation Survey Report 1997 and 1990 – 1997.

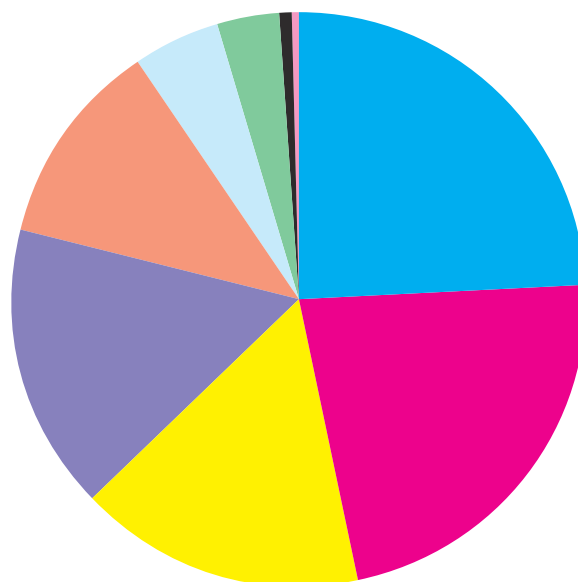


FIG 6: Percentages of bacterial species cultured from the cases of subclinical mastitis in which a pathogen was identified

Weybridge, Central Veterinary Laboratory

WILESMITH, J. W., FRANCIS, P. G. & WILSON, C. D. (1986) Incidence of clinical mastitis in a cohort of British dairy herds. *Veterinary Record* **118**, 199-204

WILSON, C. D. & KINGWILL, R. G. (1975) International Dairy Federation Annual Bulletin **85**, 422-438

WILSON, C. D. & RICHARDS, M. S. (1980) A survey of mastitis in the British dairy herd. *Veterinary Record* **106**, 431-435