infected quarters were odourless and varied from a brown coloured milk to a purulent discharge. In longer established cases, gradual separation of milk samples into a sediment and a serum-like supernatant was the most consistently observed feature. All cows were intensively treated with broad-spectrum antibiotics, with minimal effect on the clinical signs.

No pathogenic bacteria or yeast were isolated from initial milk samples. *M bovis* was detected by a monoclonal antibody-based sandwich ELISA (Ball and others 1994), and subsequently cultured from milk samples collected from the mastitic cows three weeks after the outbreak began. One of the imported Montbelliardes was also diagnosed as having *M bovis* mastitis shortly after the first seven cases. These eight cows and two cows suspected on clinical signs were segregated and milked after the rest of the herd.

Nine weeks after the outbreak began, quarter milk samples were collected aseptically from all 44 lactating cows. *M bovis* was detected in milk samples from 10 clinically infected quarters and from a further 11 subclinically infected quarters all from the eight previously identified cows. Milk SCCs ranged from 1,752,000 to 21,381,000 cells/ml in samples collected from quarters with subclinical *M bovis* infection.

Four cows developed infection in all four quarters and were dried off. Three cows resumed production of apparently normal milk but subclinical *M bovis* mastitis was later observed in two of these cows. Another infected cow continued to produce a sediment in mammary secretions from two infected quarters.

Segregation of affected cows, which included milking them last, was followed by a reduced incidence of *M bovis* mastitis in this herd. During the following three months only four new cases were identified on this farm and these included the two suspect cows already placed in the isolated group.

Four months after the outbreak the 12 cows which had *M bovis* mastitis were culled. Pathological findings included interstitial fibrosis and enlargement of supramammary lymph nodes. In one mammary gland, the milk ducts were filled with seropurulent exudate from which *M bovis* was isolated and focal areas of necrosis were observed.

Clinical signs observed in the present cases of *M bovis* mastitis were characteristic of cases previously described. These include the rapid spread to other quarters, the separation of milk samples into sediment and supernatant, and the lack of response to antibiotics (Davies and Boughton 1976). In this herd subclinical *M bovis* mastitis was found only in cows clinically affected previously, unlike other outbreaks where *M bovis* mammary infection was reported in some cows without any previous record of clinical mastitis (Bicknell and others 1983).

Other clinical problems associated with *M bovis* also developed in the herd when the mastitis was first observed in the homebred cows. Respiratory disease occurred in the calves and *M bovis* was isolated from nasal swabs collected from them. They all recovered, despite initial failure to respond to treatment. *M bovis* was also isolated from synovial fluid samples collected from the enlarged joints of one calf and one cow. Milk samples collected from the cow with arthritis were consistently negative for *M bovis*. These calves and cow remained in the herd. Purulent nasal discharges were observed intermittently in some of the adult cows.

After the first 12 cows previously diagnosed with *M bovis* mastitis were removed from the herd, only one further case of this mastitis was identified in the subsequent three months. This cow was also culled. During the following year all cows remained free of clinical signs of *M bovis* mastitis and *M bovis* was not isolated from milk samples collected from each cow in the herd in October 1996 and again in May 1997. Eradication of *M bovis* mastitis from a herd is possible if a programme of identification, segregation and culling is followed (Bicknell and others 1983). The antigen-capture ELISA made it possible to screen a large number of samples for *M bovis* and thus facilitated the elimination of *M bovis* mastitis from this herd.

Acknowledgements. – The authors gratefully acknowledge the contributions of Paul Brennan, Jim Bradley, David Kelly, Dr John Egan, the staff of the Veterinary Laboratory Service and the Veterinary Sciences Division, Stormont, and the veterinary practice.

References

BALL, H. J., MACKIE, D. P., FINLAY, D., GUNN, J., McFARLAND, E. A., REILLY, G. A. C. & POLLOCK, D. (1994) *Irish Veterinary Journal* **47**, 45
BICKNELL, S. R., GUNNING, R. F., JACKSON, G., BOUGHTON, E. & WILSON, C. D. (1983) *Veterinary Record* **112**, 294
DAVIES, A. B. & BOUGHTON, E. (1976) *Veterinary Record* **99**, 322
DOHERTY, M. L., McELROY, M. C., MARKEY, B. K., CARTER, M. E. & BALL, H. J. (1994) *Veterinary Record* **135**, 259
REILLY, G. A. C., BALL, H. J., CASSIDY, J. P. & BRYSON, T. D. G. (1993) *Veterinary Record* **133**, 550

Mastitis incidence in straw yards and cubicles

E. A. Berry

Veterinary Record (1998) **142,** 517-518

MASTITIS in dairy cows is a serious welfare issue and causes major economic losses to the British dairy industry (Esselemont and Spincer 1993). Management practices such as housing, hygiene, milking and drying off procedures are important risk factors for the occurrence of mastitis (Bartlett and others 1992, Faye and others 1994).

Recently, there has been an increased interest in the welfare issues surrounding the use of straw yards, especially in relation to lameness. The relationship between lameness and the time spent lying down in cubicles and straw yards has been studied in dairy herds by Singh and others (1993, 1994) comparing normal and lame cows under both types of system. This short communication reports the results of a survey which was carried out to investigate the effects of the type of housing on the incidence of clinical mastitis cases during the winter housing period.

This survey was carried out in England and Wales between January 1995 and May 1996 and involved 10 consultants (working for the Axient Mastitis Service). For each herd the data collected included herd size (number of milking cows), type of housing and clinical mastitis incidence for the year as a whole and for the winter housing period specifically. Only those herds keeping accurate clinical records were included. Each herd was monitored at regular intervals (ranging from monthly to three monthly visits) by a consultant and 12 months' continuous data had to be recorded, with herds starting on the survey between January and May 1995. Most herds were monitored for more than 12 months but for the purpose of this survey only 12 months data were used. The definition of a case of mastitis was any cow with abnormal milk and/or visible or palpable changes in a quarter. A new case was defined as a quarter showing changes for the first time in a lactation or in the 14 days since the last day of treatment. Clinical cases were recorded by the consultants from the herds' clinical records. About 300 herds were excluded due to incomplete or inaccurate record keeping or for leaving the study after less than 12 months.

The clinical incidence was calculated as cases per 100 cows for the whole year and for the relevant housing period specifically. None of the herds surveyed housed cows continuously and clinical records showed that they all used dry cow therapy on all cows.

E. A. Berry, Axient, Westmere Drive, Crewe CW1 1ZD

TABLE 1: Mean (se) and range of clinical mastitis cases for cubicles and yards

Number of herds	Type of housing	Mean herd size	Mean (se) clinical incidence/100 cows/year	Range	Mean (se) clinical incidence/100 cows/winter housing period	Range
355	Cubicles	110	33*(1·1)	0-163	19*(0·7)	0-52
161	Yards	101	38*(1·6)	2-231	24*(1·1)	0-107

* Values are significantly different from other values in the same row, P<0·05

For herds which reported low incidences of mastitis confirmation by tube usage and spot checks at milking time was necessary and any herds not felt to be reporting cases accurately were omitted from the survey. In total 516 herds were included in the study.

The results for mastitis incidence in cubicles and yards were analysed by Student's *t* test and are shown in Table 1.

From previous and ongoing surveys on farms visited by the Axient Mastitis Service the mean clinical incidence of cases per 100 cows per year is around 30. For those herds with a clinical incidence over 30 cases per 100 cows per year the consultants cited the following reasons as to why the herds may have a higher incidence: poor housing management (ranging from not bedding up or scraping out frequently enough, to using insufficient or wet bedding as seen on their visits), overstocking (not having equal or more cubicles to cows or less than 7·2 m^2 lying space per cow), poor building construction, failure to follow the 'five point plan' (milking machine problems, teat disinfection or culling policy) and poor dry cow management. As all herds used dry cow therapy, the term 'dry cow management' applied to environmental hygiene either at pasture or during the housing for the dry cows. In this survey 516 herds in total were monitored, and the mean winter clinical mastitis incidence was 20 cases per 100 cows which included herds using both straw yards and cubicles, and herds not housing cows over the winter.

For herds using cubicles 36 per cent had over 20 cases per 100 cows per housing period and for herds using yards 52 per cent had over 20 cases per 100 cows per housing period. Possible reasons for this are listed in Table 2. Twelve herds (3 per cent) using cubicles and 10 herds (6 per cent) using yards were cited as having poor housing management and had over 20 cases per 100 cows during the winter but less than 30 cases per 100 cows during a 12 month period.

Arithmetic means of the monthly geometric bulk milk counts were also calculated for the recording periods (Table 3). All consultants reported that over 80 per cent of herds would leave out milk from the bulk tank in order to avoid penalties on bulk milk cell counts. These penalties were incurred for bulk cell counts over 150,000 to 200,000 cells/ml depending on the buyer. The national bulk milk average for National Milk Record herds is 20,000 to 30,000 cells/ml higher than the averages reported by the dairies (M. Squires, personal communication).

Singh and others (1994) concluded that cows lay down for longer in straw yards than in cubicles and that this may have a role in the prevention of lameness. However, lameness is not the only welfare issue to be considered in housing management decisions. In this survey, clinical mastitis incidence was significantly lower in cubicles than in yards (P<0·05). In addition, 36 per cent of herds with cubicles for housing had over 20 cases per 100 cows per housing period compared to 52 per cent herds with yards for housing. As cows lie down for significantly longer periods in straw yards than cubicles (Singh and others 1993) it is possible that there are increased periods of contact with environmental pathogens. If this is indeed the case, the standards of hygiene in strawed yards will have increased importance. Seven herds in the survey had less than five cases per 100 cows per annum; all consultants reported that for these herds the standard of management was exceptionally high, although the number of herds is not high enough to carry out any significant analysis. In both types of housing, the most important reason cited for a high clinical incidence during the winter housing period, was poor housing management and it is concluded that extra attention to housing hygiene for lactating and dry cows would be beneficial to the welfare of cows with respect to mastitis.

TABLE 2: Effect of a variety of management factors on the higher than average incidence of mastitis (more than 20 cases/100 cows/year) seen in some of the surveyed herds

Factor	Percentage of herds in which the factor may have had a role: Cubicles	Yards
Poor housing management	52	54
Poor building construction	15	7
Overstocking	4	17
Failure to follow five point plan	21	8
Dry cow management	8	14

TABLE 3: Arithmetic means of three-month geometric mean bulk milk cell counts for the recording period (n = number of herds)

Clinical incidence (cases/100 cows/year)	Mean bulk milk cell count (cells/ml): Cubicles	Yards
<5	106,000 (n = 4)	110,000 (n = 3)
5-30	200,000 (n = 201)	214,000 (n = 71)
>30	219,000 (n = 150)	198,000 (n = 87)

References

BARTLETT, P. C., MILLER, G. Y., LANCE, S. E. & HEIDER, L. E. (1992) *Preventive Veterinary Medicine* **14,** 195

ESSELEMONT, R. J. & SPINCER, I. (1993) DAISY Information System Rep. No 2, University of Reading

FAYE, B., DOM, N., LECURRET, F., BAMOUIN, J. & CHASSAGNE, M. (1994) *Veterinary Research* **25,** 213

SINGH, S. S., WARD, W. R., HUGHES, J. W., LAUTENBACH, K. & MURRAY, R. D. (1994) *Veterinary Record* **135,** 251

SINGH, S. S., WARD, W. R., LAUTENBACH, K. & MURRAY, R. D. (1993) *Veterinary Record* **133,** 204

Outbreak of tetanus in lambs

M. R. Aslani, T. T. Bazargani, A. A. Ashkar, A. R. Movasaghi, A. Raoofi, N. Atiabi

Veterinary Record (1998) **142,** 518-519

TETANUS is an infectious disease caused by the toxins of *Clostridium tetani* and characterised by increasing muscular rigidity and death in affected animals. This condition occurs in all farm animals, mainly as sporadic cases, although outbreaks are occasionally observed in young cattle, young pigs and lambs (Herd and Riches 1964, Ramsay 1973, Radostits and others 1994). Infection occurs as a result of contamination of wounds or enclosed cavities by the spores of *C tetani*. In anaerobic conditions these spores convert to the vegetative form which is capable of producing toxins (Timoney and others 1988). Tetanus in sheep mostly occurs due to infections following docking, shearing, castration and vaccination or injection of pharmaceuticals, especially anthelmintics (Timoney and others 1988, Radostits and others 1994).

M. R. Aslani, Department of Clinical Studies, **A. A. Ashkar, A. R. Movasaghi,** Department of Pathobiology, School of Veterinary Medicine, Ferdowsi University of Mashhad, PO Box 91775-1793, Mashhad, Iran **T. T. Bazargani, A. Raoofi, N. Atiabi,** Department of Clinical Studies, Faculty of Veterinary Medicine, University of Tehran, PO Box 1455-6453, Tehran, Iran