

BRITISH MASTITIS CONFERENCE 1989

**THE ENVIRONMENT
AND MASTITIS**

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I N T R O D U C T I O N

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In October 1988, the first British Mastitis Conference was set up to review mastitis control in the United Kingdom and discuss the current research programmes. Delegates' response to the Conference indicated a need to continue this forum on an annual basis and it is clearly becoming a model for the growing co-operation between all sectors of the dairy industry and those involved in research.

Last year's Conference reflected the concern and interest by farmers and researchers in the problems associated with environmental mastitis and the need for more information on current progress and research from overseas.

Therefore the title chosen for the second British Mastitis Conference is "The Environment and Mastitis". It will cover why the cow's environment is so important, what measures can be taken to minimise the risk of mastitis and how problems can be tackled on the farm. It will also look at what is happening in a number of different countries to give a greater understanding of current research on both a national and international level.

WHY ENVIRONMENTAL INFLUENCES ON MASTITIS ARE IMPORTANT

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Summary

This paper categorises the environmental influences on bovine mastitis and identifies the direct and indirect ways in which they operate. It concentrates upon climatic, housing, nutritional and machine milking influences and introduces themes explored by the other papers in the proceedings.

Introduction

Mastitis is an inflammation, usually in response to microbial infection of the udder. The disease process comprises 3 elements - the cow, the pathogen and the environment in which they interact in complex ways (Figure 1).

Although research has generated considerable data on pathogens, immune mechanisms, pathology etc., this information has been of little or no use in the control of the disease. At last years conference the considerable progress made in mastitis control over the last 30 years was reviewed (1). This progress has stemmed from our slight understanding of environmental influences on disease e.g. the ways in which milking or housing influence exposure to pathogens, the incidence of teat lesions or the entry of bacteria to the mammary gland. By modifying the environment in which we place cows or by adapting management practices which impinge upon them we are constantly altering the incidence or pattern of udder disease. A fuller understanding of the interplay between the environment and disease is likely to achieve considerable improvements in the control of udder disease in the short to medium term.

The objective of our conference is to consider

- * what environmental factors feature in mastitis incidence
- * in what ways these factors may operate
- * what recommendations can be made in terms of the practices we employ upon dairy farms

In my introductory paper I shall classify the influences and identify some basic points or principles for further discussion. I shall emphasize the international aspects and differences which can be further developed by the international panel.

Classification of environmental influences

In its broadest sense the environment encompasses climate, geography and topography through to the milking parlour, housing and feeding. In Figure 2 these elements are categorised. For convenience the environmental factors can be considered as an external environment and an internal environment. The external environment is affected by climate, tradition, geography etc. while the internal effects include feeding, housing, milking and management. Generally the internal elements are more important in mastitis and certainly more manipulable in control systems.

Ways in which environmental effects operate on mastitis

The external environment

Climate, tradition and topography

Although the dairy cow is versatile and tolerant extremes of climate do modify lactation and reproduction and, possibly, mastitis (2). While mastitis outbreaks have been attributed to climatic extremes and chilling has been regarded as a contributory factor in mastitis (3;4) experiments under closely controlled conditions have generally failed to show effects (5;6). This is possibly because climate has to interact with other factors to increase mastitis risk. This is a theme that it is central to understanding the effects of environmental influences. It is the sum of stressors that is critical rather than the presence and absence of an individual stressor. Climatic stressors and many other stressors such as noise, altered herd hierarchy etc. operate on disease in subtle ways e.g. by stimulating adreno-cortical activity that leads to hormonal responses which in turn jeopardize immune function. In consequence an infection which has been dormant for some time suddenly develops into clinical disease. Most of the pioneering work in this field has been performed in rodents and small laboratory animals (7;8) and it is only relatively recently that effects on farm species have been recognized.

The susceptibility to stressors interacts with genetics. An example is given in Figure 3 which shows that Brahmin cows are much more resistant to heat stress than are Holstein cows (2). As geography and tradition are often the factors which determine distribution of breeds then they also modify national patterns of disease.

Summer mastitis provides a final example of how these external factors may influence mastitis. Summer mastitis incidence is associated with flies, which may transmit or predispose the cow to infection (9). Fly populations are determined by climate, topography, vegetation etc. and consequently incidence of summer mastitis varies markedly annually and between countries.

The internal climate

Housing systems

In general housing cows increases mastitis incidence. The effects are most marked for pathogens such as coliforms and Streptococcus uberis. Housing systems can be classified as tied (cow shed) or loose (Cubicles and yards)

and there are differences in how these systems contribute to mastitis. This will be the subject of other papers. Regardless of the type of system employed housing modifies exposure to pathogenic organisms and increases the frequency of teat damage. The type of litter material employed is important since organic materials (sawdust and straw) increase exposure to coliform or Str. uberis (10;11). There have been extensive studies, particularly in Scandinavia, of the interactions between stall length, dung removal etc. and mastitis incidence and these will not be reviewed here. Interested readers are referred to (12), (13), (14) and (15) for further detail.

In addition to direct effects there are again second order influences. Housing interacts with feeding practices which can have profound effects as described later by Smith (16). In addition tied systems restrict mobility and lead to stress because the inability of the animal to establish its position in the herd social hierarchy causes frustration. However such effects are extremely difficult to quantify experimentally.

Machine milking

In recent years much attention has been paid to the possible ways in which machine milking practices lead to mastitis and this in turn has been translated into hygiene routines and new designs of milking equipment. These will be reviewed by other contributors to the conference but a major question still surrounds the relative contribution of the milking machine to mastitis. The research on vacuum fluctuation has shown that in some herds the "impact mechanism" is a major element in mastitis while in others it is negligible (17). Schmidt-Madsen and Klastrup (18), in a herd study, in Denmark suggested that only a small part of the large variation in mastitis incidence between herds could be related to milking machine effects. It is possible however that our inability to explain the variation may be because we fail to identify, and therefore quantify, the causative mechanisms or because it is the interaction of machine and other stressors that is critical. This is in a way typified by experiments on overmilking. We all accept that overmilking is "bad" yet controlled experiments usually fail to confirm this. If overmilking is combined with inadequate pulsation or excessive vacuum fluctuation the total effect will be much greater.

Nutrition

Some feeding practices are thought to be potentiators of clinical mastitis e.g. overfeeding of concentrates, abrupt changes in feed etc. Additionally feeds high in oestrogens lead to increased clinical mastitis (19). It is likely that the latter effect is due not to increasing new infection rate but rather an exacerbation of existing infections. Johnson & Otterby (20) reported mastitis was associated with excessive energy inputs into the dry period and Radostits (21) found a relationship with calcium/phosphorus ratio. It is perhaps fair to say that, in the UK and USA at least, the significance of nutritional factors has been regarded as small. Recent work in the USA on selenium and vitamin E indicates that this has been mistaken. The evidence behind that will be discussed later by Professor Smith. I suspect that as our knowledge improves the significance of nutritional factors in infectious disease in general will substantially increase.

Mastitis - an adaptation disease?

In 1952 Selye (22) described what he referred to as the general adaptation and alarm syndrome. This proposes that stressors provoke an adreno-cortical driven non-specific demand upon the body which produces various signs and somatic dysfunction. These include ulceration of the gastrointestinal tract and involution of the spleen and thymus. These changes, and others, have been clearly demonstrated in laboratory animals and in some farm species such as poultry and pigs. Every individual has some ability to cope with the effects of accumulating stressors but these thresholds will vary. If the load of stressors exceeds the animals threshold it is postulated that disease will occur. I am sure all of us have experienced examples of this for our own health or for that of our friends and family. The stressors might take many forms within the dairy herd e.g. lactation, milking, climate, feed and nutrition etc. These elements may prove to be critical in determining the widely varying clinical mastitis rates between herds (23). Schmidt-Madsen (24) reported an association between herd mastitis level and the number of stressors operating within the herd. Perhaps this philosophy may start to explain the vexed question of why some herds have little mastitis despite the absence of control measures while others seem to suffer the reverse.

References

1. Booth, J.M. (1988) Proc. British Mastitis Conference, Stoneleigh, pp 2-7.
2. Giesecke, W.H., Van Staden, J.J., Barnard, M.L. & Petzer, I.M. (1988) Tech. Comm. 210. Dept. Agriculture, Republic of S. Africa.
3. Whittlestone, W.G., Kilgour, R., DeLangen, H. & Duirs, G. (1970). J. Milk Food Tech. 33, 217-220.
4. Ewbank, R. (1968). Vet. Record 83, 685-686.
5. Brown, R.W., Thomas, J.L., Cook, H.M., Riley, J.L. & Booth, G.D. (1977). Amer. J. Vet. Res. 38, 181-187.
6. Paape, M.J., Schultze, W.D., Miller, R.H. & Smith, J.W. (1973). J. Dairy Science, 56, 84-91.
7. Cannon, W.B. (1929). Bodily changes in pain, hunger, fear and rage. An account of recent researches into the function of emotional excitement. 2nd. Appleton, New York.
8. Selye, H. (1936). Nature, London 138,32.
9. Bramley, A.J. (1987). In Summer Mastitis. ed. Thomas, Over, Vecht and Nansen,. Martinus Nijhoff pp86-89.
10. Bramley, A.J. (1985). Proc. 24th. Ann. Meeting National Mastitis Council, Las vegas, Nevada pp .
11. Bramley, A.J. (1982). J. Dairy Res 49, 369-373.
12. Bakken, G. (1982). Acta Agric, Scand. 32,23-32.
13. Ekesbo, I. (1966). Acta Agric. Scand. Suppl.15, 74pp.

14. Saloniemi, H. (1980). J. Scientific Agric. Soc. Finland 52,85-184.
15. Dodd, F.H., Higgs, T.M. & Bramley, A.J. (1984). Vet. Rec 114,522-532.
16. Smith, K.L., Hogan, J.S. & Weiss, B.P. (1989). Proc. British Mastitis Conference, Stoneleigh.
17. Griffin, T.K., Mein, G.A., Westgarth, D.R., Neave, F.K., Thompson, W.H. & Maguire, P.D. (1980). J. Dairy Res. 47, 1-9.
18. Schmidt- Madsen, P. & Klastrup, N.O. (1980). Proc. Int. Workshop on Machine Milking and Mastitis. An Foras Taluntais, Moorepark, 137-141.
19. Pouden, W.D., Pratt, A.D., Frank, N.A. & Smith, H.R. (1958). J. Amer. Vet. Med. Assoc. 132, 337-339.
20. Johnson, D.G. & Otterby, D.E. (1981). J. Dairy Science, 64, 290-295.
21. Radostits, O.M. (1961). Can. Vet. J. 2, 201-206.
22. Selye, H. (1952) The story of the adaptation syndrome. Montreal Acta Inc. Med. Publ.
23. Bramley, A.J. (1988). Proc. British Mastitis Conference, Stoneleigh, pp 41-45.
24. Schmidt-Madsen, P. (1978). Proc. 13th. Nordic Vet, Congr., 208-211.

Figure 1. Interaction of pathogen, host and environment.

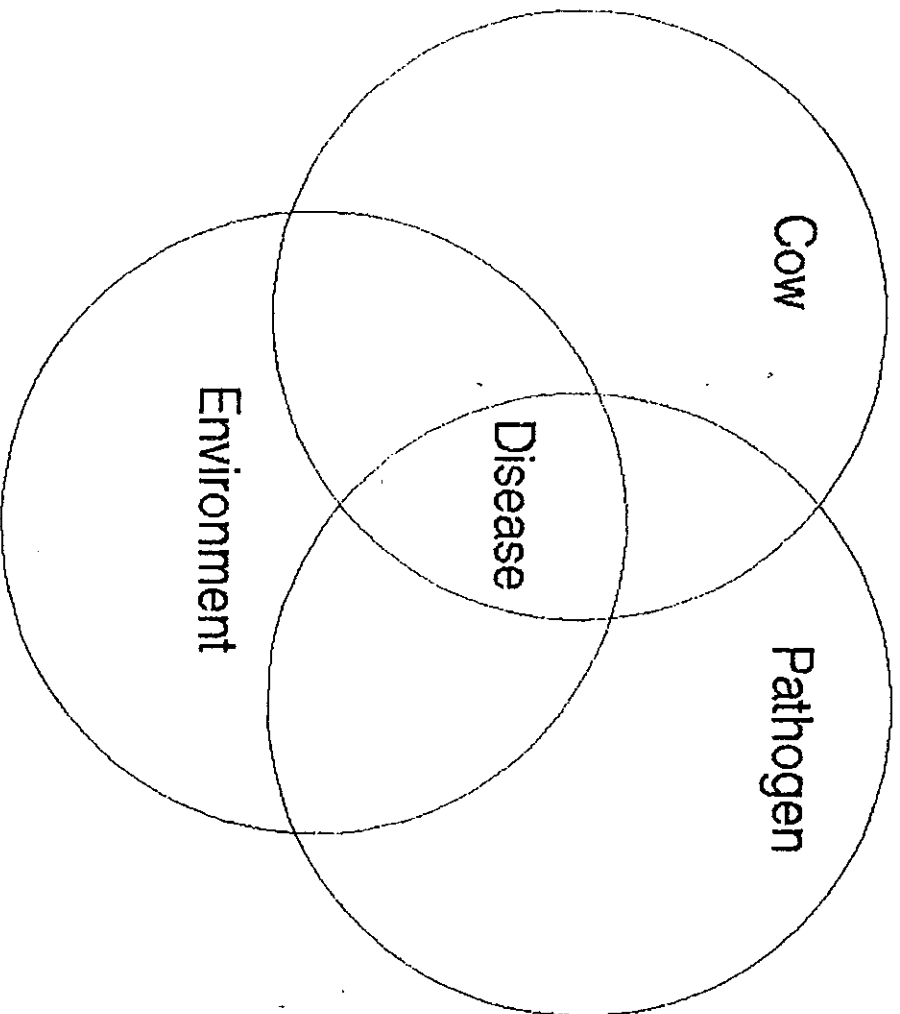


Figure 2. Environmental effects on mastitis

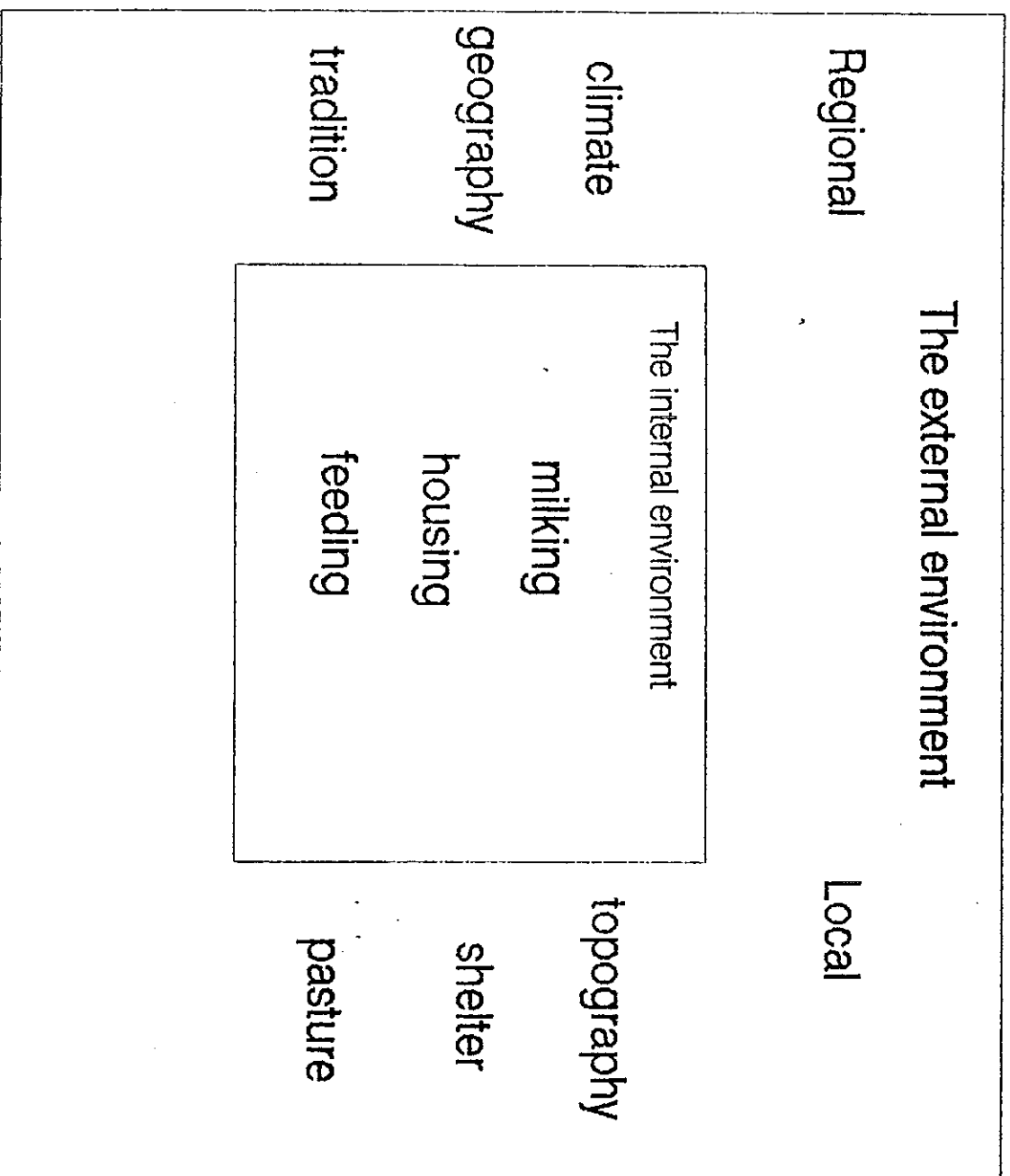
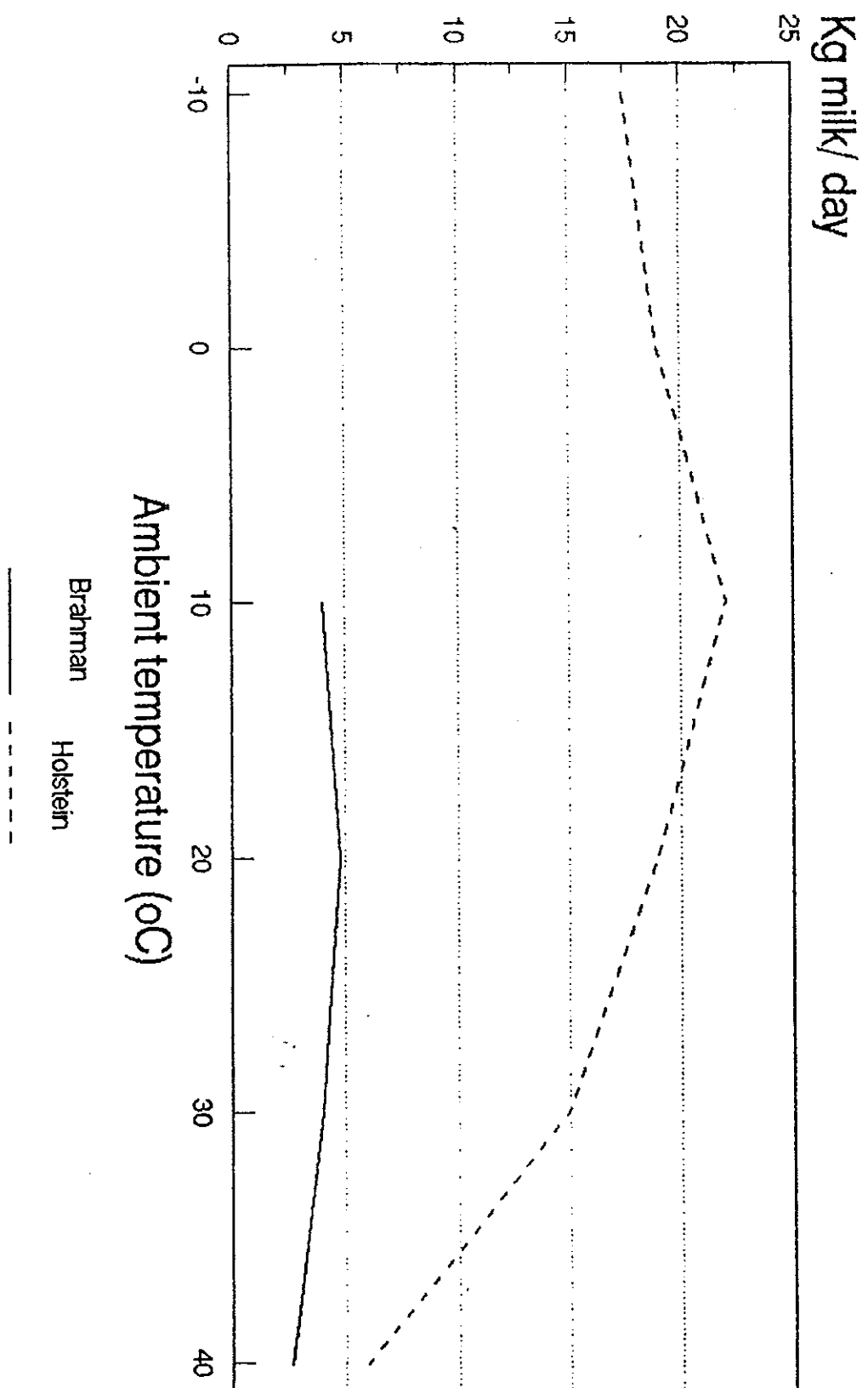


Figure 3. Milk production and temperature
Brahman v Holstein



DESIGN AND MAINTENANCE OF HOUSING SYSTEMS

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Summary

The last 30 years have seen a swing from housing cows in cowsheds to looseyards and particularly cubicles. The widespread adoption of the cubicle is evidence of its success but problems remain. Soiling and injury to cows are still far too common and increases in mastitis caused by environmental pathogens have been attributed to modern housing systems. X

In recent years the needs of the housed cow have been closely examined, new designs of cubicle division have emerged and ideas produced for modifying and adapting existing cubicles.

Introduction

Most herds in England and Wales spend between 5 and 7 months of the year confined to winter quarters. In that time much of the milk is produced and most cases of mastitis occur. It is worth reviewing the effect of housing systems on the health and performance of dairy cows.

Economic forces were responsible for the swing towards loosehousing of dairy cows which began in the 1950s. A reduction in the labour force associated with dairy farming coincided with increasing herd size, and the introduction of parlour milking provided the opportunity for considerable improvements in productivity. Looking back over the last 30 years we have seen that the prophet's predictions of dire consequences associated with such developments were never realised. Progressive improvements to machine milking technology, to milking installations, to cow housing and feeding strategies have resulted in improved herd performances. The evidence has always suggested that economic indices improve as herd size increases. The same can be said of mastitis where udder infection has been seen to decline as herd size increases(1). Yet, we have seen many problems associated with parlour milking and particularly loosehousing.

Many herds have contained (and still contain) far too many dirty cows. Some cows refuse to use cubicles and of those that do, some damage themselves, and most importantly, an increase occurs in problems associated with mastitis caused by environmental pathogens. An almost uncountable

number of papers have over the years been written on the effects of loosehousing on cow behaviour, performance, hygiene, comfort, bedding and management. This paper reviews progress to date and considers the important design features of looseyards and cubicles.

Trends in cow housing

Thirty years ago over 90% of herds were housed in cowsheds. Looseyards were the first alternative but from the early 1960s the numbers of dairy farms with cubicle or kennel housing gradually increased. Table 1 shows the current position where 80% of herds are loosehoused of which the majority are in cubicles. It is however interesting to note the regional variations which reflect herd size and the preponderance of looseyards on the Eastern side of the country.

Table 1. METHODS OF HOUSING COWS (% of herds, England & Wales)

Region	Cowsheds	Looseyards	Cubicles
Northern	27	11	62
Midlands & Western	23	11	66
Eastern	3	56	41
South Western	9	19	72
South Eastern	3	49	48
Wales	18	5	77
Total	20	16	64

Source : ADAS 1989

Looseyards

The basic requirements can be summarised as:

- . Rectangular building
- . Adequate space per cow
- . Concrete feed/loading area
- . Wide access to bedded area
- . Eaves height suitable for machinery
- . Good ventilation
- . Ample supply of bedding

A long rectangular yard is most suitable. It allows a concrete strip for feeding, watering and loafing. A wide access to the bed is provided and a bedded area that avoids unnecessary walking and disturbance to other cows. Collectively these benefits reduce poaching and help maintain a clean dry bed.

Space requirement is influenced by the size of the animals. See Table 2.

Table 2 LOOSEYARD SPACE ALLOWANCES

Body weight	Total space	Bedded area	Feed/loafing area
kg	m	m	m
600	7.5	5.5	2.0
650	8.0	6.0	2.0
700	8.5	6.25	2.25
750	9.0	6.5	2.5

For most Friesian/Holstein herds 6.0 - 6.25 m of bedded area per cow is required with a feeding/loafing area of over 2m. For larger animals a total space of nearer to 9m is needed. (2)

The space provided is important to enable cows to behave in a relatively natural way. Cows prefer to have a space of at least 1m from other cows if not protected by some physical barrier. Arguably, space is of greater importance in a looseyard than in a cubicle house where individual cubicles provide a measure of protection.

Walls should preferably be solid to a minimum of 1.8m above maximum bed height. Floors can be flat or sloped, constructed of porous material or concrete. If concrete is not used, seepage into the surrounding ground may occur with the risk of pollution to underground water sources. Feed areas obviously require concreting and should be as flat as possible.

Good ventilation of the building is vital for maintenance of a dry yard because of the moisture and heat produced by the cows and the bedding. Looseyards require large amounts of bedding. For a 180 day winter, about 1.5 tonnes of straw per cow must be provided.

Problems and opportunities.

The flexibility of the looseyard is often exploited in that too many cows can be introduced. Overcrowding leads to stressful conditions including aggression and disturbance, trodden teats and teat lesions occur and the bedding becomes wet and dirty. Dirty conditions are associated with outbreaks of mastitis caused by environmental organisms. A recent survey (3) implied a slightly higher incidence of mastitis in looseyards than in cubicles. Inadequate ventilation is often a major failing and in addition to the effect on cow health, it contributes substantially to wetness of the bedding.

The quantity of bedding required and the labour requirement for spreading the bedding and cleaning out the yard are costly and in part responsible for the relatively low numbers. However, yards can in some circumstances produce a solution to increasingly important environmental issues such as straw burning, pollution and smells associated with agitating slurry tanks and spreading slurry. A looseyard lessens the straw burning problem and it is a convenient and relatively inoffensive means of storing manure.

Well designed and managed yards are capable of producing good clean conditions for dairy cows and I have little doubt that we shall continue to see them particularly but not solely in the East.

Cubicles

It is an interesting fact of history that in 1960 two men, some thousands of miles apart 'invented' the cubicle. Mr Evans in the UK designed a cubicle measuring 2.02m x 1.11m (6'10" x 3'8") whilst Mr Owen in the USA built a slightly larger one at 2.4m x 1.2m (8' x 4'). It was a simple but revolutionary development in the housing of dairy cows. The widespread use of the cubicle is evidence of the advantages of the system. Yet, no single design has emerged as totally successful and nearly 30 years on, refusals, injuries, mastitis problems and dirty cows are still associated with the system. Cubicle size, base design, division, bedding choice and management are all important factors influencing success.

The basic requirements are:

- . Sufficient space
- . Wide passages and gateways
- . Alternative routes — *bullying*
- . Non-slip floors
- . Efficient ventilation
- . Loose box facilities
- . A dry bed.

The aim is a simple one; to allow cows to lie undisturbed with minimal risk of injury in clean comfortable conditions. Cows are sociable animals but their performance is adversely affected by overcrowding. They need enough space to lie down comfortably and walk around without conflict with other cows. It is essential that there are as many cubicles as cows. Layouts should include alternative routes between lying and feeding/watering areas so that dominant cows cannot cause obstructions. Passages and doorways must be wide enough and floors not slippery to avoid injury.

In recent years much has been written drawing attention to the need for cubicles to be large enough for today's cows and designed to allow the cow to move easily. It is worth considering the various design factors.

Base

Ideally the base should be firm, durable, free draining and easy to clean. Concrete is the most economic and durable material.

Healthy cows have surplus body heat and in well ventilated buildings where some form of bedding material is used, the amount of heat lost to the floor is not critical. Insulation of bases is therefore of no benefit. Concrete should be laid 100mm thick on consolidated hardcore and the surface finished with a wooden float. Avoid deep tamping ruts or projections.

Bitumen macadam has been used successfully but it can 'lift' if dung is allowed to dry out on its surface. Laying of the material is best done by contractor. Experience suggests that because of the less hard nature of the surface, less bedding is required.

Natural materials, e.g. chalk or soil are used but often break up and form uncomfortable and sometimes, wet beds.

ADAS studies (4) have shown that except where the bedding to be used is sand, the base should not be provided with a raised lip or kerb at the end to retain the litter. Raised lips increase bedding wetness. A front to rear slope of 100mm has been shown to reduce the soiling of beds by preventing the recumbant cow moving too far into the cubicle and is to be recommended.(5).

Size

Inadequate length is the main reason why some cows refuse to use cubicles. Some only partially lie or stand in the cubicle and have difficulty rising. Length should be suitable for the majority of cows in the herd. In practice this means it should suit the larger cows not the average. Photographic studies of cow movement (6) have shown that the forward space demand of an 800 kg cow when rising ranges from 0.7m to 1.0m.

Table 3 gives recommended dimensions for a range of cow sizes. The trend to heavier cows calls for a clear width (not centre to centre) of 1.2m in most cases. With space sharing divisions clear width can be reduced to 1.1m or 1.0m for small cows.

Table 3 CUBICLE SIZE

Body weight kg	Cubicle length m	Cubicle width(clear) m
425	2.04	1.1
525	2.12	1.1
625	2.20	1.2
725	2.28	1.2
825	2.33	1.2

Divisions

Provided the length and width are suitable for the cow size, a number of designs of division are satisfactory. However, where space is limited, many designs can be restrictive to pelvis and head and do not allow space sharing. The continued increase in cow size has resulted in this situation occurring on many farms and in response a number of alternative division designs have been introduced and attempts made to modify existing divisions.

The so called Dutch comfort division allows space sharing between adjacent cubicles and greater freedom of movement for cows when lying down or rising. Cantilever divisions can be made adjustable for width and can provide flexibility in the use of the building. Critical dimensions common to all (7) include top rail height of at least 1050mm (prevents cows from turning around) and a lower rail height of 400 mm from the base (prevents cow getting trapped or bruising their rib cages).

ADAS studies (8) have shown how cubicle divisions can be successfully modified by replacing the lower rail with nylon rope. Where cubicles are too short, removing solid fronts or adjusting front rails gives cows a 'launching space'.

Headrails and brisket boards

Headrails are necessary in most installations. They must however be easily adjustable and not interfere with cows when rising or lying down. Brisket boards have been successfully used in conjunction with headrails in controlling cow position when standing or lying down and can reduce soiling of the beds.

Resting behaviour

Bedding and its management is the subject of another paper at this Conference. However it is relevant to consider here the effect of general cubicle design on resting behaviour. The effect of division design, or the effective available space was described earlier. Floor type also has an

influence on resting time. We know that cows at grass will lie down for 14 or more hours in any 24 hour period but the type of base and quantity of bedding used has a major influence of resting time. Trials showed that cows laid for twice as long on concrete beds fitted with cow-matting or concrete generously bedded with straw that they did on bare concrete. Prolonged standing requires energy and exposes hooves to slurry for longer periods. As we in ADAS say "make the inside like the outside".

Layout

Cubicles and passageways are best in straight runs to allow easy movement of cows and cleaning of passageways. Clearways, at least two cubicles wide and preferably three, should be provided for every run of 20 cubicles. Clearways are suitable locations for water troughs. A badly positioned trough is a common cause of dirty beds. Ventilation of buildings is frequently incorrect. Many buildings imitate wind-tunnels whilst others have insufficient air openings. Protection from driving rain or severe draughts should be provided to ensure there is no reduction in the numbers of useable cubicles. Lack of outlet ventilation is particularly common.

Problems and opportunities

Today most cows in England and Wales are housed in cubicles or kennels many of which were installed 10 or more years ago. Cow size has increased during this period due to breed changes and improved diet. In addition there has been little capital expenditure on buildings since the introduction of milk quotas in 1984. As a result of these various factors, many farms have aging cubicles which are also inadequate for the cows they house. At some stage replacement or modification to existing cubicles will be necessary.

Considerable information is now available on the needs of the dairy cow and how best those needs can be met by new cubicle designs or modifications, and by improved bedding management. There remains the problem of slurry handling and storage which will become increasingly urgent as pressure on polluters grows.

The future

At the start of this paper I implied a relationship between milking systems and housing systems. Parlour milking went hand in hand with loosehousing. On the horizon now is the automated milking system. We need to consider the effect that will have on housing and managing dairy cows. Some four years ago workers at the NIRD demonstrated the benefit of daily removal of cubicle bedding on bacterial populations.

Cantilever-type divisions allowed a tractor mounted brush to remove bedding from the rear end of the cubicle. It is not difficult to envisage dairy units in the next century, or earlier, with robotic milking linked with automated systems of slurry removal from cubicle passages and mechanical removal and replenishment of bedding.

For many delegates here today, there is a need to examine more critically whether or not their housing system meets the needs of their cows. Housing systems will continue to have a major influence on cow and teat cleanliness, teat condition, TBCs of milk and mastitis caused by environmental pathogens.

References

1. WILSON, C.D., & RICHARDS, M.S. 1980. A survey of mastitis in the British dairy herd. Veterinary Record 106 431-435.
2. ADAS 1988. Design & management of looseyards. Advisory leaflet P.3136
3. WILESMITH.J.W., FRANCIS.P.G. and WILSON.C.D.1986. Incidence of clinical mastitis in a cohort of British Dairy Herds. Veterinary Record 118 119-204
4. SUMNER.J., (1980) Mastitis Control and Herd Management, Technical Bulletin 4, NIRD, Reading.
5. SUMNER. J., FRANCIS P.G., SLADE G.F. and BROOKS P.H 1982. A study of various falls on permanent based cubicles. MAFF.
6. CERMAK J., (1983) Cow cubicle design, Farm Buildings Digest 18 7-9.
7. ADAS 1988. Design and management of cubicles. Advisory leaflet P.3137
8. THOMAS, T.P., GWYNN, P.E. and WILKINSON, R., 1989 Modifying timber cow cubicle divisions to improve cow acceptability. Research and Development in Agriculture. (In press).

HYGIENE OF LITTER.

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The hygiene standard of litter is important in relation to mastitis caused by the so called "environmental bacteria", i.e. Escherichia coli, Pseudomonas aeruginosa, Streptococcus uberis, and Klebsiella species. These bacteria are able to persist in the environment, and if nutrients and moisture are present, can multiply in temperatures ranging from 8° to 45° C.

Hygiene of litter also has a considerable influence on Total Bacterial Counts (TBC's) of milk.

Exposure to potential mastitis bacteria or contact between bacteria and teat ends, has been shown to be one of the prime factors in the pathogenesis of mastitis. Litter is in contact with cow's teats for up to 14 hours in each 24 hour period, so high numbers of mastitis bacteria in litter increase the likelihood of mastitis during the housing period.

The hygiene of litter is influenced by :-

1. Type of litter material.
2. Amount of litter, and replenishment intervals.
3. Ambient temperature within buildings.
4. Moisture content of litter.
5. Cow numbers and behaviour.
6. The design and construction of the buildings.

Type of litter material.

During laboratory experiments on the ability of environmental bacteria to grow in various litter materials, it was found that bacterial multiplication was highest in old manure, and lowest in shredded newspaper and softwood sawdust. Growth in straw and in hardwood chips was intermediate between these extremes. Klebsiella species showed a 2500 fold increase in manure, an 1800 fold increase in straw, and a 100 fold increase in hardwood chips. Similarly, E. coli showed a 1000 fold increase in manure, an 180 fold increase in straw, and a 25 fold increase in hardwood chips. Both of these organisms showed small increases in paper and pine sawdust. Str. uberis multiplied in all the litter materials, but to a much smaller degree than the other bacteria tested. In all these experiments the greatest increase took place during the first 12 hours, as is shown in Fig.1.

Other similar experiments (1) in which one part of manure was added to four parts of clean litter, showed that environmental bacteria multiplied 1000 fold in 24 hours at room temperature.

Thus, manure presents bacteria with ample nutrients for growth.

Klebsiella species, E. coli, and other coliform organisms are able to use a wide variety of materials for growth. Streptococci are less adaptable than the other environmental organisms, but Str. uberis is more versatile than other Streptococci.

Sappy wood contains sugars, small amounts of nitrogen, and other useful bacterial nutrients. Some hardwoods contain fatty substances, which help bacterial growth, whilst some softwoods such as pine, contain resins and other aromatic substances which slow bacterial growth. Straw provides a moderately good environment for bacterial growth as it contains sugars and amino acids which are useable by bacteria, as well as cellulose which is not useable. Peat has been used as litter, and this material contains a good supply of nutrients which bacteria can readily utilise.

Paper contains mostly cellulose, as other nutrients are removed during the manufacturing process. Sand contains hardly any nutrients, is less absorbent and tends therefore to support lower bacterial populations than the organic litters. It presents problems related to handling both before and after use.

In spite of attempts by ADAS to define and classify various sands according to their suitability as cubicle litter, some sands classed as "good" did not always produce good cow living conditions. When sand is used as cubicle litter, a retaining lip at the rear of the cubicle is necessary, to maintain a sufficient depth of litter on the bed and provide comfortable lying conditions.

Manure contains a wide variety of bacterial nutrients, as well as a source of nitrogen. If it is mixed with litter and then moistened, it provides five-star accommodation for bacteria.

Amounts of litter, and replenishment intervals..

Investigations have shown (2) that housing cows without litter was associated with a high incidence of mastitis and teat damage. This is unacceptable on welfare as well as on economic grounds.

In calculating the total quantities of each litter needed, ADAS uses a 30 week housing period as its reference.

Cubicle housed cows.

QUANTITIES. (per cow per 30 weeks.)

Long straw	350- 450 Kg.
Chopped straw or Rape straw	180- 220 Kg.
Sawdust/Shavings/Newsprint.	250- 320 Kg.
Sand	850-1000 Kg.

Where mats or carpets are fitted to cubicles, at least 1.5 Kg litter per cow per day is necessary to keep cows clean.

Daily addition of new bedding is preferable in cubicles, although replenishment every other day can produce acceptable conditions. During an experiment on the acceptability of cubicle bases laid to various falls, Francis and Sumner (3) found that the addition of 4 Kg. long straw per cubicle three times a week, in addition to twice daily scraping of passageways was sufficient to maintain clean cows, clean teats and a low incidence of mastitis. This represents the use of 360 kg. per cow during a 30 week winter.

Yarded cows

In covered yards, at least one and a half tonnes of straw is needed per cow per winter, to enable clean and comfortable conditions to be attained. Daily additions of straw are optimal, in quantities sufficient to prevent poaching and produce clean cows. Mastitis bacteria are usually killed by the composting process in the lower layers of the bedded area, but too generous additions of straw can result in unconsolidated top layers of litter which warm up and encourage bacterial multiplication.

Bramley and Neave (4) stated that levels of coliform bacteria above 10^6 per Gram of litter, may result in a high incidence of mastitis. Laycock (5) during a study on 15 herds using on average 200Kg. straw per cow per winter, found that the arithmetic mean coli content of straw bedding from cubicles was 3.7×10^5 per Gram. These results are similar to those found during an earlier study, (6) involving the use of 260Kg. straw per cow per winter, in which over 60% of cubicle litter samples taken weekly, contained less than 10^6 bacteria per Gram.

Under most management systems, involving varying quantities of litter use, the coliform₅ content of used litter, is rarely less than 100,000, (10^5) and is usually nearer to 1 million bacteria (10^6) per gram of litter

Dodd et al (7) showed that when a cubicle management regime which comprised scraping gross soiling from cubicle beds and passageways twice daily, and replenishing sawdust litter once weekly, was replaced by one which incorporated daily sweeping of cubicle beds, daily replenishment of sawdust, and twice daily scraping of passageways, the average coliform content of used litter fell 130 fold, and the incidence of coliform mastitis cases declined markedly.

In general, increased litter use has been associated with a lowered incidence of mastitis, and this was a notable feature of the herds monitored during the Mastitis Surveillance Scheme.

We have examined bacteriologically, straw, sawdust and woodshavings stored on farms in bulk. Occasionally, these have been allowed to become damp. They have then warmed up and allowed the multiplication of coliform bacteria. Under these circumstances the litter added more bacteria to the cows' environment the moment it was first put into the cubicles or yards.

Attempts have been made to reduce the bacterial populations of litter by using disinfectants (both liquid and granular), and to change the pH of litter and slow down bacterial growth, by regularly sprinkling lime over cubicle beds. Any benefit from these actions is temporary, although the use of lime may create drier conditions in the litter.

Temperature

Cows lie down for up to 14 hours in every 24, and studies have shown (3) that high yielding cows (usually in early lactation) tended to lie down for longer periods than lower yielding cows. These lying down sessions became longer still when the ambient temperatures fell in January. During these extended lying times, the temperatures of bedding under the udder region often reached incubation temperatures. (37^o C.)

At these temperatures, and given adequate nutrients and moisture, bacteria can multiply once every 20 minutes, so the potential for high bacterial populations in litter can be immense.

Moisture Content of Litter

The moisture content of used litter varies, and a high moisture content has been associated with a high incidence of mastitis. The absorbency of all litter materials is high. Most can absorb up to 300 times their weight of water.

Relative humidities in cubicle buildings during winter, especially in December and January are often over 85%, and water slopped from drinking troughs placed near littered areas, and faeces brought onto littered areas by cows' feet, add to this moisture in the environment.

Cubicle base profiles without any lip at the rear, adequate front to rear slopes, (preferably 100mm.) and ample quantities of litter, frequently replenished, together with twice daily scraping of concreted areas, will assist in keeping litter dry.

During a number of studies in the South West of England, the use of sand as cubicle litter was found to produce a slightly drier bed than other litter materials, although a rear lip on the cubicle base is necessary in order to retain sufficient sand on the bed.

Cow Behaviour

Cows in early lactation have a number of attributes which encourage increased bacterial challenge at teat ends.

- * Cows in early lactation tend to lie down for longer periods than they do in later lactation. The increased contact time between litter and teats, and the greater warming power of these hard working cows which has been mentioned earlier, encourage the multiplication of bacteria in litter.

- * The increased activity in groups of early lactation cows, before they have been served and are settled in calf, means that, in Autumn calving herds this can create dirty conditions in the cow accommodation. Bulling cows should of course be taken out of bedded areas until they have gone off, to minimise the dirt and disturbance they cause.

- * It has been found that the faeces of cows during early lactation can contain much higher populations of coliform bacteria, than is the case in later lactation cows. In addition, cows which are receiving diets appropriate to high milk production tend to produce fluid faeces. These factors increase the possibility of high coliform populations in litter.

- * Cows which run milk whilst in the littered areas, are adding to the levels of bacterial nutrients in the litter.

The standard of management of the cow early in her lactation needs therefore to be of a high order. The efficiency with which housed cows can be managed, depends to some extent upon the design of the buildings, and this has been dealt with elsewhere at this conference.

Dry cows, especially those near to calving during the housing period, are at considerable risk. Mc. Donald and Anderson (8) showed that bacterial challenge in the last two weeks of the dry period is nearly three times as likely to cause mastitis as is the same challenge during the early weeks of the dry period. The bacterial content of litter in cubicles used by dry cows, is therefore very important, and this type of accommodation must be well designed and managed. Ideally, ample calving boxes should be available, and each should be thoroughly cleaned after each calving.

In this paper, the subject of hygiene of litter has for the sake of orderliness, been divided into a number of separate components. There is however, much interaction between them. Anything which allows a change to occur in any of the areas mentioned above is liable to set off a chain reaction in the system as a whole. Robinson et al, (9) showed, during a detailed study of six commercial dairy herds, that noticeable improvements in the environmental standards of two of the herds occurred after ADAS advice. These two had lower coliform mastitis rates than those herds in which "conditions were not perceptibly improved".

Recommendations

1. Store litter materials correctly before putting them into lying areas
2. Use cubicles without rear lips, (except when using sand as litter), and which slope 100mm. from front to rear.
3. Use plenty of litter, and replenish it often.
4. Feed and water housed cows away from bedded areas.
5. Keep cubicle beds free of faeces, and scrape concreted areas twice daily at milking times.
6. Maintain air circulation within the building, but prevent draughts at cow levels
7. Monitor the effect of diet on the consistency of faeces.
8. Pay special attention to the cleanliness of areas used by cows close to calving, and to those in early lactation.

Further research is needed on:

* The persistence of teat disinfectants.
Teat disinfectants are not strongly bactericidal for the entire period between milkings. Godinho and Bramley (10) showed experimentally that when teat disinfectant was present on the teats for 15 hours, fewer quarter infections occurred.

* The transfer of faeces onto cubicle beds by cows feet. The use of improved automatic scrapers would reduce this contamination.

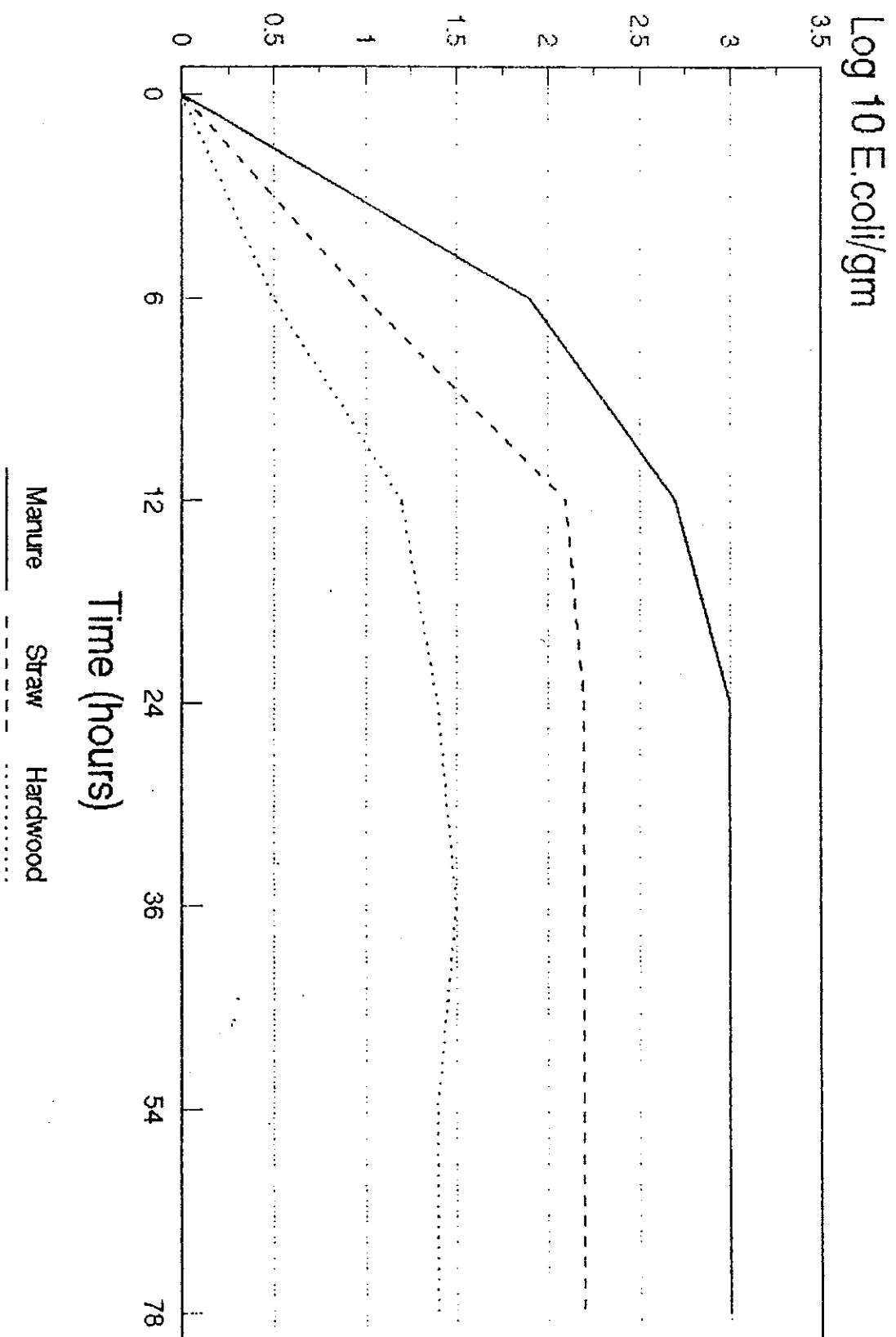
* Cubicle design.

Improvements are needed so as to increase cows' acceptance of cubicles, and to make mechanical cleaning easy and efficient. This will improve cleanliness and comfort, and may reduce lameness if cows stand in the cubicles rather than nearly in them.

REFERENCES.

1. Jasper D. E. (1980) Proc 11th Intl Congress on Cattle Diseases. Israel. pages 23-34.
2. Ekesbo I. (1966) Acta Agric. Scand. Suppl. 15.
3. Francis P.G. and Sumner J. (1979). Results of Agriculture Service Experiments. Dairy Cattle, ADAS.
4. Bramley A.J. and Neave F.K. (1975) British Vet. Journal, 131. 160-169.
5. Laycock C. (1979) ADAS Report, Burghill Road, Westbury-on-Trym, Bristol.
6. Francis P.G. Sumner J. and Joyce D.A. (1980) Proc 11th Intl Congress on Cattle Diseases. Israel. pages 35-43.
7. Dodd F.H. Higgs T.M. and Bramley A.J. (1984) Veterinary Record. 114 522-523.
8. Mc.Donald J.S. and Anderson, A.J. (1981) Am.J.Vet Res. 42 (2) 229 - 231.
9. Robinson T.C. Jackson E.R. and Marr A. (1983) Veterinary Record, 112 315 - 319.
10. Godinho K.S. and Bramley A.J. (1980) British Veterinary Journal, 136 574 - 579.
11. Zehner M.M. Farnsworth R.D. Appleman R.D. Larntz K. and Springer J.A. (1986) J. Dairy Science. 69 1932-1941.

Growth of E.coli in bedding materials



DIETARY SELENIUM AND VITAMIN E INFLUENCE THE RESISTANCE OF COWS TO MASTITIS

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Introduction.

Bovine mastitis remains the most economically devastating disease of the dairy industry. While progress on control of certain bacterial causes of the disease has been achieved, losses are still estimated to approach \$180 - \$190 per cow per year in the US (1). A fundamental concept for control of bovine mastitis is that control will likely be achieved by either decreasing the exposure of teat ends to pathogens or by increasing or optimizing the resistance of the mammary gland to infection (2). Nearly all progress in the control of bovine mastitis has been as a result of reduced exposure of teat ends to pathogens and the wide spread use of antibiotics. Our ability to modify or manipulate the resistance of the cow has developed at a much slower pace. Recent findings have suggested that resistance to mastitis can be influenced by the diets of dairy cows and suggests for the first time the possibility of achieving a degree of mastitis control by improving the natural resistance of the mammary gland.

A connection between the nutrition of dairy cows and incidence of mastitis has been suspected and debated for many years by mastitis research workers. Early efforts to understand the relationship between diet and susceptibility to mastitis were not very productive (3). In 1984 we reported (4) that low dietary intake of vitamin E and selenium during the dry period was associated with increased incidence of clinical mastitis and clinical cases of longer duration. Since this first report, a number of other studies have appeared which support the concept that vitamin E and selenium are important for optimum mammary health.

What are selenium and vitamin E ?

Selenium is an essential micronutrient present in tissues throughout the body. Selenium is important physiologically (5) because it is an integral component of the enzyme glutathione peroxidase (GSHpx). Tissue concentrations of Se are highly correlated with GSHpx activity and directly related to Se intake (6). Plants grown on Se deficient soils do not provide adequate dietary Se. In general forages grown on poorly drained acid soils are Se deficient. Selenium deficient soils are geographically wide spread in the US and Europe, and approximately two-thirds of the dairy cattle in the US are in areas with Se deficient soils.

Vitamin E is the generic name for a group of lipid-soluble compounds known as tocopherols and tocotrienols (7). Vitamin E is a powerful antioxidant that is present in cellular membranes throughout the body. Physiologically, α -tocopherol is the most important form of vitamin E and the total vitamin E content of a diet is not as important as the α -tocopherol content. The most commonly used form of vitamin E for dietary supplementation is d,l α -tocopherol acetate. Throughout the remainder of this paper we will use the term vitamin E to mean α -tocopherol.

Forages are a major source of vitamin E in dairy cow diets (8,9). High levels of vitamin E are found in fresh green forages; however, the vitamin E content of stored forages is decreased significantly and the decrease is progressive with length of storage. The ensiling process is particularly destructive to vitamin E and corn silage is notoriously low in vitamin E.

Why are selenium and vitamin E important?

Vitamin E and the Se containing enzyme GSHpx are an integral part of the antioxidant system present in all cells (10,11). Both are important for optimum cell

function because they help maintain low cellular and tissue concentrations of reactive oxygen molecules and lipid hydroperoxides. During the metabolism of oxygen within cells large quantities of superoxide and hydrogen peroxide are produced and these reactive oxygen species can severely damage membrane lipids, DNA, cellular proteins, and enzymes. The specific function of GSHpx is the conversion of hydrogen peroxide to water and lipid hydroperoxides to the corresponding alcohol. Vitamin E is a very efficient scavenger of both reactive oxygen species and lipid hydroperoxides, converting both to nonreactive forms (7).

Vitamin E and GSHpx function at two different levels within the cell. GSHpx functions in the cytosol of the cell, while vitamin E is an integral component of lipid membranes (10). Vitamin E protects the polyunsaturated fatty acids (PUFA), enzymes, and transport proteins located in membranes from reactive oxygen species. The PUFA are present in all cellular membranes, but their concentration varies considerably from tissue to tissue. Membrane PUFA are extremely susceptible to attack from reactive oxygen species and the higher the concentration of membrane PUFA, the more susceptible the cell and tissue to oxidant damage (12).

An important PUFA in cellular membranes is arachidonic acid (AA). Arachidonic acid can be metabolized to prostaglandins, thromboxane, and prostacyclin by the enzyme complex cyclo-oxygenase and to the leukotrienes by the lipoxygenase enzyme complex. Evidence clearly suggests that AA metabolism is altered in animals deficient in vitamin E, Se, or both (12). GSHpx participates directly in AA metabolism and vitamin E may function to control peroxidation of AA or its unstable metabolites. The AA metabolites are important for polymorphonuclear neutrophil (PMN) function and the amplification of the inflammatory response following pathogen invasion of tissues.

Pathogen invasion of the mammary gland triggers an influx of PMN and other white blood cells. The production of leukotriene B₄ by macrophages and PMN is important for the initiation and amplification of this response (13). Phagocytosis of the invading pathogen results in a "respiratory burst" within the PMN (14). During the "respiratory burst" there is increased oxygen metabolism within the cell and increased production of reactive oxygen species. The reactive oxygen species are produced to kill the engulfed pathogen. Thus, phagocytosis is accompanied by an intracellular increase in peroxides which is necessary to kill the pathogen, but potentially dangerous to the cell and surrounding tissue. Accumulation of hydrogen peroxide in PMN is generally associated with reduced intracellular kill of pathogens.

Clearly, the speed with which PMN can be mobilized following pathogen invasion and the efficiency of intracellular kill are events of critical importance to protection of the mammary gland from infection (13). Evidence suggests that vitamin E and Se play essential roles in these events and that dietary deficiencies of either would lead to impaired PMN function and increased incidence of intramammary infection in dairy cows.

Selenium and vitamin E responsive diseases in dairy cattle.

Historically, Se and vitamin E deficiencies have been associated with nutritional myopathies, commonly called "white muscle disease" (11,12). This condition is caused by a buildup of reactive oxygen species in muscle cells that eventually destroy the cell membrane. The disorder is typically seen in the US when dietary Se concentrations are 0.02 -0.03 ppm, but can also be the result of low dietary vitamin E.

Retained placenta was added to the list of vitamin E - Se responsive diseases following a report by Trender et al. (15) in 1969 that vitamin E and Se injections during the dry period reduced the incidence of retained placenta. Work conducted in Ohio confirmed these observations but led to the conclusion that there was considerable variation in response among herds (16). Additional work revealed that the amount of vitamin E being injected was very small relative to dietary intake.

Harrison et al. (17) conducted an experiment in which vitamin E was supplemented (1000 IU per cow per day) during the dry period and Se (50 mg) injected at 21 days prior to calving. There were four groups of approximately 20 cows each and the groups received: 1) no supplemental vitamin E or Se; 2) supplemental vitamin E only; 3) Se injected only; or 4) both. Compared to the controls, there was a significant reduction in incidence of retained placenta, metritis, and cystic ovaries for those cows

receiving both the supplemental vitamin E and Se injection. Clearly, both are required for optimum reproductive performance around calving.

Retrospectively, the incidence of clinical mastitis in these cows (4) was examined. Those cows fed vitamin E during the dry period had significantly fewer cases of clinical mastitis during the subsequent lactation and clinical cases were of shorter duration in cows receiving both supplemental E and the Se injection. The results suggested that vitamin E and selenium could influence mammary health.

Evidence that selenium and vitamin E influence mammary health.

A role for vitamin E and Se in mastitis control should have been suspected following a 1979 report by UK research workers. Boyne and Arthur (18) reported that PMN function was compromised in Se deficient cattle, suggesting the possibility of increased infectious disease. Equally important, they found that PMN function was compromised at Se concentrations considerably above those known to cause "white muscle disease".

Following our first observation of a positive role for vitamin E and Se on mastitis (4) we conducted an experiment with two groups of first lactation heifers (19,20). Diets were either unsupplemented or supplemented with Se (.3ppm) and vitamin E (1000 IU per day total intake) from 60 days prepartum and continuing throughout lactation. Plasma vitamin E and Se were higher at calving and throughout lactation in the supplemented group. Supplemented heifers had significantly fewer quarters infected at calving, reduced prevalence of infection throughout lactation, fewer cases of clinical mastitis, infections of shorter duration, and lower milk somatic cell counts (SCC) when compared to unsupplemented heifers.

Erskine and co-workers (21) studied 32 dairy herds in Pennsylvania. Sixteen herds had consistently high SCC and 16 were consistently low. Cows in high SCC herds had significantly lower Se and GSHpx compared to cows in the low SCC herds. There was a significant negative correlation within all 32 herds between percent quarters infected and herd mean GSHpx concentration in whole blood. No differences in plasma vitamin E between the two groups of herds were detected. However, blood samples for analysis were obtained during summer months when the majority of cows were on pasture.

We recently studied the incidence and cause of mastitis in 9 well managed dairy herds (22). Herds chosen had a long history of post-milking teat dipping and dry cow therapy of all cows. Herds were studied for a one year period. All herds had eliminated *Str. agalactiae* and prevalence of *S. aureus* infected quarters was less than 1%. The major mastitis problem in these herds was clinical mastitis and the overall incidence was approximately one new clinical case per two cow lactations. The major cause of clinical mastitis was the environmental pathogens.

The vitamin E and Se status of these 9 herds was determined and relationships to mastitis incidence investigated (23). Diets of dry cows and the ration fed to the top producing 1/3 of the lactating herd were analyzed three different times during the year for vitamin E and Se. Vitamin E and Se status of cows within a herd was determined by bleeding 10 cows, 3 different times during the year. Cows chosen for bleeding were the 10 cows closest to calving at the time of bleeding. There was a significant negative correlation between plasma Se and both incidence of clinical mastitis and SCC in bulk tank milk. No significant relationship between plasma vitamin E and mastitis was found. However, dietary content of vitamin E was negatively correlated with incidence of clinical mastitis.

Pennsylvania researchers have studied mammary PMN function in cows deficient in Se but adequate in vitamin E (24). PMN from Se deficient cows have an increased accumulation of hydrogen peroxide, decreased viability and a reduced ability for intracellular kill of mastitis pathogens. In addition, Erskine et al. (25) infused viable *E. coli* into mammary quarters of both Se supplemented and Se deficient cows. All were vitamin E sufficient. Resulting infections were more severe, of longer duration, and associated with greater milk loss in Se deficient cows than in Se supplemented cows. Milk SCC in the infused quarters increased more slowly and peak *E. coli* numbers were 100 fold higher when compared to supplemented cows.

We recently investigated PMN function in cows whose diets were either unsupplemented or supplemented with vitamin E, Se or both vitamin E and Se (26). PMN from vitamin E supplemented cows had improved intracellular kill for both *E. coli* and *S. aureus* compared to unsupplemented cows or cows supplemented with Se only. Plasma vitamin E concentration was not correlated with intracellular kill but whole blood Se was positively correlated with intracellular kill of *S. aureus*.

In addition to the above work, a report from Australia (27) showed that supplementing the diet of dairy cows with selenium reduced the rate of new intramammary infection during lactation and the incidence of subclinical mastitis, when compared to cows fed unsupplemented diets. Atroshi and coworkers (28,29,30) in Finland reported that vitamin E in plasma and milk was lower in mastitic cows than in healthy cows and that both mastitic cows and dairy goats had lower erythrocyte GSHpx than healthy cows and goats.

Does my herd have a problem?

Diagnosis of nutritional muscular dystrophy in a dairy herd clearly indicates a severe deficiency in Se, vitamin E or both. If cattle are receiving enough of these nutrients to prevent the onset of nutritional muscular dystrophy, but less than optimal amounts to prevent other diseases, the incidence of retained placenta, metritis, or mastitis may increase. These might be particularly good indicators in problem herds when other recommended control measures appear to be properly implemented (31).

The dietary intake of Se and vitamin E should be evaluated when a deficiency is suspected, but dietary content alone may not be sufficient to determine the problem. Many factors interfere with absorption of the nutrients from the intestinal tract. Sulfates or nitrates in water may tie up Se and prevent intestinal absorption, and high dietary calcium may decrease intestinal absorption of Se. High concentrate feeding may increase pre-intestinal destruction of vitamin E. The duration of storage and the conditions under which feeds are stored influence the vitamin E actually being fed.

Although the dietary content of Se and vitamin E should be determined, we recommend analysis of blood for Se (or GSHpx) and vitamin E. If marginal deficiencies exist, they are most obvious immediately pre- and postcalving; therefore, we recommend sampling some of the animals in the herd during the immediate pre- or postpartum period.

Selenium can be determined in either whole blood or plasma. Glutathione peroxidase should be determined in whole blood. Whole blood Se values are a measure of Se stores representing Se intake at a previous time. Plasma or serum Se is more indicative of the current Se intake. Vitamin E is generally determined in plasma or serum.

What Are Acceptable Target Levels?

There is increasing evidence that whole blood Se should be .2 $\mu\text{g/ml}$ and should not exceed 1.0 $\mu\text{g/ml}$ (31,32). Values in the range of .1 to .15 $\mu\text{g/ml}$ are marginal and values below .1 $\mu\text{g/ml}$ are deficient. We use .07 $\mu\text{g/ml}$ Se as the dividing line between sufficient and insufficient concentrations in plasma or serum.

The optimal blood or tissue concentration of vitamin E is not clearly established. However, summary of a number of studies regarding the vitamin E content of blood from cattle suggests the following. Values greater than 4 $\mu\text{g/ml}$ appear to be adequate; values from 3 to 4 $\mu\text{g/ml}$ are marginal; values from 1.5 to 3.0 are low; and values less than 1.5 $\mu\text{g/ml}$ are deficient (31).

Our Feeding Recommendations

Our recommendations are for typical Holstein cows, housed in confinement in areas of known Se deficient soils. The concentration of Se in the total diet should be .3 ppm. This equates to 3 mg/cow/day during the dry period and 6 mg/cow/day during lactation. Selenium is generally supplemented as sodium selenite. In addition, in herds that are very low in Se, we recommend the injection of 50 mg of Se at 21 days prior to calving.

We are of the opinion that Holstein dairy cows need 1000 IU of vitamin E/cow/day during both the dry period and lactation. The exact supplemental amount of vitamin E will depend on the quality and amount of forage being fed. It is not uncommon in the US for dry cows to be fed poor quality forage and thus we recommend they be supplemented with 1000 IU/cow/day. Forage quality generally improves during lactation and we recommend supplementing lactating cow diets with 400 to 600 IU of vitamin E/cow/day.

Are Selenium and Vitamin E the Answer to Your Mastitis Problems?

Maybe and maybe not! While we are convinced that vitamin E and Se influence cow resistance to mastitis, these dietary constituents do not influence the exposure of teat ends to pathogens. Serious mastitis control begins with a good program of post milking teat-end disinfection, total dry cow therapy and a clean dry environment for cows. The degree of success of these hygienic measures may to some degree depend on the resistance of the herd to disease. There is a growing body of evidence that dairy cows fed diets low in vitamin E and Se are more susceptible to disease (33).

References

1. Current Concepts of Bovine Mastitis. Third Edition. The National Mastitis Council, Arlington, Virginia. 1987.
2. DODD F H (1983). *Journal of Dairy Science* 66, 1773.
3. INTERNATIONAL DAIRY FEDERATION (1987). Document 217, 21.
4. SMITH K L, HARRISON J H, HANCOCK D D, TODHUNTER D A, & CONRAD H R (1984). *Journal of Dairy Science* 67, 1293.
5. ROTRUCK J T, POPE A L, GANTHER H E, SWANSON A B, HAFEMAN D G, & HOEKSTRA W G (1973). *Science* 179, 558.
6. SCHOLZ R W, & HUTCHINSON L J (1979). *American Journal of Veterinary Research* 40, 245.
7. PUTNAM M E, & COMBEN N (1987). *Veterinary Record* 121, 541.
8. KIRMAE A, & CARPENA C (1973). *Acta Agriculture Scandinavia* 19 (Suppl. 1), 162.
9. SCHINGOETHE D J, PARSONS J G, LUDENS F C, TUCKER W L, & SCHAVE H J (1978). *Journal of Dairy Science* 61, 1582.
10. HOEKSTRA W G (1975). *Federation Proceedings* 34, 2083.
11. ARTHUR J R (1985). Proceedings of a Meeting on "Selenium and Ruminant Health". Glenfield, New South Wales, Australia. pp 1.1.
12. RICE D A, & KENNEDY S (1988). *Proceedings of the Nutrition Society* 47, 177.
13. CRAVEN N, & WILLIAMS M R (1985). *Veterinary Immunology and Immunopathology* 10, 71.
14. BABIOR B M (1984). *Journal of Clinical Investigation* 73, 599.
15. TRINDER N, WOODHOUSE C D, & RENTON C P (1969). *Veterinary Record* 85, 550.

16. JULIEN W E, CONRAD H R, & MOXON A L (1976). *Journal of Dairy Science* 59, 1960.
17. HARRISON J H, HANCOCK D D, & CONRAD H R (1984). *Journal of Dairy Science* 67, 123.
18. BOYNE R, & ARTHUR J R (1979). *Journal of Comparative Pathology* 89, 151.
19. SMITH K L, CONRAD H R, AMIET B A, & TODHUNTER D A (1985). *Kieler Milchwirtschaftliche Forschungsberichte* 37, 482.
20. SMITH K L (1986). *The Value of Vitamins in Animal Nutrition*. Roche Symposium, London, England. pp 3.
21. ERSKINE R J, EBERHART R J, HUTCHINSON L J, SCHOLZ R W, & SPENCER S B (1987). *Journal of the American Veterinary Medical Association* 190, 1417.
22. HOGAN J S, SMITH K L, HOBLET K H, SCHOENBERGER P S, TODHUNTER D A, HUESTON W D, PRITCHARD D E, BOWMAN G L, HEIDER L E, BROCKETT B L, & CONRAD, H R (1989). *Journal of Dairy Science* 72, 1547.
23. WEISS W P, HOGAN J S, SMITH K L, & HOBLET K H (1989). *Journal of Dairy Science* (In Press).
24. GRASSO P J, SCHOLZ R W, EBERHART R J, & ERSKINE R J (1987). *Journal of Dairy Science* 70(Suppl.1), 166.
25. ERSKINE R J, EBERHART R J, & SCHOLZ R W (1988). *Journal of Dairy Science* 71(Suppl.1), 150.
26. HOGAN J S, SMITH K L, WEISS W P, TODHUNTER D A, & SHOCKEY W L (1989). *Journal of Dairy Science* (Submitted).
27. RYAN D P (1985). *Proceedings of a Meeting on "Selenium and Ruminant Health"*, Glenfield, New South Wales, Australia. pp 9.1.
28. ATROSHI F, TYOPPONEN J, SANKARI S, KANGASNIEMI R, & PARANTAINEN J (1986). *International Journal of Vitamin and Nutrition Research* 57, 37.
29. ATROSHI F, SANKARI S, & LINSTROM U B (1985). *Archives of Experimental Veterinary Medicine* 39, 520.
30. ATROSHI F, PARANTAINEN J, SANKARI S, & OSTERMAN T (1986). *Research in Veterinary Science* 40, 361.
31. SMITH K L, HOGAN J S, & CONRAD H R (1988). *Veterinary Medicine* 83, 72.
32. ERSKINE R J, EBERHART R J, GRASSO P J, & SCHOLZ R W (1989). *Proceedings of the Annual Meeting of the American Association of Bovine Practitioners*. pp 119.
33. SMITH K L, HOGAN J S, TODHUNTER D A, & SCHOENBERGER P S (1989). *Proceedings of the Annual Meeting of the American Association of Bovine Practitioners*. pp 122.

WIDE BORE MILKING SYSTEMS

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Summary

Since the introduction of quota's in 1984, a major rethink has been taking place in the dairying community in an endeavour to maintain or improve margins by increasing the efficiency of the dairying operation. This quest for improved efficiency has also taken place in the basic design of the milking machine with a more "back to basics" approach in machine milking. A more indepth look has been taken into traditional pipeline layouts and milk and air pipeline diameters which has led to the increasing use of wide bore milking systems. The effects of these changes in design are discussed.

Introduction

The emphasis of this paper is on the milking machine and its effect on milking efficiency and udder health. Reference will be made to the British Standard BS5545 and the comparison with American practises and the extent to which these have been introduced into the U.K.

At the outset it should be stated that the aims of the British Standard and the U.S standard vary very little - if at all. Each aims at vacuum stability and efficient consistent pulsation in order to milk cows cleanly, comfortably and efficiently. It might serve well at this stage to draw attention to the basic scope of BS5545. In Part Two it is stated in the Scope that the standard "Specifies the minimum performance and certain dimensional requirements for the satisfactory functioning of milking machines" ---- The emphasis here should be placed on the word minimum and it is important that the stated dimensional requirements particularly on pipeline diameters are considered in the same light to prevent restriction on design.

With the return of confidence in the Dairy Sector recently, there is a groundswell of sales of new milking parlours and conversion of existing parlours to the direct to line design. These in the main, can be looked upon as wide bore systems using 2" (50mm) or 3" (76mm) stainless steel milklines. It is necessary to slope this milkline in the direction of the receiver vessel and to have sufficient pipeline volume for the milk to flow along the bottom of the line (Fig 1A) with adequate space above the milk to allow for the free, uninterrupted passage of air flowing from the claw air bleeds, cluster changing etc., to the vacuum pump.

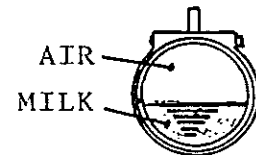
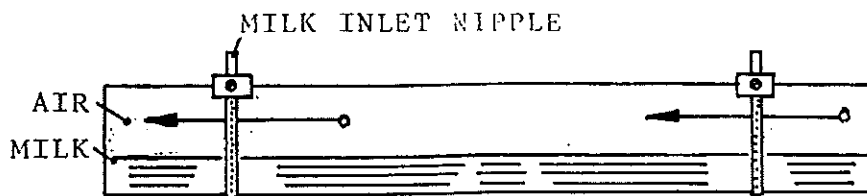


FIG 1A

From this it will be appreciated that the milk line is serving two major purposes -

1. To supply vacuum to the cluster in order to extract milk from the udder.
2. To convey this milk to the receiver.

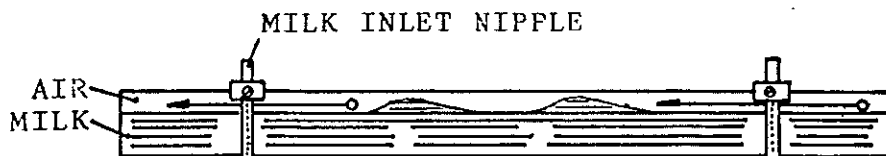


FIG 1B

Any restriction to the air flow in this line (Fig 1B) due to plugging of milk or flooding of the line is transmitted to each cluster in the form of reduced vacuum level. Therefore milking vacuum stability is affected.

This logic can be followed in every part of the installation. Wherever there is a restriction to the normal air flow in a milking machine, there necessarily follows a drop in vacuum. Whether this affects the complete machine or just individual parts is dependent upon the location of the restriction - but it nevertheless creates the basic problem of vacuum instability. Therefore when talking of wide bore milking machines, it is essential to consider all parts and pipes in the machine - not just the milkline. Starting at the "working end" - the udder, (Fig. 2). The short milk tube through which the milk extracted from each teat passes - must be of sufficient bore to accept this milk flow from the teat with minimum restriction. Removing the milk cleanly from each teat is of little value if this milk blocks the manifold, or claw piece. Therefore, it is essential, to have good volume claws which allow vacuum stability to the teat cups and which do not flood. The pipe from the claw to the milkline must likewise be of adequate bore for the same reasons.

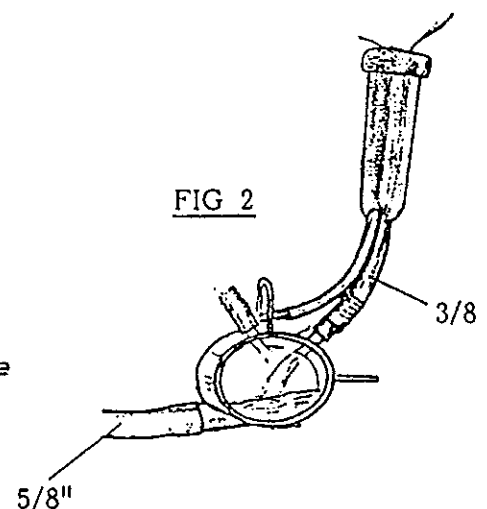


FIG 2

All the foregoing assumes that -

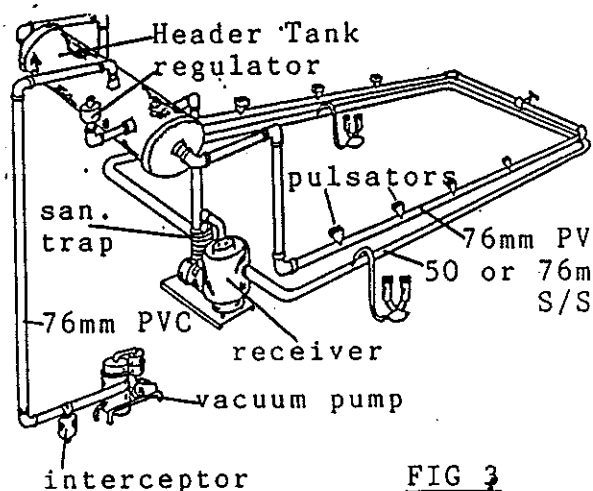
- a) the vacuum pump extracting this air is of adequate capacity for the size of installation.
- b) the pipelines conveying the air from the different parts of the installation are of adequate bore and correctly plumbed to allow free passage of air
- c) that the vacuum regulator is of good design and correctly sited.

One of the major innovations brought into Europe by Surge from the USA has been the use of large bore PVC air pipelines. These are used for the vacuum supply line from the vacuum pump to the Header or Balance tank. The use of 3" bore PVC has a number of advantages.

1. It is heavy walled and therefore requires a minimum of bracketing to prevent sagging.
2. It is smooth bore - minimising air flow turbulence.
3. It is non corrosive and will not therefore start to restrict over a period of years.
4. The pipeline and fittings are relatively inexpensive.
5. The heavy wall may be drilled and threaded.

The large diameter allows for high volumes of air flow with minimal vacuum drop. Comparing a 2" galvanised line with a 3" PVC line, the velocity of air flowing along the 2" is in the region of 50% faster than in the large bore line. Likewise in a 1 1/2" galvanised line the air flow velocity is approximately 2 1/2 times greater than in a 3" line. Slow moving air creates less turbulence and is thus easier to control.

A Header or Balance tank - usually around 45 gallon capacity can be located at the head of the milking parlour or cowshed pipeline to act as a manifold (Fig 3). Within reason, this allows the vacuum pump to be sited some convenient distance from the parlour. The stable vacuum thus provided in the Header tank can then be tapped to each part of the machine e.g. milk receiver and milkline and pulsation line, in the knowledge that the vacuum levels thus supplied will be equal. Vacuum operated ancillary equipment, such as feeders, Automatic cluster removers and entry and exit gates may also have their supplies tapped from separate entries in the Header tank, thus minimising possible interference with the milking vacuum system.



In wide bore systems it is a good policy to use similar large diameter looped PVC lines as pulsation lines and this practise has caused many raised eyebrows in the UK. The reasoning for this use is to provide effective liner movement in pulsation, on the basis that each time a pulsator operates on the vacuum stroke, it is extracting a large volume of air over a very short period of time. When this is multiplied by the number of pulsations per minute (50 to 60) and the number of milking units, the airflow "shocks" into the line are such as to create instability in the pulse line with consequent detrimental effects on the pulsation characteristics and liner movement. The use of large bore pulsation lines creates a large volume path for the air flow, greatly reducing its velocity and smoothing out the "shocks".

The vacuum pumping requirements issued as recommendations by the US Milking Machine Manufacturers are greatly in excess of the minimum standards in BS5545. This is mainly due to the fact that, included in the total is a figure of 50% of the plant consumption calculation as Reserve Capacity. This leads, in the case of larger installations, to the call for vacuum pump capacities sometimes two to three times greater than called for in BS5545. The Surge UK design has adhered more closely to the BS5545 than to US recommendations in respect to vacuum pumping capacity and air flow requirements. However, from the point of view of pipeline materials, diameters layout and plumbing the US recommendations have been adapted to suit local requirements.

As previously indicated a high proportion of new parlour sales in the UK are of the direct to line system. However, the vast majority of milking parlours in existence are of the recorder jar type. Very many of these would benefit from the adaption to the wide bore milking machine principle. The same design principles apply to the vacuum and pulsation systems as previously described for the direct to line - i.e. large bore looped pulsation line, large volume claw and large bore short milk tubes and long milk tubes.

Where a recorder jar is fitted, it is necessary to ensure that there is no cause for restriction to air flow created by its fittings and connections. (Fig. 4). There must be an unrestricted path for air and milk to flow from the claw to the jar. Partial restrictions caused by poorly designed jar sprayers and milk transfer valves will be detrimental to vacuum stability in the jar and thus in the claw. The use of 5/8" tubing from the claw to the jar and from the jar to the vacuum/wash line is of great advantage. However, the continued use of existing 1" or 1 1/4" vacuum wash lines will have the effect of throttling air flow and this should be increased in bore to 40mm to 50mm dependent upon the size of the installation.

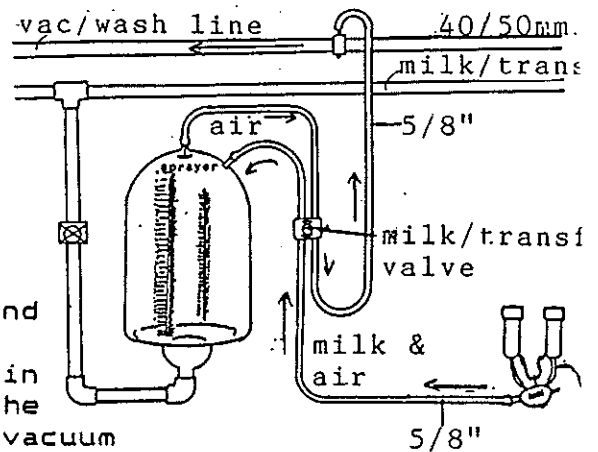


FIG 4

The most important factor in adapting an existing recorder installation is that it must be designed as a whole, not approached in a piecemeal manner. Little or no benefit will be gained by half doing a job.

The acid test for proving the efficiency of any milking system is the Dynamic test carried out when it matters most at milking time. This is not to underestimate the importance and value of the static milking machine test which is essential on a routine basis, particularly when a machine is a borderline case. The dynamic test indicates what is happening in the claw or liner during the actual milking operation. It is advantageous in measuring vacuum stability in the claw to carry out the measurement actually in the claw - preferably above the milk level if possible. (Fig 5.)

If the point of measurement is flooded with milk a damping effect is likely to occur causing the recording equipment to provide an incorrect reading and this can prove misleading making a poor system appear better than it is in fact.

No standards have yet been devised for either test equipment or interpretation of results and, therefore, conclusions drawn from any test tend to be subjective.

However, the aim should be toward optimum vacuum stability at this most important part of the milking machine. It is therefore logical to assume that the flatter the recording the better. A graph adhering as close to the milking vacuum level as possible and minimal irregular fluctuations, implicated in teat end "impacting" (Thiel cc & Mein GA, NIRD Machine Milking Technical Bulletin No 1 1979.) is that which should be sought.

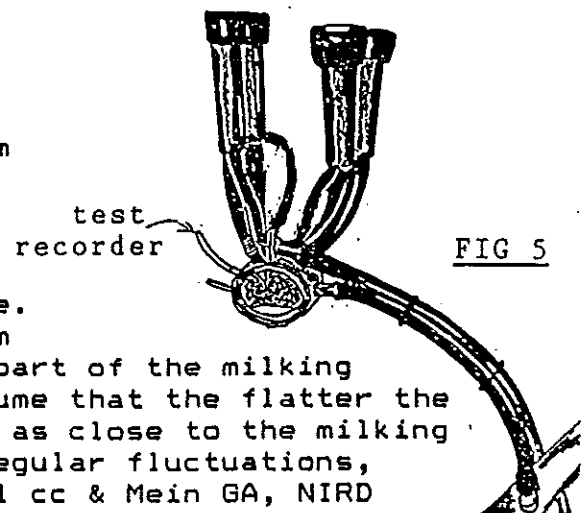
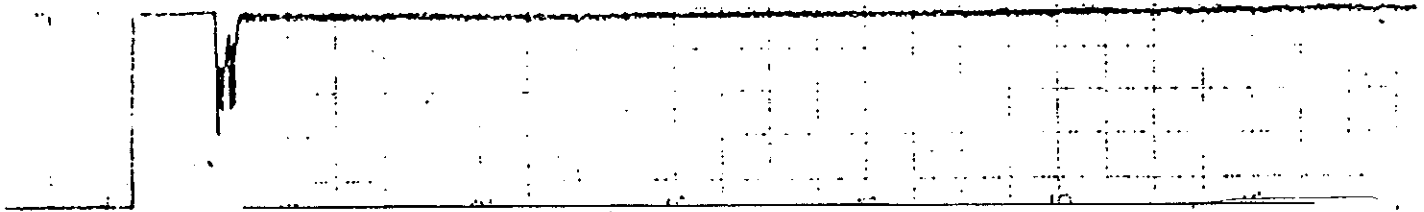


FIG 5

Comparisons of dynamic test recordings on basically similar types and sizes of installation will be seen in Figs 6 & 6A, and 7 & 7A. To the best knowledge available, the graph at the claw in 6 must be preferable to that in 6A. Both plants have effective reserves well in excess of BS5545 requirements, but in Fig. 6A this is obviously not available where it most matters - at the claw.

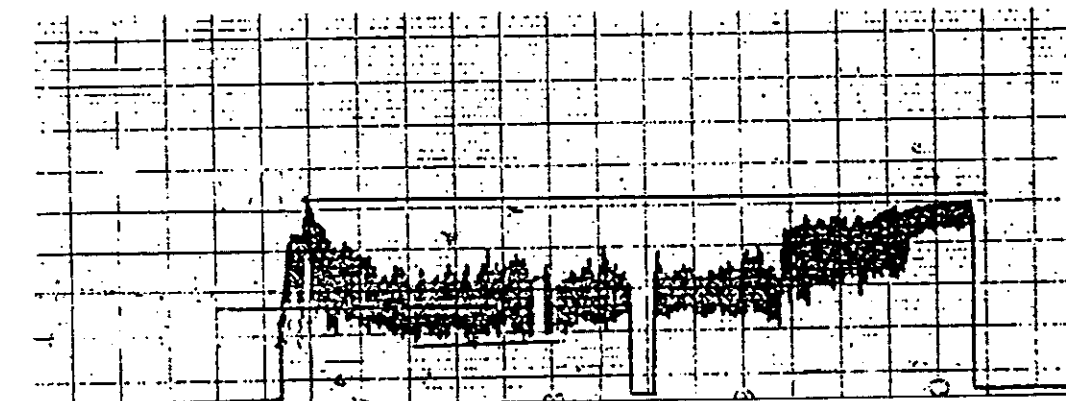
FIG 6.



10 low level jars. Effective Reserve 750 ltrs. Main vacuum line 76mm PVC Air pipeline (milking vacuum) 40mm. Claws 600ml.

OR

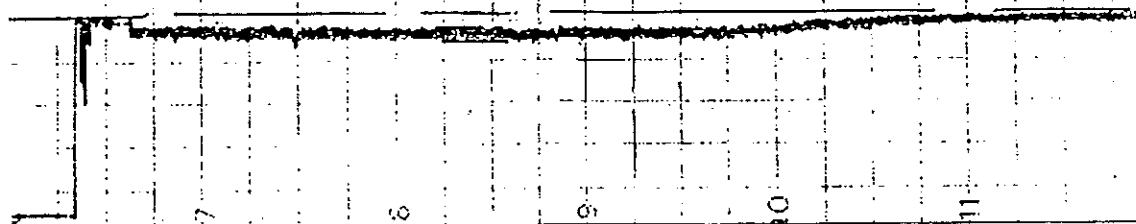
FIG 6A



12 low level jars. Effective Reserve 600 ltrs. Main vacuum line 32mm G.I. Air pipeline (milking vacuum) 32mm. Claws 60ml.

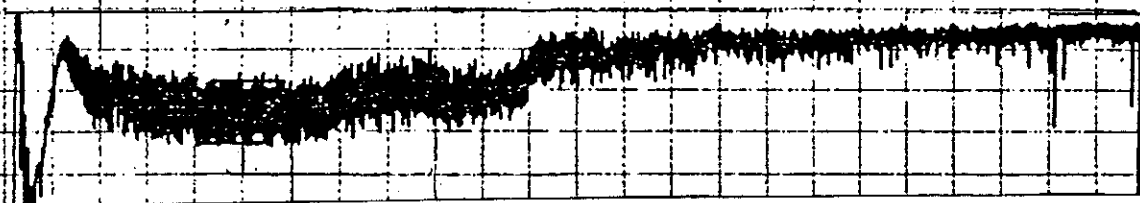
Similarly the two plants in Figs 7 & 7A which are both 10 unit high level jar systems both have effective reserves higher than required, but the design and plumbing which allowed the more stable vacuum at the claw in Fig.7 would obviously be the system to aim for.

FIG 7



10 unit high jars. Effective Reserve 580 ltrs. Main vacuum line 76mm. Air pipeline 59mm. Claws c. 300ml.

FIG 7A



10 unit high jars. Effective Reserve 470. ltrs. Main vacuum line 40mm G.I. Air pipeline 25 and 32mm. Claws 100ml.

An independent survey of the effects of modifying a large number of existing installations to the wide bore milking system as previously described, carried out in 1987, brought to light a number of improvements. Among these were improved teat-end condition, reduced clinical mastitis, reduced vet bills and use of antibiotics, reduction in milking time, greater vacuum stability and reduced "liner slip", more contented cows and dairymen and in many cases reports of increased yields.

Many of the plants modified had suffered from a history of udder health problems and had not met BS5545 standards.

In summary it should be emphasised that mastitis is a complex problem with many causes, of which the milking machine is one. Dairymen should continue to pay great attention to housing, dry cow therapy, the culling of chronic cases and teat disinfection after every milking. They should also place great importance on the design and operation of the milking machine in the drive towards greater milking efficiency. There should be greater emphasis placed on dynamic testing for which standards of interpretation are required, and milking machines should be installed which are of sufficient capacity to ensure that they continue to operate efficiently and effectively over long periods of time taking into account the adverse conditions to which they are subjected. Correctly maintained wide bore milking systems go a long way toward attaining that goal.

MACHINE MILKING AND MASTITIS

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INTRODUCTION

Whilst mastitis infections occur in cows which are suckled or hand milked it is generally accepted that the milking machine is an important factor influencing the level of infection in machine milked herds. If no bacteria penetrate the teat canal there will be no new infection. The milking machine is involved both in the transfer of bacteria on to teat surfaces and the penetration of the teat canal by bacteria.

The milking machine can act as a simple vector for the transfer of pathogens between the teat surfaces of cows or from an infected quarter in one cow to the teat skin of another. Tracer bacteria placed on the liners at the milking of one cow can persist on the liners for the next 6 cows to be milked (1). Forces within the machine can be produced which allow milk from the clawpiece to be thrown back against the teat orifice either in plugs or droplets. This occurrence is commonly referred to as impact. Where these impacts occur with sufficient force milk will be driven back through the teat canal. If this milk contains pathogens new infections can be produced. During the milking process, using a machine of conventional design, milk can cross the clawpiece from one liner to another. Pathogens from infected quarters can therefore be transferred from quarter to quarter on the same cow, either by contact or impact, in addition to the spread between cows.

Poor design or installation of the machine and its inefficient use by an unskilled operator can increase the frequency and magnitude of impacts and can also lead to physical damage to the teat and teat orifice and thus reduce the resistance of the teat canal to bacterial invasion. This teat damage and reduced resistance to penetration by bacteria can pre-dispose to infection. Ensuring the correct design and operation of the milking installation in the light of research results, practical field experience and anecdotal evidence is therefore an important aspect of mastitis control.

Techniques of machine testing have been developed to ensure that the plant is operating within the desired parameters. The employment of these techniques, particularly the increasing use of dynamic testing ie whilst the machine is being used to milk cows, are essential tools in assessing the operation of the plant and to identify faults which may be a contributory cause of high levels of infection when investigating problem herds.

This paper considers the major machine factors which may influence of rates of new infection and discusses the role of machine testing, both static and dynamic; in identifying machine factors which may be important in the spread of infection within the herd. The comments relate to machines of conventional design.

EFFECT OF MACHINE FACTORS ON NEW INFECTION LEVELS

A number of design and operating parameters of the milking machine are known to influence the level of mastitis infection. Many of these same parameters

contribute in a part otherwise of the same mastitis

will also effect the speed of milk extraction. Frequently the optimum design and operating parameters are a compromise between maximising milk extraction rate and minimising new infection levels.

VACUUM LEVELS

Operating vacuum levels above the normally accepted range of 40-50 kilopascals have been associated with increased teat orifice eversion, erosion and oedema of the teats. As there is undisputed evidence that high vacuum levels lead to increased teat injury it is reasonable to expect that this will also lead to a rise in new infection rates. However experiments have shown that whilst high vacuum levels (67kPa) have increased the incidence of teat orifice erosion the rate of new infections did not increase (2). However where high vacuum levels are used in the presence of other machine malfunctions - particularly inadequate liner collapse and excessively wide pulsator ratios - the risk of infection is increased (3). There are a significant number of field observations which attribute increased levels of clinical mastitis to high vacuum levels. However the experimental evidence is not clear cut and although the majority of results show minor increases in infection levels with increasing vacuum these increases are relatively small and then only occur at very high vacuum levels.

There does not appear to be any definite relationship between increasing vacuum levels within the range 40-55 kPa and new infection rates where all other operating parameters are satisfactory.

PULSATION

Pulsation of the liners was introduced at an early stage in the development of machine milking to overcome the effects of constant vacuum and aid the circulation of blood and thereby reduce the congestion of the tissue at the tip of the teat. The collapsed liner does not cut off vacuum from the teat apex but will exert a pressure on the teat. In order to prevent oedema a compressive force of around 10 kPa on the teat is required. The pressure exerted on the teat will be affected by liner tension, liner wall thickness and the vacuum difference across the liner.

The teat sphincter is forced closed by the collapsing liner (4) and this may theoretically lead to damage to the teat canal but there is no experimental evidence to confirm that damage does occur or of the effect that this may have on infection levels. On the contrary the absence of pulsation has been shown to increase new infection rates (5). In a series of experiments at NIRD it was shown that if pulsation is stopped completely in one half of the milking cluster the new infection rate for quarters milked with non-pulsating liners increased. This is supported by field observations which suggest that the complete breakdown of the pulsation system can result in serious mastitis problems.

Although pulsation has been shown to be important in reducing infection levels the opening and closing of the liner causes pressure changes below the teat which results in milk, frequently contaminated with pathogens, returning from the clawpiece to the teat end as the liner opens. The operating parameters the pulsator must therefore be such as to minimise the effect of the reverse flow of milk and to maximise the apparent beneficial effects of the liner closed phase of the cycle.

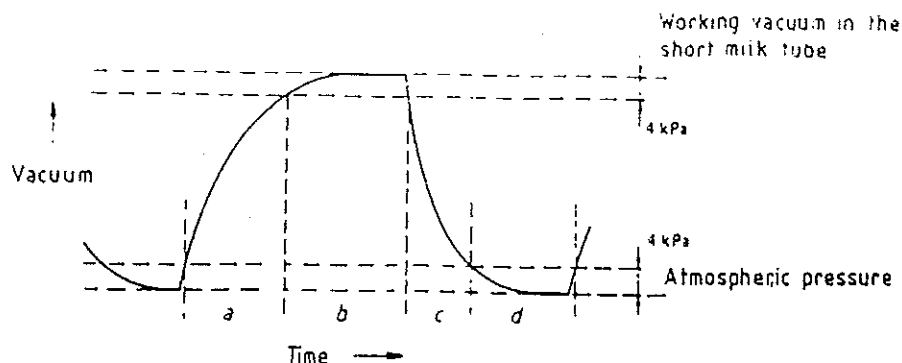


FIG 1
PULSATION CHAMBER VACUUM RECORD

The characteristics of a pulsation system which may influence the level of mastitis infection are rate, speed of liner opening and closing, ratio, duration of the liner closed phase and whether the liners pulsate simultaneously or alternately in pairs.

Pulsators are normally designed to work in the range of 50-60 cycles per minute. Where, in operation, pulsators significantly exceed this speed it is probable that the liner closed (d) phase will be either incomplete or of sort duration.

In order to minimise the effects of the reverse flow of milk during pulsation a slow liner opening (a) phase is preferable. There are also sound arguments for a relatively slow liner closing (c) phase to prevent trauma to the teat tissue from sudden increases in the compressive force of the liner. "Snappy" liner movement should be avoided and a and c phases of around 20% are thought to be desirable.

There is ample experimental work to suggest that the duration of the liner closed (d) phase is of major importance in reducing levels of infection. In one such experiment to investigate the effect of different liner closed times when the liner was more than half closed for 0.00, 0.17, 0.34 and 0.51 seconds per pulsation cycle, 20/40, 11/40, 4/40 and 5/39 quarters developed new infections (6). Arising from these experiments the requirement that the d phase of the pulsation chamber record should be at least 15% of the cycle and should be not less than 150 millisecond in any one cycle was included in the British Standard for Milking Machine Installations.

The pulsator ratio is defined as the duration of the sum of the a and b phases divided by the duration of the complete pulsation cycle expressed as a percentage. Conventionally the terms wide and narrow ratio have been used to describe the relative duration of milking to non-milking phases.

The effect of increasing the pulsator ratio is to increase the time during which the liner is opening and fully open. This will increase the speed of milk extraction. However as the ratio is increased the proportion of each cycle during which the liner is closing or closed is decreased thus increasing the risk of teat damage and oedema. With pulsator ratios in excess of 70% it becomes increasingly difficult to achieve a satisfactory d phase. Pulsator ratios within the range of 50-70% are normally regarded as satisfactory. Towards the upper end of the range problems with teat damage may be experienced particularly where operating vacuum levels exceed 50 kPa.

There has been much discussion regarding the relative merits of simultaneous and alternate pulsation systems. With alternate pulsation there is evidence of significant cross contamination between opposing pairs of liners where one pair opens as the other pair closes ie with a 50% ratio. This problem can be minimised by using a wider ratio with alternate pulsation. A simultaneous system will ensure that the pulsation characteristics applied to all 4 teats are identical. However as all 4 liners are open together peak milk flow through the clawpiece will be higher than with alternate pulsation. This may give rise to flooding of the clawpiece and liners where a small capacity clawpiece is used and there is an excessive milk lift. This problem becomes more acute as yield levels and milk flow rates increase but can be mitigated by increasing the capacity of the clawpiece and the diameter of the long milk tube.

The arguments are finely balanced and provided an alternate system has a ratio wider than 50% and a simultaneous system does not result in flooding of the clawpiece and liners there is no evidence to support one system compared with the other in terms of increased infection levels.

VACUUM FLUCTUATION

It is important to define the term vacuum fluctuation. Two types occur within a milking machine. Firstly cyclic vacuum fluctuations which occur in the liner and clawpiece due to the effect of the liner volume change produced by the cyclical opening and closing phases of the liners. The amplitude of the cyclic fluctuation will be influenced by milk flow rate, volume of air omitted through the clawpiece air bleed, height of milk list and whether the pulsation system is simultaneous or alternate.

Secondly irregular fluctuations which occur randomly when for various reasons the plant fails momentarily to maintain its working vacuum level. Factors which can contribute to irregular fluctuations are low vacuum reserves which cannot compensate for sudden air admissions, excessive quantities of air admitted by the operator, restricted air flow through the pipework and poor regulator response. The irregular fluctuation which occurs within the plant will also occur within the liner and clawpiece and will be superimposed on the cyclic fluctuation occurring there.

The most substantial evidence of a relationship between vacuum fluctuations and new infections were provided by Moorepark experiments in the late 1960s (7). A five-fold increase in new infections was seen in animals milked with a machine with induced irregular fluctuations compared to one with a stable vacuum. Further work carried out at NIRD showed that neither large irregular fluctuations nor substantial cyclic fluctuations alone increased infection rates. However combinations of large irregular and substantial cyclic fluctuations did produce a large increase in new infections. The results also suggested that infections were most likely to be initiated towards the end of milking. The mechanism by which the new infections occurred were thought to be the penetration of the teat canal by bacteria carried on droplets of milk travelling with sufficient velocity to be driven through the teat orifice. The velocity of the milk droplets impacting on the teat will be determined by the magnitude of the vacuum fluctuation occurring as the liner opens.

The impact theory as a transfer mechanism of bacteria was tested by fitting shields at the base of the liner to deflect droplets of milk travelling towards the teat. Following experimental work which indicated a reduction in infection levels where shields were used a trial on commercial

farms showed a 10% reduction in new infections in shields versus unshielded quarters. In herds where the design and operation of the machine was such as may produce significant irregular fluctuations the reduction due to shields approached 50% (8).

Although effective in preventing bacteria being transferred by impacts during milking, shields do not prevent transfer of bacteria in milk to the teat surfaces of uninfected quarters by movement of milk across the clawpiece. The use of one-way valves in the clawpiece will prevent both impacts and cross flooding. Further studies both at NIRD and Moorepark examined the effect of local air admissions such as would occur during liner slip into the cluster and which would produce irregular fluctuations in the liner. New infection rates were greatly increased by liner slip and by sudden admission at take-off.

Research evidence therefore indicates the importance of maintaining a stable milking vacuum free from irregular fluctuations, minimising cyclic vacuum fluctuations and using a liner design which will reduce slip.

TESTS ON MILKING INSTALLATIONS

With the knowledge that the operation of the milking machine can influence the level of mastitis infection it is important to be able to assess the performance of the equipment both as a preventative measure as part of the the mastitis control routine and when investigating problem cases. British Standard 5545 Milking Machine Installations (Revised in 1988) sets out in Part 2 the specification for Construction and Performance and in Part 3 Methods for Mechanical Testing. Although detailed, the requirements in Part 2 are aimed at ensuring that an installation which meets all the requirements will operate with a vacuum free from significant irregular vacuum fluctuations and that the liner will both open and close for the required duration.

The various requirements for the Standard are reviewed and amended in relation to research work and field experience. The last revision was in 1988 when a number of changes were made and new requirements were included eg. amendments to the requirements for the design of milking pipelines and the inclusion of the requirement for recorder jar vacuum recovery time. In order to ensure efficient milking and minimise the risk of new infection the installation of new plant or major updates of existing equipment should be in accordance with the details set out in the Standard. Compliance with the Standard is not mandatory and the onus is on the purchaser to request that the installation will meet the requirements as part of the contract. Following initial installation it is important that the plant receives proper maintenance and that it is tested at least annually to ensure that it remains in good operating condition. Faults can develop gradually over a period and may not be evident to the operator. Testing services are provided mainly by the MMB, ADAS and the major milking machine manufacturers and their dealers. Part 3 of the Standard sets out the requirement for the test equipment and detailed procedures. It is important that the full procedure is followed if a test is to be of value. The tests specified in Part 3 are carried out between milkings and are referred to as static tests. A visual inspection is carried out to check that the construction requirements are met and subsequent tests cover vacuum level measurements at various points in the plant, vacuum pump extraction capacity, measurement of air leaks into the system, vacuum reserves, regulator performance, clawpiece air bleeds, recorder jar vacuum recovery tests and detailed analysis of the performance of the pulsation system.

Whilst static testing will ensure that the basic requirements of the plant are satisfactory there is increasing interest in carrying out additional observations and tests whilst the equipment is being used for milking - referred to as dynamic testing. In addition to taking continuous measurements of vacuum levels both in the recorder jar and milking pipeline and near the teat observations of operator technique are also made. Dynamic testing can often reveal faults which will not be present during static tests. Problems which can be identified include the effect of vacuum operated components such as feeders and gates which would not operate during the static test on vacuum stability, the admission of excessive volumes of air by the operator for which even an adequate reserve cannot compensate and poor regulator performance when under load. Currently the equipment and procedures for carrying out dynamic testing tend to vary between operatives and proposals to specify the requirements as part of BS 5545 are currently under consideration.

Continuous vacuum recordings using a single channel pressure transducer recorder are taken both at a point where normally a stable vacuum supply is expected and near the clawpiece or in the short milk tube for a period during which the effect of all operations carried out are recorded. When investigating more difficult cases a multi-channel recorder can be used to carry out simultaneous vacuum records at different points in the plant.

Where ringing or excessive blueing around the tops of teats is a problem recordings can be made of the vacuum levels in the liner mouth piece. Where the liner fails to maintain contact with the teat high vacuum levels can be present. It is important to observe that cluster placement and support is such that it hangs evenly on the udder to prevent uneven milking and incomplete removal of milk from some quarters which may predispose to infection.

INTERPRETATION OF DYNAMIC TESTS

In the absence of definitive standards for many of the measurements made during dynamic tests the interpretation is somewhat subjective. Irregular vacuum fluctuations in the source of steady vacuum in excess of 3-4 kPa may be regarded as unsatisfactory. As fluctuation of this magnitude have not been directly associated with increases in new infection it is important to distinguish between optimum operating conditions which remove risk and those which may be associated with new infections.

The amplitude of the cyclic fluctuations recorded at the clawpiece will indicate the start and end of milk flow. This can demonstrate poor let down and over milking. Although attempts have been made experimentally to demonstrate that over milking increases the incidence of mastitis these met with limited success. It has to be concluded that over-milking, whilst regarded as bad practice, is not a major influence on mastitis.

A wave pattern in the recording can indicate alternate flooding and clearing of the system at peak milk flow rates. In some cases this can be corrected by increasing clawpiece capacity, air admission and long milk tube diameter.

Liner slip will be indicated by a momentary change in the cyclic pattern caused by the sudden inrush of air. Liner slip is a function of liner design, cluster weight and vacuum level and stability. Air can also be admitted during machine stripping - the effect in terms of new infection would be similar to that produced by liner slip.

Where the amplitude of the cyclic fluctuation is excessively wide this may affect liner wall movement and prevent satisfactory liner collapse. The absence of the expected amplitude of cyclic fluctuation may indicate failure of the liner to respond to the pressure changes in the pulsation chamber. This will occur when the barrel length of the liner is too short to accommodate the teat and still leave sufficient liner to produce an effective collapse. Failure to collapse can be confirmed by visual observation using a perspex shell.

In many cases the results of the dynamic tests simply confirm what an experienced adviser could predict from the results of the static tests and observations of the operator. However the visual evidence of the recording can be helpful in demonstrating the presence of problems. As experience is gained with dynamic testing and data is accumulated it may be possible in the longer term to set more definitive standards.

REFERENCES

1. WOOLFORD et al 1980. Aspects of milking machine design related to intra-mammary infections. Proc. Int. Workshop Moorepark p45.
2. DODD F H and KINGWILL R G 1960. The effect of the level of vacuum on the incidence of mastitis. Report NIRD p25.
3. JACKSON E R 1970. An outbreak of teat sores in an commercial dairy herd associated with milking machine faults. Vet Record 87 2-6.
4. WILLIAMS et al 1981. Biological responses of the bovine teat to milking. J Dairy Research 48.7.
5. BRAMLEY et al 1978. Investigations on the effect of continuous vacuum milking on new infections. Proc. 17th Annual Meeting National Mastitis Council p 291.
6. REITSMA et al 1981. Effect of duration of teat cup liner closure per pulsation cycle on bovine mastitis. J. Dairy Sci. 64 2240.
7. NYHAN J F 1968. Proceedings of the symposium on Machine Milking p71.
8. GRIFFIN et al 1980. Effect of deflector shields fitted in the milking machine teat cup liner on bovine udder disease. J. Dairy Research 47 1-9
9. NATZKE R P 1978. The relationship between over-milking and new infection. Proc 17th Annual Meeting National Mastitis Council 256-261

INVESTIGATING A HERD MASTITIS PROBLEM

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The following paper describes a mastitis problem occurring on a dairy herd, the steps to investigate it and the corrective action implemented.

The Farm

This is a 220-cow herd, which was established in August 1986 by purchasing stock from a variety of sources. The milking parlour is a Fullwood 18/18 direct to pipeline herringbone, with automatic cluster removers (ACRs), computer flow-meters recording at each milking and computer-operated feeders.

The cows are housed in four straw yards, each yard being filled in calving date order. There is no interchange of cows between yards.

During the winter of 1988/89 cows were fed a ration of silage and approximately 11.0kgs of 20%CP concentrate, plus 15kgs fodder beet from December 1988. The herd is milked three times daily and aims for a yield of around 7000 litres.

The Problem

Calving started in July 1988 and the fresh calvers were housed on full winter rations. There was an increased incidence of clinical cases of mastitis from August 1988 onwards, but cell count remained normal, with a rolling 12-month mean of 254,000 cells per ml (Table 1). Total bacterial counts were very variable. Some were acceptable values, others were extremely high. The herd dropped from Band A to Band B in October, 1988.

Clearly the cost of the outbreak was considerable. A 1986 value for the cost of a single case of mastitis was £40 (1). A case was defined as one cow affected once and a new case occurred if more than five days elapsed between treatments. At an average of 14 cases per month between October 1988 and January 1989, this represented a monthly loss of around $14 \times £40 = £560$. The cost of dropping from Band A to Band B in the peak milk production month of October 1988 cost $134,593 \text{ litres} \times 0.205\text{p} = £275$. Summing the two items, this came to a monthly total of $£560 + £275 = £835$, a figure which clearly could not be tolerated.

Identifying the Cause

The standard approach to such a problem is a bacteriological examination of milk samples, to identify the causative organism(s). Once these are known, and the source of infection has therefore been identified, then control measures can be implemented. However, a careful analysis of records can provide similar information. As it is, perhaps, a novel approach, this will be dealt with first.

The herd is recorded on the DAISY system. Information on all health events is collected at the weekly routine visit and print-outs supplied as necessary. Figure 1 shows that the incidence of mastitis in the herd was quite high. Sixty four cows were affected representing 28% of the herd. There were 96 cases of mastitis on a rolling 12-month basis, i.e. 44 cases per 100 cows p.a. (target = 25 cases/100 cows/year). Figure 2 gives the monthly distribution of cases, with a clear increase from October onwards. Figure 3 shows that it was not a down-calving mastitis problem, but there was an increased incidence at 60-90 days of lactation. Figure 4 indicates that all parities, cows and heifers, were equally affected. Figure 5 gives the number of cases (70) which were 'first time' and the number (20) which needed a second or subsequent (4) treatment. The proportion of cases needing a second or subsequent treatment was reasonably low (20 ex 94 = 21%). This is typical of an environmental mastitis problem, where all cows are equally exposed to the same challenge of infection. For comparison figure 6 shows a herd where 'contagious' mastitis (viz. staphylococci and Streptococcus agalactiae) is a problem. Note the high percentage of repeat treatments and the number of cows which have had four, five or even six cases over a 12-month period.

Although calculated by a computer, much of the information can be very easily extracted from manual records. In fact several herds are still on a manual recording system and such systems were used for several years before computer facilities were available. Careful recording of mastitis data in order to identify the extent of the problem and its cause is therefore a very important part of correction.

Bacteriology was carried out on a proportion of clinical mastitis cases and on bulk milk, the latter in an attempt to identify the organisms responsible for the high total bacterial count. Results of mastitis bacteriology are given in table 2. Bulk milk was tested by our own laboratory, where mixed growths, including Str. uberis were often obtained. Swabs were taken from a variety of sites within the milking plant. The dump line was found to be contaminated, but even when this was thoroughly cleaned the problem still persisted. Consequently ADAS carried out daily TBC sampling from 1-14 November and 22-28 November, 1988. Values obtained ranged from 800 to 60,000 bacteria per ml, which indicated that plant sterilisation was not at fault. Detailed analyses of milks showed that Strep. uberis was the organism commonly responsible for high TBC's and was often the predominant organism in low counts. On one occasion, in January 1989, a TBC of 61,000 per ml coincided with four clinical cases of mastitis.

The combined information from analysis of records, mastitis bacteriology and bacteriological examination of high TBC milk all indicated that environmental mastitis and Str. uberis in particular, was the cause of the problem.

Corrective Action

The knowledge that both clinical mastitis and high TBC were from the same source, prompted rapid action. The straw yards were an obvious starting-point.

1. Stocking density. The yards measure 100' x 40', with a 100' x 30' bedded area, and were designed for 50 cows. At the time of the problem there were 54 cows per yard, an increase of 8%.

2. Bedding routine. Each yard was bedded daily, adding two round bales per yard, with barley straw being preferred to wheat as it is more absorbant. With the increased incidence of mastitis, straw use was increased to three bales per day. The yards which gave the problem were often those with cows at 6-12 weeks after calving, presumably because their higher proportion of 'bulling' cows made the yards dirtier (see Figure 3). These yards were given 3 bales per day until mastitis decreased.

The straw beds seemed to be particularly hot beneath the surface, and it was thought that perhaps earth floors, allowing drainage of excess moisture, led to a drier bed, which then heated excessively. Bed temperatures were checked and were around 40°C, which is considered acceptable although there was one 'hot spot' of 48°C. (On the recording day the ambient temperature in the building was 6°C with an external temperature of 4°C.

The yards were cleaned out and rebedded every six weeks, although it was felt that mastitis incidence was already increasing slightly towards the end of the six weeks.

3. Milking routine. Cows were 'dry milked', that is the teats were dry wiped with paper towel and only the few badly contaminated teats were washed and dried. As environmental mastitis was the major part of the problem, there seemed to be little point in introducing washing for all cows. Milk sediment was less than 1.0mg/l.

Cows were not routinely foremilked to check for mastitis. This discussed, although it was felt that the disadvantages of continually handling the teats and of increased milking times were not warranted. However, in-line mastitis detectors were installed at a readily visible point in the long milk tube, to help detect cows shedding infection. A spray was used for post-milking disinfection.

The dipping of teats prior to milking was discussed, as recently-published work in the United States had indicated that this could reduce the incidence of environmental mastitis by up to 50%. Unfortunately there were no predipping preparations commercially available in Great Britain, and because of the very high cost and possible risks of inhibitory substances contaminating the milk if the standard iodine post-milking dip was used, predipping routines were not introduced.

4. Milking plant. This was carefully checked by bacteriological examination at numerous points, by a dynamic machine test (which revealed a very stable working vacuum of 47kPa) and by an evaluation of the circulation cleaning. The latter showed all metal pipeline and claw components achieving at least 77°C. The installation and component design gave few opportunities for cross-infection.

5. Mastitis susceptibility. There had been a suggestion that cows low in vitamin E/selenium were more susceptible to mastitis. Six cows were blood-sampled on 20 September, 1988 and again on 24

February, 1989. All had normal copper and selenium levels, although deficiency problems had been encountered in previous years.

CONCLUSION

A combined problem of increased incidence of mastitis and a high TBC was found to be related to Str. uberis infection. The most important control measures were the increased use of straw in the yards, regular cleaning and early detection of clinical mastitis. Should the problem recur, the following would be very useful:

- a. from the Milk Marketing Board, early notification of high results by phone, rather than unavoidable postal delays.
- b. also from the MMB, details of the organism(s) involved in a high TBC result
- c. from the dairy hygiene manufacturers, trial work on the value of predipping preparations.

REFERENCE

1. Blowey R.W. (1986) Vet. Rec. 119, 551.
2. DAISY - The Dairy Information System (1989)
Reference Manual, Department of Agriculture,
Early Gate, Reading. RG6 2AT

Table 1: Monthly incidence of mastitis total bacterial counts and milk somatic cell counts - June 1988 - February 1989

	June '88	July	August	Sept	Oct	Nov	Dec	Jan '89	February
Number of clinical cases	2	3	5	5	12	12	14	18	5
Number 1st time cases	1	3	5	4	12	9	9	14	4
Monthly cell count	221	194	232	235	232	240	180	361	263
Monthly TBC	4	10	7	17	29	16	8	11	19

Table 2: Results of Bacteriology on individual mastitis cases

DATE	COW NO.	MASTITIS ORGANISM
9.9.88	530	<i>S. uberis</i>
12.9.89	422	<i>E. coli</i>
14.10.88	6	<i>S. uberis</i>
19.10.88	19	<i>E. coli</i>
21.10.88	A 19	<i>Staph</i>
18.11.88	210	<i>E. coli</i>
2.12.88	354	<i>Staph</i>
2.12.88	24	<i>S. uberis</i>
2.12.88	119	<i>Bacillus spp.</i>
2.12.88	19	<i>E. coli</i>
2.12.88	77	<i>Staph and Strep uberis</i>
6.1.89	383	<i>Strep uberis</i>

Figure 1. Herd disease incidence

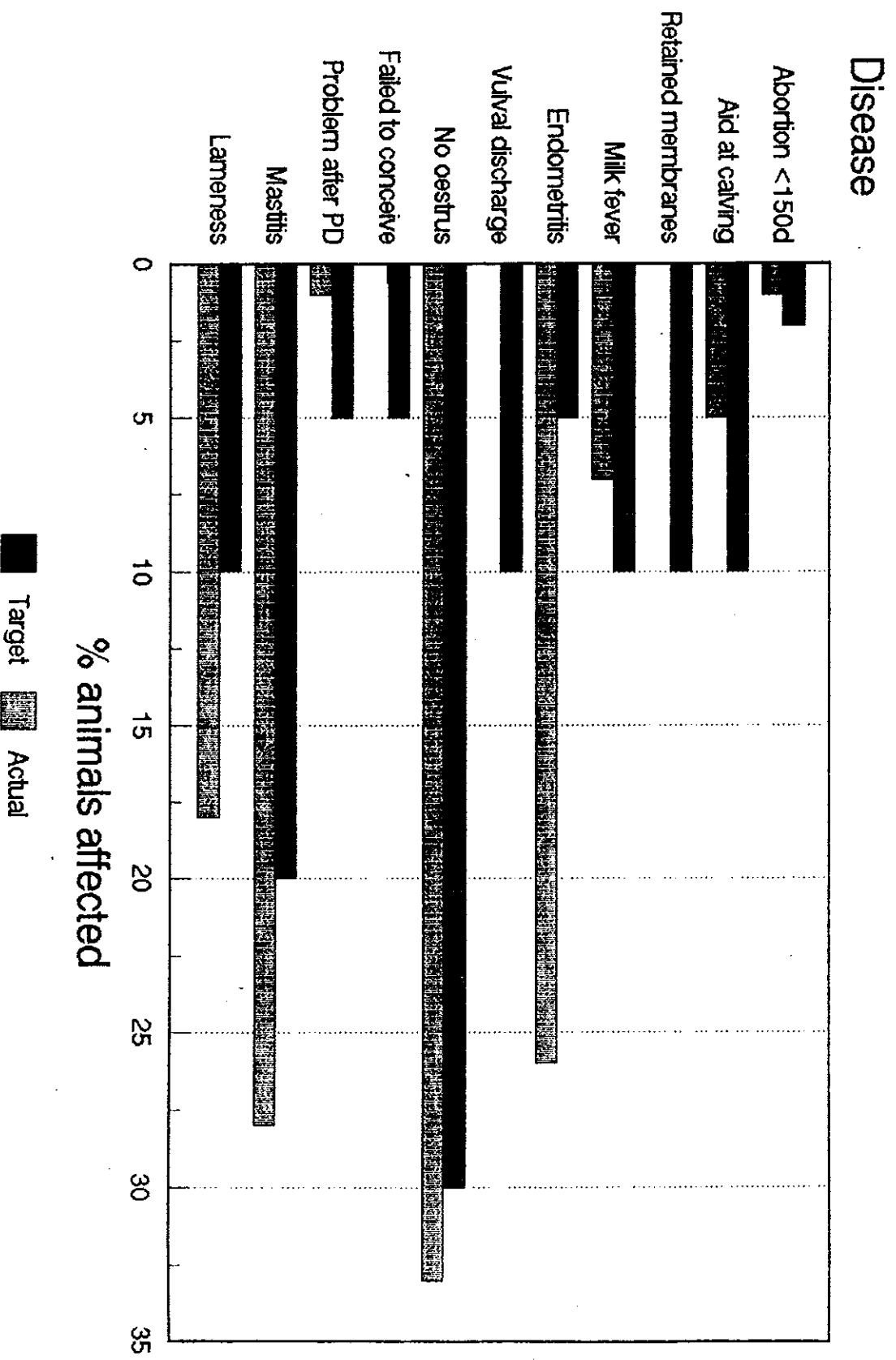


Figure 2. Monthly mastitis incidence

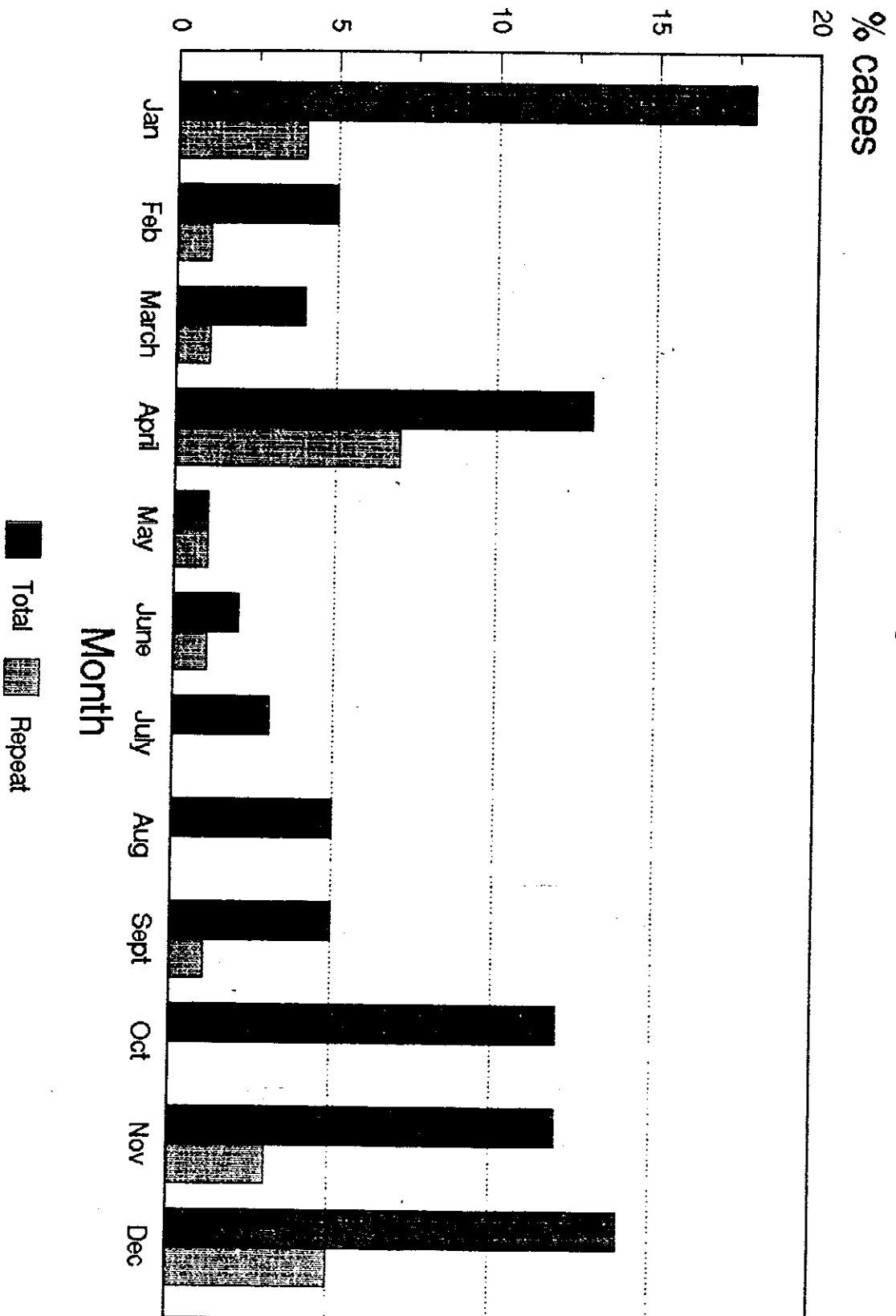


Figure 3. Mastitis - lactation stage

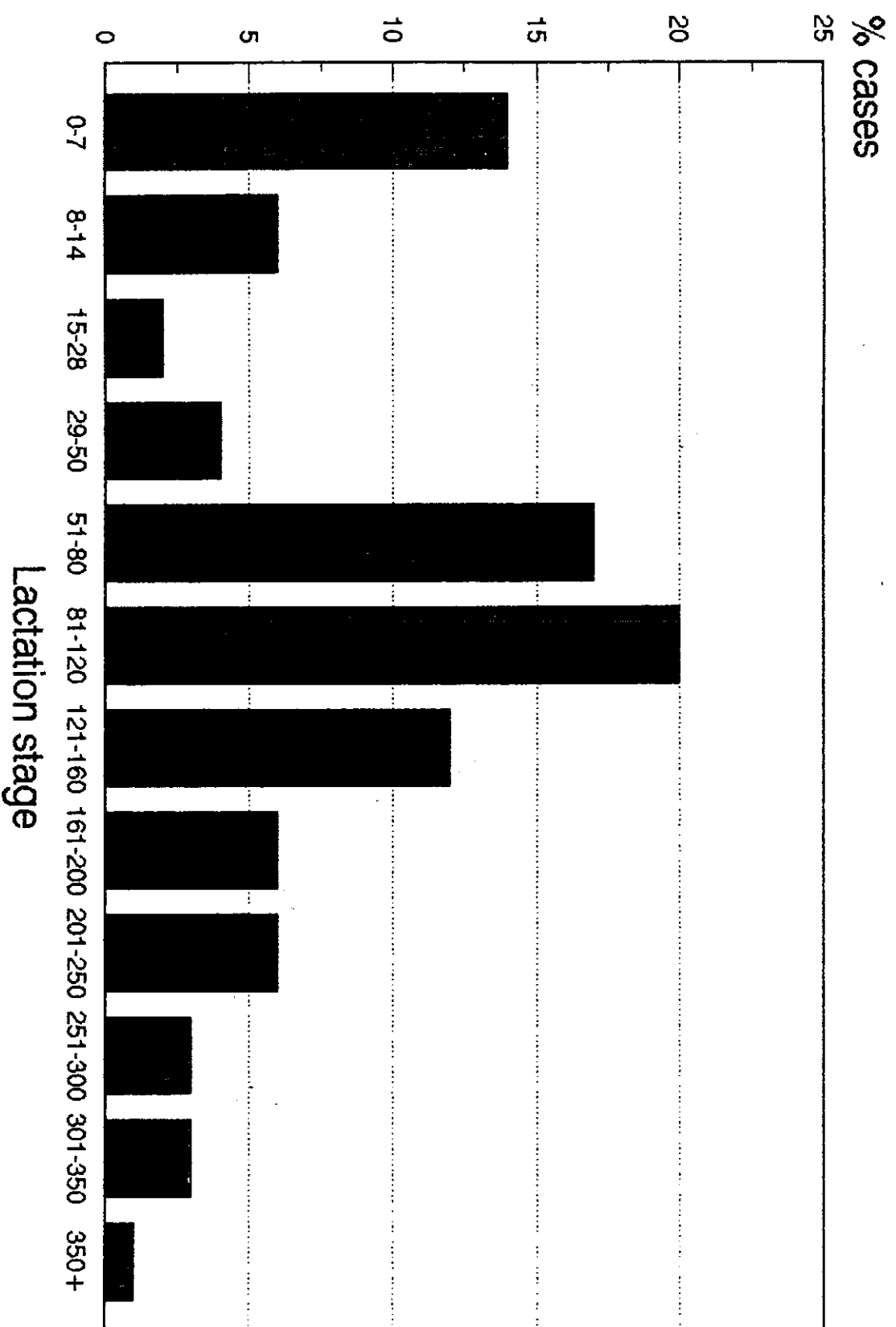


Figure 4. Mastitis - lactation age

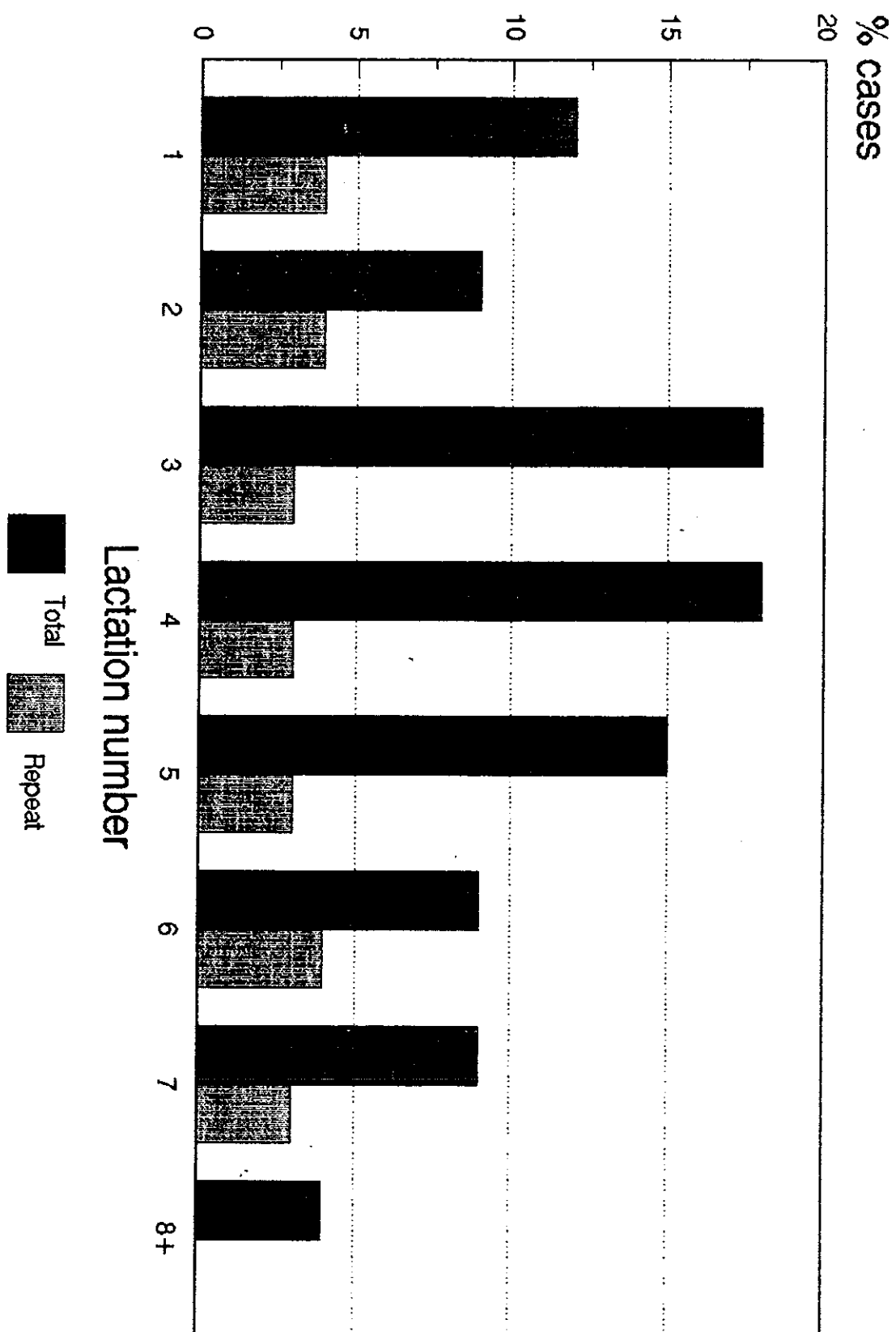


Figure 5. Incidence of recurrent cases

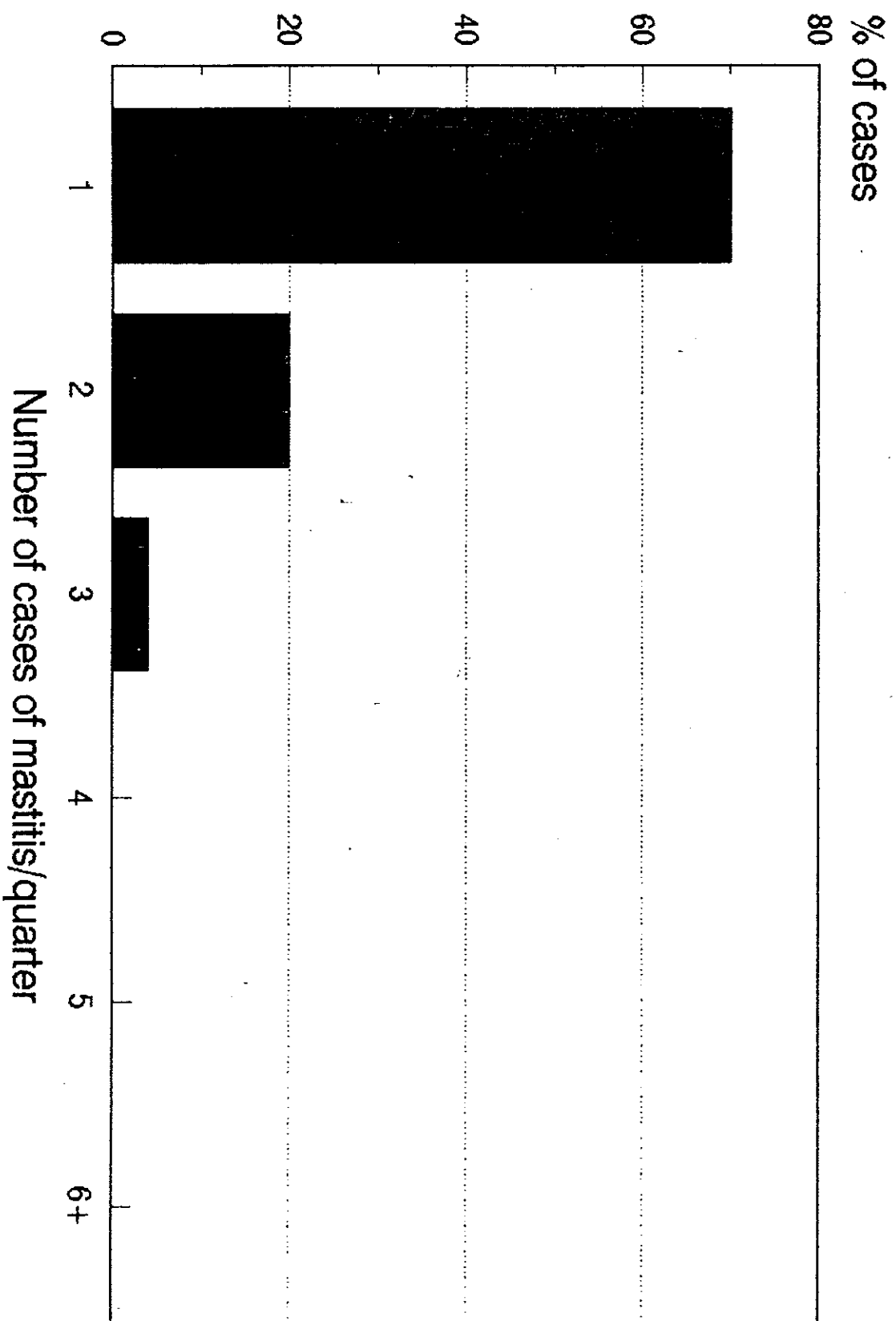


Figure 6. Mastitis cases - recurrence

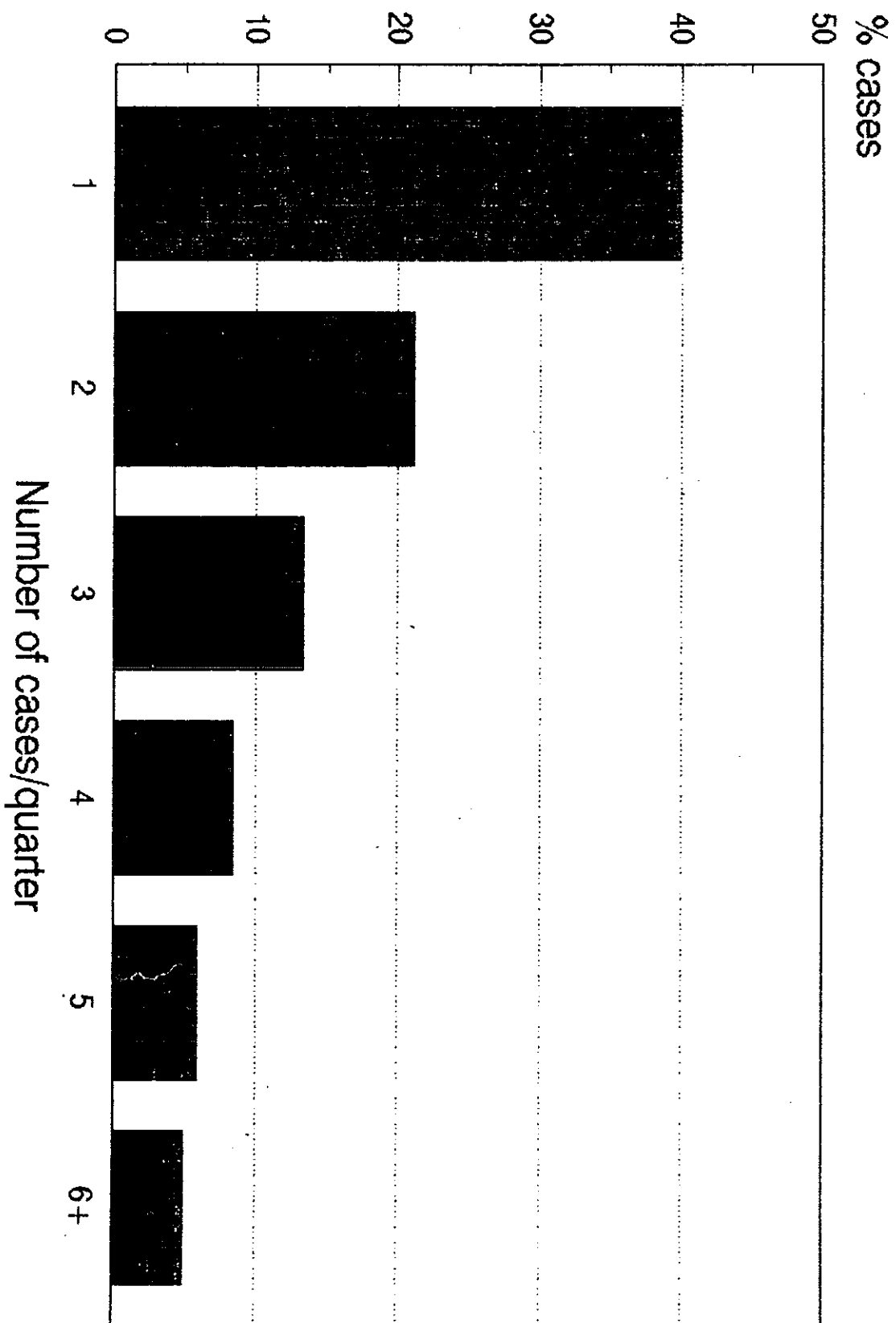
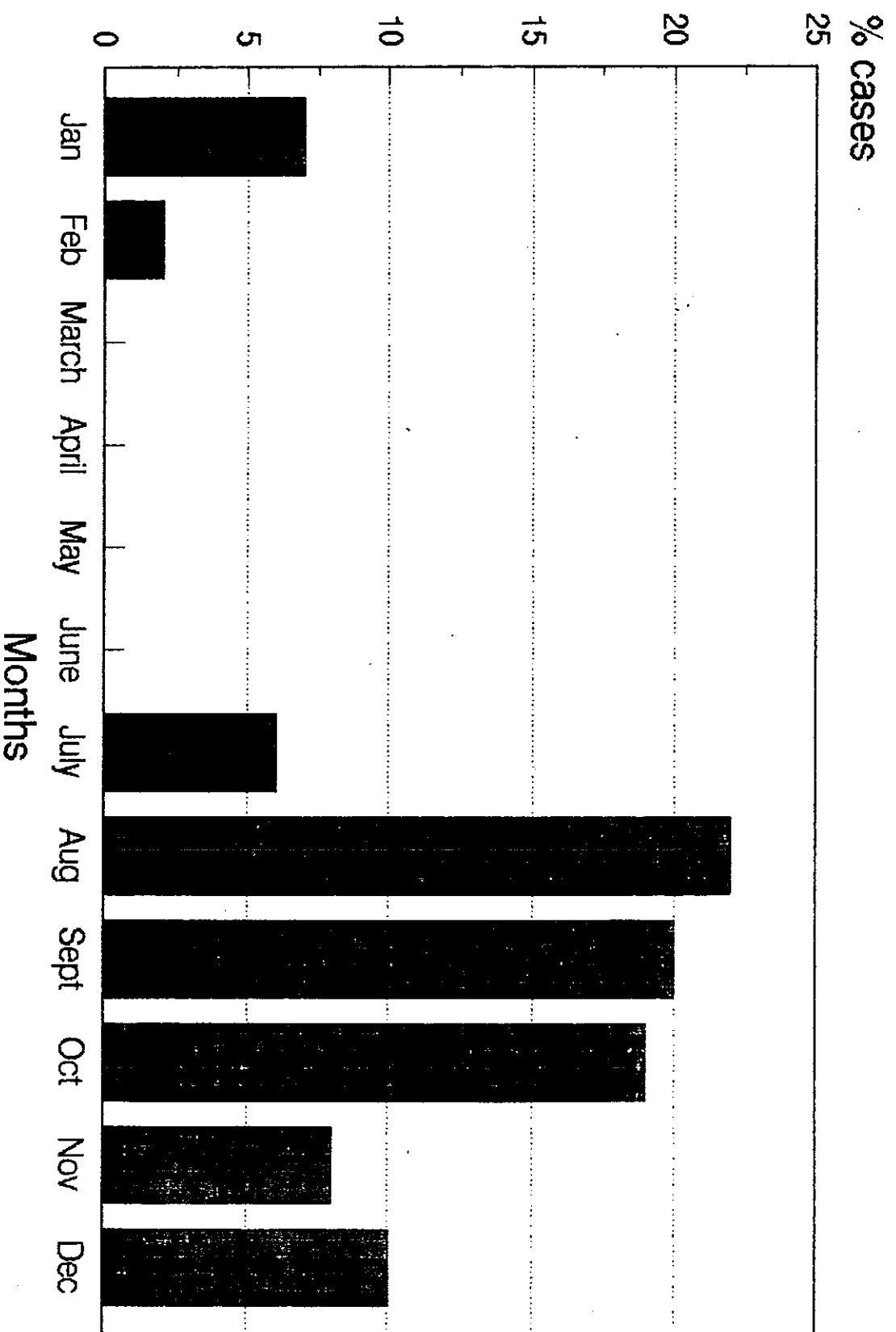


Figure 7. Seasonal distribution of cases



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