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A Review of Milking Machine-Induced Teat-End Lesions and Susceptibility to Mastitis

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Abstract

Milking machine-induced teat lesions are commonly thought to be related to increased susceptibility to new intramammary infections. Many of the mechanisms of dimensional and physiological changes in the teat during milking, such as congestion and edema, are quite well understood. Loss of keratin from the teat canal during milking is believed to lead to increased susceptibility to penetration and colonization by bacteria, but this effect is counterbalanced by the apparent protective effect of the proliferative response to mechanical stress on the teat. This leads to increased regeneration of keratin in the teat canal and hyperkeratosis of the teat-end. Severe teat-end lesions clearly increase susceptibility to new intramammary infections, while some degree of mild hyperkeratosis exerts a protective effect. There is conflicting evidence as to whether moderate hyperkeratosis of the teat-end affects susceptibility to new mastitis infections. Machine-induced teat-end changes may increase susceptibility to mastitis in the presence of other management failures, such as poor milking hygiene.

Résumé

On croit que la susceptibilité aux nouvelles infections intramammaires s'accroît lorsque des lésions sont causées aux trayons par les machines à traire. Les changements physiologiques et dimensionnels du trayon lors de la traite, tels la congestion et l'œdème, sont pour la plupart bien compris. La perte de kératine dans le canal du trayon durant la traite pourrait quant à elle faciliter la pénétration et la colonisation du trayon par des bactéries mais cet effet semble être compensé par la protection apparente reliée à la réponse proliférative suivant le stress mécanique sur le trayon. Ceci permet une régénération accrue de la kératine dans le canal du trayon et l'hyperkératinisation du bout du trayon. Les lésions graves du bout du trayon accroissent nettement la susceptibilité aux nouvelles infections intramammaires bien qu'un certain degré d'hyperkératinisation aurait un effet protecteur. L'effet de l'hyperkératinisation modérée du bout du trayon sur la susceptibilité aux nouvelles infections intramammaires n'est pas bien établi. Les changements causés au bout des trayons par les machines à traire peuvent accroître la susceptibilité à la mammite lorsqu'ils sont associés à d'autres déficiences dans la gestion, comme par exemple une pauvre hygiène de traite.

Introduction

Mastitis continues to be one of the most important diseases on dairy farms. In one US study the lactation frequency for clinical mastitis in second-lactation cows was 0.22, and in first-lactation cows it was 0.27.[5] Losses from subclinical mastitis are estimated to cost $110/cow averaged across all dairy herds in the US.[15] Moreover, the major risk factor for antibiotic residues in bulk milk is the contamination of milk from the treatment of clinical mastitis, or the use of antibiotics in dry-off treatments at the end of lactation.

Because virtually all mastitis infections are caused by pathogens gaining entry to the mammary gland via the teat canal, the teat is a critical part of the cow's defenses against intramammary infection (IMI). The milking machine, especially when not functioning properly, may be involved in the etiology of mastitis by transmitting infections between cows or quarters, or by changing the condition of the teat and teat-end in such a way that new IMI are more likely to occur.[20,22,52] Since the teat-end defends against bacterial penetration and possible resulting infection of the mammary gland, it may be considered as the first-line of defense against IMI.[43] The type and extent of teat tissue damage may

influence the degree of bacterial contamination of the teat orifice and penetration of bacteria through the teat canal.

The purpose of this paper is to review the literature on the role of mechanically induced teat-end lesions in the development of mastitis in dairy cows.

Teat and Teat Canal Conformation

The teat of the cow has been characterized as a membranous tube that forms the passageway through which milk is removed.[16] Its shape varies from cylindrical to conical.[16,50,55] The length of the teat varies considerably and reported values range between 3 and 18 cm.[20,50] On average, the front teats are 22.5% longer than the rear teats.[20] The shapes of the tip of the teat have been categorized as conical, pointed, rounded, disk shaped, flat, or funnel shaped.[1,4,20,32] The teat wall contains an abundance of elastic connective tissue with layers of muscle fibers,[55] heavily permeated by blood vessels and lymph vessels, and profusely innervated. The teat-end includes the teat canal or streak canal, surrounded by a circular sphincter muscle.[55] The teat canal acts as an elastic valve-like opening.[55] Its length may vary between 8 and 18 mm.[20,48,50] Cells that line the teat canal contain keratin, a waxy material that includes chemical substances such as cationic proteins, xanthine oxidase, and long chain fatty acids with bactericidal or bacteriostatic properties.[12,27,28] Bacteria which enter the teat canal adhere to the keratin cells and are flushed out of the teat with the cells during the milking process. The keratin material also helps to seal the teat-end between milkings and to inhibit the growth of bacteria.[7,12] The physiological process of formation of a keratin plug in the teat canal after drying off appears to constitute a major defensive mechanism preventing the establishment of new IMI in the dry period,[1] although up to 22% of teats may still not have sealed by six weeks into the dry period.[13] The teat also contains a structure known as Fuerstenberg's rosette[5] that is located at the proximal end of the teat canal. It is made up of loose folds of membrane that smooth out as milk accumulates in the udder. This membrane aids in blocking the escape of milk between milkings, but is susceptible to damage by mastitis infusion nozzles or teat cannulae.

Effect of Machine Milking on the Teat

A number of changes are induced in the teat by machine milking. These include changes in the dimensions of the teat and its structures, physiological changes such as congestion and edema, loss of keratin from the teat canal, and longer term changes such as hyperkeratosis of the teat-end. There has been considerable discussion and investigation of the role that these machine-induced tissue effects play in susceptibility to bacterial colonization of the teat canal and to new mastitis infections.

Teat Dimensions

When the teat is exposed to vacuum, the teat sinus will expand until the surface of the teat is supported by the barrel of the inflation or until the connective and elastic tissues within the teat are fully stretched. The teat increases in length during machine milking.[18,26] Hillerton and co-workers documented an increase of 5 mm in teat length, an increase which was not affected by overmilking,[26] although in other studies teat length continued to increase linearly when milking was continued after milk flow dropped to 0.2 kg/min.[18] Teat length also increases as cows age from first to third lactation.[20] Not only does teat length increase during milking, but so does the length of the teat canal. Ultrasonographic scanning of teat tissues before and after milking revealed that the teat canal length deviated most at two hours after milking, compared with before milking.[18,43] Teat canal length and teat-end diameter, which were both increased after milking, took a considerable time (> 8 hrs) to revert to their original length, which may have implications for teat health when milking intervals are shortened.[43] The teat-end is less pliable than the rest of the teat, but the fact that on a relative basis teat canal length is increased more than teat width may suggest that the teat-end is under greater stress in length than across its width.[43] These dimensional changes increase with higher vacuum levels[23] and with yield per milking, most likely as a result of increased milking times in high-yielding cows.[18]

Teat canal diameters measured at three locations (distal, middle and proximal) were reported to have average open values of 0.49, 0.64 and 0.97 mm, respectively.[20] The teat canal diameter increases with increasing lactation number and advancing lactation.[20] Using ultrasound scanning techniques, the teat canal diameter of rear teats took three hours to recover after milking, and the front teats took eight hours.[43] Larger teat canal diameters are a risk factor for high somatic cell counts[31,40] and the post-milking delay in closure of the teat canal is important because of the increased penetrability of teats after milking.[43,51] The diameter of the teat canal is also influenced by teat-end shape.[20] Teats with disk or cone-shaped ends had wider teat canals than those with pointed or round ends.[1,20] It has been reported that mastitis infections are more likely to occur if the distal end of the teat canal has a wide diameter,[1] and flat or inverted teat-ends may be associated with higher SCC, through a dependency on increased teat canal diameter.[31] However, Chrystal and Seykora reported that teat-end shapes did not significantly affect somatic cell scores, but suggested that in their study the widespread use of pre- and post-milking teat dips

may have negated any influence by teat-end shape on somatic cell scores.[10]

When a vacuum of 50 kPa (15 in Hg) is applied to the teat during milking, there is congestion at the teat-end after 0.1-0.3 seconds.[20] This congestion reduces the teat canal open diameter by about 30% and reduces milk flow rate. The occurrence of congestion after about 0.1-0.3 sec of flow in a pulsation cycle, and the ensuing effect on flow rate, explains why pulsation rates of 50-60 cycles/min are regarded as optimal.[35]

Teat wall thickness also varies with different settings of the milking machine, such as vacuum level, pulsation and type of liners used.[22,58] Teats milked with wide-bore liners generally show an increase in teat wall thickness as a result of increased congestion and edema compared to teats milked with narrow-bore liners, although narrow-bore liners tend to have more teat-cup liner slips.[38] Ultrasound scanning also revealed that teat wall thickness took six hours for recovery after milking.[43]

Milking Machine Induced Lesions of the Teat

Visible or palpable teat lesions occur mainly at the teat-end, and less frequently on the teat barrel.[20]

Teat Chaps / Abrasions

An edematous ring or "garter mark" forming at the base of the teat during milking is a consequence of the vacuum in the mouthpiece chamber of the liner and the design of the liner.[25,40] Linear skin cracks, extending transversely around the teat base, which are initiated by milking machine action, can be aggravated by environmental factors to form chaps.[17] These teat abrasions are usually confined to the area where the mouthpiece of the inflation contacts the teat.[46]

Congestive and Bruising Effects on Teat Tissues

The effects of vacuum on the fluid dynamics of the teat result in some of the most significant machine-mediated lesions.[54] During machine milking, blood and tissue fluids pool in the portion of the teat exposed to vacuum.[23,29] When teats are machine-milked using generally accepted milking machine vacuum and pulsation settings, some congestion and edema of the teat-end occurs.[3,18,22,23] Although thickening of the teat-end due to congestion and edema may be within physiological limits,[29] it has been proposed that the changes in the tissues of the teat-end around the streak canal may reduce the resistance of the teat to bacterial invasion.[43]

The primary function of pulsation is to periodically squeeze the teat and reverse the pooling effects. If the compressive forces applied during inflation collapse are inadequate to force the collected blood and tissue fluids from the teat, congestion, pain and edema will occur.[26] Petechial hemorrhages at the teat-end, and lesions such as "black spot" or "black pox" at the opening of the teat canal, are attributable to pulsation failures,[20,41] such as those resulting from the use of short teat-cup liners,[34] overmilking, or insufficient collapse of the liner during the massage phase.

Hemorrhages can also occur in the teat cistern and Fuerstenberg's rosette, but less frequently than in the teat wall.[20] Metaplasia, which is an abnormal transformation of fully differentiated cells of the epithelial lining of the teat cistern, can be seen in 20.8% of teats.[20] These changes most likely reflect continued mechanical stress on the tissue of the teat. Cows milked by machine have more than twice the frequency of metaplasia compared to non-milked or hand-milked cows. In addition, metaplasia is reported to be a feature of the epithelial lining of the teat in infected quarters.[44]

The forces applied to the teat tissue during machine milking are controlled by the pulsation characteristics of the machine. The pulsation cycle is conventionally described as having four phases: a) increasing vacuum phase; b) maximum vacuum phase; c) decreasing vacuum phase and d) minimum vacuum phase.[47] These phases direct the physical forces transmitted from the milking machine through liner movement to the teat tissue. Variation of these measures from generally accepted norms has been associated with udder disease. For example, Osteras *et al* reported a positive association between a shorter d phase and a high incidence of teat lesions requiring veterinary treatment. They suggested that in high line milking systems the d phase should be at least 250ms, and preferably 300ms.[47] Effective pulsation, as defined by Williams and Mein,[56,57] requires that liners apply a compressive load, also known as over-pressure, of about +10 kPa (3 in Hg) to the teat, for a minimum time of 0.25sec at a rate of about 55-60 cycles/min.

Effective pulsation at the distal end of the teat is needed to minimize the risk of teat-end damage. A milking vacuum of 40-50 kPa (12-15 in Hg), combined with a relatively low pulsation chamber vacuum (<10 kPa; 3 in Hg) and a short duration of liner closure (< 0.25 sec/pulsation cycle),[50] can result in bruising of the teat-end by the slapping action of the liner.[17] Similarly, teat-cup liners mounted in the shell under higher tension put greater compressive loads on the teat, resulting in mechanical stress on the teat-end.[9,37] Moreover, increasing milking duration and over-milking increase the probability of teat tissue damage.[25] It has been shown that over-milking for five minutes at only four milkings causes measurable tissue damage.[20] An increase in teat apex thickness of more than 5%, as measured by a cutimeter,[24] was positively associated with infection and with new mastitis infections.[58]

Keratin Dynamics and Hyperkeratosis

Changes in the Amount of Keratin in the Teat Canal

Keratin is lost from the teat during milking, more

so when teats are milked with pulsation than without.[8,33] To a certain extent, this keratin loss is offset by effective pulsation in which the collapsed liner causes a compressive load on the teat. This has a component acting vertically upwards, which may conserve the keratin in the teat canal.[34] Up to 40% of the keratin content of the teat canal is lost during milking,[8] but is replaced relatively quickly.[9,34] In experiments in which the full complement of keratin in the teat canal is mechanically removed, keratin weights return to their original value in less than three days.[6]

Keratin loss is affected by milk production, as greater shearing forces within the teat canal caused by increased milk flow remove more keratin.[6] Milking with pulsation removes 32% of the keratin that lines the teat canal at each milking.[33] Williams *et al* postulated that the shear stress on the teat canal wall caused by normal milk flow velocity of eight meters/second gave rise to extrusion of teat canal keratin.[57] On the other hand, Lacy-Hulbert and her colleagues[33] proposed that keratin loss is controlled by liner compression rather than by the rate of milk flow through the teat canal, and that, as well as pulsation being responsible for keratin loss, it is also a positive stimulus to the rate of keratin regeneration.

Keratin regeneration is also affected by liner tension. Capuco *et al*[9] showed that at the end of experimental periods of both 10 and 30 days, the quantity of keratin was increased 10-20% in teats that were milked using liners under a higher tension compared with teats that were milked using liners under a lower tension. In the same experiment, histopathological examination of the teat-end indicated a mild increase in the extent of hyperplasia and hyperkeratosis caused by milking with liners under high tension or overmilking. This correlated with a worsening of teat-end scores.

Hyperkeratosis

Teat-end lesions and hyperkeratotic abnormalities at the teat-end are very common and are seen in virtually all herds. Generally, machine induced teat-end lesions appear in three forms: as a smooth or roughened raised ring of tissue, or callus, around the orifice; as an actual loss of epithelium; or as formation of a scab.[52] A variety of terms have been used to describe these lesions, such as eversions, erosions, prolapses, fibrous rings and epithelial hyperplasia,[20,52,53] although some of these expressions, such as teat-end eversion or prolapse, do not accurately describe the common proliferative lesions. The epithelium of the teat canal consists of three layers, the most superficial being the *stratum corneum*, separated from the deepest layer, the *stratum germanitivum*, by the *stratum granulosum*. This epithelial structure is supported by a papillary layer, which consists of connective tissue. The direction of the papillae in this layer determines the direction of keratinization in the teat canal. Keratinization of the teat-duct orifice is a normal physiological adaptive process during the initiation of lactation.[41] However, repeated application of mechanical force to the epithelium stimulates keratinization. Keratinization which has progressed beyond a certain point is considered pathological keratinization or hyperkeratosis, since callus type lesions do not progress from mild to severe, or to erosions and scabs without continued mechanical stress.[14,20] The term hyperkeratosis, as applied to the teat-end, describes a thickened smooth keratin ring or extending fronds of keratin around the teat orifice.[19] A generally accepted field scoring system for teat-end hyperkeratosis has been developed (Table 1).[45]

It is believed this callused condition of the teat-end is due to the compressive forces applied to the teat by the inflation during mechanical milking.[54] When milk flows through the teat canal, it creates a shear stress that tends to drag the keratin that lines the teat canal out of the teat. During effective pulsation, however, these forces are interrupted and partially counteracted by the

Table 1. A field scoring system for teat-end hyperkeratosis.

N	No ring The teat-end is smooth with a small even orifice. This is a typical status for many teats soon after the start of lactation.
S	Smooth or Slightly rough ring A raised ring encircles the orifice. The surface of the ring is smooth or it may feel slightly rough, but no fronds of old keratin are evident.
R	Rough ring A raised, roughened ring with isolated fronds or mounds of old keratin extending 1-3 mm from the orifice.
VR	Very Rough ring A raised ring with rough fronds or mounds of old keratin extending 4 mm or more from the orifice. The rim of the ring is rough and cracked, often giving the teat-end a "flowered" appearance.

Adopted from Ohnstad *et al*.[45]

collapse of the inflation.[35] Although milking with liners under high tension can be shown to stimulate an increased keratin content of the teat canal after 10-30 days,[9] it is doubtful that hyperkeratosis is entirely related to machine milking.[4,20,36,38,45,54] This condition can be found in hand-milked cows and beef cows.[20,54] However, hyperkeratosis is more prevalent in herds that are over-milked, and is more prevalent at the peak of lactation than during stages of low production.[54] Neijenhuis *et al* noted that cow factors such as teat-end shape, days in milk, milking speed and parity were also associated with the degree of teat-end callosity, and the probability of the callosity ring becoming rough.[41] These authors also observed that pointed or rounded teat-ends showed more callus than inverted teat-ends, and longer machine-on time resulted in a higher probability of the callosity ring becoming rough. There is evidence that the total time per day when milk flow is less than 2.2 lb (1.0 kg)/min has a profound effect on the level of hyperkeratosis,[45] leading to recommendations and milking practices that limit machine-on time.[11] Other cow factors such as teat position, teat length and milk production have also been shown to have a relationship with callused teat-ends.[2,53] Mein *et al* concluded that the major factors influencing the development of hyperkeratosis are long pointed teats, slow milking, high-producing cows, stage of lactation, parity, weather conditions, chemical irritation and cluster removal time.[36]

Hyperkeratosis and Susceptibility to Mastitis

An association between teat-end condition and clinical mastitis is often assumed in the field. Severe teat-end lesions (erosions or scabs) are positively associated with the prevalence of subclinical mastitis.[30,52,53] Petechial hemorrhages, black spot and black pox are associated with increased new infection rates.[34] However, the relationship between various degrees of hyperkeratosis of the teat-end and the rate of udder infection is more controversial. Some studies have found no correlation between teat-end hyperkeratosis scores in well managed herds.[19] Other studies have suggested that quarters with teats with smooth or rough chronic rings of very mild or mild severity tend to have a lower level infection than those with normal teat-ends,[52,53] although there was a much higher level of infection in quarters with ulcerative or scabbed teat-end lesions, traumatized teats or teats that leaked milk. A more recent study has produced similar results, indicating there is a significantly lower risk of clinical mastitis in quarters with mild or moderate smooth rings compared to quarters having no ring or rough rings.[41] On the other hand, in the same study mid-lactation cows suffering clinical mastitis had higher teat-end scores than healthy cows.[41]

In a longitudinal study of 15 herds with a total of 2157 cows, conducted to examine the relationship between teat-end callosity and incidence of clinical mastitis, the level of teat-end callosity thickness was higher in the clinical mastitis quarter than in the lateral healthy quarter within a cow before, and particularly during and after, occurrence of clinical mastitis.[42] Hamann measured the changes in teat-end thickness in relation to different milking systems, and investigated this in relation to infection risk.[21] In his study, there were marked differences in the development of teat-end thickness between different milking systems. A marked increase in teat-end thickness (20%) in one system or decrease (-5%) in another system were combined with increased new infection rates.[21] Capuco *et al* studied the effect of tension of teat-cup liners on teat-end condition and investigated the quantity of keratin in the teat canal.[9] Udder health was evaluated by bacteriological analysis and somatic cell counting. Across these short-term studies udder health was not adversely affected by milking with liners under high tension, although milking using liners under high tension stimulated proliferation within the teat-end, as evidenced by hyperplasia and hyperkeratosis.[9]

Finally, in an interesting study comparing teat-end hyperkeratosis scores, somatic cell counts and milk mastitis pathogens between disinfected and non-disinfected teats of the same cows, Gleeson *et al* showed that among cows with disinfected teats no correlation could be found between teat-end scores and somatic cell counts. Among non-disinfected teats there was a significant correlation.[19] This suggests that there is an important interaction between teat-end hygiene and cleanliness and the presence of hyperkeratosis in the risk of acquiring intramammary infections.

Conclusions

The teat-end is a critical part of the cow's defenses against IMI, guarding against bacterial penetration, colonization of the teat canal and infection of mammary tissue by mastitis organisms. The milking machine causes changes to the teat and teat-end which are, for the most part, quite well understood. A number of changes in the dimensions of the teat and its structures occur which are influenced by vacuum level and duration of milking. Some of these changes persist for hours after removal of the inflation at the end of milking. The application of milking vacuum to the teat leads to congestion and edema of the teat-end, and when pulsation failures occur, to hemorrhage of the teat-end and such lesions as "black spot."

The keratin which lines the teat canal has important properties which protect the teat from bacterial penetration and colonization. Keratin is lost from the teat during milking as a result of liner compression and the shearing forces engendered by milk flow. The me-

chanical forces applied to the teat during milking that are responsible for keratin loss are also a positive stimulus to the rate of keratin regeneration.

Research on the influence of the milking machine on teat condition and susceptibility to new IMI provides some guidelines for the veterinary practitioner, but leaves a number of questions unanswered. For instance, although replenishment of teat canal keratin lost during milking is thought to be important in the prevention of new IMI, and there is some information on the rate of keratin regeneration, there is not sufficient information available to recommend minimum intervals between milkings.

Although mild hyperkeratosis provides some protection against the establishment of new infections, at some point the increasing severity of these changes puts the udder at greater risk. Grade S teat-ends (smooth or slightly rough ring) appear to be less susceptible to new IMI than teats with no visible ring (Grade N). On the other hand, more severe teat-end hyperkeratosis (Grade R or Grade VR) may not *per se* increase the risk of new infections, but is likely to make the teat-ends more difficult to clean and disinfect during pre-milking udder preparation. If there are deficiencies in teat cleaning and sanitization, then rough hyperkeratotic teat-ends are likely to increase the risk of new infections. Severe, machine-induced teat-end lesions such as black spot, abrasions and hemorrhages are, on their own, clear risk factors for new IMI.

Development of severe hyperkeratotic lesions can be minimized by making sure that the d phase (massage phase) is at least 250ms, that cluster removal occurs when milk flow reaches 2.2 lb/min (1.0 kg/min), and that teat skin is kept in good condition by avoiding chapping due to cold weather or chemical irritation from teat dips. Vacuum levels; liner bore, length and tension; and liner replacement schedules are also important factors which are under management control, and should be reviewed when investigating causes of hyperkeratotic teat-ends. Attention to these items may help to minimize the influence of factors that cannot be controlled, including teat shape, stage of lactation, parity and production levels.

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Abstract

The Tuberculin Test

Monaghan M.L., Doherty M.L., Collins J.D., Kazda J.F., Quinn P.J.
Cattle Practice 13(4):337-345, 2005

Tuberculin tests in general use today rely on the response to intradermal injections of tuberculin with assessment of the injection site for swelling at 72 hours post injection. Estimates of the sensitivity of tuberculin tests range from 68-95% while specificity is estimated to be 96-99%. The sensitivity of the test is affected by the potency and dose of tuberculin administered, the interval post-infection, desensitization, deliberate interference, post-partum immunosuppression and observer variation. Specificity is influenced by sensitization as a result of exposure to *M. avium*, *M. paratuberculosis* and environmental mycobacteria and by skin tuberculosis.

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with MetaStim®

Serious Protection

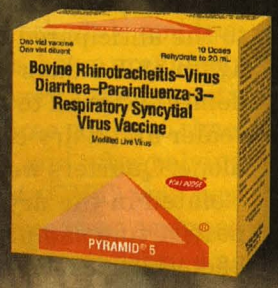
Type I & II BVD Protection

Two specifically selected strains offering broad protection and increased immune response.

Proven Safety

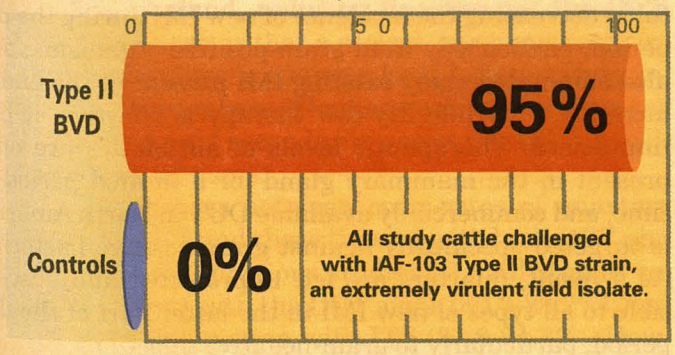
Licensed by the USDA for Sub-Q administration and proven safe when used according to label directions.

Stimulating Results Against BVD



Type II Respiratory Challenge Results[1]

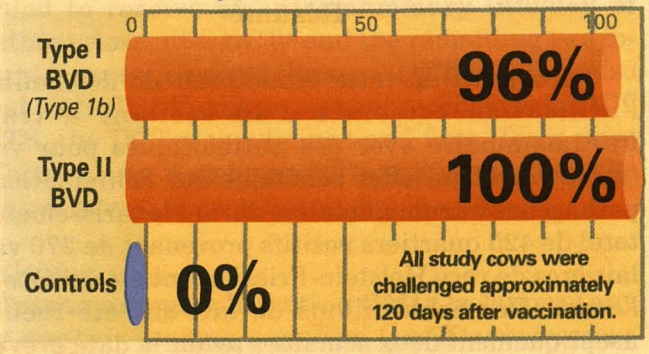
Protection from Disease
(sum of all clinical parameters)



***95%** of PYRAMID 5 vaccinated calves showed no clinical Type II BVD signs following virulent challenge.*

BVD Fetal Challenge Results[1]

% fetuses protected from persistent infection



Up to 100% fetal protection against Type I and Type II BVD challenges.

1. Data on file.

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