ERADICATION OF VIRULENT FOOTROT FROM NEW SOUTH WALES

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Introduction

Before 1788 there were no domestic livestock in Australia. Inevitably, when farm animals were imported from Europe, some of their endemic diseases were imported with them. Footrot of sheep was one of these and there are reports of its presence in the Sydney region from the early nineteenth century. As sheep spread across the country so did footrot and by the early part of the twentieth century it was endemic in all environments favourable to its development and persistence. Following the introduction of superphosphate fertilizer, the development of improved pastures and increased stocking rates, footrot became more prevalent and severe. It was recognized as a major constraint on production and money began to be invested in research designed to remove or reduce this constraint. The first phase of this research (Beveridge 1941) culminated in the identification of the bacterium responsible for transmission of the disease and the finding that this organism (Fusiformis nodosus; now Dichelobacter nodosus, Dewhirst et al 1990) has a very limited capacity to survive other than in affected animals. Other organisms constantly present in lesions, including fusiforms and treponemes, had a secondary role. The principle on which eradication of footrot could be achieved was established - the removal all cases of disease in a flock either by successful treatment or removal and the placement of healthy animals in an environment free of affected animals for at least seven days.

From that time individual flock owners applied this principle - the Beveridge plan - and achieved complete eradication of footrot from their flocks. Nevertheless some who attempted eradication failed and research into the epidemiology of the disease, better treatments and prevention methods continued. It became apparent from 1960 that there was a spectrum of virulence in isolates of D nodosus (Thomas 1962, Egerton and Parsonson1969, Stewart et al 1982) and that clinical presentations of footrot were often benign. It was also found that the strains from benign disease in sheep also affected the feet of cattle. Cattle were therefore a reservoir for the benign form of the disease (Egerton and Parsonson 1966, Laing and Egerton 1978, Wilkinson et al 1970)

The role of the environment in promoting or limiting the development of footrot was evident from the first studies on footrot in Europe - so much so that many believed that excessive exposure to water was alone sufficient to cause the disease. Research in France and England eventually demonstrated it was contagious. Roberts and Egerton (1969) demonstrated the role of a wet environment in predisposing the interdigital skin to invasion by organisms other than D nodosus. They suggested that these organisms were essential in the pathogenesis of footrot and acted in synergy with D nodosus. An ambient temperature sufficient to maintain continuous blood flow to the skin of the digits was also necessary. Graham and Egerton (1968) suggested that by consideration of rainfall and temperature, periods suitable
for transmission of footrot could be predicted and management of footrot programs arranged accordingly.

Initially treatment of footrot cases depended on the topical application of antibacterial agents. For optimum effect all necrotic tissue in the sensitive laminae needed to be exposed by removing overlying horn. This was done either with sharp knives or secateurs. Inevitably considerable pain was inflicted on sheep and this process sometimes caused unnecessary damage to hoof structures. Demonstration that parenteral antibiotics were effective in the treatment of footrot removed the need for the intensive paring associated with topical treatments (Egerton et al, 1968). Cost and the reduced efficacy of parenteral drugs when used in wet environments were limiting factors in the use of this approach.

In those flocks where control rather than eradication was the objective, walk through foot bathing was found to achieve reasonable reduction of prevalence. Timing of footbathing to coincide with predictable periods of transmission increased the efficacy of this approach.

Earlier the most common drug used in footbaths was formalin but this has largely been replaced by solutions of zinc sulphate with or without wetting agents. Vaccines against D. nodosus became available in the latter part of the twentieth century and offered another method of reducing prevalence. The duration of immunity conferred by multivalent (serogroup) vaccines is limited due to antigenic competition and revaccination needs to be done to precede anticipated periods of transmission. Where there are limited numbers of strains present in a flock, vaccines based on those strains are highly effective prophylactically and therapeutically (Egerton et al 2002).

In 1987, in New South Wales, the major sheep growing state in Australia, some sheep owners considered that sufficient knowledge and resources were available to establish and execute a program designed to eradicate

footrot from the state. Successful eradication by individuals and the prior success achieved in controlling footrot in the New England Footrot Protected Area established in 1969 supported this idea. A benefit cost study by Carmody (1981) demonstrated considerable community and social benefit from the eradication of footrot in this northern area of the state. Regulatory powers were put in place to prevent the re-introduction of footrot into the area and quarantine and eradication programs were required when footrot was detected. Walker (1997) described results achieved in the first eight years of the NSW footrot earication program. This paper describes the program and progress since then.

The sheep industry in NSW

In 1988 when the program started there were approximately 56.4 million sheep in 45,399 flocks in NSW. These flocks were distributed in three definable sheep raising regions based on rainfall. These are: high rainfall sheep and cattle grazing districts of the highlands and their western slopes (>600 mm. annual rainfall), sheep and grain districts in the slopes and eastern plains (400-600 mm) and the low rainfall, pastoral areas of the western plains (<400mm; Figure 1). These regions are not absolute in their definition - they fluctuate with the cycles of seasonal conditions. Climatically the highlands and slopes are more favourable for footrot. In normal seasons, footrot, if introduced to the dry western plains, may persist in some animals but does not transmit to others. In the sheep and grain districts some seasons are favourable for the disease others not.

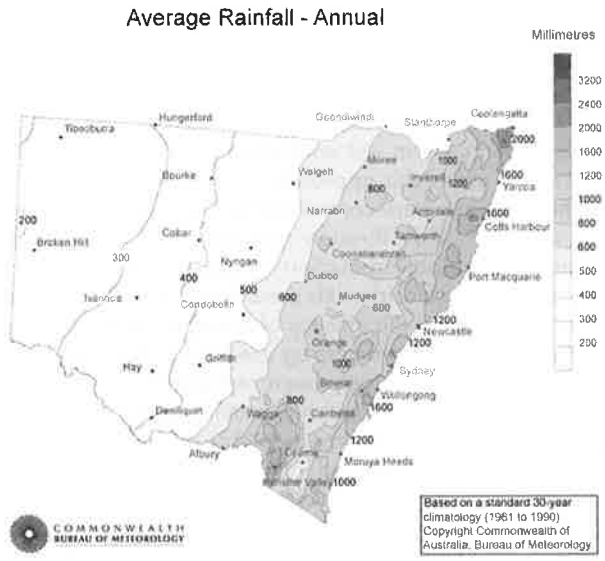

Figure 1. Rainfall Distribution NSW

The majority of flocks are Merino but many different breeds mostly of European origin are also kept for lamb production and for cross breeding with Merinos. Commercial flock owners normally purchase rams from studs either located in NSW or in other states. At the outset of the program studs were recognized as an important potential source of footrot for flock owners and representatives of the stud industry were included in the planning process for the program. There is considerable movement of sheep around the state depending on seasonal conditions. Throughout the state there are many markets which offer sheep for sale. These too were recognized as a source of infection for unsuspecting owners. Even before the program commenced there was legislation preventing the sale of sheep with footrot in markets but this did not always prevent affected sheep being sold. Again, at the outset, the need for continuing cooperation of the livestock agents responsible for trading in these markets was realized and they too were directly involved in the planning and supervision of the program.

Animal health administration in New South Wales

Under the Australian constitution the state governments have responsibility for agriculture and thus for animal health. Legal powers to control various aspects of livestock industry are delivered through Stock Diseases Acts or their equivalent. While each state pursues this responsibility independently there is some coordination of animal disease control and there is similar legislation for this in each state. While there have been national programs for the eradication of bovine tuberculosis, brucellosis and contagious pleuropneumonia, programs for footrot vary state by state.

In the Australian context there is an aspect of animal heath management which is unique to NSW. The state is divided into regions known as Rural Land Protection Boards (RLPBs). These Boards are managed by Directors elected by animal owners from their respective Board areas. Collectively the Boards are represented by a State Council and this Council is influential in the development of animal health policies in NSW. Funds for the support of the work of these Boards are derived from a levy determined by the number of livestock on individual farms in the Board areas. These funds are used to employ a District Veterinarian, para-veterinary and administrative staff. The District Veterinarians provide herd and flock health services to all levy or rate payers in their areas (strictly speaking, care of individual animal cases is left to private veterinarians).

Administration of the Footrot Strategic Plan

Policy on endemic disease control is determined by the Department of Agriculture and its advisory systems. Thus the policy for the eradication of footrot in NSW is ultimately the responsibility of NSW Agriculture but the development of an acceptable policy and its execution involved many other components of the sheep industry. At the outset a Footrot Steering Committee (FSC) was formed and the importance of industry participation was emphasized by the appointment of the Chairman of the State Council of the RLPB of NSW to chair that committee. Other members (Table 1) included representatives of relevant industry organisations including the veterinary research community. The committee meets at least twice

each year, makes recommendations to the Animal Health Committee of NSW Agriculture on changes in policy and communicates directly with RLPBs and other industry groups. From the outset the Steering Committee was advised by a technical sub-committee chaired by the executive officer of the FSC and consisting of representatives of field veterinary staff, para-veterinary organizations, diagnostic and research organizations. Recommendations from this group are discussed at meetings of the FSC.

Table 1 Structure of Footrot Strategic Plan Steering Committee

Organisation	Members
Rural Lands Protection Board	2*
NSW Agriculture	2**
NSW Farmers Federation	1
Merino Stud Breeders of NSW	1
Australian Veterinary Association	1
NSW Stock and Stations Agents Association	1
University of Sydney	1

* Includes chair of committee
** Includes executive officer of committee

Principal components of the Footrot Strategic Plan

The Footrot Strategic Plan was developed with the objective of improving the productivity and welfare of sheep and goats in New South Wales by the progressive eradication of virulent footrot.

Target for eradication: Virulent footrot was identified as the target for eradication. It was accepted that benign footrot was not a serious disease and that, because it had a reservoir in cattle, it would not be a target for eradication. Immediately then it became essential that the diagnosis of the clinical form of footrot present in a flock was accurate. From the beginning of the program diagnosis at the flock level was the responsibility of a veterinarian.

Definitions of eradication: Eradication at the flock level is defined as the complete elimination of virulent footrot from all animals in a flock. For an affected flock to achieve footrot free status it needed to undertake an approved program of eradication and to maintain its freedom from disease during and after a subsequent period favourable for the development of cases.

In 1991, RLPBs, or areas (parts) within Boards, were designated as Protected (<1%), Control (1-10%) or Residual Areas (>10%) depending on flock prevalences of footrot. Regulatory control of footrot was instituted when areas changed from Residual to Control or Protected status. Affected farms in Control and Protected status areas were quarantined and owners legally required to proceed with eradication. Owners of affected flocks knowing or suspecting the presence of footrot were required to report the presence of the disease to a Stock Inspector with regulatory powers under the Stock Disease Act. Reporting was not compulsory, and vaccination was permitted, in Residual areas (but not allowed in Control and Protected areas). The goal of the Strategic Plan is to have all New South Wales achieving Protected Area status throughout all RLPBs by the end of 2005.

Diagnosis of footrot in a flock: In New South Wales the decision on whether or not virulent footrot was present in a flock was based on clinical findings and the professional judgment of a veterinarian. The occurrence of disease which resulted in underrunning of the horn of the heel of the hoof of any sheep in the flock initiated action. Where there was unequivocal evidence of many sheep with severe underrunning infections, as was frequently the case early in the program, virulent footrot was diagnosed immediately. Where doubt existed about the severity of the disease in the flock the policy included the requirement to examine at least 100 sheep to determine the prevalence and severity of the disease. Emphasis was given to the need to record findings from these examinations. The lesion scoring system defined by Stewart and Claxton (1993) was used for this purpose and where doubt existed re-examination of the flock was required. Where there was uncertainty field officers had access to in vitro tests for assessing the virulence of isolates of D nodosus. These tests, based on the quantitative and qualitative nature of proteases elaborated by the organism (Palmer, 1993), were used as an aid to diagnosis but the results of laboratory tests alone were, under NSW policy, insufficient to support a diagnosis of virulent or benign footrot.

Declarations and accreditation: At the beginning of the program there were many flocks in NSW free of virulent footrot. Provision was made for the owners of these flocks, wishing to sell sheep, to sign a declaration of freedom from the disease (a Footrot Vendor Declaration). Such a declaration had legal status and if found to be incorrect had serious consequences for the seller. It was made possible also for flocks to achieve formal accreditation of freedom from footrot either following successful eradication or of pre-existing freedom from infection. This involved certification by private veterinarians and as flock prevalence decreased the scheme lapsed because of the costs involved. The stud industry embraced the Plan enthusiastically and introduced an approved system of self-declared freedom from footrot. These declarations were used in advertising stud sheep for sale.

Funding: The footrot eradication plan in NSW has essentially been funded by industry. The salaries of District Veterinarians within RLPBs are paid by ratepayers and these veterinarians are responsible for managing disease control programs in their district. If, as sometimes happened early in the program, owners elected to consult a private veterinarian for diagnosis and planning of eradication they paid the cost of his/her fees. All the costs of labour, drugs and vaccines were paid for by owners. NSW Agriculture provided some laboratory diagnostic services and initially the salary of a veterinarian who was administratively responsible for the program. This veterinarian organized the accreditation scheme for contactors (see below), collected, analysed and maintained data from the RLPBs and was also the executive officer of and secretary to, the Steering Committee.

Quality Control: From the outset it was determined that audits would be made annually of randomly selected areas to determine the accuracy of the claimed flock prevalence status of those areas.

Execution of the Strategic Plan

Farmers' groups: Because it was known that the most likely source of footrot was infection from a neighbouring farm the development of groups of neighbouring cooperating farmers was recognized as essential to the success of the plan. A pilot group established by one of us (RIW) in the middle of the endemic area in southern NSW in 1985 had successfully eradicated footrot from 24 of 25 farms before the statewide plan was launched. The success of this initial group indicated the importance of stimulating the commitment of individuals to collective action. This in turn reinforced the essentially voluntary nature of the program. There is a system throughout rural NSW of voluntary bush fire groups and the footrot groups were in part modelled on these. The collective commitment of farmers to footrot eradication helped also to eliminate some of the secrecy and social stigma previously associated with footrot.

Thus within each RLPB an early priority for the District Veterinarian was to promote the establishment and working of groups. Each of these groups became the focus of delivery of technical advice on the planning and execution of eradication plans for individual farms within the group. While the principles of eradication remain constant the needs and resources on different farms necessitate local modifications of those principles. The numbers of farmers groups and the flocks involved varied through the program and are summarised in Table 4 in the results.

Livestock contractors: Footrot eradication is based on inspection and re-inspection of the feet of all sheep in flocks at times of the year when transmission is not expected to occur. This inspection is necessary to identify cases either for removal, treatment or response to treatment. Other routine sheep husbandry practices on NSW farms like castration, tail docking and vaccinating are often done by contractors. Early in the program groups of these contractors were trained and accredited for footrot eradication work. An amendment was made to the Veterinary Surgeons Act to allow them to undertake diagnosis and treatment of cases of footrot. Diagnosis of the form of footrot in a flock remained the responsibility of a veterinarian. It was also necessary for a supervising veterinarian to prescribe antibiotics used in treatment of cases identified by contractors.

Footrot officers: Many RLPBs in the endemic area appointed, and paid for, one or more special footrot officers to assist DVs with footrot work within their district. These para- veterinary staff were trained in the principles and techniques of footrot eradication and under supervision worked with farmers' groups and individual owners in planning and execution of eradication plans. Statewide, their training was the responsibility of the senior veterinary officer of the Strategic Plan.

Extension and education: At the initiation of the plan and for the next five years considerable effort was put into ensuring that the principles and techniques used for eradication were known to all industry participants. The University of Sydney and NSW Agriculture organized and presented a series of training workshops for veterinarians both from the RLPBs and from rural private practice, for stud owners and for farmer groups. These workshops which included work on affected flocks were supplemented by facts sheets, illustrative material and manuals subsidized by veterinary chemical industry organizations. Veterinarians who completed these workshops were accredited for flock diagnosis, the preparation of eradication plans and the supervision of livestock contractors.

Attainment of Control or Protected Area status: RLPBs, on the advice of their District Veterinarian, submitted applications to the FSC, supported by appropriate prevalence data to justify designation of all or part of their Board as a Control or Protected area.

Progress towards eradication

Reduction of flock prevalence: In 1988 the sheep population of NSW was estimated to be about 50,000,000. Estimates of flock prevalences provided by District Veterinarians of virulent footrot in different RLPBs ranged from 0 - 75%. Overall there were about 5,000 affected flocks among the 45,000 which existed at that time i.e. a flock prevalence of about 11% (Scott-Orr 1986). The estimated flock prevalences in the high, medium and low rainfall zones at this time were 11-50%, 1-10% and <1% respectively. At the start of the FSP in 1988 and for the following three years seasonal conditions were good and wool prices were high. The removal of a price support scheme in 1991 resulted in a collapse of wool prices and this coincided with the onset of a long period of drought. Sheep and flock numbers fell dramatically. Although wool prices have returned to their pre 1991 values the sheep population has not recovered (Table 2).

There are now (October, 2003) 30 million sheep in 25,158 flocks and 236 of these (<1 %) are known to be infected with footrot. Thus the flock prevalence has been reduced tenfold and Protected Area status reached for the majority of the state. However, some Boards in the high rainfall zone remain as Control Areas and the flock prevalence still exceeds 4% in some of them.

Table 2: Sheep Numbers, Flock Numbers in New South Wales and Wool Prices 1988-2003

Year	Total Number Sheep* (million)	Number of Flocks*	Wool Market Indicator (cents per kg clean)**
1988	56.4	45,399	1003
1991	53.7	41,244	628
1993	44.2	40,891	488
1996	37.2	40,367	619
1999	36.0	32,378	524
2001	34.5	28,036	764
2002	32.5	26,363	841
2003	30.0	25,158	937

*Number of sheep and number of flocks based on Rural Lands Protection Board Annual Reports
**Wool Market Indicator based on Australian Wool Exchange data

The major impact of the Program has been in the high rainfall, high risk region of the state. Here the flock

prevalence has been reduced in some Boards from almost 50% to < 4%. Holbrook, Albury and Gundagai Boards were estimated by Locke and Coombe (1994) to have flock prevalences of 49, 45 and 48% respectively in 1988. In June 2003 these figures were reduced to 4% in the case of the Gundagai Board and < 1% in the case of Hume which consists now of the combined Boards of Holbrook and Albury. The western arid regions, far less favourable for footrot have continued with a very low flock prevalence (< 1%; see Figure 2; Table 3).

Table 3: Number of Footrot Infected Flocks in NSW 1988 - 2003

Numbers	1988	1991	1994	1999	2003
Infected Flocks	3820	6179*	3319	588	236
Flocks in State	45399	41244	40750	32378	25158
Flock Prevalence	8%	15%	8.1%	2%	1%

* increase in flock prevalence associated with better disease intelligence and more responsible reporting by sheep owners

The most striking changes in flock prevalence were achieved in the period 1991-1999. During much of this period there were dry seasons and wool prices were low. For these reasons many owners elected either to dispose of all sheep in affected flocks or to dispose of affected animals rather than to attempt eradication by treatments which cost more than the value of the sheep.

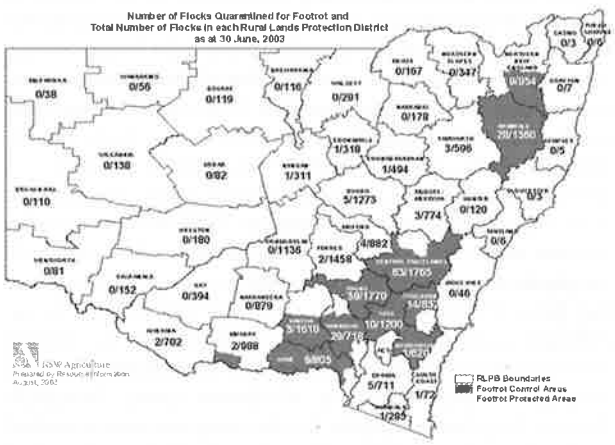

Figure 2

The farmers groups which made a major contribution to reduction of prevalence through cooperative action in defined local areas were mostly located in the high prevalence higher rainfall zone. The proportion of total flocks involved in group activity was never greater than 30% of all flocks in the state but their impact was greater because of their concentration in the high risk areas. As the prevalence within these groups was reduced to acceptable levels by 1999 and because of administrative changes in NSW Agriculture less emphasis was given to their support and to data collection within them.

Table 4: Groups, membership and flock prevalences

Date	Groups	Members	Infected Flocks (FP%)
Mar 1991	68	1841	342 (18.57)
Mar 1992	163	4575	543(11.86)
May 1993	243	7005	659(9.40)
May 1994	268	7675	761(9.91)
June 1995	300	8639	777(8.99)
June 1996	312	7894	415(5.25)
Oct 1997	356	9543	368(3.85)
Jul 1999	359	9733	298(3.06)

State wide summary: At the beginning of the program 20 of the 57 Boards which existed at that time were eligible for declaration as having Protected Area status. The other 37 were classified as Residual Areas i.e. with a flock prevalence above 10%. Within about ten years this number had increased to 33 and there were no Boards with a full Residual status (Table 5). In June 2002 there are no remaining Residual Areas and only some parts of 8 Boards have Control Area status. The goal of achievement of Protected Area status for all parts of all Boards by 2005 now seems achievable.

Table 5 Change of Footrot Status of Rural Lands Protection Boards

Status	1988	1991	1999	2003
Protected (whole)	20 (35%)	32 (56%)	33 (69%)	37 (77%)
Protected (part)		5 (9%)	9	8
Control (whole)			4 (8%)	3 (6%)
Control (part)			9	8 **
Residual (part)			5*	
Residual (whole)	37 (65%)	20 (35%)		

* 1999 the 5 part Residual Boards included 1 x R/P, 1 x R/C and 3 x R/C/P
** 2003 the 8 part Control Boards also have part Protected Area status

Virulent footrot in goats: Strains of D nodosus virulent for sheep cause footrot in goats. This disease is transmissible to sheep (Ghimire et al, 1999). It is unusual in NSW for sheep and goats to be grazed together so the risk of goats acting as a reservoir of infection for sheep is not high. Nevertheless where VFR is diagnosed in goat herds owners are obliged to undertake eradication. The methods applicable to sheep have been used successfully in infected goat herds.

Discussion

The impetus for a footrot eradication program originated with some sheep owners who had the foresight to recognize that a state flock without footrot would be of great benefit. It would lead to increased profitability of the industry, improve the welfare of sheep, facilitate trade within the industry and remove a source of continuing conflict between neighbouring farmers with and without the disease. The success of the FSP so far is a direct consequence of the enthusiastic and continuing support of

the whole sheep industry.
The flock prevalences in each of the Boards at the beginning of the program were estimates only (Scott-Orr, 1986). Later these estimates were revised and a structured survey was made of a sample of flocks in south-western NSW to test the accuracy of these estimates (Locke and Coombes, 1994). Overall the flock prevalences within the Boards included in the survey were considerably higher than those previously estimated. However a succession of years between 1988 and 1991, favourable for footrot, may have accounted for this. Also extensive publicity about the FSP preceded this survey and coincided with increased optimism in the farming community about the likelihood of success of eradication.
Although progress towards eradication has been substantial, more work is needed to meet the objective of the Strategic Plan i.e. for each Board to achieve Protected Area status by end of 2005. When that goal is reached there will be a continuing need for surveillance and the action necessary to ensure that flock prevalence is kept below 1%.

The impact of footrot on the productivity and welfare of sheep has been greatly reduced. There are no current estimates of losses due to footrot in NSW but the estimated annual cost of the disease was reduced from $42.6 million in 1991 to $13 million in 1996 (Walker 1997). There have been substantial benefits achieved other than direct reduction of costs. Trade of sheep within and between states is now far less likely to be complicated by the presence of footrot and a chronic source of conflict between neighbouring farms has been largely eliminated. In some areas the groups formed to manage footrot on a local basis have adopted that approach to other endemic diseases of sheep.
Given the scale of the program and the numbers of flock owners and sheep involved progress towards eradication has been relatively free of complications. A major reason for this has been because of the voluntary nature of the campaign and the strong commitment of the sheep industry to succeed. The infrastructure provided by the Rural Lands Board Protection system ensured the program was delivered at a local level and industry support was maintained. Regulatory enforcement other than mandatory quarantine in Control and Protected Areas has not been common and to date there have few prosecutions of recalcitrant owners.
Perhaps the most challenging aspect of the program has been the difficulty experienced from time to time in distinguishing between benign and virulent footrot. Most observers agree that there is a complete spectrum of clinical expression possible in flocks of sheep. There is no absolute cut-off between benign and virulent diseases and there is a tendency to attribute a major influence to the environment in which the disease occurs. In West Australia where a footrot eradication campaign has been in place for many years, virulent footrot has been defined as the disease associated with the presence of strains of D nodosus which are thermostable in the gelatin gel test (Palmer 1993). While the correlation between presence of these strains and virulent disease is strong it is not absolute. In NSW there are well documented circumstances where there is no evidence of severe clinical disease in some flocks where these thermostable strains exist. In the limited investigation done so far this disease does not increase in severity when affected flocks are moved to more favourable environments (Hall et al 2001, Abbott and Egerton. 2001). By contrast, in West Australia movement of an infection from one part of the state had a major impact on presentation of the disease (Depiazzi et al, 1998).
As the prevalence of the most virulent infections has been greatly reduced the relative importance of such infections as a diagnostic problem has become greater. It is of interest that the predictive value of a negative gelatin test is much higher than that of a positive test where there is no clinical evidence of virulent disease. The challenge for the program moving towards 2005 will be the continuing ability to effectively deal with the less virulent strains and at the same time maintaining support of the States' sheep industry. Once eradicated an ongoing biosecurity awareness program will be necessary to ensure the disease is not reintroduced. If identified it will become increasingly important that prompt action is taken to eliminate the disease.

Summary

It has been known for many years that ovine footrot is eradicable from flocks. The bacterium which transmits the disease, D. nodosus, is an obligate parasite so identification and removal of all cases of the disease results in its elimination from the environment. This principle has been applied to individual flocks in many countries but its application to regions, states or countries is unusual.
In New South Wales, a major sheep producing state of Australia, a program was started in 1988 to eradicate the virulent form of footrot. This program was essentially voluntary and was funded by owners of sheep throughout the state. Policy for the program was determined by an industry wide group and was implemented by District Veterinarians who, in New South Wales, are employed by livestock owners in about 50 Rural Lands Protection Boards. Legislative support for the program was provided by the New South Wales Department of Agriculture but legal regulatory action has rarely been applied.
At the commencement of the program there were about 45, 000 sheep flocks in the state of which 11% were believed to be affected. The flock prevalence(FP) of the disease was highest in the higher rainfall areas of the state and in some Boards this approached 50%. Since the program started there has been a major decrease in the sheep population of the state and in the number of sheep flocks but there are still about 30 million sheep in 25, 000 flocks. The flock prevalence of footrot, state wide, has been reduced to <1% and the most marked reduction has been in the areas most favourable for footrot. The highest flock prevalence in some parts of some Boards is about 4%. The objective of the program is to achieve Protected status (FP<1%) for all parts of all Boards by 2005. Based on progress so far there is confidence that this can be achieved.

References

Abbott K A and Egerton J R (2001). Ovine footrot: differences in optimal conditions for transmission and lesion development affect the consistency of clinical diagnostic criteria. In Aust Sheep Vet Soc Proc No 11, Melbourne, Pg 1-4.

Abbott K A and Egerton J R (2003). Eradication of virulent footrot of lesser clinical severity (intermediate footrot). Australian Veterinary Journal. 81:688-693.

Beveridge, W I B (1941) Footrot in sheep: a transmissible disease due to Fusiformis nodosus (n.sp.) Council of Scientific and Industrial Research. Bulletin 140.

Carmody M J (1981). An economic valuation of the footrot eradication program on the northern tablelands of NSW. Agricultural Economics Bulletin No. 25. Department of Agriculture and Business Management, Unversity of New England, Armidale.

Depiazzi L J, Roberts W D, Hawkins C D, Palmer M A, Pitman D R, McQuade M D, Jelinek P D, Devereaux D J and Rippon R J (1998) Severity and persistence of footrot in merino sheep infected with a thermostable strain of Dichelbacter nodosus at five sites.Australian Veterinary Journal 76:32-38.

Dewhirst F E, Paster B J, La Fontaine S and Rood J I (1990). Transfer of Kingella indologenes(Snell and La Page 1976) to the genus Suttonella gen.nov. as Suttonella indologenes comb. Nov.; transfer of Bacetroides nodosus (Beveridge 1941) to the genus Dichelobacter gen. Nov. as Dichelobacter nodosus comb . nov.; and assignment of the genera Cardiobacterium, Dichelobacter and Suttonella to Cardiobacteriaceae fam. nov. in the gamma division of Protobacteria based on 16S ribosomal ribonucleic acid sequence comparisons. International Journal of Systematic Bacteriology 40: 426-433.

Egerton J R and Parsonson I M (1966). Isolation of Fusiformis nodosus from cattle. Australian Veterinary Journal 42:425-429.

Egerton J R and Parsonson I M (1969) Benign footrot- a specific interdigital dermatitis of sheep associated with infection by less proteolytic strains of Fusiformis nodosus. Australian Veterinary Journal 45: 345-349.

Egerton J R, Parsonson I M and Graham N P H (1968) Parenteral chemotherapy of ovine footrot Australian Veterinary Journal 44:275-283.

Egerton J R, Egerton J R, Ghimire S C, Dhungyel O P, Shrestha H K, Joshi H D, Joshi B R, Abbott K A, and Kristo C (2002). Eradication of virulent footrot from sheep and goats in an endemic area of Nepal and an evaluation of specific vaccination. Veterinary Record 151: 290-295.

Ghimire S C, Egerton J R and Dhungyel O M (1999).Transmission of virulent footrot between sheep and goats. Australian Veterinary Journal 77:450-453.

Graham N P H and Egerton J R (1968). Pathogenesis of ovine footrot: role of some environmental factors Australian Veterinary Journal 44: 235-240.

Hall E, Cheetham B, Gaden C and Green G (2001). Gelatin gel stable strains of footrot which express as clinically benign. Conference Proceedings, Australian Sheep Veterinary Society. Melbourne Convention Centre, Melbourne Victoria. 13-18 May. pp 5-10.

Laing E A and Egerton J R (1978). The occurrence, prevalence and transmission of Bacteroides nodosus infection in cattle. Research in Veterinary Science 24: 300-304.

Locke R H and Coombes NE (1994). Prevalence of Virulent Footrot in sheep flocks in southern New South Wales. Australian Veterinary Journal 71: 348-349.

Palmer M A (1993). A gelatin test to detect activity and stability of proteases produced by Dichelobacter (Bacteroides) nodosus. Veterinary Microbiology 36:113-122.

Roberts D S and Egerton J R (1969). The aetiology and pathogenesis of ovine footrot. II The pathogenic association of Fusiformis nodosus and F. necrophorus. Journal of Comparative Pathology 79: 217-227.

Scott-Orr H (1986) The current footrot situation in New South Wales in Footrot in Ruminants- proceedings of a workshop, Melbourne 1985. CSIRO Division of Animal Health, Australian Wool Corporation, pp 13-17.

Stewart D J, Clark B L and Jarrett R G (1982). Observations on strains of Bacteroides nodosus of intermediate virulence to sheep. Advances in Veterinary Science, Australian Veterinary Association, pp 74-76.

Stewart D J and Claxton P D (1993. Ovine footrot clinical diagnosis and bacteriology. In: Australian Standard Diagnostic Techniques for Animal Diseases. Standing Committee on agriculture and resource management. Sub-committee on animal health laboratory standards. CSIRO Parkville, Australia. Eds. Corner L A and Bagust T J. pp 1-27.

Thomas J H (1962). The differential diagnosis of footrot in sheep. Australian Veterinary Journal 38:159-163.

Walker R I (1997). The NSW Strategic Plan and Eradication of virulent footrot. Armidale Proceedings IV International Congress for Sheep Veterinarians, University of New England, Armidale, Australia 2-8 February pp 114-120.

Wilkinson F C, Egerton J R and Dickson J (1970). Transmission of Fusiformis nodosus from cattle to sheep. Australian Veterinary Journal 46: 382-384.

FOOTROT IN SHEEP: CONTROL VERSUS ELIMINATION

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Farmers in Britain have attempted to eliminate footrot (FR) in sheep, caused by Dichelobacter nodosus (Beveridge, 1941; Roberts and Egerton, 1967), for over 40 years. The recommended techniques for managing

flock foot health are routine trimming of feet, regular foot bathing and vaccination. The recommended treatment for diseased sheep includes parenteral antibiotic, topical foot spray and paring the diseased foot (Morgan, 1987; Bagley et al., 1987). It is also recommended that diseased sheep are isolated and culled if they recurrently become diseased and that sheep new to the flock are quarantined and only introduced to the flock when they are free from FR.

Despite the recommendations above the prevalence of footrot remains high and in 1994 was estimated to be approximately 10% of the national flock lame at any one time (Grogono Thomas and Johnson, 1994). This had not changed by 2000 (Wassink et al., 2003).

This study set out to test the hypothesis that traditional techniques to manage footrot in sheep were unsuccessful either because a) they did not work per se or b) because farmers were not able to carry out these methods to a high enough standard to achieve efficacy.

In November 2000 a postal survey was conducted in England and Wales as part of an investigation into improving the treatment and control of FR in sheep using existing techniques. A total of 210 farmers with 70,000 sheep in total responded. Negative binomial regression analysis indicated that the isolation of new stock, and the separation and treatment of clinically diseased sheep using all current recommendations were associated with very low prevalences of FR (2% in the previous 12 months). However, flock control measures had no impact on the reported prevalence of FR, except routine foot trimming, which was associated with an increase in the prevalence of FR (16%). The majority of farmers (60%) considered that individual treatments for diseased sheep were 'good' or 'excellent'. They were generally less confident that routine flock management controlled foot rot. These poor results from routine treatments may have led farmers to accept a prevalence of FR of 5 - 10% as 'not a problem' (Wassink et al, 2001; 2003 and unpublished data).

In a follow up study of 80 of these farmers, 80% had not changed their foot trimming or footbathing practices for more than 5 years. This strengthened the cause and effect relationship that was observed in the cross sectional study that flock interventions made FR disease worse.

Two hypotheses are supported by our findings

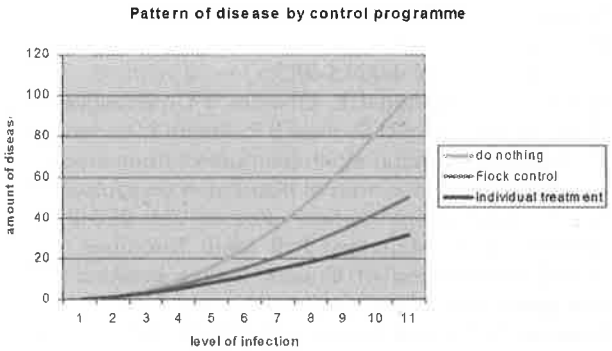
1) We hypothesise that FR is an infectious disease which, given the climate in Britain, it is not possible to elimi nate nationally. Rather than repeatedly trying to remove D. nodosus (making feet susceptible) through gathering, foot bathing and foot trimming it is better if sheep's feet are left undisturbed and continually exposed to D. nodosus and so mount an effective immune response, which generally prevents disease. Only when sheep are diseased should treatment be used to a) prevent suffering and b) decrease the con centration of D. nodosus and so prevent a high dose which might be more likely to produce disease in other sheep. This is an exciting hypothesis because if true it would relieve farmers of much of the time burden associated with foot care whilst increasing the health of the sheep.

2) Our second hypothesis is that the actions of gathering, foot trimming and footbathing increase the success of transmission of D. nodosus through physical damage to the feet (Cross, 1978). The act of gathering may increase close contact with D. nodosus, which occurs in the faeces; gathering leads to increased faecal soiling of the feet.

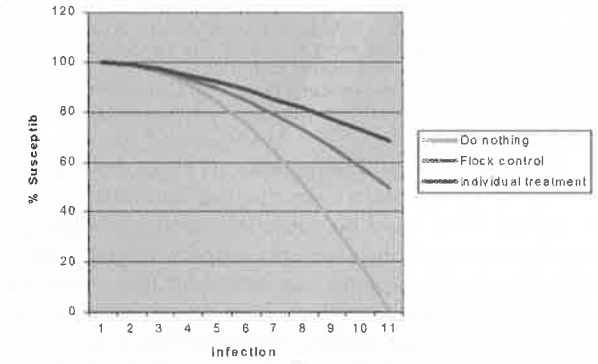
Discussion

Much of our knowledge of control and elimination of footrot has come from Australian literature. Many parts of Australia are now free from FR through an intensive programme of inspection, vaccination, footbathing and culling repeatedly lame sheep (Scott-Orr H, 1990, 1995). The programme of elimination starts on each farm when the hot, dry season commences so that FR does not persist on the land. The programme has been successful in many areas of the country when there is a minimum of six weeks dry, hot weather. It has been less successful where there is rainfall during the six-week period (Walker personal communication, 2001).

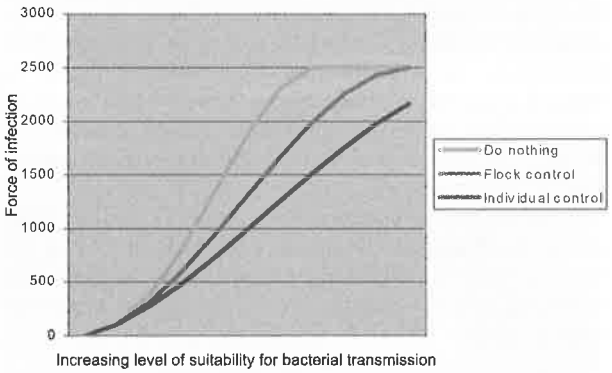
The UK does not have many summers with six weeks of dry weather and these certainly cannot be predicted so the programme of elimination is usually focused on the winter months, when D. nodosus may not survive the cold weather. However, the weather is more temperate in the UK and in some winters in the south of England there may not be a frost. As a result many farmers have worked very hard to eliminate D. nodosus but have been unsuccessful. Elimination appears an unlikely option for the whole of the UK. We therefore propose an alternative strategy to control FR in the UK rather than to eliminate it. This strategy is hypothesised below.



a) Amount of disease



b) Proportion susceptible by level of infection from sheep / environment



c) Theoretical force of infection (proportion susceptible x proportion diseased) by level of infection

Diagram a is the theoretical difference in disease postulated by doing nothing versus flock management (trimming and footbathing) versus individual management of diseased sheep (Wassink et al., 2003) in environments of low (x axis = 1) to high (x axis = 11) levels of infectious pathogen. This results in different proportions of susceptible sheep (diagram b) and different rates of successful transmission of the pathogen (force of infection, diagram c). Where there are low levels of the pathogen - either few diseased sheep or low environmental contamination, any of the three strategies are successful. As the level of contamination increases (e.g. introducing diseased sheep, gathering sheep, increasing stocking density,) then doing nothing soon leads to a high proportion of infected (diagram c) and diseased (diagram a) sheep. Flock control remains fairly successful whilst contamination levels are low but enhances transmission and disease as the level of infection increases. At high levels of infection individual treatment leads to the lowest levels of disease, susceptibility and the slowest force of infection. It should be stressed that this is theoretical and requires testing.

References

Bagley, C. V., Healey, M. C. & Hurst, R .L. (1987) Comparison of treatments for ovine footrot. Journal of the American Veterinary Medical Association 191, (5) 541-545.

Beveridge, W. I. B. (1941) Footrot in sheep: a transmissible disease due to infection with Fusiformis nodosus (n. sp.): Studies on its cause, epidemiology and control. Commonwealth Scientific & Industrial Research Organisation (C.S.I.R.O). Australian Bulletin 140, 1-56

Cross, R. F. (1978) Influence of environmental factors on transmission of ovine contagious foot rot. Journal of the American Veterinary Medical Association 173, (12) 1567-1568.

Graham, N. P. H. & Egerton, J. R. (1968) Pathogenesis of ovine foot-rot: the role of some environmental factors. Australian Veterinary Journal 44, 235-240

Grogono-Thomas, R. & Johnston, A. M. (1997) A study of ovine lameness. MAFF Final Report. MAFF open contract OC59 45K

Morgan, K. (1987) Footrot. In Practice, July 1987, 124-129

Roberts D. S. & Egerton J. R. (1969) The aetiology and pathogenesis of ovine foot-rot. II. The pathogenic association of Fusiformis nodosus and F. necrophorus. Journal of Comparative Pathology 79, 217-227

Scott - Orr (1990) The NSW footrot strategic plan - a co-operative approach to disease control. Aus. Advances in Vet. Sci.

Scott - Orr (1995) NSW footrot control - a success story. AusSheep Vet. Soc.

Wassink G. J., R. Grogono-Thomas, L. J. Moore, and Green L. E. (2003). Risk factors associated with foot rot in sheep The Veterinary Record 152 351 - 358

Wassink G. J., Grogono-Thomas R., Moore L. J., and Green L. E. Risk factors associated with scald in sheep The Veterinary Record in press

GUIDE TO ULTRASOUND EXAMINATION OF THE BOVINE CARPUS

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Introduction

The carpus in cattle is frequently affected by a number of different diseases. Al-though studies of various regions of interest as well as pathological cases have been published (Kofler 1995, 2000), a complete ultrasonographic survey of the carpal re-gion in cattle is lacking. The aim of this study was therefore to create a standardised ultrasonographic examination procedure of the carpal region based on the anatomi-cal structures.
Material and Methods
All examinations were carried out with a real-time ultrasound unit Sonoline Prima (Siemens), equipped with a 5/7,5-MHz multi-frequency linear transducer. For a better imaging of surface-near structures, Aquaflex®-Gel-Pads (Parker Laboratories, Inc., NJ) were used.
Initially, 8 isolated cadaver specimens were prepared to show the topographic anat-omy, and directly scanned to recognize the typical ultrasonographic appearance. Later in 13 cadaver limbs, frozen sections of the carpus were made, prepared and photographed.

Plane 1: At the levelt of the distal physis of the radius.
Plane 2: At the level of the palpable joint space of the antebrachiocarpal joint.
Plane 3: At the level of the palpable intercarpal joint space, distal to the Os carpi ac-cessorium.
Plane 4: At the level of the palpable carpometacarpal joint space, proximal to the Os metacarpale quintum.

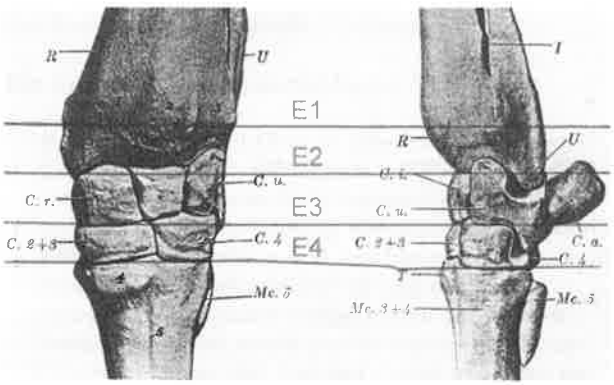

Fig. 1: Bony structures of the carpus (Sisson and Grossman 1938) and examination planes.
Planes 1-4 are marked as E1-E4. Legend: R = Radius; U = Ulna; I = Spatium interosseum ante-brachii; T and 4 = Tuberositas ossis metacarpalis III; C.r. = Os carpi radiale; C.i. = Os carpi intermedium; C.u. Os carpi ulnare; C.a. = Os carpi accessorium; C.2+3 = Os carpale secundum et tertium; C.4 = Os carpale quartum; Mc.3+4 = Os metacarpale tertium et quartum; Mc.5 = Os metacarpale quintum; 5 = Canalis metacarpi proximalis.

In accordance with Mettenleiter (1995) and Budde (1997), the carpal region was di-vided into four horizontal planes. They could be easily identified by the palpation of prominent bony structures in both young and adult cattle (Fig. 1). Each plane was examined from eight different sides of the carpal joint, from dorsal, dorsolateral, lateral, lateropalmar, palmar, mediopalmar, medial, and dorsomedial. The resulting 32 planes were scanned horizontally and vertically, moving the trans-ducer distally step by step. By comparing each ultrasound image with the corre-sponding 32 anatomical sections, the relevant structures could be verified clearly. With corresponding illustrations, all muscles and their tendons could be imaged properly. In three cadaver specimens, the carpal joint pouches were filled with saline solution to determine the best transducer positions for the diagnosis of pathological joint conditions.
The examination procedure was used in 11 healthy cattle of both sexes aged 9 months to 9 years. Furthermore, 24 carpi from cattle with diseases of the carpal re-gion were examined and compared with the ultrasound images of healthy cattle.

Results

A concise picture of each of the 32 carpal planes could be produced using this ex-amination procedure. The topography of each ultrasonographic picture could be explained in detail by means of illustrations and by comparing it to the corresponding frozen anatomical section (Fig. 2). By doing so, a good comparison of the structures in the ultrasonographic images and the anatomical parts could be made.
The carpal flexor and extensor tendons, partially their tendon sheaths, the medial and lateral collateral ligament, the ligamentum accessoriometacarpeum and the sur-faces of the carpal bones could be imaged without major problems. The only excep-tion was the musculus abductor pollicis longus. This muscle had to be examined in an oblique direction. With this ultrasound system, it was not always possible to dis-tinguish it clearly from the surrounding tissue.

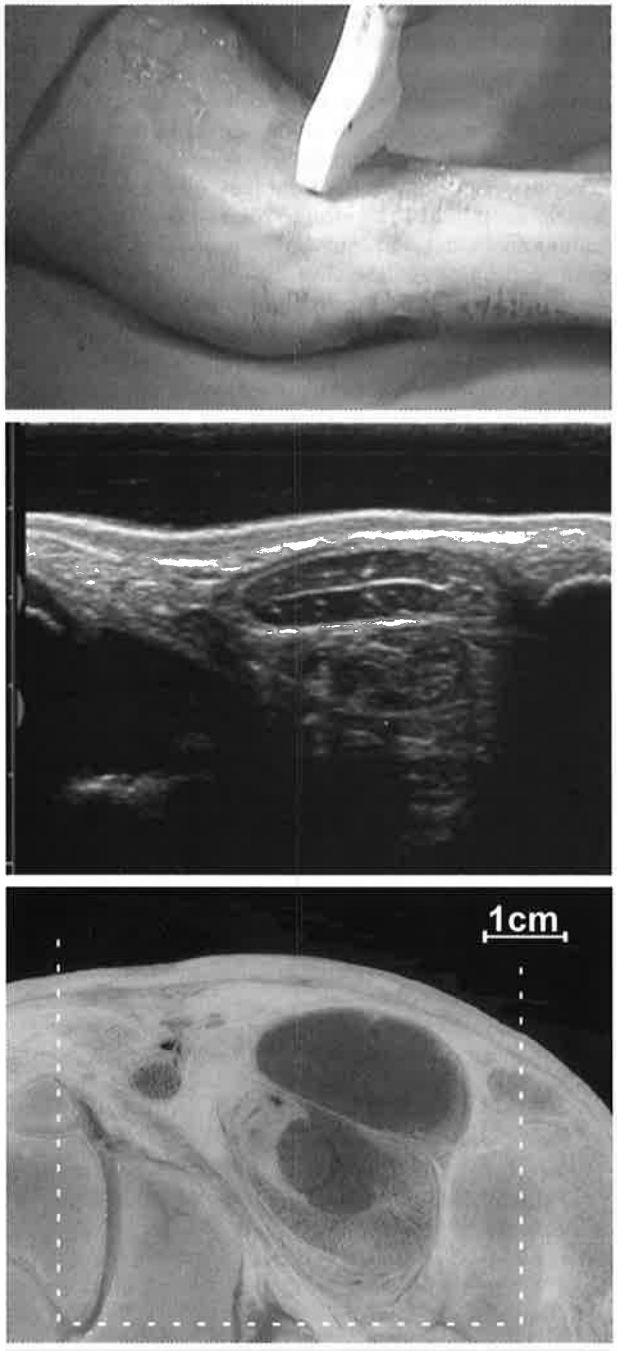

Fig. 2: Example images of one examination plane: Plane 3 - mediopalmar - horizontal.
Upper left: transducer position. Upper right: ultrasound image. Lower left and right: anatomical sec-tion and drawn illustration of the same region as in the ultrasound image.
R = radius; Ci = Os carpi intermedium; Ca = Os carpi accesso-rium; Flexdigsup = Musculus flexor digitorum superficialis (o: sur-face near, t: deep part); Flexdigpro = Musculus flexor digitorum profun-dus; Flexcarprad = Musculus flexor carpi radialis; Flexcarpuln = Musculus flexor carpi ulnaris; MSB = medial col-lateral ligament; Haut = skin;

Ultrasonographic imaging of the larger vessels and the median nerve, running over the palmar and medial aspects of the carpus, required some practice and was not possible in every case. The antebrachiocarpal joint space and the intercarpal and carpometacarpal joint spaces could be clearly defined as interruptions of the echo-genic bone surfaces, whereas the joint pouches could only be detected around the dorsal and the lateral aspects. The joint capsules could not be identified. In young animals the cartilaginous growth plates of the dis-tal physes of radius and ulna could be seen as ane-chogenic zones interrupting the hyperechogenic bone surface.
The experimental filling of the joints showed that the joint pouches of the carpus could be illustrated most easily close to the puncture sites of joints described in the liter-ature. This worked out best when holding the transducer vertically.

Discussion

For a reliable ultrasound examination, a thorough knowl-edge of anatomy is prerequi-site (Kofler 2000). To get accurate information out of every region of interest, how-ever, typical images of each single plane together with the corresponding anatomical sections and drawn illustra-tions are very helpful. Based on these reference images, an ultrasonographic examination of both healthy and dis-eased carpal joints can be performed easier.

References

Budde K. Sonographische Diagnostik im Bereich des Karpalgelenkes beim Pferd. Diss vet. med. Bern, 1997.
Kofler J. Application of ultrasonic examination in the diagnosis of bovine locomotory system disorders. Schweiz Arch Tierheilk 1995;137:369-80.
Kofler J. Ultrasonographic examination of the carpal region in cattle - normal appearance. The Veterinary Journal 2000;159:85-96.
Mettenleiter E. Das Tarsalgelenk des Pferdes - Sonographischer Atlas. Stuttgart; New York: Schattauer 1995.
Sisson S, Grossman JD. The Anatomy of the Domestic Animals. 3rd Ed. Philadelphia and London: W. B. Saunders Company 1938.
Tnibar M, Kaser-Hotz B, Auer JA. Ultrasonography of the dorsal and lateral aspects of the equine carpus: tech-nique and normal appearance. Vet Radiol & Ultrasound 1993; 34(6):413-425.

TWO CLINICAL CASES OF LAMENESS IN RUMINANTS FROM SOUTH AFRICA

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1)Corrosive effects of concrete on buffalo hooves
A request from the attending Veterinarian to travel 400 kilometres to examine a very lame, valuable buffalo bull was fulfilled. A number of buffalo from a disease breeding facility had been purchased a few weeks previously. The animals had been placed under roof in concrete pens similar to the previous lodgings. The new facilities were over 1 year old.
On arrival, the buffalo bull was crawling around on his elbows and knees. Due to his aggressive behaviour it was decided to bleed and examine hooves of some of his herd mates. It was very noticeable that the hooves of the darted buffalo were freshly and severely worn. The bull was heavily sedated and examined. All 8 claws had been worn down to the sensitive lamina with a few areas covered by a paper thin sole that was readily and simply pealed away. Prognosis was ZERO and the owner was advised to euthanasia on welfare grounds. Due to the value and extreme reluctance of the owner, and against veterinary advice, it was decided to try and treated subsequently the less severely claws on all 4 feet were blocked. The blocks were attached solely to the hoof walls with cotton wool padding between the exposed sensitive lamina and the blocks. Painkillers and antibiotics were administrated systemically and the bull was placed in a rapidly erected outside camp with relatively soft soil.
A better history revealed that due to the bull's aggressive nature he had, with the previous owners, been kept in an adjoining camp (without concrete flooring) that was wet and damp in certain areas. Consequently, his hooves were softer than the other buffalo and were not adapted to concrete. The new facilities, although over 1 year old, had never been used nor hosed down and were well protected from the elements. The concrete was therefore relatively fresh and very abrasive and together with the aggressiveness of the buffalo bull with the continual pacing and mock charging and his relatively soft, non-adapted hooves, disaster was eminent.
The case is well illustrated.

2)Progressive complications of the claws of a Holstein Bull
Occasionally requests to attend complicated hoof problems of valuable bulls at a large semen producing concern arise. A 6-year-old Holstein bull with a very disjointed history was one such request. The initial complaint was of a severe all round lameness and that the claws seemed to be peeling away from the coronets. The first visit revealed a lame bull with severe chronic interdigital dermatitis but as the resident veterinarian was unavailable it was not certain if this was the correct animal. Curative trimming was attempted and along with the severity of the lesions and sincethe bull was unproven as regards semen quality and genetics the prognosis was guarded. Two weeks later, a second visit to the same bull was met with a significant deterioration with a severe weight bearing lameness, especially of the front feet. All 8 claws showed severe, deep, black "V", eroded ridges typical of interdigital dermatitis but with "golf-ball" like swellings in zone 6, axial to the ridges, involving the heels. These swellings were hard and painful on palpation. Curative trimming with reducing ridge wall and cap of the "golf-ball" swellings was attempted plus the right fore lateral claw was blocked to try and ease the extremely painful medial claw. Daily phenylbutazon per os was recommended. It was then discovered that the bull had been placed in a formalin footbath daily for over 3 weeks.
A third visit, 2 weeks later, found the bull much improved with an increased appetite and more ambulatory but with pronounced the left hind leg lameness. Both hind feet exhibited lesions as described previously but also with large necrotic scabs between the claws and just above the bulb region. Removal of the sensitive scabs left raw, well-circumscribed lesions that possibly could be confused with digital dermatitis but probably were the aftermath of excessive formalin exposure. Both black feet produced very large tylomas in addition to the already existing typical tylomas which were extremely painful and had to be removed surgically. Topical oxytetracycline and a light bandage were applied with good results. The later tylomas were possible due to severe irritation from the corrosive effect of the formalin and complicated by secondary infection from the interdigital dermatitis.
The case is still pending and is well illustrated.

NEOPLASMS OF THE EXTREMITIES IN CATTLE - CLINICAL FINDINGS, SURGICAL TREATMENT AND OUTCOME IN 10 CASES

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Summary

This paper presents ten cases of true neoplasms located on bovine extremities. Neoplasms are rarely diagnosed in cattle. Clinical presentation, ultrasonographic findings, the results of the necropsies and pathohistological findings are presented. The diagnoses were a synovial sarcoma, a chondrosarcoma originating at the cartilage of the right scapula, metastases of an adenocarcinoma of unknown primary, a fibrosarcoma, two

fibromas as well as a fibropapillomas, two squamous cell carcinomas of the distal phalanx and a carcinoma of the convoluted glands.

Introduction

Contrary to humans with the highest frequency of neoplasms followed by dogs, horses and cats, tumours are less common in cattle and have not been well documented in literature. There exist only a few reports about neoplasms originating from the extremities of cattle (Alton and Kofler, 1998). Neoplasms of the cartilage are extremely rare in cattle (Richardson and Acland, 1982). Several studies on neoplasms showed an incidence of chondrosarcoma on bovine species of less than 0.1% of all tumours recorded (Anderson et al., 1969; Shortridge and Cordes, 1971). Benign neoplasms on the limbs can also disturb normal movement mechanically and thus cause lameness. Malignant neoplasms destroy the surrounding tissues by infiltrating growth. Benign limbneoplasms can also disturb normal movement mechanically and thus cause lameness. Cartilaginous neoplasms, whether malignant or benign, are relatively rare in all domestic species. Chondrosarcoma is more common than its benign counterpart. Only a few cases of bovine chondrosarcomas have been described in detail, all of which being recorded as originating in the sternum and the ribs.

Material and Methods

The case records comprising clinical, radiographic and ultrasonographic findings as well as the results of necropsies and pathohistological findings from 10 cattle, ranging from 6 months to 8 years old, with various neoplasms originating on the limbs are presented. All patients underwent routine clinical examination. Blood samples were taken during hospitalisation. Depending on the size and the localisation of the tumour, ultrasonographic and radiographic examination was done. Ultrasonography was carried out with the Sonoline Sienna (Siemens) unit, equipped with a 7.5 MHz linear and a 3.5 MHz convex transducer. Radiography was performed with the Super 100 CD (Philips). Digital radiographic examination was done with the Fuji FCR AC - 3 unit (Fuji). Biopsies and/or fine-needle aspiration cytology were performed in some cases. Tissue samples obtained either by biopsy or during necropsy were fixed in 7 per cent neutral buffered formalin and sections were stained with haematoxylin and eosin (HE).

Results

A 6-months-old male calf had a congenital synovial sarcoma. After the first operation done by the referring veterinarian the tumour had immediately recurred. The calf presented with a partly crude, partly soft-to-elastic tumour double the size of a human fist midway between the right hock and stifle joints on the dorsolateral aspect of the tibia, the surface being ulcerated and partly necrotic. On patho-histological examination a malignant synovial sarcoma was diagnosed.

An 8-year-old Simmental cow presented with anorexia and a firm, painful swelling of the right shoulder region. Weight loss and respiratory distress had occurred within the last weeks. Clinical examination revealed a poor body condition. The normal bone structure of the right shoulder was totally destroyed, ultrasonography showed many irregularly shaped, hyperechoic small reflexes. A chondrosarcoma, which had already spread to the lungs, was diagnosed.

An 8-year-old Simmental cow was referred a subcutaneous phlegmon involving a melon-sized, crude tumour on the caudolateral aspect of the right hind limb. The patho-histological examination revealed metastases of an adenocarcinoma of unknown primary. These were found in the muscles of the right hind limb, the lung and the pleura.

A 3.5-year-old Simmental cow had been referred with the history of a slowly growing neoplasm of two years duration. On the lateral aspect of the elbow a tumour nearly spheroid, possibly having its origin in the skin, was diagnosed. Pathohistological examination was consistent with the suspected diagnosis of a fibropapilloma. The cow was discharged two weeks after surgical treatment.

A 1.5-year-old Simmental heifer presented with a tumour of the claws of the right hind limb. After the first operation done by the referring veterinarian the tumour had immediately recurred. After pathohistological examination a diagnosis of fibrosarcoma was made.

Two cows in this study were diagnosed with a fibroma after pathohistological examination of the tissue samples.

A 6-year-old Brown Swiss cow presented with a painless growth double the size of a human fist localised on the left tuber coxae. In the case of a 6-year-old Simmental cow the melon-sized tumour was situated on the left calcaneal region.

A 3 year-old Simmental showed a severe lameness of the right frontlimb. Radiography revealed extensive osteolysis of the medial distal phalanx. The first clinical diagnosis of an apical pedal bone necrosis was followed by amputation and pathological examination of the claw. Macroscopically, the horn capsule showed no lesions. The pathoanatomical examination of the sagittally transected claw revealed a soft consistency of the pedal bone and a yellowish crumbling material, had replaced most of the pedal bone. Histologically a poorly keratinized squamous cell carcinoma was identified. The cow was discharged five weeks after surgery.

A similar tumour was found in a four years old Simmental cow. This cow presented a moderate lameness of the right front limb caused by white line disease and necrosis of the pedal bone.

A six year -old Simmental cow presented a severe lameness of the right hindlimb and a severe swelling of the coronary region. The diagnosis of a purulent necrotising arthritis of the distal interphalangeal joint was followed by amputation. On pathohistology a carcinoma of the convoluted glands was diagnosed. Four weeks after surgery the cow was discharged from the clinic.

5 cattle showing tumours on the claws, the elbow and cal-

caneal region were surgically treated with good success: digital amputation allowed removal all the affected tissues in this area and the fibropapilloma of the elbow region and the fibroma of the calcaneal region were well demarcated from the underlying tissues.

Discussion

According to relevant literature, tumours are less common in cattle than in other species. In the authors opinion this might be due to the fact that most cattle are slaughtered before reaching a higher age, which is probably a predisposition for the development of neoplasms. Neoplasms of the extremities present with a slowly developing growth. In some cases, lameness is not caused by pain or destruction of the joint itself or adjoining structures, but by mechanical obstruction alone. Infiltration of surrounding tissues, metastases in regional lymph nodes or other organs as well as anorexia, loss of milk yield, emaciation and perhaps fever are signs of malignancy. Benign tumours, on the other hand, are characterised by solitary appearance, slow growth, good demarcation from the surroundings, absence of metastases, good general condition and appetite.
Diagnoses were made after clinical, radiographic and ultrasonographic and most importantly the patho-histological examinations of tissue samples fixed in 7 per cent neutral buffered formalin. It is most important to take the samples from various parts of the suspected neoplasm in the zone between sound and diseased tissue. Surgical excision of neoplastic bone and cortical or osteochondral allograft is used in small animals and humans (Brown and Cruess, 1982; Hanson and Markel, 1992) but is not practicable in cattle and horses. In these species weight bearing on the affected limb after extensive resection of neoplastic bone can lead to instability and pathological fractures. Although reconstruction and use of prothesis after surgery of malignant bone tumours is performed in humans (Wada et al., 1999) this technique is not feasible in large animals. Chemotherapy should be only performed in pets and not in production animals.
Surgical intervention is of limited use in malign neoplasms, because the animals will not be referred until the disease has already spread to vital organs. In benign cases, the outcome of the operation depends on the size and site of the tumour and the treatment costs.

References

Alton, K., Kofler, J. (1998): Plattenepithelkarzinom der Klauenmatrix eines Rindes. Tierärztliche Praxis 26, 73-77.
Anderson, L.J., Sandison, A.T., Jarett, S.H. (1969): A British abattoir survey of tumors in cattle, sheep and pigs. Vet. Rec. 84, 547 - 551.
Brown, K.L.B., Cruess, R.L. (1982): Bone and cartilage transplantation in orthopedic surgery. J. Bone Joint Surg. 64, 270 - 279.
Hanson, P.D., Markel, M.D. (1992): Bone and cartilage transplantation. Vet. Comp. Orth. Trauma 5, 163 - 169.
Richardson, D.W., Acland, H.M. (1983): Chondrosarcoma in a cow. Cornell Vet. 73, 137 - 143.
Shortridge, E.H., Cordes, D.O. (1971): Neoplasms in cattle: a survey of 372 neoplasms examinated at the Rua Kura Veterinary Diagnostic Station. New Zeal. Vet. J. 19, 5 - 11.
Wada, T., Usui, M., Isu, K., Yamawakii, S., Ishii, S. (1999): Reconstruction and limb salvage after resection for malignant bone tumour of the proximal humerus. A sling procedure using a free vascularised fibular graft. J. Bone Jt. Surg. B 81 (5), 808 - 813.

TOE ULCER AND NECROSIS OF THE DISTAL PHALANX IN A PÉRÉ DAVID'S DEER

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Introduction

Ruminants kept in a zoological garden under conditions simulating the wild are difficult orthopaedic patients. Examination and treatment is only possible under general anaesthesia and the necessary changes of bandages until the wound is healed poses a problem. Repeated general anaesthesia carries a high risk in ruminants, quite apart from the costs. In many cases it is not even possible to separate the patients from the herd during convalescence. Even if it seems possible to shut the patient in a stable, the wild animal may suddenly go beserk, breaking its neck in the attempt to break free.

Material and Methods

A 2-year-old Péré David hind of the wild park Gänserndorf presented with a severe lameness of the right front limb. Not being domesticated, it had to be narcotised using a blow pipe (Immobilon®). The clinical examination including x- rays were performed under general anaesthesia. The operation was performed after the application of a tourniquet under additional regional intravenous anaesthesia using 10 ml of procaine-hydrochloride (Minocain 2%, Atarost, Germany). A tourniquet of rubber tubing was applied directly to the middle of the metacarpus.

Results

The right lateral claw showed a hole at the tip of the toe. The claw itself was partly exungulated. There was no swelling of the soft tissues proximal to the coronary band. The loose parts of the claw horn were removed. The corium was necrotic with a superimposed infection. The distal third of the pedal bone showed a possible pathological fracture with bone necrosis, yellow to greenish in colour. The x-ray findings were consistent with a diagnosis of pathological fracture showing marked osteolysis of the bone structure. The diseased bone was removed using a hammer and mallet. The resulting wound cavity was lavaged with isotonic saline solution with diluted 0.1% polyvidon-iodine-solution.
Ampivet® (100 mg/ml Ampicillin-trihydrat) was used as a local antibiotic treatment. This was supplemented with a long acting penicillin given intramuscularly (Duplocillin®, Mycoform, Netherlands). A protective wound bandage up to the middle of the metacarpus was applied. The bandage was changed three times having stayed in place for a week. At no time did the wound show any signs of infection and healed very fast. Four weeks after the operation the hind was successfully reintegrated in the herd.

Follow-Up

Three months later a slight lameness recurred. The hind was in heat at that time and therefore pestered by the male deer. An orthopaedic control examination of the claw showed normal findings. After a separation for three days, she was reintegrated into the herd showing no signs of a lameness. The hind is still alive and well.

Discussion

Reports on the orthopaedic treatment of wild ruminants are rarely found in the relevant literature with a preponderance of case-reports (Butt et al., 2001; Cruz et al., 1999; Kaneps, 1996; Toews et al., 1998). Treatment and surgery was performed as described in cattle (Kofler, 1999). The long-term prognosis depends not so much on the medical problem itself as on the post-operative management. As soon as lameness persists, the herd instinctively tries to rid itself of the animal in question because of a possible liability in a conflict with a predator. The lame deer is attacked by the males of the herd and are not strong enough to fight for their share of the feed leading to death by starvation. If the deer can be separated, one has to take care that it is returned as quickly as possible to its natural habitat. In our case, the wound healed quickly enough for the animal to be turned out in time to get the winter coat. Another problem with residual lameness is the public opinion of the visitors. With the best intentions, visitors might report the zoo to the authorities leading to expensive law suits.

References

Butt, T.D., Cruz, A.M., Bailey, J.V., Crawford, W.H. (2001): Outcome of limb amputations in wapiti: 13 cases (1995-2001). Can. Vet. J. 42(12); 936-939.
Cruz, A.M., Bailey, J.V., Fretz, P.B. (1999): Management of limb fractures in wapiti (Cervus elaphus): 22 cases (1993-1997). J. Am. Vet. Med. Assoc. 15, 214(12);1829-1832.
Kaneps, A.J. (1996): Orthopedic conditions of small ruminants. Llama, sheep, goat, and deer. Ve.t Clin. North Am. Food Anim. Pract., 12(1); 211-31.
Kofler, J. (1999): Clinical study of toe ulcer and necrosis of the apex of the distal phalanx in 53 cattle. Vet J., 157 (2); 139-147.
Toews, A.R., Bailey, J.V., Theore, C. (1998): External skeletal fixation for treatment of comminuted fractures in wapiti: 5 cases. Can. Vet. J., 39(6); 370-372.

TREATMENT OF INFECTED WOUNDS AND ABSCESSES IN BOVINE LIMBS WITH LIGASANO® -POLYURETHANE-SOFT FOAM DRESSING MATERIAL

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Introduction

Reports on the treatment of wounds in bovine patients and wound healing abnormalities in bovines are rare compared with the corresponding literature in horses.
The treatment of traumatic injuries with subsequent infection of the digital flexor tendon sheath and the therapy of traumatic exungulation and bone sequestration in limbs was described in cattle by some researches (1, 3, 5, 7, 10). Other authors reported on the surgical therapy of infected abdominal incisions after abomasopexy, cesarean section and colic surgery in cattle (3, 6, 9).
In cattle, regarding the treatment of wounds beside the surgical techniques and the possibilities that are available, it is necessary to incorporate also economic aspects for the prognosis. So, if there are dressing materials available that are not expensive and have been proven in human medicine, they should be applied also for wound therapy in cattle (11-12).
Polyurethane-soft foam dressing material is frequently used in human patients since many years for treatment of infected wounds, as postoperative wound dressing material, for the prophylaxis of decubital ulcers and for mechanical wound debridement (11, 12). This

polyurethane-soft foam dressing material has been used also with good results for therapy of various large sized wounds in equine and bovine patients since about 5 years at our clinic.
The objective of this report is present this special polyurethane-soft foam dressing material for the treatment of wounds in bovine limbs, to show its most important indications and to illustrate the healing progress and the outcome of selected clinical cases which were treated with Ligasano® polyurethane-soft foam dressing material.

Material and Methods

For this study 23 bovine patients were selected which were treated between 2001-2003 at the clinic for infected cuts and laceration wounds (n=14) on the limbs with involvement of digital joints, for large abscesses in the tarsal and thigh region (n=7), intertrigo on the disto-medial crural region (n=1) and for deep puncture wounds leading to purulent tenosynovitis of the digital flexor tendon sheath (n=4) by carrying out a total resection of both digital flexor tendons, and open surgical wound management.
After routine wound cleaning, wound debridement or surgical intervention with subsequentwound lavage, Ligasano®-polyurethane-soft foam dressing material (Ligamed medical products, Cadolzburg-Wachendorf, Germany) was applied as a direct wound dressing instead of conventional cotton gauze swabs, or as drainage material in all cases. Ligasano®
is available sheets 59 x 49 x 1 cm, and can be cut to size and shape for any wound, and can also be sterilised.

Results

Depending on the wound depth Ligasano® can be applied as a sheet onto the wound, or as a roll pushed into the wound, to cover both the surface and margin, so that they are in full contact with this material under moderate pressure. Its porous and alveolar surface structure causes mechanical wound cleaning and a mechanical stimulation of the wound surface resulting in an increased exudate and decreased fibrinous adhesions and adhesions of bacteria. The large suction effect of this bulky porous wound dressing material ensured good drainage of exudate, avoiding the accumulation of exudate and resultant maceration of the surface.
Ligasano® stimulated the formation of healthy granulation tissue over a level wound surface.
In all cases of digital joint and purulent digital flexor tendon sheath infections, starting with surgical treatment, no purulent exudate was observed during the postoperative healing period. Also when this material was used for drainage of large abscessses incised on the lateral aspect of the tarsus, distal crural and caudal thigh region, only a slightly purulent exudate was noted and rapid secondary closure by granulation tissue could be seen.
The adhesion of this material to the wound surface, even when mainkained in place for several dayswas not as great as with gauze. The detachment of a gauze dressing usually caused extensive bleeding. In contrast the Ligasano® dressing could always be easily detached from the wound when soaked with saline solution.

Discussion

In contrast to many other wound dressings Ligasano® can be cut in one piece from sheets59 x 49 x 1 cm appropriate to the particular the wound. In all the above mentioned indications for Ligasano® treatment either no or a slight purulent exudate was observed. Especially in treatment of purulent tenosynovitis with complete resection of the superficial and deep digital flexor tendons of one digit the healing period was much shorter (about 14 days) then reported previously (5, 8).
The therapeutic success with Ligasano® was so convincing in these cattle, that currently this material is used more or less exclusively as primary dressing material in bovine orthopaedic patients in our clinic and conventional cotton gauze swabs (1, 2, 8) have been discarded.

References

1 Anderson, D.E., St-Jean, G., Morin, D.E., Ducharme, N.G., Nelson, D.R., Desrochers, A. (1996): Traumatic flexor tendon injuries in 27 cattle. Veterinary Surgery 25, 320-6.
2 Bailey, J. (1997): Wounds. In Lameness in cattle. eds Greenough P.R., Weaver A.D., 3rd Edn, Philadelphia, W.B. Saunders, pp. 181-8.
3 Bienek, A., Grunert, E. (1997): Sonographische Verlaufskontrolle der Wundheilung nach Sectio caesarea beim Rind. Deutsche Tierärztliche Wochenschrift 104, 423-7.
4 Hirsbrunner, G., Steiner, A., Martig, J. (1995): Knochensequestration des Röhrbeines beim Rind. Tierärztliche Praxis 23, 251-8.
5 Kofler, J. (1994): Sonography as a new diagnostic tool for septic tenosynovitis of the digital flexor tendon sheath in cattle - therapy and long term follow-up. Deutsche Tierärztliche Wochenschrift 101, 215-22.
6 Seger, T., Grunert, E., Ahlers, D. (1994): Zur Entstehung einer gestörten Heilung der Bauchwandwunde nach Schnittentbindung beim Rind. Deutsche Tierärztliche Wochenschrift 101, 309-11.
7 Stanek C. (1981): Beitrag zur primären, totalen Exungulation beim Rind. Berliner Münchener Tierärztliche Wochenschrift 94, 126-8.
8 Stanek, C. (1997): Tendons and tendon sheaths. In: Lameness in cattle. eds Greenough, P.R., Weaver, A.D., 3rd ed, Philadelphia: W.B. Saunders, pp. 188-90.
9 Tulleners, E.P., Donawick, W.J. (1983): Secondary closure of infected abdominal incisions in cattle and horses. J. Am. Vet. Med. Assoc. 182 (12):1377-9.
10 Valentino, L.W., St-Jean, G., Anderson, D.E., Desrochers, A. et al. (2000): Osseous sequestration in cattle: 110 cases (1987-1997). J. Am. Vet. Med. Assoc. 217, 376-83.
11 Weber, G., Galli, K. (1980): Therapeutische

Anwendung von Ligasano®-Schaumstoff. Deutsches Ärzteblatt - Ärztliche Mitteilungen 77 (25), 1621-5, Sonderdruck.
12 Zemlin, C. (2001): Studie über die Anwendung von Ligasano als Primärwundverband bei diabetischen Fussläsionen. Lazarus 16, 24-5.

CLINICAL ASPECTS OF AN ABNORMAL PRESENTATION OF ACTINOBACILLOSIS IN A DAIRY HERD

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Introduction

Actinobacillosis is a well-recognised condition in ruminants (Swarbrick, 1967) caused by infection with the Gram-negative bacterium Actinobacillus lignieresii. Classically, the best known conditions of actinobacillosis in cows are "wooden tongue" and local infections of the pharyngeal region in cattle (Gillespie and Timoney, 1981). Other forms of actinobacillosis in cattle are described in different publications. In this poster, a presentation of Actinobacillus lignieresii infection is described on the hind legs, which gave problems in two 1-2 years old animals. Although this form of actinobacillosis has been described earlier e.g. by Weaver and others, it has never been reported before in the Netherlands.

History

Two Holstein-Friesian yearlings (18 and 20 months old) were presented to the local practitioner with "potato sized" nodules on one hind leg, which started to bleed when touched. The farmer had moved from another dairy farm to this location last winter, where cows had never before been kept. The housing of the milking cows was new build cubicles, while the young stock were housed in a reconstructed existing building.
The lesions were first noticed in spring and had increased during the last 2 months.

Clinical inspection and pathology

Clinical inspection, revealed that the two yearlings were in good condition, but smaller than animals of the same age in the group. None of the other animals showed lesions. Nodules were present on the right hind legs of both animals and located on the dorsal and lateral surfaces of the tarsus, metatarsus, ferlock and on phalanx I (see Pictures). The round-shaped nodules had a maximum diameter of 12cm, and had a broad contact place with the skin. The nodules were firm and painful, and bled easily. It seemed that the nodules were located on skin or subcutis without contact with bone. The regional lymph nodes were not considered enlarged.
One non-pregnant animal was slaughtered and the affected hind leg presented to the pathologist for further investigation.
Macroscopica examination revealed three well defined, protruding round processes in the skin with a firm wall and a fibrous aspect on cross-section. The subcutis surrounding these masses was oedematous.
At histological examination the diagnosis of actinobacillosis was made, based on the characteristics of the macroscopical and histological aspects of the process. The specific microscopic features included: microabscesses with granules, consisting of colonies of bacteria with a circle of surrounding eosinophilic-like bodies.
It was not possible to isolate the bacterium. This rarely happens and could have its origin in the fact that the process is often presented to the pathologist in a very late phase, and/or the animals have been treated with antibiotics.
The other yearling was treated with streptomycin (25 mg/kg b/w.) for twelve days, following post-mortal diagnosis. The leg lesions resolved satisfactorily.

Conclusion

The diagnosis usually can be made based on clinical and post-mortem examination. Frequently, it is not possible to isolate the bacterium.

THE ISOLATED HAEMOPERFUSED COW LIMB AS A MODEL FOR STUDYING THE PATHOGENESIS OF BOVINE LAMINITIS

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Introduction

According to many authors, laminitis is regarded as the most important predisposing factor for the development of lameness (GREENOUGH, 1985; BOOSMANN, 1991; LISCHER, 1994; COLLICK, 1997). Nevertheless, the pathomechanisms of laminitis in cattle are still poorly understood. Many theories, whose experimental proof are still pending, exist. The knowledge on the pathophysiology of equine laminitis derives extensively from animal experiments. Animal experiments, however, are problematic under ethical and economical considerations.

Isolated limb perfusion models represent a potential alternative for animal experiments. A number of different isolated organ models, including an isolated porcine limb model already exist. These models are used by the pharmaceutical industry for development and testing of drugs. Examples are the isolated perfused pig heart (LUEGER et al, 2002) and the isolated perfused bovine udder (KIETZMANN et al, 1993).
The aim of our work was to develop and characterise an isolated haemoperfused cow limb model, based on a pre-existing porcine limb model (WAGNER et al, 2003). This work is part of the fundamental research work done under the Lamecow project. The first developmental phase will be followed by an experimental phase, i.e. a series of studies on the effects of bioactive molecules on the claw tissue.

Material and methods

The perfusion apparatus and regularisation mechanism were taken from the porcine limb model. All parameters were under similar physiological conditions, only perfusion flow and pressure had to be calculated based on values given in literature for equine and porcine limbs, since there are no cattle specific values published. ROBINSON recorded in 1974 measurements of equine limbs. He found pressure values of 93-154 mmHg and flow values of 24-116 ml/min. SCOTT et al recorded in 1978 pressure values of 78-130 mmHg and flow values of 25,5-80ml/min in equine limbs. For the pig comparable values were published (flow values of 100-250 ml/min and a pressure value of 100 mmHg (NOGUEIRA et al, 1999; WAGNER et al, 2003).
Isolated limbs and blood were obtained from routinely slaughtered healthy cows older than 24 months from local abattoirs. Immediately after slaughter the arteria metacarpea dorsalis III and arteria digitalis palmaris communis III were cannulated and injected with an oxygenated electrolyte solution supplemented with glucose, heparin and insulin until the outflow solution was clear. Then the limb was transported to the laboratory at 4°C, where it was connected to the perfusion apparatus. The perfusion apparatus developed by Vitrotec Entwicklungs GmbH, Berlin, Germany, comprised two circuits, a perfusate and a dialysate circuit. They are connected through a capillary dialyzer (Hemoflow F7 low flux, Fresenius) for oxygenation and exchange of metabolites.
First perfusions were conducted with a pressure of 100 mmHg and a resulting flow of 230-250 ml/min. However, in some limbs the resulting flow was abnormal (400 ml/min), and no perfusion would have been possible. Therefore later limbs were perfused with a constant flow of 200 ml/min. The resulting pressure might not exceed 150 mmHg.
The limbs were perfused with a mixture (=perfusate) of bovine albumin (4%)/ erythrocytes (10%) in 630 ml dialysate, with a resulting haematocrit of 8% and an haemoglobin concentration of 2-3 g/dl. The dialysate consisted of NaCl, KCl, MgCl, CH3COOH, NaHCO3 and H2O. An oxygen/room air mixture produced an oxygen saturation of 98-100 %. The pH value was adjusted over the CO2 - and HCO3 content. Glucose, as a nutrient, and insulin were added half an hour later. The parameters were recorded from the arterial, venous and dialysate half-hourly. In addition lactate was examined to check the adequate oxygen supply, pyruvate was measured to characterise the metabolism and the increase of weight was determined during the perfusion to detect possible development of oedema within the claw. When neither the pressure exceeded 150 mmHg nor the potassium content increased above 5 mmol/l, it was possible to perfuse limbs 6-8 hours. After each perfusion samples were taken from all regions (segments) of the claws for light and electron microscopic examination of tissue integrity. Samples were fixed in formaldehyde or Karnovsky´s solution and routinely processed for light and electron microscopy.

Results

The first perfusions were conducted with a pressure of 100 mmHg and a flow of 230-250 ml/min over a total time of 4 hours. The light microscopic analysis did not show cell degeneration, but vascular dilations and interstitial oedema were located in all regions of the claw. The following limbs were perfused with a lower and constant flow of 150- 200 ml/min. The resulting pressure varied from 30 to 117 mmHg. The light microscopic analysis indicated only few thromboses in a couple of smaller vessels. There was no indication of cell and/or tissue damage. When the 4 hour perfusions provided satisfactory results, the perfusion period was extended to 5 hours. The light microscopic analysis showed pressure-related damage limited to the wall region, where we found vascular dilations and cellular necrosis in the apical third of the dermal lamellae. In all other regions of these claws however, there was no histological indication of cell damage. The potassium content in no perfusion exceeded 5 mmol/l.

Discussion

For perfusion of the isolated pig limb it was an acceptable method to adjust the pressure to 100 mmHg. The resulting flow amounted generally to 230-250 ml/min. After the first perfusion it seems, that these parameters could be transferred. However the light microscopic analysis indicated vascular dilations and oedema showing that the exposure to the vessels was too intensive.
The limbs evinced a sizable variance in the vascular diameters, so that in some limbs a pressure of 100 mmHg effected a very pathological flow (> 400 ml/min). To achieve reproducible results in spite of the different vascular diameters, a constant flow interval had to be defined and the resulting pressure recorded (unless > 150 mmHg). With very large vessels the flow interval contained pressures of 30-50 mmHg. But these low pressures, according to current knowledge, were not physiological and microscopic analysis also suggested that the perfusion is not adequate. Therefore it is recommended one should eliminate these limbs in the slaughterhouse

already.
The degenerative alterations in perfusions 9 and 10 suggest an outflow disturbance. During a perfusion there is the possibility of vascular obliteration initiated by thromboses. If a bigger vessel is occluded, the pressure will increase. Occlusion of a small vessel remain undetected until light microscopic analysis.
Morphological evaluation of tissue integrity turned out to provide the most valuable information whereas the pyruvate/lactate ratio was less valuable. In all perfusions the light microscopic analysis supplied the most authentic information about the tissue vitality and integrity in the perfused claws.
During 8 to 10 further perfusions the developmental phase will be finished. Subsequently, the model will be used to study the effects of reduced flow rates and reduced oxygen levels on the tissue.

This work was funded by the EU Lamecow project QLRT-2001-00969.

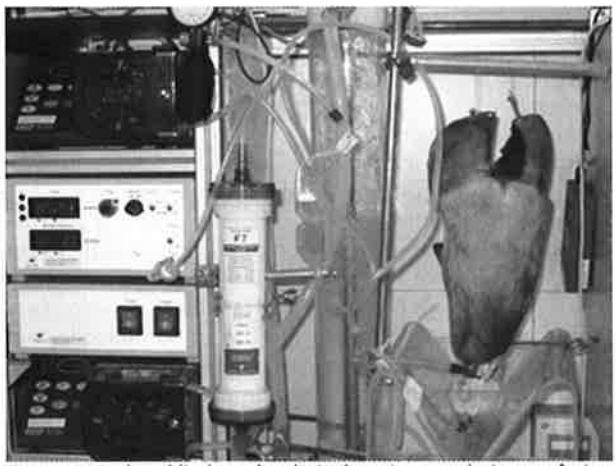

Figure 1: Isolated limb and technical equipment during perfusion

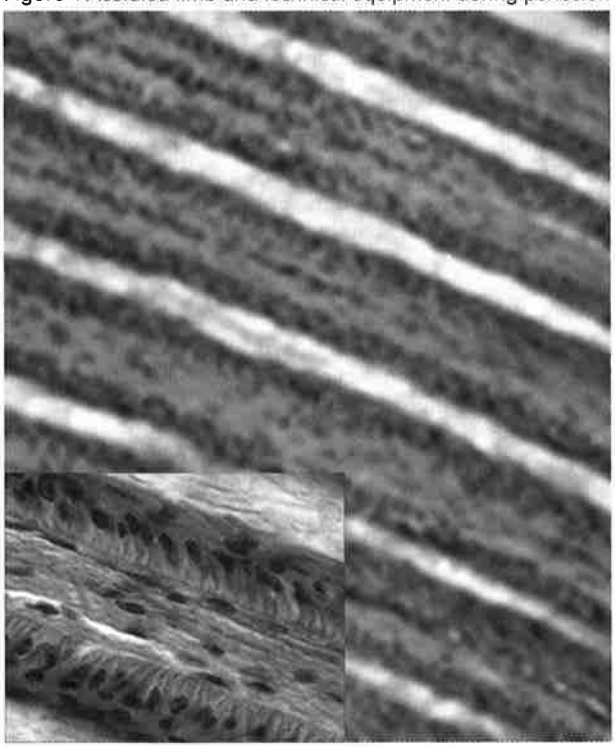

Figure 2: Micrograph from the wall region of a perfused limb showing intact dermal and epidermal laminae. H&E stain, x 125. Insert: Physiological cell morphology in the strata basale and spinosum. X 250.

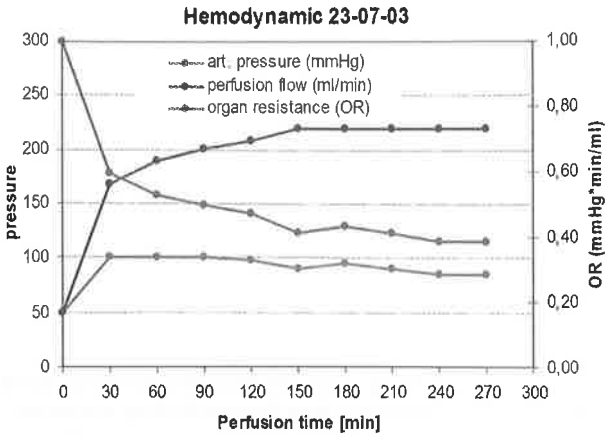

Figure 3: Time course of blood pressure, perfusion flow and organ resistance during a limb perfusion

References

Boosman R. et al. Bovine laminitis: clinical aspects, pathology and pathogenesis with reference to acute equine laminitis. Vet. Quart. 1991; 13(3): 163-71
Collick DW. Pododermatitis Circumscripta. In Lameness in Cattle. (Ed. Greenough, PR, Weaver, D.) WB Saunders Company, Philadelphia 1997: 101-104
Greenough PR. The subclinical Laminitis Syndrome. Bov. Pract. 1985; 20: 144-149
Kietzmann M et al. The isolated perfused bovine udder as an in vitro model of percutaneous drug absorption, skin viability and percutaneous absorption of dexamethasone, benzoyl peroxide and etofanamate. J Pharmacol Toxicol Methods. 1993 Oct; 30 (2): 75-84
Lischer C, Ossent P. Klauenrehe beim Rind: eine Literaturübersicht. Tierärztl. Prax. 1994; 22: 424-32
Lueger A et al. The influence of magnesium on the electrophysiological effects of erythromycin in the isolated Guinea pig heart. Cardiovasc. Drugs Ther. 2002 Jul; 16 (4): 327-33
Nogueira AC et al. The isolated normothermic hemoperfused porcine leg as a model for pharmacological and toxicological investigations. Altrex 1999; 16: 90-94
Robinson NE et al. Vascular Responses in the Equine Digit. Am J Res 1975 Aug; 36 (8):1249-53
Scott EA et al. Electromagnetic Measurements of Metacarpal and Digital Blood Flow in the Pony. Am J Res 1978 Nov; 39 (11): 1853-55
Wagner SM et al. The isolated normothermic hemoperfused porcine forelimb as a test system for transdermal absorption studies. J Artif Organs. 2003 Sept; 6 (3): 183-91

CELLULAR COMMUNICATION CHANNELS IN BOVINE CLAW EPIDERMIS AND THEIR FUNCTIONAL ROLE FOR HORN FORMATION

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Introduction

General: The causes of claw diseases can be attributed to various factors, however, most frequently they are associated with housing systems, nutrition, genetic factors, breeding influences and physiological state. Increasingly intensified milk production in the last three decades has led to an exaggerated rise in the occurrence of non-infectious claw diseases. Relatively little is known about the cellular and molecular processes behind these disorders. To date few investigations have dealt with the complex interactions of proteins and other signal-producing molecules with cellular structures. On the contrary extensive studies have been done with human skin in this field. A number of them involve GJIC (Gap Junctional Intercellular Communication), may to play a crucial role in maintaining tissue homeostasis including differentiation and stratification processes.
GJ and connexins (figure 1): In almost all tissues the transmission of information between cells takes place via gap junctions. These channels directly connect the cytoplasm of neighbouring cells. They are formed through concentrations of certain proteins, so-called connexins. The connexins belong to a multigene family composed of at least 20 members in humans. Based on similarities in sequences they were divided into 2 groups, and connexins. The current nomenclature refers to the molecular weight of the protein. Six connexins together build up so-called hemichannels, or connexons, which are then integrated into the gap junctional plaques of the cell membranes. Because most of the cells express more than one connexon type, there are homomeric (consisting of a single connexin type) and heteromeric connexons (consisting of various connexins). In order to build a functional channel, it is necessary that the extracellular domains of connexons from neighbouring cells interact with each other. Depending on the combination of homomeric or heteromeric connexons, homotypical, heterotypical or heteromeric gap junctional channels can be formed, which allow the passage of low weight molecular substances (1 kD) such as ions, metabolites and second messengers. In human skin 10 different transcripts for connexins (Cx26, Cx30, Cx30.3, Cx31, Cx31.1, Cx32, Cx37, Cx40, Cx43 and Cx45) have been discovered so far. Seven of them (Cx26, Cx30, Cx31, Cx32, Cx40, Cx43 and Cx45) have been approved on the protein level by immunohistochemical methods (Di et al, 2001). Best studied in human skin are Cx26 and Cx43. Gap junctions of human keratinocytes mainly consist of Cx43 (Guo et al, 1992) which can be found in all living epidermal layers in both interfollicular epidermis and epidermal appendices. Cx26 is mostly found in the appendices where it is co-localised with Cx43 (Salomon et al, 1994), rare in interfollicular epidermis but conspicuously expressed in the skin of palms and soles (Salomon et al, 1994, Lucke et al, 1999). Cx31 and 37 occur in human skin suprabasal in spiny cell and granular cell layers (Richard, 2000).

Materials and Methods

Tissue: Tissue samples were obtained from 5 regions of bovine hooves of slaughtered dairy cows provided by local abattoirs. Claws were sectioned on a band saw and samples were cut out. Subsequently tissue samples were either fixed in neutral buffered formaldehyde for 24 hours or embedded in Tissue-Tek , fresh snap frozen in liquid nitrogen and stored at -80 C. Dehydration for wax embedding was done in a Histokinette (Shandon) and the tissue was embedded in paraffin wax. 4-5 m sections (cryosections 8 m) were deposited on Capillary Gap Microscope Slides 155 m (ChemMate , DakoCytomation, Hamburg). Cryosections were fixed in ice cold acetone for 8 min. before immunostaining.
Immunohistochemistry: All reagents were from the ChemMate series (DakoCytomation, Hamburg). Slides with sections were washed with Buffer 1 and positioned into a immunostainer (TechMate -Horizon, DakoCytomation, Hamburg). The automatically run staining protocol included the following steps: rinsing with buffer, incubation with the primary antibodies (Rabbit anti-Mouse Cx26, Rabbit anti-Mouse Cx31, Rabbit anti-Mouse Cx37, ADI, USA; Mouse anti-Mouse Cx43, Chemicon, USA) for 35 min, rinsing with buffer, peroxidase block, incubation with the secondary antibody Goat anti-Rabbit and Mouse coupled with peroxidase labelled dextran (ChemMate EnVision /HRP, Rabbit/Mouse, Dakocytomation, Hamburg), rinsing with buffer, incubation with the chromogen diaminobenzidine, washing and counterstaining with haematoxylin. Cover slides were mounted with Canadabalm. Specimens were examined with a Olympus BX51 light microscope equipped with a Colour viewII digital camera (SIS).
Electron microscopy Samples were fixed in Karnovsky´s solution, rinsed with Cacodylatbuffer, postfixed with osmic acid solution (OsO4, 1%), dehydrated and embedded in epoxy-resin. Semithin sections were prepared and stained with Toluidine blue. Ultrathin sections of selected areas were stained with uranyl acetate and lead citrate. Samples were examined with a Zeiss EM 10 transmission electron microscope.

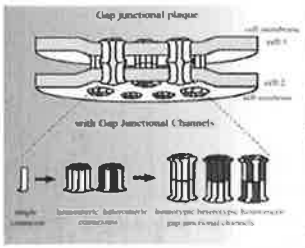

Figure 1: Schematic drawing of a gap junctional plaque and the arrangement of connexin proteins. Illustration modified according to Richard 2000.

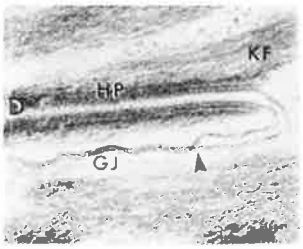

Figure 2: Electron micrograph of a gap junction (GJ) and a desmosome (D) between 2 neighbouring cells in the stratum spinosum (sole). Cell membranes have converged to 1 nanometer (arrowhead). Desmosomal adhesion disk (HP), keratin filaments (KF); Figure according to Muelling 1993

Results

Connexin 43 expression in bovine claw epidermis (figures 3 and 4) To assess whether keratinocytes from bovine claw epidermis express connexin proteins we performed immunohistochemical analysis using four commercially available anti connexin antibodies (Cx26, Cx31, Cx37 and Cx43). We found a punctuate staining pattern for Cx43 at the plasma membrane from keratinocytes implying that the protein occurred at gap junctional plaques between adjacent cells. In the same position gap junction formation was confirmed by electron microscopy (figure 2). Basal cells expressed high levels of Cx43 protein. In suprabasal layers (stratum spinosum) Cx43 expression declined to a weak staining in the stratum granulosum and in terminal differentiating cells. The stratum corneum from bovine claw epidermis did not contain immunohistochemically detectable levels of Cx43. The staining pattern described above was present in epidermal tissue from all the five regions of the bovine claw (Coronary, Wall, Sole and Bulbar region). We were not able to detect unequivocal levels of Cx26, Cx31 and Cx37 by immunohistochemistry in bovine keratinocytes.

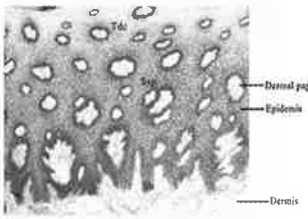

Figure 3: Connexin 43 protein in bovine claw epidermis, expression pattern in the distal bulbar region; Stratum basale (Sb), Stratum spinosum (Ssp), Terminal differentiating cells (Tdc)

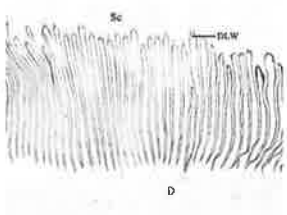

Figure 4: Connexin 43 protein in the epidermis of the wall region of the claw. Dermis (D), Dermal lamella from the Wall region (DLW), Stratum corneum (Sc)

Discussion

As part of investigations of cellular biological causes for lameness in dairy cattle, we examined the expression of Cx26, 31, 37 and 43 in bovine hoof tissue immunohistochemically and showed that Cx43 was expressed strongly in the stratum basale of the claw epidermis. Cells of the stratum spinosum have been found to express lower levels and terminal differentiating cells showed only weak expression of Cx43. Cx26, 31 and 37 could not be detected.

Because antibodies against bovine connexin proteins are not yet available, we used a mouse anti-mouse Cx43. A synthetic peptide corresponding to position 252-270 of the mouse Cx43 represents the immunogen and its sequence is identical in bovine. Cx 26, 31 and 37 antibodies were anti-mouse from rabbit and actual antibody crossreactivity with these connexins from other species was not known. Therefore further investigations are required to verify our findings and to examine these connexins by other immunological methods, PCR and in situ hybridisation techniques, but it is likely that connexins exist in bovine epidermis in a distribution similar to human epidermis (resp. epidermal appendices). Tissues will show specific patterns of connexin expression depending on their state of differentiation and function. There is evidence for changing patterns of connexin expression during human fetal skin development (Arita et al, 2002). Intercellular communication was shown to be intimately involved in regulating epidermal wound repair (Goliger and Paul, 1995) and changes in normal expression of connexins are associated with changes in the proliferation and differentiation program which keratinocytes undergo in psoriasis (Labarthe et al, 1998). To date a number of connexin gen mutations have been described to cause several epidermal dysfunctions (Richard, 2000), which supports the importance of gap junctions in epidermal differentiation. Therefore connexins in bovine claw epidermis are supposed to play a similar role in maintaining tissue homeostasis, controlling growth and development and consequently they must be involved in physiological and pathological processes of claw horn formation.

This work was founded by the EU Lamecow project QLRT-2001-00969

References

Arita K, Akiyama M, Tsuji Y, McMillan JR, Eady RA, Shimizu H. Changes in gap junction distribution and connexin expression pattern during human fetal skin development. J Histochem Cytochem 2002: 50: 1493-1500

Di WL, Rugg EL, Leigh IM, Kelsell DP. Multiple epidermal connexins are expressed in different keratinocyte subpopulations including connexin 31. J Invest Dermatol 2001: 117: 958-964

Evans WH, Martin PEM. Gap junctions: structure and function. Mol Membr Biol 2002: 19: 121-136

Goliger JA, Paul DL. Wounding alters epidermal connexin expression and gap junction-mediated intercellular communication. Mol Biol Cell 1995: 6: 1491-1501

Labarthe MP, Bosco D, Saurat JH, Meda P, Salomon D. Upregulation of connexin 26 between

keratinocytes of psoriatic lesions. J Invest Dermatol 1998: 111: 72-76
Lucke T, Choudhry R, Thom R, Selmer IS, Burden AD, Hodgins MB. Upregulation of connexin 26 is a feature of keratinocyte differentiation in hyperproliferative epidermis, vaginal epithelium and buccal epithelium. J Invest Dermatol 1999: 112: 354-361
Matic M, Evans WH, Brink PR, Simon M. Epidermal stem cells do not communicate through gap junctions. J Invest Dermatol 2002: 118: 110-116
Richard G. Connexins: a connection with the skin. Exp Dermatol 2000: 9: 77-96
Salomon D, Masgrau E, Vischer S, Ullrich S, Dupout E, Sappino P, Saurat JH, Meda P. Topography of mammalian connexins in human skin. J Invest Dermatol 1994: 103: 240-247
Traub O, Hertlein B, Kasper M, Eckert R, Krisciukaitis A, Hulser D, Willecke K. Characterization of the gap junction protein connexin 37 in murine endothelium, respiratory epithelium and after transfection in human HeLa cells. Eur J Cell Biol 1998: 77: 313-322
Wiszniewski L, Limat A, Saurat JH, Meda P, Salomon D. Differential expression of connexins during stratification of human keratinocytes. J Invest Dermatol 2000: 115: 278-285

IMMUNOHISTOCHEMICAL DETECTION AND LOCALISATION OF GROWTH FACTORS IN BOVINE CLAW TISSUE

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Introduction

Horn production and the maintenance of functionally important structures are continual processes in bovine claw tissue. These are dependent on the proliferation and differentiation of horn-producing cells of the epidermis and the synthesis and breakdown of structural macromolecules and adhesion molecules in the basement membrane and dermis. A range of chemical messengers have been identified in integumental tissues such as skin and hair follicle (e.g. Stenn et al, 2001) but their presence and role in the bovine claw are poorly understood. The aim of the present study was to investigate the expression and localisation of two signalling molecules, basic fibroblast growth factor (bFGF) and transforming growth factor-β (TGF-β) in bovine claw epidermis and dermis. These growth factors have been shown to influence proliferation and morphogenesis in a range of tissues of the integument.

Materials and Methods

Hind right lateral claws were obtained immediately after slaughter from crossbred female beef cattle. The claws were transported to the laboratory on ice. Tissue blocks containing a small amount of horn and soft tissue epidermis and dermis were prepared, frozen in liquid nitrogen and then stored at -80 °C. These were then sectioned by cryostat at a thickness of 10 μm and then mounted onto Vectabond (Vector Laboratories) coated glass slides. The sections were subjected to immunohistochemical examination using primary antibody systems as follows: rabbit anti-bovine bFGF (Sigma, cat no. F3393) at a dilution of 1:1500 and mouse anti-human TGF-β (Serotec, cat no. MCA 797) at a dilution of 1:3000. Detection was achieved using the second antibody Vectastain Universal Elite ABC kit (Vector Laboratories) with appropriate anti-mouse and anti-rabbit negative controls. Sections were treated as appropriate with supplied blocking serum and 3 % hydrogen peroxide in methanol to deplete endogenous peroxidase activity. Sections were counterstained with hematoxylin.

Results

Examination of the immunostained histological sections established the location of bFGF consistently along the dermal-epidermal border and predominantly in the basal and suprabasal epidermal cells in the solear sections tested. Typical examples (highlighted by arrows) of vertical (Figure 2) and horizontal (Figure 3) sections show clear signals, which may be compared with the absence of stain in the IgG control (Figure 1). In contrast, the signals for TFG-β were localised in papillary dermis in representative solear (Figure 4), coronary (Figure 5) and laminar region (Figures 6, 7) sections.

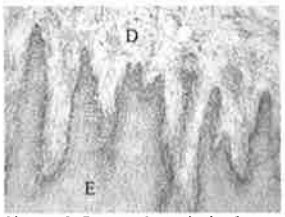

Plate 1: IgG control, vertical sole section, and hematoxylin, x100

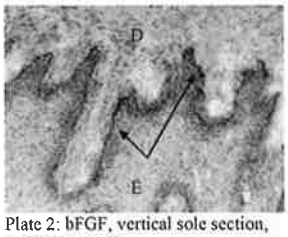

Plate 2: bFGF, vertical sole section, and hematoxylin, x100

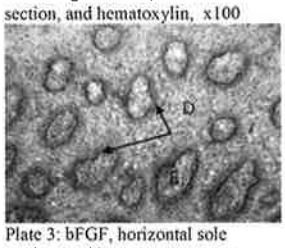

Plate 3: bFGF, horizontal sole section, and hematoxylin, x100

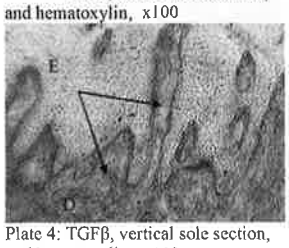

Plate 4: TGFβ, vertical sole section, and hematoxylin, x100

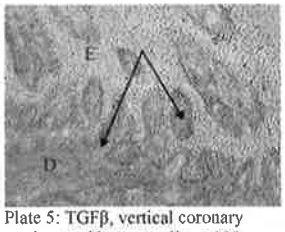

Plate 5: TGFβ, vertical coronary section, and hematoxylin, x100

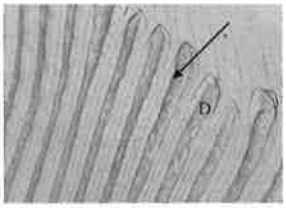

Plate 6: TGFβ, vertical laminar section, x100

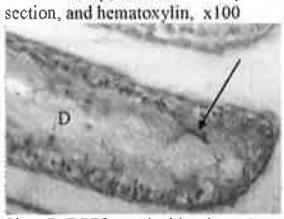

Plate 7: TGFβ, vertical laminar section, and hematoxylin, x400

D=dermis
E=epidermis

Discussion

It is concluded that the claw tissues studied express the growth factors in locations typical of those in other integumental tissues. For example, bFGF expression has been previously described in keratinocytes in skin, in which mitogenic, angiogenic and morphogenic effects have been recognised (Arbiser et al, 2000). Similarly, members of the TGF-β superfamily have been identified as components of the extracellular matrix produced by dermal fibroblasts. These signalling molecules produce downstream effects on synthesis of collagens and other proteins in extracellular matrix and act as inhibitors of keratinocyte proliferation (Yamasaki et al, 2003). The properties of extracellular matrix in dermal tissue, the proliferation of keratinocytes and interactions between the two are important in the function of claw tissue. Results from the present study can be used to assist investigation into mechanisms of regulation of these processes that are essential to maintain good health in the bovine claw.

References

Arbiser JL, Byers R, Cohen C and Arbeit J (2000). Altered basic fibroblast growth factor expression in common epidermal neoplasms: Examination with in situ hybridisation and immunohistochemistry. Journal of the American Academy of Dermatology 42: 973-977.

Stenn KS and Paus R (2001). Controls of hair follicle cycling. Physiological Reviews 81: 449-494.

Yamasaki K, Toriu N, Hanakawa Y, Shirakata Y, Sayama K, Takayanagai A, Ohtsubo M, Gamou S, Shimizu N, Fujii H, Miyazono K and Hashimoto K (2003). Keratinocyte growth inhibition by high-dose epidermal growth factor is mediated by transforming growth factor beta autoinduction: A negative feedback mechanism for keratinocyte growth. Journal of Investigative Dermatology 120: 1030-1037.

Acknowledgements

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VALIDATION OF LAME LIMB IDENTIFICATION THROUGH THERMAL IMAGING

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Abstract

The limb temperature of 50 dairy cows was recorded through using a thermal camera. The locomotion of each cow was then observed and scored. Of the 25 cows found to have detectable hind limb lameness a significantly higher temperature was found on surface of the limb in which lameness was detected. This increased temperature found on the lame limb was detected not only in the region of the horn capsule but also further up the limb on the metatarsus and at the tarsal joint.

Introduction

Thermography records the surface temperature of an object and is used in many industrial situations. It also has the potential to be used as a non-invasive assessment tool to detect areas of heat and swelling expressed on the skin of animals. As the collecting of images can be carried out remotely from the animal requiring little or no restraint the technique avoids temperature artefacts associated with capture and restraint.

Deterioration in the locomotion of dairy cattle was found to be among the most important determinants of cattle welfare in a survey of cattle welfare experts (Whay et al. 2003). However, in both a research and a commercial context reliable lameness detection remains a challenge. The sensitivity of locomotion scoring and lameness detection is difficult to validate other than through assessment of repeatability of trained observers. In addition it is known that in comparison with an observer trained in lameness detection UK dairy farmers only perceive about a quarter of lameness cases within their herds (Whay et al. 2002).

The pathological changes in the claws of cattle showing

signs of lameness may be detectable through thermographic imaging. A pilot study was carried out to investigate whether such changes in the surface temperature of the limbs of lame cows were detectable using thermal imaging.

Methods

Thermal images and surface skin temperature recordings were taken from 50 lactating dairy cows during May 2003 following Spring turnout on two farms in the South West of England. The measurements were made using a thermal imaging camera (ThermaCAMTM E2, FLIR Systems). Temperature recordings were taken of the lateral aspects of both hind-limbs at the metatarsal joint, mid metatarsus and the abaxial aspect of the lateral claw horn capsule. The lowest, average and maximum surface temperatures were recorded from each region. The cows were then walked on a flat concrete surface and their locomotion scored on a scale of 0 - 3 (0 - sound, 3 - severe lameness) and where detectable the lame limb was identified.

The thermography data recorded from the limbs of the dairy cows was corrected against the average temperature of the udder on the same side. This allowed a correction for variations in skin temperature due to changes in ambient temperature. A general Linear Model was then used to examine the relationship between the points on each limb where temperature was recorded and between the limb which was identified as being lame through locomotion scoring.

Results

Of the fifty animals observed 36 animals were scored as having some abnormality of locomotion of which 25 had clear hind-limb lameness where one limb could be identified as causing the limp. Analysis of the results found that at all three points measured on the limb the average and maximum temperature was significantly higher on the side of the lame limb than the sound limb ($P<0.01$) whereas measurement of minimum temperature was found to be a less consistent measure (Table 1). At the level of the hocks and the metatarsus the temperature on the lame limb was elevated by an average of 0.8°C while at the claw the temperature was raised by between 1 and 2°C. Fewer significant associations were found between lameness and the minimum temperature recorded at each region of the limb.

Table 1. The effect of lameness on the limb temperature recorded at the tarsal joint, metatarsus and claw horn capsule

		Difference in surface temperature recorded between lame and sound limb		
		minimum temperature	maximum temperature	average temperature
Region of limb where surface temperature was recorded	Lateral aspect of tarsal joint	n /s	n /s (0.051)	**
	Lateral aspect of metatarsus	*	**	***
	Lateral claw horn capsule	n /s	**	**

Discussion

The results demonstrated that an increase in temperature of the lame limb was detectable. Also that this temperature difference between the lame and sound limb extends further up the limb than may have been expected. A strong correlation between prevalence of lame cows and hock lesions (hair loss, swelling and ulceration) at the herd level have been reported by Whay et al. (2003). Findings supported by Klaas et al. (2003) who speculated that lame cows lie down more frequently than healthy cows and have difficulties in rising or changing position, so are more likely to develop pressure lesions. A combination of lameness causing lesions and increased lying time possibly associated with lesions on the hocks (lateral aspect of tarsal joint) may account for the widespread increase in limb temperature although this has not been confirmed.

The result showed an increased surface temperature on the lame limb of the cow this offers validation of identification of the lame limb using visual locomotion scoring. Work in the future will include characterising these changes in temperature in more detail and examining their association with specific lesion types.

References

KLAAS, I.C., ROUSING, T., FOSSING, C., HINDHEDE, J. AND S□RENSEN, J.T. (2003) Is lameness a welfare problem in dairy farms with automatic milking systems. Animal Welfare 12, 599-603

WHAY, HR, MAIN, DCJ, GREEN, LE, WEBSTER,AJF (2002) Farmer perception of lameness prevalence. 12th International Symposium on Lameness in Ruminants, Orlando. 355-358

WHAY,HR, MAIN,DCJ, GREEN,LE, WEBSTER,AJF (2003) Assessment of the welfare of dairy cattle using animal-based measurements: direct observations and investigation of farm records. Veterinary Record 153. 197-202

WHAY,HR, MAIN, DCJ, GREEN,LE, WEBSTER,AJF (2003) Animal Based measures for the assessment of welfare state of dairy cattle, pigs and laying hens: consensus of expert opinion. Animal Welfare 12. 205-217

TREATMENT AND OUTCOME OF INTERDIGITAL NECROBACILLOSIS (INTERDIGITAL PHLEGMON, FOOT ROT) IN 43 COWS

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Introduction

Interdigital phlegmon is perhaps the disease with the most synonyms: infectious pododermatitis, interdigital necrobacillosis, foot rot , foul in the foot, foot abscess, panaritium are often mentioned. Traumatic lesions of the interdigital skin, caused by rough floor, uneven ground, stones, straw or pieces of wood are the most common causes. Maceration of the skin by wet weather conditions, faeces and urine may predispose the claw to injuries (1-12). Pain, leading to mild or severe lameness and moderate to severe swelling of the interdigital space are major signs of the disease. Interdigital phlegmon has a worldwide occurence with massive economical losses in milk and meat production. Fusobacterium necrophorum and Bacteroides melaninogenicus are mostly isolated from this infection. Clark et al. (1986) were able to reproduce foot rot by experimental cultures of F. necrophorum alone, as they injected the agent through the skin into the dermis. Interdigital phlegmon is usually sporadic, but may be endemic in high intensity beef or dairy cattle production units (1, 3, 8, 10).

Material and Methods

In this retrospective study clinical and radiographic findings, treatment and outcome of 43 cases (1998-2003) with interdigital phlegmon are presented. All these patients had been referred by the local veterinarians to the clinic, with an advanced stage of an interdigital phlegmon. All patients underwent routine clinical and orthopaedic examination. The age of the animals ranged from 3 to 9 years (mean age 5 y). Thirty were Simmental cows, 7 were Holstein Friesian, 3 were Brown-Swiss and 2 were Crossbreds.
Clinical signs included moderate or severe lameness with marked swelling of the coronary region and the soft tissues of the interdigital space. In cases of advanced infection a characteristic fetid smell caused by the interdigital necrotizing lesions was noted.
Radiographic examination was performed in cases of severe lameness and obvious circular swelling over the whole coronet from dorsal to abaxial to rule out infection of the distal or/and proximal interphalangeal joint or involvement of the remaining claw.
The patients were prepared for surgical resection of the infected and necrotic tissues and/or digital amputation with an intravenous regional anaesthesia using 20 ml of procaine-hydrochloride (Minocain 2%, Atarost, Germany). A tourniquet of rubber tubing was applied directly in the middle of the metatarsus or metacarpus.

Results

Twenty-two cows showed a severe lameness (grade 3 and 4 out of 4), thirteen cows presented a moderate lameness (grade 2 of 4) and in 8 cases only a slight lameness (grade 1 of 4) was noted. In 19 cows the left hindlimb was affected, in 20 cases interdigital phlegmon was located at the right hindlimb. Two cows had infections of both hindlimbs. The right frontlimb was affected in 2 cows. Radiography was performed in 15 cattle with a severe lameness. Radiographic signs of bone and/or joint infection were widening of the joint space, loss of joint and bone architecture as well as reactive periostal new bone formation.
Surgical resection of the affected soft tissues of the interdigital space was performed in 19 out of 43 cattle. Starting from the causative penetrating wound the infected and necrotic soft tissue structures of the interdigital space were resected completely. In one case the infected and necrotic tissue reached the axial capsule of the distal interphalangeal joint showing a serous, slightly turbid effusion: in this case the distal interphalangeal joint was opened from this track and was lavaged with 2000 ml isotonic saline solution with diluted 0.1% polyvidon-iodine-solution.
Although surgery and parenteral antibiosis was done infection spread to the distal interphalangeal joint in one case and led to amputation of the affected claw.
In 23 cows (out of 43) a purulent arthritis of the distal interphalangeal joint and in 1 cow a purulent-necrotising arthritis of the proximal interphalangeal joint had developed from the interdigital phlegmon. 12 of the 24 cows were euthanized or slaughtered due to the bad condition, the poor prognosis or economic reasons.
Amputation of the infected claw was performed in 11 cases, another one was treated by resection of the distal interphalangeal joint. In addition, infections of the digital flexor tendon sheaths occurred in five cases (out of 24). Three of them were euthanized after diagnosis: one cow had already developed an infection of the fetlock joint, another one showed a severe swelling of the remaining hind claw because of a deep sole ulcer. In one case amputation was refused by the owner. In 2 cows the deep and superficial flexor tendons and the infected flexor tendon sheaths were resected.
One out of these 24 cases showed a severe interdigital phlegmon with a purulent arthritis of the distal interphalangeal joint, multiple abscesses on the lateral digit and distal limb and a purulent endocarditis and was euthanized.
Systemic and local antibiosis was used for 3-5 days. For systemic antibiosis the cattle were treated with 1 mg ceftiofur (Excenel , Pharmacia & Upjohn) per kg body weight or 10 mg oxytetracycline (Engemycin10% , Intervet,

Vienna) per kg body weight. When amputation of the claw was performed 20.000 IU benzyl-penicillin and 20 mg streptomycin per kg body weight (Peni-strepto , Virbac Laboratoires) were given for 5 days.
Depending on the surgical technique clinic hospitalisation ranged from 2-28 days (mean 10.4).

Discussion

The first observed signs of foot rot are varying degrees of lameness, from barely noticeable to an extensive condition in which cows get more or less recumbent (1, 12). Symptoms like widening of the interdigital space due to a moderate or severe swelling along with a reddening of the skin and the coronary band, heels and necrotic and demarcated interdigital tissue could be detected in all cases. If the foot rot condition is severe and is not treated in the early stages, complications may result such as septic arthritis of the distal or/and proximal interphalangeal joint, septic inflammation of the digital flexor tendon sheath, other deep infections of the foot as local abscesses and haematogenous spread of infectious material with subsequent purulent endocarditis, abscesses in the lung, liver or kidneys (1, 12). In this study 24 cows showed a purulent arthritis of the distal and proximal interphalangeal joint, in 5 cases an inflammation of the flexor tendon sheaths and in one case a septic endocarditis.

References

1 Berg JN, Franklin, CL (2000): Interdigital phlegmon / interdigital necrobazillosis / acute foot rot of cattle: considerations in etiology, diagnosis and treatment. Proc. 11th International Symposium on Disorders of the Ruminant Digit, Parma, Italy, 3-7. September 2000, p. 27-30.
2 Bergsten Ch (1997): Infectious diseases of the digits. In: Greenough, PR, Weaver, AD (eds): Lameness in cattle. 3rd ed., Philadelphia, WB Saunders, pp. 89-94.
3 Berry, St. (2001): Diseases of the digital soft tissues. Vet. Clin. North America: Food Animal Prac. 17, 35-37.
4 Clark BL, Emery DL, Stewart DJ (1986): The aetiology and prevention of interdigital necrobacillosis (foot abscess) in cattle. In Stewart, DJ et others (ed.): Footrot in ruminants. Proc. Workshop, Melbourne, 1985, p. 275-8.
5 Clark BL, Stewart DJ, Emery DL (1985): The role of Fusobacterium necrophorum and Bacteroides melaninogenicus in the aetiology of interdigital necrobacillosis in cattle. Australian Vet. J. 62, 47-9.
6 Clark, B.L., Emery, D.L., Stewart, D.J., Dufty, J.H., Anderson, D.A. (1986): Studies into immunisation of cattle against interdigital necrobacillosis. Aust. Vet. J. 63, 107 - 110.
7 Henke CL, Howie NM, Smith FJ, Flaus L, Kausche FM (1994): Efficacy of Ceftiofur-Sodium (Excenel*) for the treatment of acute foot-rot (interdigital necrobacillosis) in cattle. Proc. 18th World Buiatrics Congress, Bologna, Italy, August 29 - September 2, 1994, Vol. 2, 1355-8.
8 Iselin U, Lischer CJ, Zindel F (1998): Erfahrungen mit der Prophylaxe des Panaritiums bei Alprindern in Graubünden. Abstracts Schweizer Tierärztetage 1998 in Interlaken, 4.-5. September 1998, S. 116-7.
9 Kausche, FM, ROBB, EJ (2002): A comprehensive review of Ceftiofur Na (Excenel sterile powder) and Ceftiofur HCl (Excenel RTU suspension) in the treatment of acute bovine foot rot. Proc. 22nd World Buiatrics Congress, Hannover, Germany, August 18-23, 2002, p.83.
10 Monrad J, Kassuku AA, Nansen P, Willeberg P (1983): An epidemiological study of foot rot in pastured cattle. Acta Vet. Scand. 24, 403-17.
11 Morck DW, Olson ME, Louie TJ, Koppe A, Quinn B (1998): Comparison of ceftiofur sodium and oxytetracycline for treatment of acute interdigital phlegmon (root rot) in feedlot cattle. J. Am. Vet. Med. Assoc. 212, 254-7.
12 Stokka, GL, Lechtenberg, K, Edwards, T, MacGregor, S, Voss, K, Griffin, D, Grotelueschen, DM, Smith, RA, Perino, LJ (2001): Lameness in feetlot cattle: Interdigital necrobacillosis. Vet. Clin. North America: Food Animal Prac. 17, 189-190.

CHANGES SEEN IN THE SOLE HORN CORRELATED WITH HORIZONTAL GROOVES IN A DIGIT OF DAIRLY CATTLE

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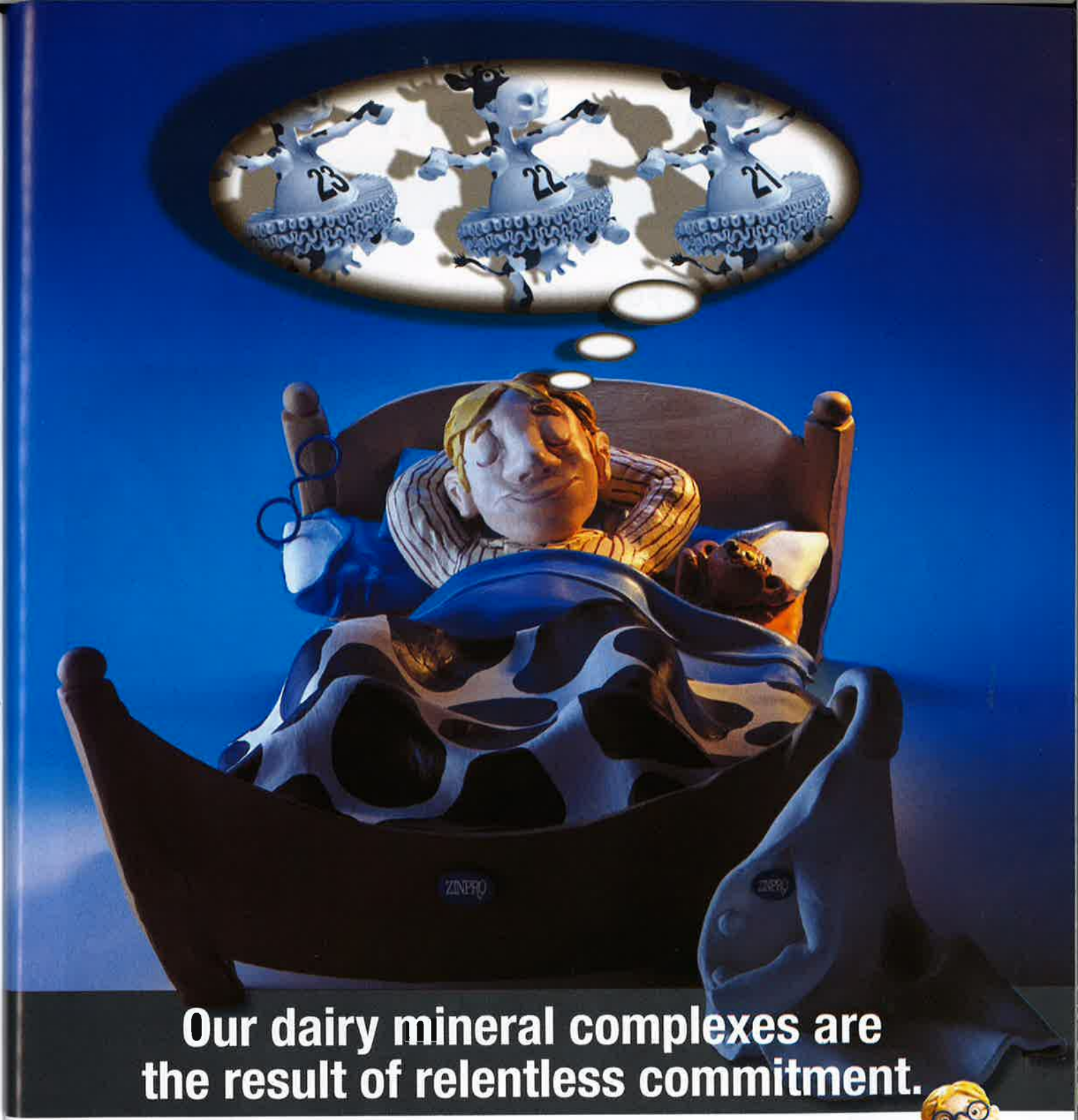
Abstract

In a digit of dairy cattle obtained from a slaughterhouse, three horizontal grooves were observed on the wall. In the vertical section of the sole, two horizontal thin layers were seen, which had yellowish color.
The first horizontal groove formed nearest under the digital band was connected to the first horizontal thin layer in the vertical section of the sole. It showed that the horizontal groove and the thin layer in the sole were formed at the same time. From this observation, the growth of the sole could be estimated.
The surface of the heel horn was detached at the groove on the heel bulb.
The relationship between horizontal grooves and the sole changes will be discussed.

Authors index



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Research, commitment, results. Give us a call or visit **www.availa4.com** to find out what our scientists have discovered lately.

Product of Minnesota

HCA ZIN0671

Kovex Foam

PREMIUM • PROGRAM

Active Foam for animal foot hygiene

- Protection of healthy animals by preventing cross contamination risks
- Optimal contact time
- Effective at all temperatures
- Easy to use and less stressfull for the animals
- Excellent ecological profile
- Noticeable time saving compared to traditional foot bath

ECOLAB

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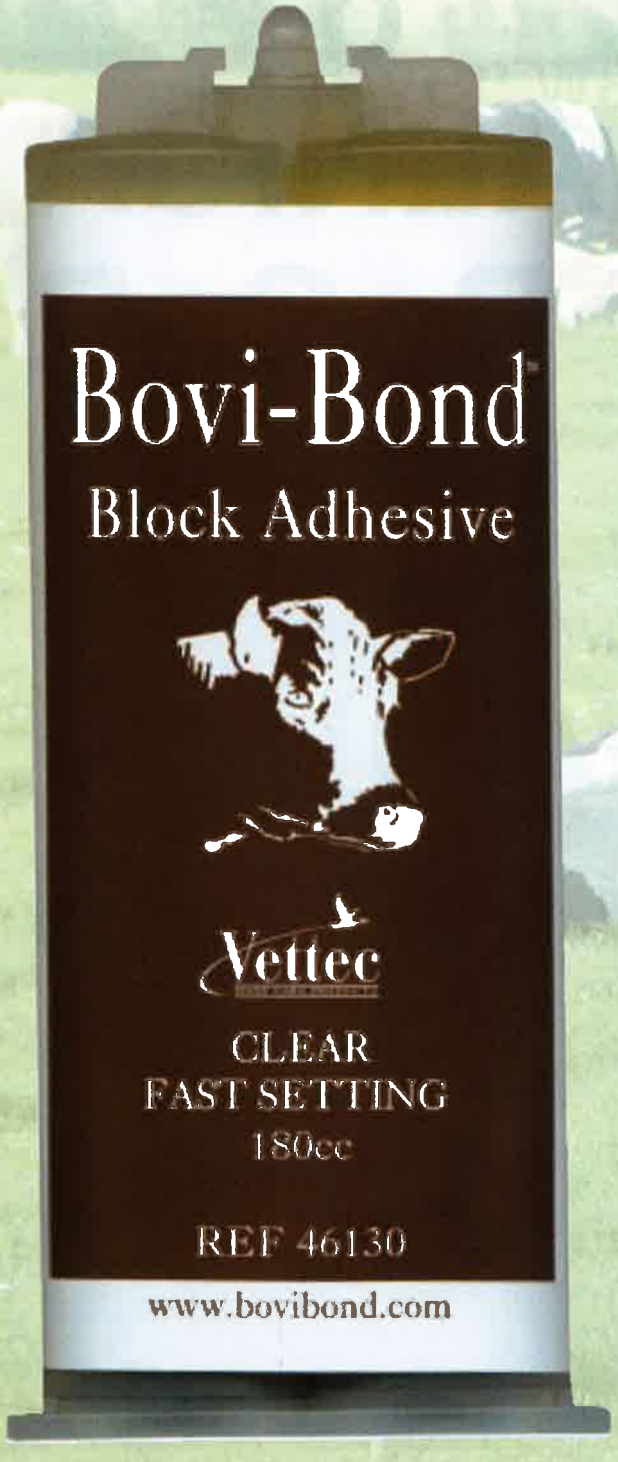
www.kovexfoam.com

Bovi-Bond is a state of the art fast setting adhesive used to glue wood or rubber blocks to bovine hooves. This strong adhesive sets in thirty seconds and secures a solid bond. This unique chemistry possesses no odors and is non toxic. Simply dispense **Bovi-Bond** onto the block or the hoof and position the block against the hoof. No mixing is required and no waste is produced. **Bovi-Bond** may be stored cold or warm and possesses a one year shelf life.



- ✓ *Fast Setting*
- ✓ *No Mixing*
- ✓ *No Waste*
- ✓ *Easy Application*
- ✓ *Fewer Steps*
- ✓ *Non Toxic*
- ✓ *No Methyl-methacrylate-monomer*
- ✓ *No Fumes*

Clean and Dry the hoof

Apply bovi-bond

Position block

Finished hoof

Vettec
Hoof Care Products

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What a clever warrior!

Excellent penetration to the site of infection

LINKOMICIN F

Lincomycin, neomycin and dexamethasone

Intramammary suspension

One of the main problems in treating mastitis is poor penetration of the antibiotic to the site of infection. Antibiotic medicines often fail to reach the target. The most persistent pathogen causing mastitis is ***Staphylococcus aureus***. There are few antibiotics capable of reaching it, and even fewer that can successfully overcome it.

Linkomicin F is one of them. Use it and feel the joy of victory.

COMPOSITION 10 ml of suspension contain 200 mg of lincomycin (in the form of hydrochloride), 200 mg of neomycin (in the form of sulphate), and 1 mg of dexamethasone-21-phosphate. **ACTION** Lincomycin is a lincosamide antibiotic. It is bacteriostatic in action against gram-positive pathogens and is especially effective against *staphylococci* and *streptococci*. Neomycin is an aminoglycoside antibiotic acting bactericidally against gram-negative microorganisms including *E. coli*. Dexamethasone-21-phosphate shortly supresses the inflammatory reaction and edema of the mammary gland. Linkomicin F is an oily suspension rapidly and easily penetrating into milk and gland parenchyma. The combination of the two antibiotics in Linkomicin F covers a broad spectrum of causative organisms, both gram-positive and gram-negative. **INDICATIONS** Acute and subacute mastitis caused by: *Streptococcus agalactiae, Streptococcus dysgalactiae, Streptococcus uberis, Staphylococcus aureus, E. coli*. **CONTRAINDICATIONS** Not known. **DOSAGE AND ADMINISTRATION** The content of the syringe is applied into the affected quarter after each milking, best at 12-hour intervals. Cows should not be milked for at least 6 hours after the drug application. In moderate lactating animals the Linkomicin F is applied every 24 hours, in high lactating animals it is applied after milking every 12 hours for 3 to 5 succesive days. Treatment lasts for 3 to 5 days and may be prolonged if necessary. **WITHDRAWAL PERIOD** Animals must not be slaughtered for human consumption during treatment and within 2 days following the last administration of the drug product. Milk from treated animals must not be used for human consumption during treatment and for 72 hours following the last administration of the drug product. **STORAGE** The drug should be stored at a temperature up to 25 °C. Keep out of reach of children. **AVAILABILITY** On prescription only. **PRESENTATION** Boxes of 20 syringes containing 10 ml of the suspension each. **PRODUCED BY** Lek Pharmaceutical and Chemical Company d.d., Verovškova 57, Ljubljana, Slovenia. More details on the medicine are available from the manufacturer: **Lek Animal Health,** Verovškova 57, **SI -1526 Ljubljana, SLOVENIA, Tel.:** +386 1 580 29 51, **www.lek.si/animal health/**, veterina@lek.si

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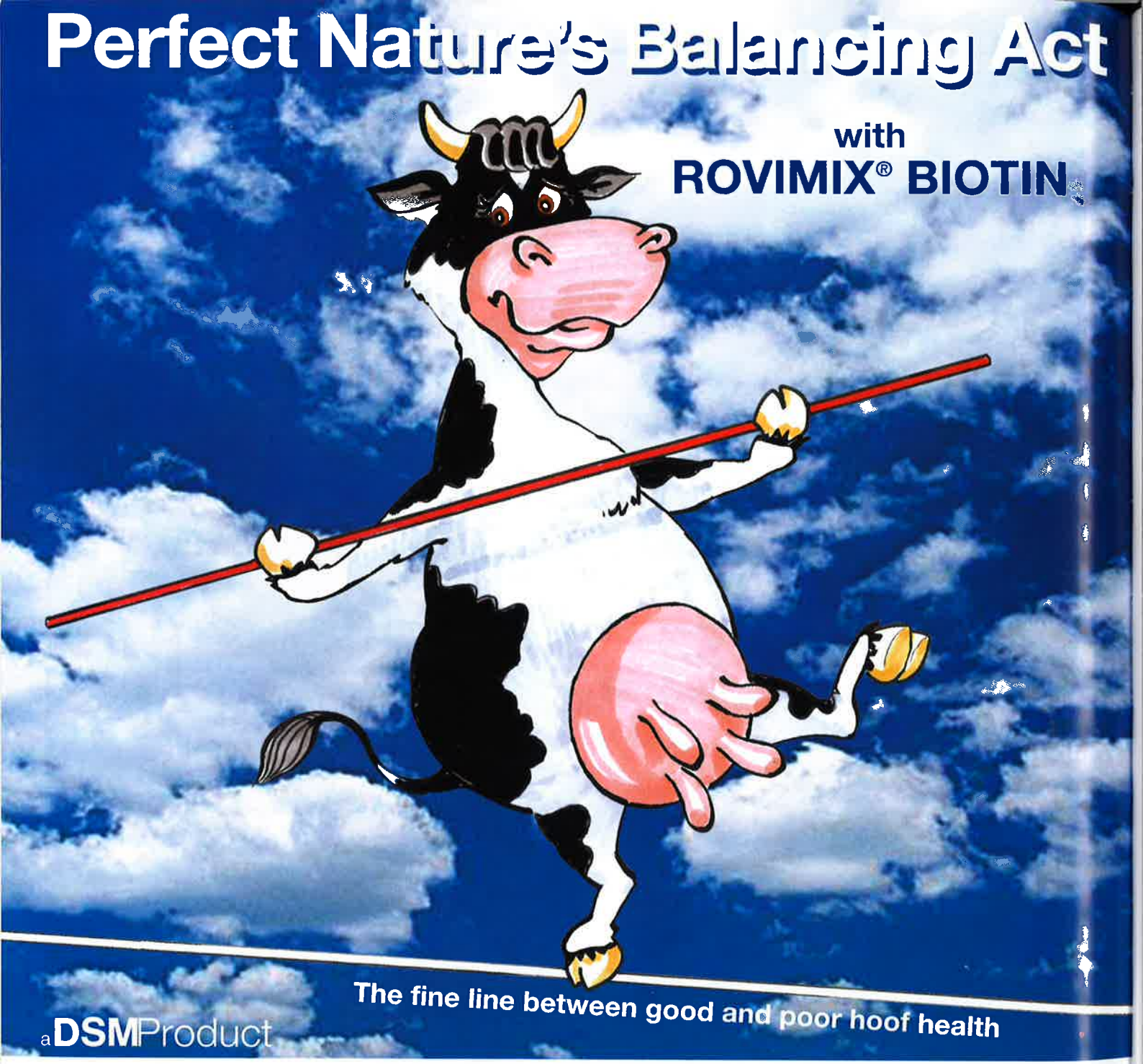
Perfect Nature's Balancing Act

with
ROVIMIX® BIOTIN



The fine line between good and poor hoof health

a DSM Product

Across the world, farmers are seeing the benefits of biotin for dairy cows

- Helps improve horn quality, hoof health and animal welfare
- Highly cost-effective and contributes to better profitability
- Easy to add to dairy feeds and minerals
- New data! Biotin improves milk production too

20mg per day is all it takes

Neat feet, Biotin feet

Ask for ROVIMIX® H-2 (Biotin) - the only scientifically proven source of Biotin for improving hoof health and milk production

http://www.dsmnutritionalproducts.com

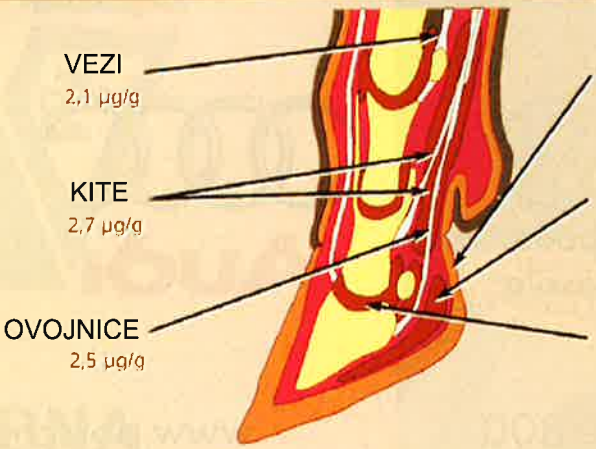
Unlimited. **DSM**

Terramycin*/LA

VISOKA KONCENTRACIJA V CILJANIH TKIVIH

VNETJE PARKLJEV

Koncentracija Terramycin LA v tkivih parklja



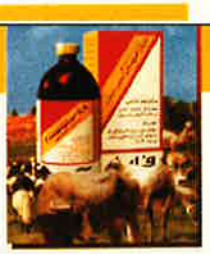
IRIS www.iris.si

Pfizer Animal Health

Terramycin LA: Zdravi lezije in ustavi širjenje infekcije!

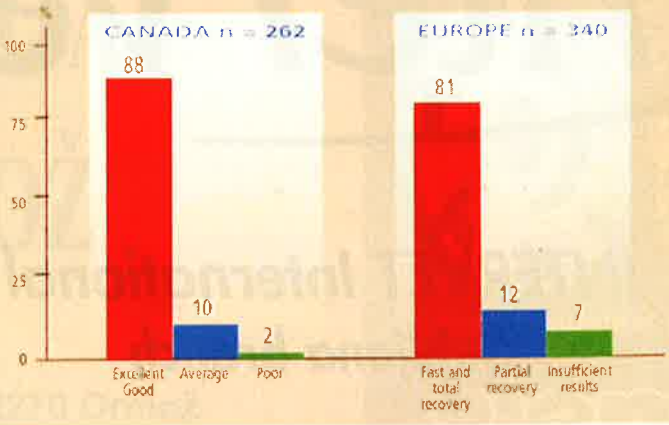
Terramycin*/LA

DOKAZANA TERAPIJA POVSOD PO SVETU



VNETJE PARKLJEV

Zdravstvena ocena pri vnetju parkljev



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Pfizer Animal Health

Terramycin LA: Zdravi vnetje parkljev hitro in učinkovito → Zdravilo obnovi proizvodnjo mesa in mleka!

ZASEBNA VETERINARSKA AMBULANTA

VETERINARSKA POSTAJA ORMOŽ

Ljutomerska 25, 2270 Ormož
Tel.: 02 740 43 00, Fax: 02 740 43 01
E-pošta: veterina@veterina-ormoz.si
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