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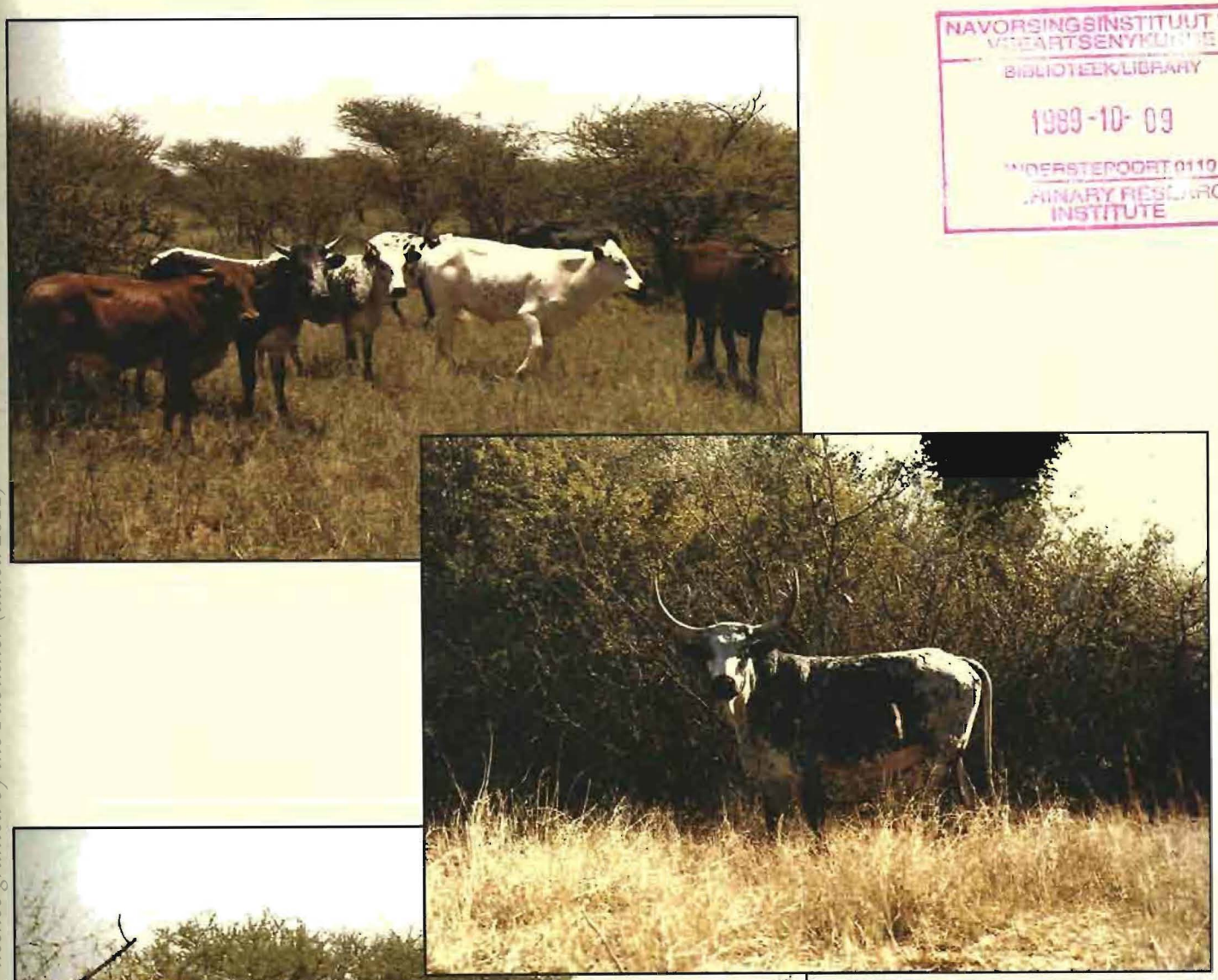

Nguni cattle

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Contents • Inhoud

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IMPORTANT REQUIREMENTS FOR FUTURE ANIMAL PRODUCTION-ORIENTATED RESEARCH WITH PARTICULAR REFERENCE TO VETERINARY SCIENCE*

I had intended to start my address by thanking you for the honour you have bestowed on me to deliver the Theiler Memorial Lecture. Then I encountered this remark made by Theiler in 1935 in Gutsche's biography[2], which brought me down to earth, but did not diminish my gratitude:

Honours kept coming - "a sure sign of advancing senility", he told Alfred.

As theme for this lecture I have selected what I regard as 3 cardinal interrelated developments which will have a profound effect on animal production-orientated research requirements and the future education requirements for such research. I shall try to motivate this theme by means of facts, figures and my own thoughts, some of which are speculative, but will hopefully be convincing. Since this occasion commemorates Sir Arnold Theiler, particular attention will be paid to veterinary aspects of the theme.

THE FUTURE HOME OF ANIMAL PRODUCTION-ORIENTATED AND OTHER AGRICULTURAL RESEARCH

The correct home for agricultural research, which includes veterinary and other animal production-orientated research, in the South African household has occupied the minds of many; from the researcher at the bench, middle and top management levels, to politicians, in the past as well as in recent years. The so-called "Kolb Report" is the most recent and, at the moment, most tangible addition to the published line of thought.

I shall confine myself today to thoughts on and recent developments with regard to the home for research being done by the Department of Agriculture and Water Supply, which includes the Veterinary Research Institute, Onderstepoort and other animal production-orientated research being done by the Department.

It is clear from Gutsche[2] that Theiler felt strongly that the Onderstepoort Research Institute should be unfettered from the bureaucratic system applying to the Department of Agriculture.

Theiler was not prepared to accept these restrictions and proposed "a very strong protest demanding the right of Onderstepoort to be put outside general arrangements by reason of the nature of its work, by its traditions, by its standing in the scientific world and its outstanding success .... It has the right to have its own way - the psychology of scientific workers should not have to give way to the general machine-like way of working of the Treasury, the Auditor-General and the Agricultural Department".

*Theiler Memorial Lecture given at the 5th Faculty Day of the Faculty of Veterinary Science, University of Pretoria on 5 October 1988

Many have shared that view. It must, however, be realised that the account for Onderstepoort's research budget and those of other research institutions within the Department is being paid by the taxpayer. In pure government service, bureaucracy is a given fact. The Treasury, Commission for Administration and Auditor-General are essential for orderly government administration. These institutions are like the Rock of Gibraltar. There may be room for improvement in their *modus operandi*, but they cannot be wished away. Any move away from the "hated" bureaucracy will mean having to find funds, to a greater or lesser extent, elsewhere.

This is particularly relevant now in view of the Government's avowed policy of privatisation and deregulation. This can be interpreted to be a green light for state funded agricultural research to move out of the government service per se. Such a development and the anticipated advantages that will accrue, can also be construed as being a contribution to the State's declared policy to "restructure and rebuild agriculture" in order to achieve a better agricultural dispensation for the farmers and thus for the country as a whole.

A move towards a semi-state, also known as parastatal institution, which will probably be known as an Agricultural *Research Council (ARC), is* on the cards for research being conducted by the Department of Agriculture and Water Supply. The Onderstepoort Veterinary Research Institute, being its largest single component, will be implicated.

The Department of Agriculture and Water Supply has been instructed by the Minister of Agriculture to investigate the possibility of establishing an ARC and to report back before the end of 1988 on its likely structure, probable personnel requirements and estimated financial implications. Sources of possible funding must also be provisionally explored. This is of course excellent news to me since I have been propagating this move for the Onderstepoort Institute by written word and verbally ever since I was in a position to do so.

It must be clearly appreciated that a parastatal body such as an ARC will of necessity obtain a considerable proportion of its funds from the State. The amount is, however, most unlikely to be increased annually, particularly in respect of running expenses. This means that it will be eroded by inflation, and an annual escalation in private funding will have to be sought by the ARC to compensate for this loss, not to mention providing for growth.

The most important message is that the products of research will have to be marketed in future in a format sought by the consumer.

In this case I am referring to the marketing of technology generated by research in a package which the market seeks. In the case of veterinary and other animal production-orientated research the potential market extends from "conception to consumption", in other words from the producer to the consumer. Into this reasoning can also be read that research will not stop at the farm gate, as has been the policy hitherto, although exceptions were made. This means that the farmer is not the only client for the products of agricultural and veterinary research, but that all the farming-orientated industries are also potential clients.

Corrollaries of this reasoning are that:

- more personnel may be appointed if the necessary funds can be found
- the ARC will be in a better position to compete for highly-qualified personnel
- there will be a shift away from the "publish or perish" concept to "publish and perish".
- it will consequently be more important to file a patent than to publish a paper
- there will inevitably be a shift away from fundamental to more applied research
- evaluation of the progress of research will feature much more strongly
- the consumer of research results will have much more say - researchers have hitherto enjoyed a remarkable degree of freedom from external or even internal pressures

It must be clearly understood, however that a parastatal body is no Utopia.

Probably the most important potential problem which will have to be firmly addressed is the fact that "he who pays the piper calls the tune". Special care will have to be taken that fundamental research, which is the backbone of long-term scientific progress, is not an eventual casualty of the system. Many of the big scientific breakthroughs come from "fortuitous observations" made during fundamental scientific research. For this reason, State funding can, in my opinion, never be entirely dispensed with.

FUTURE RESEARCH REQUIREMENTS FOR PROGRESS IN ANIMAL PRODUCTION

I think it can be positively stated that Theiler was aware of the fact that animal production and animal health cannot be divorced from one other. In other words, the ultimate objective of any research on diseases of animals must be to keep animals healthy so that they can produce optimally. In 1928 Theiler said:

"The Institute at Onderstepoort, South Africa which was founded and organised by me, did not deal with all the aspects of animal health as I propose should be done in Australia. It dealt almost exclusively with disease. ..... Animal health ..... is national wealth"[2]

The food requirements for a self-sufficient South Africa, a situation that would

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be expedient for strategic and economic reasons, for the coming century will be determined by its population growth. The latter can only be described as awesome. It has been predicted that the total human population will number about 47 million people by the year 2000, most of whom will have adopted a Western food consumption pattern. In the average Western diet, animal products provide 70% of the protein, 35% of the energy and most of the required minerals[3].

One expert has estimated that the increase in the requirement for beef will be 50%, for mutton 30%, and for dairy products 60% by the turn of the century[4], to name but a few products. Another predicts a 70% increase for beef and 75% for mutton[3]. Irrespective of which of these predictions is correct, it is clear that these goals can only be attained by a considerable increase in animal production.

Theiler's[2] view on South Africa's potential for beef production is clearly overoptimistic:

"The last hindrance to cattle farming in South Africa has now been cleared up and this country will now become one of the biggest producers of meat"[2].

It is obvious that we cannot increase our cattle, sheep, goat and game numbers dramatically because we already have a full house under extensive conditions. The potential for increase by greater intensification is, with the exception of poultry and pigs, also limited. A dramatic increase in the productivity of our ruminant livestock is the only alternative left to reach the abovementioned animal production goals. To reach a goal of 70% more beef, for example, we will have to slaughter 3,7 million cattle annually instead of the current 2 million head. How will we reach this figure?

There is considerable scope for the increase of animal production by merely improving the management of our national herd to obtain better nutrition, a quicker growth rate, increased fertility and better immunity against diseases. A key to much of this lies in persuading the South African farmer to keep fewer, but more productive livestock.

If the weaning rate of our national beef herd could be increased, for example, from the current estimated less than 50% to 65%, which is not unrealistic, 1,95 million instead of 1,5 million calves could be weaned annually. This means that both an increased fertility and a lowered mortality rate are required. A higher turnover of cattle (the current turnover is 26%) will be possible because 450 000 more cattle could be available for slaughter each year. Because the veld cannot accommodate more animals, better selection and earlier culling of females must result. Increased feedlotting has already ensured that the ratio of young to older animals being slaughtered is increasing, namely from 44% in the A category in 1983 to 53,5% in 1987 (R.T. Naude, ADSRI, Irene, personal communication, 1988). Some further growth in the feedlot industry would be essential to absorb the higher number of animals available for slaughter. This is again dependent on a low grain price structure. There is likewise much room for improvement of the current 70% weaning rate of sheep.

The government's soil conversion scheme, which involves the conversion of about one million ha of marginal cash crop lands to artificial pastures over a period of 5 years, can also make a contribution of about 300 000 LSU, consisting of both sheep and cattle, to the required shortfall. It is, however, likely that most of these pastures will be used initially to relieve the grazing pressure on already overburdened natural pastures.

It therefore seems unlikely that we will be able to reach a 50% increase in beef production, for example, by a more rapid turnover of animals. A 70% increase by this method seems a pipe dream. The same arguments would apply to mutton, etc.

To reach the required increases in the abovementioned commodities by the year 2000, increased production per animal by higher quality animals is indicated. Performance and progeny-testing are extremely useful tools in this regard. We don't need excessively large animals, but cows that each wean a calf weighing 200kg every year. Performance-testing, which is being run by the State at present, does not nearly meet the national requirement. For example only 25% of the beef breed bulls acquired for breeding purposes annually, are being performance-tested at present under the largely government-sponsored scheme. Privatisation of performance-testing seems unavoidable.

However, more rapid methods to increase the performance of our livestock than the relatively slow progress provided by conventional selection methods will have to be sought and exploited if we are serious about reaching the abovementioned animal production targets. Biotechnology at both the molecular and cellular levels of genetic manipulation offer the choicest fruits in this regard.

Although Theiler and his contemporaries could not have foreseen biotechnology as we know it today, their awareness of the importance of progress achieved by pure science is a measure of their intellectual greatness. The following quotations by Theiler (1905) and Schonland (1908), respectively, from Gutsche[2], illustrate this point:

"We may assert with confidence that the time is not far distant when South Africa will not be devastated by ravaging diseases. And this point will be won not only by the advance of our particular branch of science but by the advance of science in general".

"Your work" he told Theiler, "illustrates in a particularly happy manner the fact that progress in applied science must go hand in hand with progress in pure science.....".

The theoretical possibilities open to exploitation by the variety of powerful biotechnological techniques which have been and are constantly being developed, seem to be infinite. Our manpower and financial resources are, however, so limited, that it would be wise to concentrate them on those problems which are unique to South Africa, and for the rest to make as much use as possible of knowledge generated elsewhere.

I regard the following objectives as the highest priorities for the RSA:

1. A dramatic breakthrough in respect of ruminant digestion which will enable ruminants to utilise the millions of tons of available low grade roughage such as veld grass and crop residues more efficiently. This should enable us, for example, to market younger animals off the veld or other roughage, in a finished condition and to eliminate winter nutritional stress in females. Development by recombinant DNA technology of ruminal micro-organisms (super bugs) that can effect this miracle is, for example, no longer such a far-fetched idea.

2. Embryo transfer technology must be made freely available on a country-wide scale. This technology includes all the techniques associated with embryo transfer such as multiple ovulation, recovery and freezing of embryos obtained thus or from ova fertilised in vitro, embryo splitting, embryo fusion, sexing of embryos and gene transfer. England is already offering beef embryos salvaged as ova from abattoirs and fertilised in vitro as an alternative to AI. Moreover, Australia and New Zealand are importing Angora embryos, against our wishes, from South Africa for the benefit of their countries. Is this technology being exploited to multiply genetically superior small stock in South Africa to any significant extent?

3. Gene transfer is a technique which has great theoretical potential for use in Africa, and hence the RSA, because it will enable us to produce animals with specific adaptational genotypes that are tailor-made to make them highly productive under African environmental conditions. The genotypes concerned have already been provided by natural selection.

   I am thinking here of transgenic animals that are resistant to diseases such as trypanosomiasis and tick-borne diseases. Perhaps even more important is resistance to the plethora of species of African ticks. We have already determined that an indigenous Sanga breed such as the Nguni has large numbers of individuals that are highly resistant to our cattle ticks. This genetically determined characteristic would be most useful in faster growing breeds. Once established, such transgenic animals can be multiplied by the abovementioned embryo transfer technology.
   An Australian genetically engineered vaccine against the blue tick, *Boophilus microplus*, is on the cards and vaccines against other tick species are bound to follow suit. However, I am prepared to predict that ticks will find a way to circumvent the immunity thus induced.

   Their ability to manifest resistance to acaricides is an indication to me that there will also be individuals in a population endowed with the ability to avoid an immune response directed at specific antigens. The more far-sighted, longer term approach of developing cattle with transgenically induced tick-resistance should therefore not be neglected. In the meantime the use of tick-resistant breeds of cattle in the appropriate environment should be

exploited much more fully.

The ultimate aim regarding worm and blowfly control in sheep should be genetically determined resistance. Resistance to chemical control by the parasites concerned, and the increasing pressure against pesticide residues in animal products and against pollution, will make this approach inevitable. Transgenic animals, and their multiplication by embryo transfer technology, again come into the picture as a means of bringing a resistance gene(s) into a breed more quickly than by classical selection for resistant animals.

Mice, equipped by gene transfer with a human growth hormone gene, grew more than twice as fast as litter mates. This has opened up the way for similar research in other species. I doubt, however, whether elephantine cattle or sheep will be an advantage production-wise under extensive South African conditions.

Transgenic fodder plants and grasses can also make an important contribution to increased animal production. A sulphur-containing lucerne cultivar which increases wool production has, for example, been developed in Australia. More digestable indigenous grasses, which are already drought resistant, or the introduction of drought-resistance genes into more nutritious grasses would be most useful as improved artificial pastures, for example, under the crop withdrawal scheme, or even natural pastures, which would amount to radical pasture improvement.

4. Recombinant-DNA technology has made it possible to produce vast quantities of growth hormone in vitro, which has been shown to substantially increase growth in pigs and sheep and to induce considerably higher milk yields in cattle. It still remains to be seen whether the product will eventually be released for general use. Public pressure against its use seems to be triumphing in Europe, and the USFDA appears to be dragging its feet too.

5. Genetically engineered vaccines against local diseases must feature strongly in any research programme aimed at increasing animal production. Particularly relevant are those diseases against which it has not been possible to develop vaccines, such as snotsiekte, jaagsiekte and cysticercosis of cattle, or instances where existing vaccines are either not very effective, or for various reasons, impractical to use or dangerous to produce. Important examples of the latter are vaccines against heartwater, redwater, anaplasmosis (current vaccines consist of infected blood, with obvious disadvantages), bluetongue (which is a live attenuated vaccine consisting of 15 serotypes, also with obvious disadvantages) and FMD (which consists of inactivated virulent viruses).

6. Recombinant-DNA technology will also provide a series of diagnostic probes for a variety of diseases and parasites which could revolutionise the diagnosis of diseases and carrier states. The detection of measles in live cattle; crush-side diagnoses of diseases such as heartwater, redwater, and anaplasmosis; detection of the carrier states of *Theileria lawrencei* in buffalo, jaagsiekte in sheep, bluetongue in cattle and sheep destined for export, equine viral arteritis and various other viral diseases in imported stock, biliary fever and horsesickness in horses destined for export, and heartwater infection in ticks, are a few examples which come to mind.

Biotechnology therefore has much potential for making a significant contribution towards increasing the productivity of our extensively-farmed livestock. It is, however, longterm research and consequently unlikely to have a dramatic effect before the year 2000. Therefore our imports of the commodities referred to initially, are likely to increase. It may well be necessary to fill in the balance with pork and chicken.

FUTURE EDUCATION REQUIREMENTS FOR PROGRESS IN ANIMAL PRODUCTION

I am convinced that much of the progress required in animal production will, in the medium and longer term, depend on research in which biotechnology in its various guises, will feature strongly. Suitably-trained manpower will have to be found for this purpose.

From the above exposition it is also clear that a veterinary qualification is not a prerequisite for any of the research work required. Whereas experienced veterinarians should be in their element with the required improvement in the management of livestock, and likewise those with appropriate post-graduate training with embryo transfer technology and all its permutations, the work can also be done by non-veterinarians. Much of the basic research on embryo transfer technology in this country is already in the hands of non-veterinarians who have been trained in a variety of biologically-orientated natural sciences.

Applicable research approaches for which there is currently the greatest need and which offer the greatest chances of success are:

- Biotechnology, which includes embryo transfer technology and all its ramifications, gene transfer, molecular biology, r-DNA technology, microbiology and immunology.
- Genetically-determined resistance to disease and parasites, which can be achieved relatively slowly by conventional methods or probably faster by means of gene transfer.
- Physiology, particularly that of ruminant nutrition and digestion.
- Chemical and molecular pathology, and pathogenesis of disease.
- Ecology, under which umbrella I include studies on the epidemiology of disease, ecosystems, pasture sciences and plant poisonings, livestock and game management, and pollution-orientated sciences, which would include the use of industrial byproducts to produce food and energy.

These research approaches all require a sound background knowledge of the basic, biologically-orientated sciences such as biochemistry and molecular biology, microbiology, physiology, biology, genetics and ecology.

Throughout his career, even before his association with the brilliant biochemist, Harry Green, Theiler had what can be described as a yearning for the "pure sciences", as is once again illustrated by this quotation from Gutsche[2]:

"Theiler concluded his address with a virtual expression of his own philosophy: Foster by all means the pure sciences. They are, in the hands of experts, the medium of solving the many economic problems of South Africa."

Veterinarians receive some training in the abovementioned subjects at the undergraduate level, but it is not nearly sufficient to equip them for a research career in which the abovementioned scientific approaches feature strongly. I have a problem with the concept of providing for these diciplines at the post-graduate level because it is like placing the cart before the horse. Moreover, it will be necessary to rope in expertise from all possible institutions, irrespective of whether the required tuition is offered at undergraduate or post-graduate levels.

In my opinion veterinarians would be wise to take note of recent developments in a sister profession. Members of this profession can be likened to the dinosaurs. Like the dinosaurs, they excelled in every respect. They eventually filled every niche and every cavity. They became so specialised that they could not adapt and, lo and behold, eventually virtually worked themselves out of a job. Now they have gone back to the drawing board to try to find a way out of their predicament.

If the basic sciences are neglected in favour of clinically-orientated ones, veterinarians will at best be at a disadvantage and at worst be unable to contribute towards the advanced research required to increase animal production. This will not only curtail the job opportunities for veterinarians, but also lower the high status that they have always enjoyed as researchers in this country. The technological revolution currently on our doorstep may even decrease the demand for veterinary services in the farm animal industry, because scientists with a more basic training would be in a better position to provide for the whole spectrum of sophisticated biotechnological techniques required to improve animal production.

Thus the veterinarian's slice of the animal production market is bound to shrink even further. Eventually the basket containing the currently fairly lucrative companion animal market may be virtually the only one left. May I remind you that the saying goes: Don't put all your eggs into one basket.

CONCLUSION

In conclusion I wish to say that the purpose of my talk was not to show that Theiler was prophetic. I used Theilerian quotations to give perspective, colour and spice to my views and arguments. I have no doubt, however, that Theiler was remarkably far-sighted, as was aptly diagnosed by Smuts (Gutsche[2]) when he unveiled the statue we all know so well in 1939:

"Theiler had had a great capacity for

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application but in addition he had insight into the nature of things which was given to very few people. Rutherford, Einstein and others like them had had that insight into the significance of the situation before them. It is the grace of God, genius - something you get in some unaccountable way".

Let those of us who, quite naturally, sometimes have doubts about the revolutionary developments envisaged, take heart. Theiler would undoubtedly have given these changes his blessing.

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ERYTHROCYTE GLUTATHIONE PEROXIDASE ACTIVITY AS AN INDICATOR OF SELENIUM STATUS IN AN INTENSIVELY-MANAGED BEEF HERD

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ABSTRACT

A survey of erythrocyte glutathione peroxidase (GPx) activity was undertaken in a herd of crossbred cattle after 3 cases of white muscle disease had been diagnosed. All animals examined, appeared to be deficient in selenium, relative to control animals sampled. Oral administration of sodium selenite or sodium biselenite was followed within 30 d by an increase in erythrocyte GPx activity. Despite an apparent improvement in the selenium status of cows after supplementation with selenium, no difference was seen in the conception rate after artificial insemination.

Key words: Bovine, white muscle disease, glutathione peroxidase, oral selenium supplementation, conception rate

Ehret W.J.; Meltzer D.G.A.; Mulders M.S.; Collett F.A. Erythrocyte glutathione peroxidase activity as an indicator of selenium status in an intensively managed beef herd. *Journal of the South African Veterinary Association* **(1989) 60 No. 3, 130-133 (En) Johannesburg City Health Department, P O Box 1477, 2000 Johannesburg, Republic of South Africa.**

INTRODUCTION

The importance and interrelationship of selenium and vitamin E in animal nutrition has been well-documented. These 2 chemically different entities, with antioxidant properties, are necessary for normal cellular function and both play an important role alone or together. Specific deficiency diseases have been found to respond to one or both of these substances[2] Clinical signs depend on the severity of the deficiency and the species affected, and may vary from reduced performance to sudden death. Nutritional muscular degeneration occurs principally in young calves, lambs and foals. There are two major syndromes : an acute form in which the animals may die suddenly with or without premonitory signs[2 4 15], or the more common form seen in rapidly growing calves known as white muscle disease (WMD). This form has also been recorded in yearling cattle[2]. It has been suggested that WMD seen in selenium-deficient calves may be the result of oxidative stress due to changes in exercise patterns and high concentrations of unsaturated fatty acids in the diet[3 12]. Blood et al.[2], however, question this, pointing out that the myopathic agent, if any, in these cases has not been identified and that selenium is protective.

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Improvement in general condition and growth rate have been reported in deficient young cattle and positive responses in terms of rate of conception and retained placenta have been ascribed to supplementation with selenium[5 6 8 13]. These findings are contradicted by several authors[3 9 12 14], giving credence to the proposal of Suttle[12] who pointed out that despite biochemical evidence of deficiency, animals need not necessarily be affected functionally.

The concentration of selenium in blood is closely correlated to the activity of glutathione peroxidase (GPx), a selenium-containing enzyme in the red blood cell. Determination of the latter has been widely used to establish the selenium status of domestic stock[1 8 11 15].

This study was undertaken 1) to establish the selenium status of a herd of crossbred cattle after 3 calves were diagnosed as having white muscle disease; 2) to institute an economically-effective method of selenium supplementation; and 3) to examine the effect of selenium supplementation on conception rate.

MATERIAL AND METHODS

The study was conducted on the Johannesburg City Council's crossbred beef herd run on their Northern Farm as an ancillary operation to the purification of sewage and waste water effluent which is mainly domestic in origin. The foundation of the herd, which averages 2 740 head, consists primarily of various *Bos taurus* breeds, in particular Simmentaler, Hereford and Charolais. The breed composition of individual animals varies markedly. The cattle graze irrigated, predominantly rye grass pastures for most of the year. The irrigation cycle on the pastures averages 75-90 mm every 10-14 d. The soil is shallow (10-45 cm in depth), is underlain by decomposed granite, sandy, acid (pH 5,5 - 6,0) and is in many places stony. Drainage is poor. The average annual rainfall is 660 mm. Limited use is made of natural veld during the latter half of summer and autumn. During the winter months the animals are fed on dry land *Eragrostis curvula* hay, rye grass silage produced on the farms and they have free access to a commercial protein lick. Depending on the year, the cattle may be completely off the pastures for 4 to 6 weeks during winter. During 1984, the year in which the selenium supplementation trials were performed, hay and rye grass silage feeding commenced at the end of May and continued until the middle of July when the animals were put out onto the irrigated pasture. A commercial phosphate and salt lick with a copper content of 0,0625% is available throughout the year.

Breeding takes place by means of artificial insemination, during October and November, in which mainly semen from bulls from within the herd is used. Part of the nutritional requirements of these bulls is provided by a commercial supplement.

The herd is well managed, accurate records are kept, and all disease conditions or losses are examined and monitored by a veterinarian. Conception results and production figures have been good and the losses acceptable for the intensive system of management followed. During the 10-year period prior to the start of this investigation the pre- and post-natal calf losses (up to weaning at 210 d of age) were 8,1% and 4,0% respectively. More than 90% of the stillbirth and post-natal calf losses were examined post mortem by a veterinarian. Confirmed diagnoses of WMD were made in 3 calves only. No problem with retained placentas was experienced and no other obvious clinical manifestation indicating a possible selenium/vitamin E deficiency was noticed.

In October 1983, 3 young bull calves aged 66, 67 and 71 d (out of a total population of 770 calves born that year) all from a herd of 172 primiparous cows, developed clinical signs of muscular lameness and respiratory distress. Two of the calves responded well to parenteral selenium and vitamin E therapy (BO-SE, Burns Biotec), but the other calf died within 24 h of treatment. After white muscle disease had been confirmed histologically, the animals of this herd were all given parenteral selenium/Vit E (BO-SE) at the recommended rate.

An initial survey of the erythocyte GPx activity in the herd was undertaken using the method of Paglia & Valentine[10] as modified by Allen et al[1]. Heparinised blood samples were transported on ice

to the laboratory where the assay was performed within 24 h. Enzyme activity was expressed as enzyme units/10^{10} RBC. Blood samples were collected from various classes of animals: the dams of the 3 calves that developed WMD, prior to their response to BO-SE treatment; other cows from the herd in which the cases of WMD had been diagnosed and which had been treated with BO-SE 43 d earlier (WMD herd); 2 groups of breeding heifers, one grazing pastures only, the other on pasture plus concentrate supplement; mature cows with calves at foot; and breeding bulls. Comparative GPx activity levels were measured in blood samples from 16 cows (control animals) kept at the Faculty of Veterinary Science, University of Pretoria. These animals were being fed concentrates and had access to veld grazing. Thereafter the effect of oral selenium supplementation was examined in 3 trials.

First selenium supplementation trial

During December 1983, 10 of the 172 cows in the WMD herd and 10 of the 82 breeding heifers grazing pastures only were selected at random from their respective herds. The former animals were weighed and blood samples were collected prior to the per os administration of sodium selenite at an estimated rate of 0,1 mg kg^{-1} elemental selenium. A further 0,2 mg kg^{-1} was administered 3 months later using sodium biselenite. Blood samples were collected every month for the next 8 months and erythrocyte GPx activity measured.

Second selenium supplementation trial

During March 1984, 30 of the 165 mature, non-pregnant, non-lactating cows were allocated at random to 2 groups. One group was given 100 mg of elemental selenium as sodium biselenite per os (estimated dosage 0,2 mg kg^{-1}); the other acted as undosed controls. Blood samples were collected on the day of treatment and at monthly intervals thereafter for 6 months.

Conception rate trial

During August 1984, 190 cows that had been given 100 mg selenium 6 months previously were divided into 2 equal groups according to calving dates. One group was given a further 100 mg of elemental selenium as sodium biselenite per os; the other was not dosed and served as a control group. The interval between calving and the start of the breeding season of the treated cows was 34 - 93 d and that of the control animals, 30 - 98 d. Blood samples were collected for GPx determination from 15 randomly-selected animals from both of these groups, prior to treatment and at monthly intervals thereafter, for 6 months from the controls and 11 months from the treated cows. All cows were inseminated artificially by the same person during October and November 1984 using semen from a single bull, and were examined for pregnancy by rectal palpation during February of the following year.

Analysis of variance and Student's t-tests were performed using a Hewlett Packard HP41C calculator

RESULTS

The results of the survey done on the herd and the comparative GPx activity measured in the control animals are shown in Fig. 1.

An analysis of variance of the data shows that the mean erythrocyte GPx activity measured in the control animals was significantly greater than that of the Johannesburg herd ($F = 63,66$, $p < 0,001$). Within the herd the mean GPx activity of the bulls was significantly greater than the mean activity measured in the BO-SE-treated cows and heifers on pastures and ration ($p < 0,001$), both of which in turn were greater than that of the rest of the animals ($p < 0,01$).

Selenium supplementation

First selenium supplementation trial

The results of this trial are shown in Fig. 2. The initial GPx activity levels of the primiparous cows that had been treated with BO-SE were significantly greater than those measured in the heifers ($t = 5,28$, $p < 0,001$). In both groups, GPx activity was significantly higher 1 month after the oral administration of selenium ($t = 4,82$, $p < 0,001$) but then declined markedly until

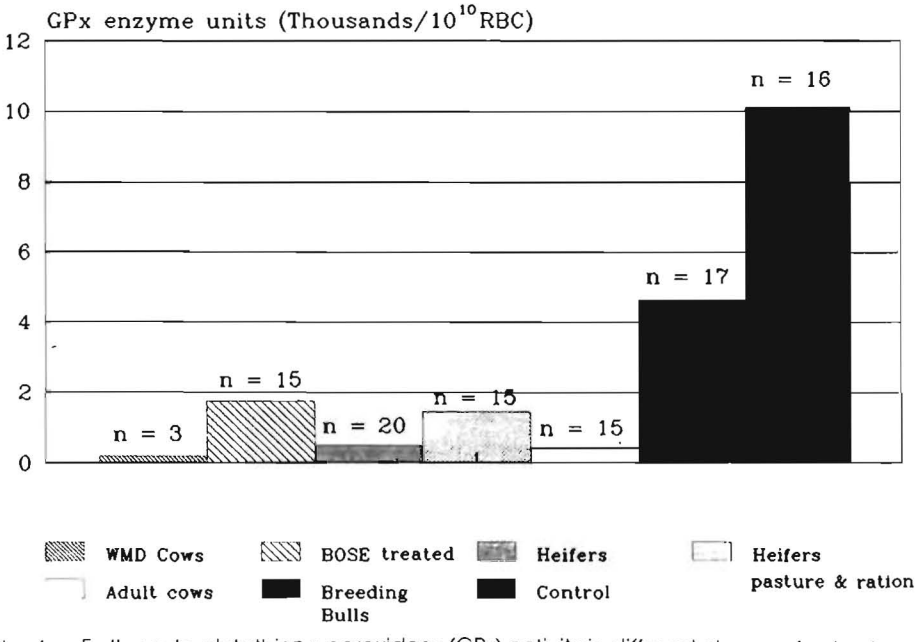

Fig. 1: Erythrocyte glutathione peroxidase (GPx) activity in different classes of animals from the Johannesburg herd and in control animals

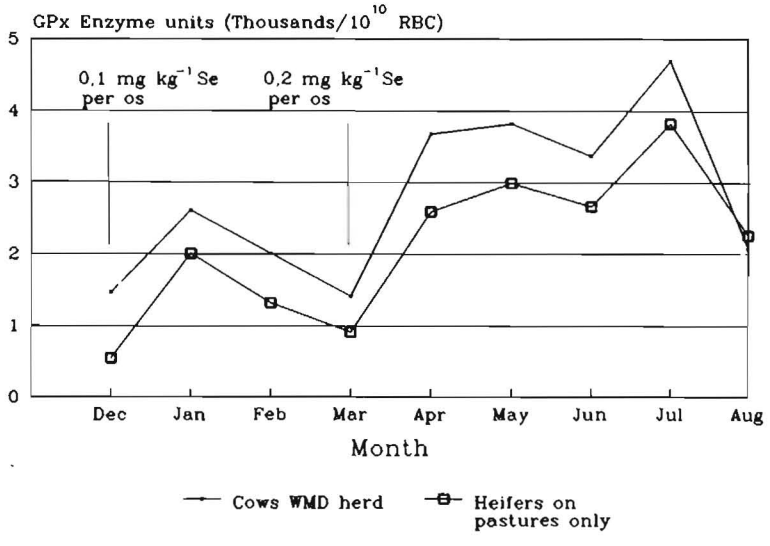

Fig. 2: Erythrocyte glutathione peroxidase (GPx) activity in animals after the per os administration of selenium

the second dose was given in March. This was followed once again by an increase in GPx activity which increased even further during July and then fell during the following month.

Second selenium supplementation trial

The results of this trial are shown in Fig. 3. One month after treatment, GPx activity of the treated animals had increased significantly (paired $t = 5,15$ $p < 0,001$) and it remained higher than that of the control animals throughout. A peak in GPx activity was also evident in both treated and control animals during July.

Conception rate trial

GPx activity measured in these animals is shown in Fig. 4. Once again an increase of GPx activity was measured during July. The conception rate results are summarised in Table 1.

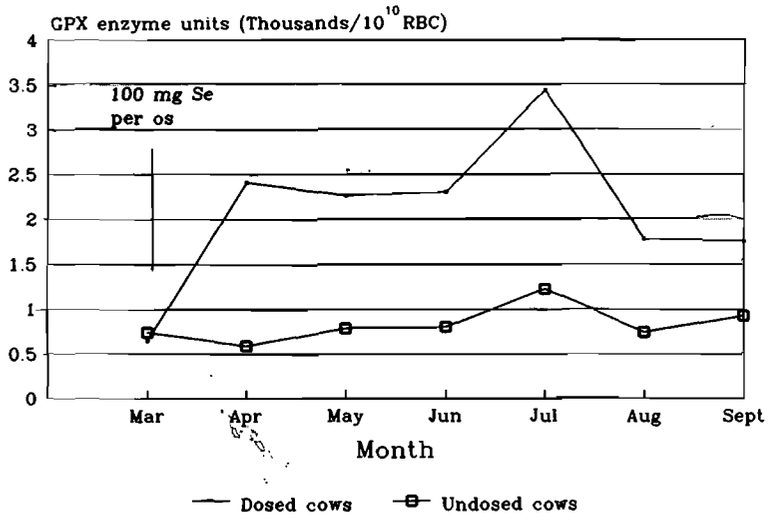

Fig. 3: Erythrocyte glutathione peroxidase (GPx) activity in cows after the administration of 100 mg selenium per os

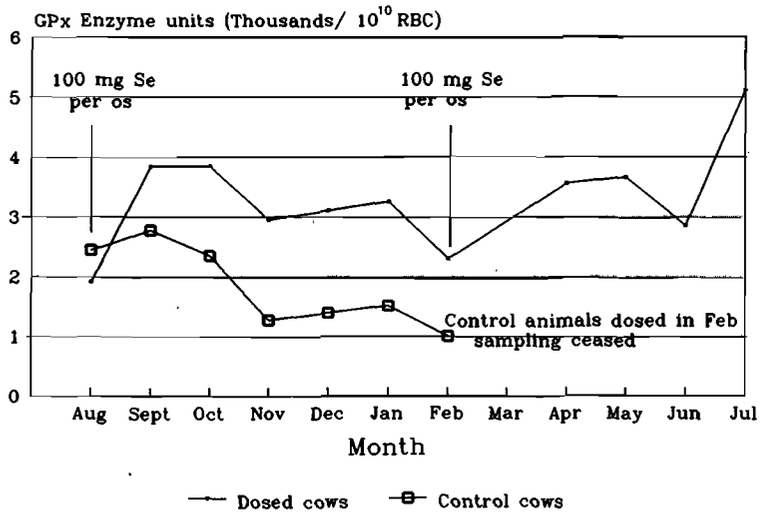

Fig. 4: Erythrocyte glutathione peroxidase (GPx) activity in selenium-dosed and control cows in which the conception rates were compared

Treatment	Conception rate	
	First insemination	Overall
Selenium dosed	39.2%	76.3%
control	52.7%	78.5%

Table 1: Conception results of selenium supplemented and control (undosed) animals bred by AI to bull No L399.

DISCUSSION

Allen et al.[1] reported erythrocyte GPx activity levels which ranged from 1 600 enzyme units per 10^{10} erythrocytes in untreated cattle to 8 067 in animals treated with parenteral selenium and vitamin E. They did not indicate, however, whether they regarded the GPx levels in the untreated animals as being deficient. Other GPx activity levels reported for cattle were not regarded comparable to those obtained in this study as a different method was used[2,15].

The low erythrocyte GPx activity, relative to that of the control animals, measured in all classes of animals in the Johannesburg herd, indicated that the animals had a deficient intake of selenium. Leaching by constant irrigation and the low pH of the soil were probably factors causing plant growth deficient in selenium and appeared to explain the 3 cases of WMD diagnosed at the commencement of the present study[2]. The occurrence of WMD appears to have been typical of the course described by Blood et al[2]. The fast-growing calves, from dams deficient in selenium, were on fresh spring pastures.

The significantly higher GPx activity in the erythrocytes of the bulls appears to be the result of the concentrates fed, but their activity levels were nevertheless significantly lower than the reference values measured in the control animals.

Oral sodium biselenite is less expensive than parenteral selenium. Parenteral selenium supplementation is recommended at a dosage of ca. 7 mg/100 kg elemental selenium (BO-SE package insert). Sodium biselenite has a bioavailability of 30-50% per os[6]. Using an estimated bioavailability of elemental selenium of 35% the per os supplementation rate, a dosage equivalent to that of the parenteral route was estimated to be 20 mg/100 kg. The estimated cost per 100 kg using the BO-SE formulation was 147,7 c while that of the bioequivalent amount of sodium biselenite was 1,53 c.

The significant increase in GPx activity in the blood samples taken a month after oral selenium administration is similar to that found by Sheppard & Millar[11], who reported an increase in GPx activity 30 d after supplementation with selenium in sheep. The initial 0,1 mg kg^{-1} dose resulted in a short-term response which appeared to be inadequate and a decision was made to double the oral dosage given. Animals dosed in March showed a similar increase in GPx activity, 30 d later, which increased further during the winter months. The increase in GPx activity seen in July in all of the animals appeared to coincide with the supplementary feeding which commenced at the

end of May. While the selenium concentration of the pastures and the winter feed was not determined during the study, it is possible that the use of supplementary feeding resulted in a greater selenium intake and an increase in GPx activity. The fall in GPx during August in trials 1 & 2 appears to be related to the return of the animals to lush irrigated pastures with a lower selenium content[2,7].

While the good overall conception rate of the animals in this trial are comparable with results in other intensively-managed herds, the higher conception rate to first insemination in the undosed cows cannot be explained. However, this result together with the good health record of the herd, as a whole, appears to indicate that despite their poor selenium status the animals were not functionally deficient. The minor outbreak of WMD was, however, taken as evidence of the need to supplement the diet of these animals with selenium.

Oral dosage with selenium salts was followed a month later by a significant increase in the GPx activity measured in the erythrocytes of these animals. The cost of oral supplementation per head is considerably lower than when injectable forms of selenium are used. The improved selenium status which followed this treatment was not found to improve conception rates in treated animals, probably because they were not functionally deficient.

ACKNOWLEDGEMENTS

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UNUSUAL HEPATIC PARENCHYMAL CRYSTALLOID MATERIAL AND BILIARY MICROLITHS IN GOATS

M G COLLETT* and A M SPICKETT**

Abstract

Investigation into an outbreak of suspected photosensitivity in Boer goats grazing green oats *(Avena sativa)* led to the finding of microscopical parenchymal crystalloid material and biliary microliths in the livers of 3 ewes that were killed for necropsy. Neither *Tribulus terrestris* nor *Panicum* spp. occurred on the farm. Further investigation resulted in the isolation, from leaf spots on the oat leaves, of the fungus *Drechslera campanulata*, cultures of which have been shown to be highly toxic to sheep, goats and calves.

The hepatic parenchymal crystalloid material, which did not invoke any inflammatory reaction, occurred intracellularly in hepatocytes and extracellulary in sinusoids and central veins. Histochemically, this material reacted positively for calcium and free fatty acids and ranged from non-birefringent, grey spicules to birefringent, glass-like sheaved crystals. A distinctive feature in the livers of the 3 goats was the presence of periductal concentric fibrosis and cast-like biliary microliths which occasionally contained a core of birefringent crystalloid material morphologically and histochemically different from that seen in the parenchyma.

An aetiological relationship between the presence of highly toxic *D. campanulata* isolates on green oats and the hepatopathy with biliary microliths and calcium-free fatty acid crystalloids is possible.

Key words: *Avena sativa*, biliary microliths, calcium-fatty acid crystalloids, *Drechslera campanulata*, goats, liver crystalloid material, oats, photosensitivity

Collett M.G.; Spickett A.M. Unusual hepatic parenchymal crystalloid material and biliary microliths in goats. *Journal of the South African Veterinary Association* **(1989) 60 No. 3, 134-138 (En) Department of Pathology, Faculty of Veterinary Science, University of Pretoria, Privat Bag X04, 0110 Onderstepoort, Republic of South Africa.**

INTRODUCTION

Conspicuous crystalloid material in bile ducts, Kupffer cells and hepatocytes are features of the histopathology of 4 known hepatotoxicities of sheep and goats, namely geeldikkop *(Tribulosis ovis)*[5 8 17] and dikoor *(Panicum* spp. photosensitivity)[5 17] in South Africa and lechuguilla *(Agave lecheguilla* Torr.)[10] and sacahuiste *(Nolina texana* S. Wats.)[11] poisonings in Texas. These diseases can be grouped together and referred to as "crystal(loid)-associated cholangiohepatopathies"[3].

Microscopical parenchymal crystalloid material and biliary microliths, reminiscent of those seen in the above diseases, were seen in the livers of 3 Boer goats that were slaughtered for diagnostic investigation in the field outbreak of photosensitivity (at Klipheuwel, near Stellenbosch, in the western Cape Province) referred to by Schneider et al[15]. This finding spurred detailed examination of the grazing for toxic plants and fungi, specifically with respect to those known to cause hepatogenous photosensitivity. This exercise culminated in the isolation of the cereal phytopathogen *Drechslera campanulata* (Lév.) Sutton from green oats *(Avena sativa* L.) in the pasture[16]. The experimental mycotoxicosis produced when cultures of *D. campanulata* were dosed to sheep, goats and calves has been reported[6 15].

In this paper, the histopathology of the livers, kidneys, gall and urinary bladders of the 3 goats, as well as details of the light microscopical appearance, histochemistry and ultrastructure of the hepatic crystalloid material and biliary microliths, are presented.

MATERIALS AND METHODS

Necropsies: The 3 Boer goats mentioned above (Goats 1, 2 and 3) all ewes, were pale to dark brown in colour with a few isolated small white patches. Necropsies were performed immediately following slaughter. Goats 1 and 2 (an adult and a yearling respectively) were necropsied on the same day while Goat 3 (an adult) was necropsied 5 weeks later. Goat 1 had anasarca of the eyelids, lips, submandibular region, ears, throat and forelimbs and was also icteric (total serum bilirubin 56 μmol l^{-1}). Anasarca was less severe in Goat 2. On the unpigmented areas of the face, ears and back of Goat 3, the skin was thickened and covered with hard, dry, necrotic crusts. The livers of Goats 1 and 2 were slightly enlarged and paler than normal, while that of Goat 3 appeared normal. The only other macroscopical feature worth mentioning was that, in Goats 1 and 2, the gall bladder mucosa was oedematous and roughened and the surface was covered with a layer of material (1 - 2 mm thick) having the colour and consistency of cream.

Microscopy: Samples of liver, gall bladder, kidney and urinary bladder from Goats 1, 2 and 3 were collected in 10% buffered formalin and processed routinely for light microscopy. Tissue sections were stained with haematoxylin and eosin (HE) and examined microscopically with polarised and non-polarised light. Additional liver sections were stained with alizarin red S for calcium[1], Von Kossa's method for calcium[9], Gomori's reticulin method[9] and Pizzolato's method for calcium oxalate[9]. Cryostat sections of formalin-fixed liver at a thickness of 10 - 20 μm were stained with oil-red-0 for lipids[12], Holczinger's method for free fatty acids[1], Schultz's method[12] and Okamoto's method[12] for cholesterol and cholesterol esters and the fluorescent (morin) method[12] for calcuim. Formalin-fixed liver specimens from a confirmed case of geeldikkop in an Angora goat and from a normal sheep were processed in the same way and stained with the above methods for comparative purposes. All of the above sections were also examined by means of polarised light microscopy.

For examination with the scanning electron microscope (SEM), specimen blocks measuring approximately 4 x 2 x 2 mm were blade-cut from formalin-fixed liver samples (from Goats 1, 2, 3 and the same normal sheep). The specimens were mounted upright on a stage with 2% methyl cellulose and quench frozen in liquid nitrogen slush. They were then placed under vacuum on a cold stage and freeze-fractured using an Emscope SP2000 cryopreparation apparatus. The fractured surface was gold-sputtered at 25 mA for 3 min and viewed and photographed at 5 - 10 kV in an ISI 100 SEM equipped with a cold stage.

Measurements: Linear measurements of crystalloids and microliths were performed on photographs and on-line microscopic images by means of a semi-automatic image analyser (Videoplan, Kontron Bildanalyse).

Farm visit: The farm was visited a few days after the first 2 goats were necropsied (and histopathologically examined) so that the possible role of toxic plants and/or fungi could be investigated. The camp in question had a well established green oats pasture and, on closer inspection, it was apparent that a large percen-

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tage of the oats plants had leaf spots due to infection by a fungal pathogen, which resulted in the leaves turning yellow and dying. Other plants in the grazing included seredella (*Ornithopus sativus* Brot.) and Cape spinach (*Emex australis* Steinh.), while a few Port Jackson willows (*Acacia saligna* (Labill.) Wendl.) occurred at one end of the camp. Neither *T. terrestris* nor *Panicum* spp. was found on the farm. At the time of the farm visit it was observed that, of the pasture plants, the goats grazed the green oats almost exclusively.

Mycology: Ten pasture specimens were taken for mycological culture as well as spore counts, which were performed according to the method routinely used in New Zealand for predicting facial eczema danger periods[4]. The spore counts revealed an insignificant *Pithomyces* count (5 000 g^{-1} of leaves) in one specimen only. *D. campanulata* was the predominant fungus isolated from leaf spots on the green oats leaves, although two other related fungi, namely *D. avenae* (Eidam) Scharif and *Bipolaris spicifera* (Bain) Subram., were also cultured[15,16].

RESULTS

The liver lesions in Goats 1 and 2 were similar. Diffuse cloudy swelling of hepatocytes, resulting in partial occlusion of sinusoids, was evident. Widespread single cell necrosis and anisonucleosis, as well as the presence of occasional mitotic figures and eosinophilic cytoplasmic droplets, were also observed.

A characteristic feature in HE-stained sections of the liver of both goats (particularly Goat 2) was the presence of parenchymal crystalloid material which occurred both intracellularly within hepatocytes and extracellularly in sinusoids (Fig. 1 - 3). The crystalloid material varied from aggregates of non-birefringent, fine, grey, acicular, feathery spicules 6 - 12 μm in length (referred to hereafter as non-birefringent crystalloids) (Fig. 1), to larger (12 - 28 μm), birefringent, colourless, glass-like, rhomboid, sheaved crystalloids (birefringent crystalloids) (Fig. 2 & 3).

Non-birefringent crystalloids occurred more frequently centrilobularly, as well as within the lumen of central veins (Fig. 1). Clumps of birefringent crystalloids were commonly sited midzonally (Fig. 2) causing disruption and fragmentation of the reticulin framework. They also occurred in the vicinity of central veins, sometimes subintimally causing distortion of the vessel wall, and also intravascularly (Fig. 3). Both types of crystalloid material were occasionally seen periportally. Hepatocytes adjacent to cells containing crystalloids were frequently displaced, their nuclei appeared pycnotic and the cytoplasm showed increased eosinophilia (Fig. 2). Crystalloid material did not invoke an inflammatory reaction.

Using the SEM the morphology of these crystalloids ranged from parallel pillars with small globules dispersed and adherent to the surface (Fig. 4 & 5) (possibly corresponding to the non-birefringent form) to well-defined sheaves of rhomboid plates or spikes (possibly the birefringent form) (Fig. 6 & 7). Lengths of both forms varied between 6 and 30 μm.

No lesions were found in liver specimens from the normal sheep viewed under the light microscope and SEM.

In addition to the 2 types of parenchymal crystalloid mentioned so far, a distinctive feature in the lumens of many small, medium and large bile ducts in the liver of Goat 1 was the presence of cast-like, amorphous, pale eosinophilic microlithic material measuring 45 - 252 μm in diameter (Fig. 8 - 11). Some bile ducts were completely occluded by these microliths which often appeared to be

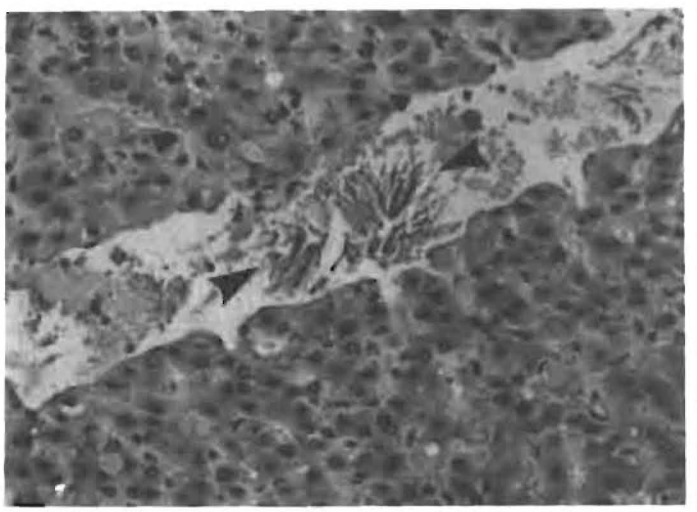

Fig. 1: Aggregates of feathery spicules regarded as immature crystalloids (arrows) in central vein. HE x 100

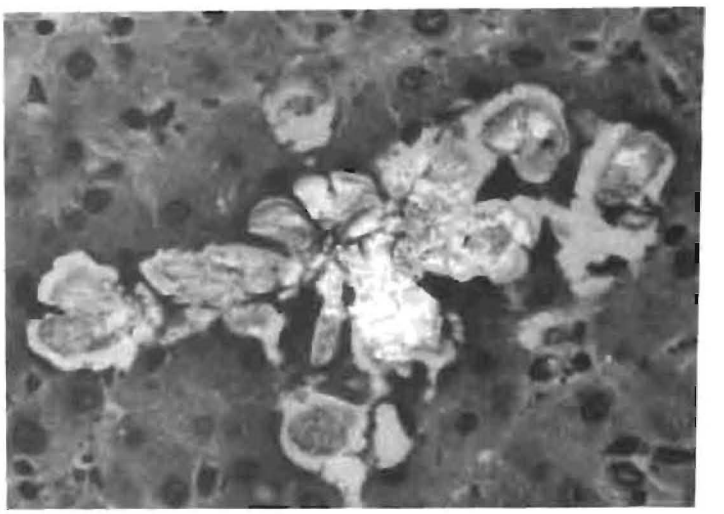

Fig. 2: Group of birefringent crystalloids in midzonal region. Polarised light. HE x 400

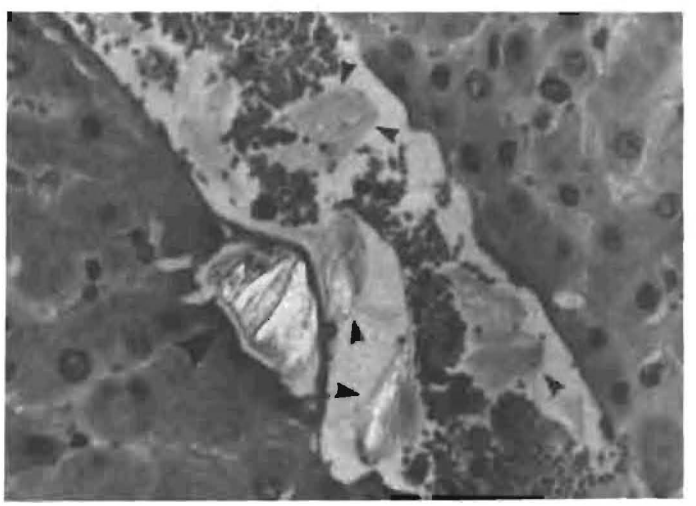

Fig. 3: Birefringent crystalloids (considered mature) beneath the intima of central vein (large arrow) and within the lumen (small arrows). Polarised light. HE x 400

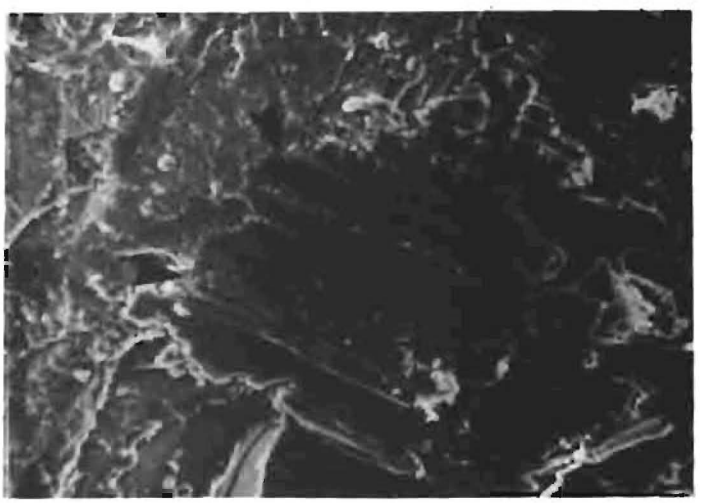

Fig. 4: Crystalloids arranged as parallel pillars (arrows). SEM x 700

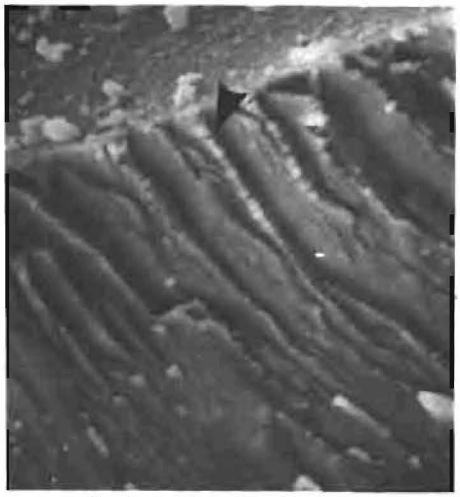

Fig. 5: Crystalloids arranged as ragged pillars with small globules (arrow) on ridges. SEM x 1800

Fig. 6: Rhomboid plates stacked together (arrow) - considered to be mature, birefringent crystalloids (compare Fig. 3). SEM x 1300

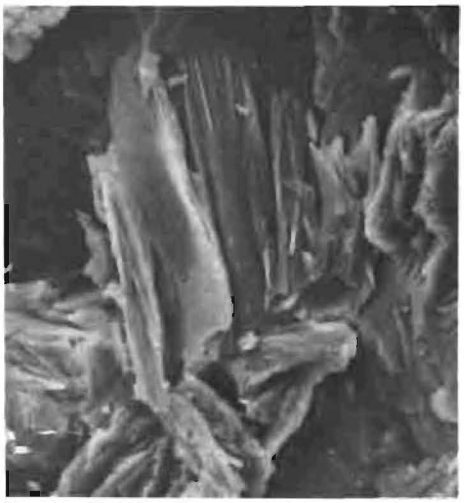

Fig. 7: Detail of crystalloid arrangement similar to that in Fig. 6. SEM x 1680

composed of a core and an outer concentric layer of faintly eosinophilic, radiating, non-birefringent crystalloid material (Fig. 8 & 10). Occasionally, birefringent crystalloid material was present in the cores of some microliths (Fig. 8), while others contained a few small cholesterol-like clefts. This microlithic crystalloid material was morphologically different from the 2 types of parenchymal crystalloid material already referred to (compare Fig 2 & 3 with Fig 8).

In many instances, the microliths only occupied a portion of the bile duct lumen, yet segments of bile duct epithelium adjacent to microliths showed pressure atrophy or even necrosis (Fig. 8 -11). In HE sections of the liver of Goat 2, biliary microliths were rare and in none of the latter was microlithic crystalloid material seen resembling that in Goat 1

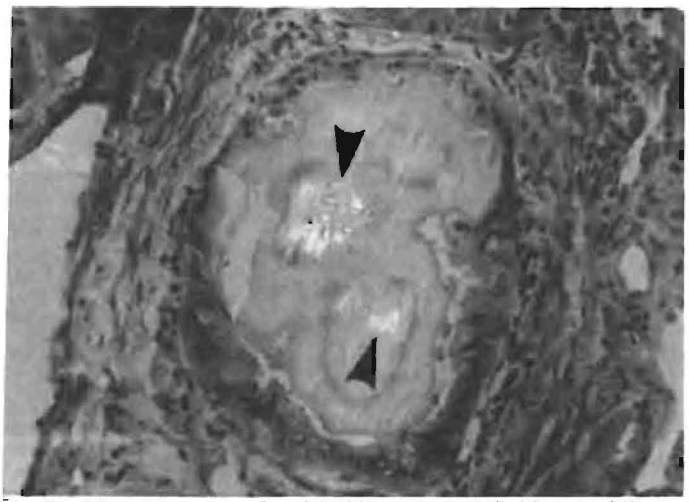

Fig. 8: Microlith containing birefringent crystalloid material (arrows) in medium-sized bile duct Polarised light HE x 100

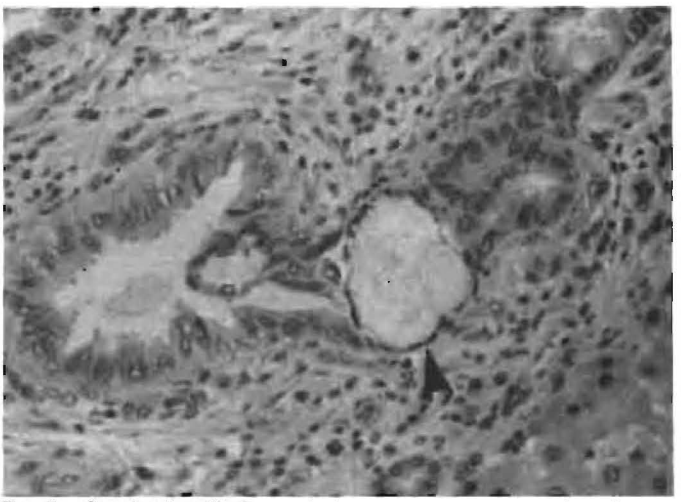

Fig. 9: Small microlith (arrow) obstructing a portion of a bile duct Note concentric fibroplasia around the bile duct on left. HE x 100

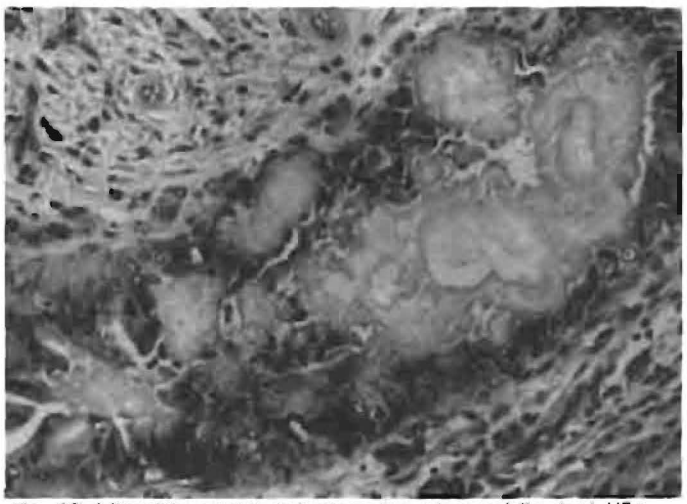

Fig. 10: Microliths and debris clogging a larger bile duct HE x 100

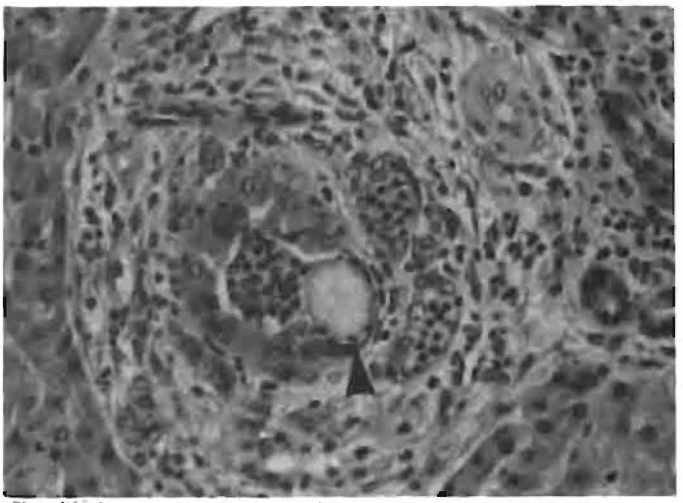

Fig. 11: Small microlith (arrow) compressing the epithelium of a small bile duct. Note concentric fibroplasia and cholangitis. HE x 100

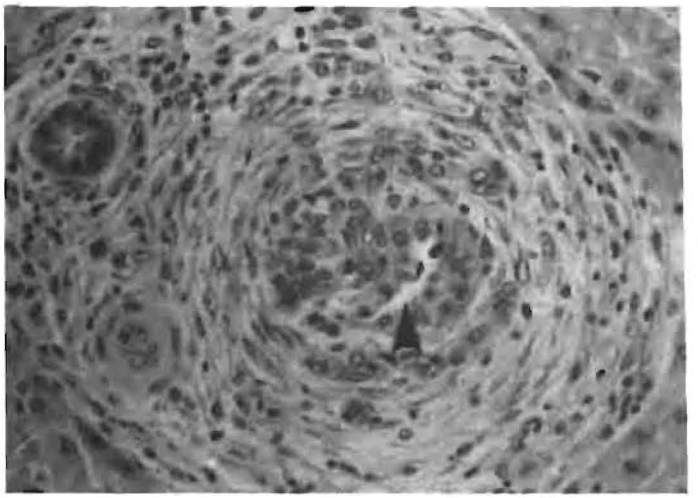

Fig. 12: Concentric fibroplasia about a bile duct. Note near obliteration of bile duct lumen (arrow). HE x 100

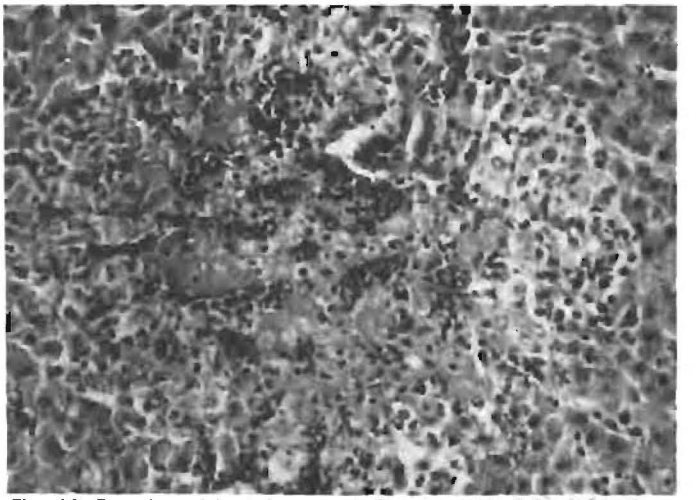

Fig. 13: Focal periductal coagulative necrosis (bile infarct). HE x 100

Attempts at visualising the microliths with the SEM failed.

Periductal concentric fibrosis and oedema were conspicuous in the portal regions of the livers of both goats (Fig. 9 -12). The lumens of some of the affected bile ducts were obliterated by constrictive fibrosis (Fig. 12). Areas of focal, periportal coagulative necrosis with neutrophil infiltration ("bile infarcts") were also observed (Fig. 13). Some bile ducts contained necrotic cell debris and neutrophils, others had segmental epithelial necrosis while a focal purulent cholangitis occurred in Goat 1, as did a diffuse sinusoidal leukostasis.

The comparative histochemistry and sizes of the 2 forms of parenchymal crystalloid, biliary microliths and geeldikkop crystalloids are presented in Table 1.

In Goat 3, the liver lobules were well demarcated due to increased eosinophilia of peripheral hepatocytes associated with darkly-stained, almost pycnotic nuclei. As in Goats 1 and 2, single cell necrosis and anisonucleosis of the hepatocytes were present. Although the majority of bile ducts appeared normal, focal areas of mild bile ductular proliferation were observed. An occasional portal reaction, consisting of infiltrations of lymphocytes and macrophages, was present. Isolated biliary microliths containing crystalloid material similar to that seen in Goat 1 were seen, while the other 2 types of parenchymal crystalloid material were not detected.

The gall bladder mucosa of Goats 1 and 2 was oedematous and focal erosions were present. Nephrosis, characterised by cloudy swelling of proximal convoluted tubular epithelial cells and the presence of numerous protein globules and granular casts in convoluted tubule lumens, was severe in Goat 1 and milder in Goats 2 and 3. The urinary bladders of the 3 goats appeared normal.

DISCUSSION

The icterus in Goat 1 was probably predominantly obstructive due to the biliary microliths and periductal fibrosis. Although phylloerythrin levels were not determined, the macroscopical and microscopical findings in the necropsied goats are indicative of hepatogenous photosensitivity.

The biliary microliths and constrictive biliary fibrosis are comparable to those seen in cases of geeldikkop and facial eczema (pithomycotoxicosis) respectively. Geeldikkop has been induced experimentally by the simultaneous ingestion of *Tribulus terrestris* L. plants and sporidesmin from the fungus *Pithomyces chartarum* (Berk & Curt.) M.B. Ellis[8].

The biliary microliths found in the goats differed morphologically from the crystalloid material in geeldikkop and dikoor in that they are more amorphous and cast-like with less birefringent crystalloid material. The geeldikkop crystalloid material appears cholesterol-like in HE sections but is apparently not composed of either cholesterol or common bile salts[8]. Histochemically, the crystalloid material in the goat biliary microliths also reacted negatively for cholesterol and its esters.

The aetiology and pathogenesis of the crystalloid material in geeldikkop and dikoor are unknown and the same applies to those in the goats in this investigation. Recently, however, Bridges et al.[2], in a study of dikoor crystalloid material, found that the solubility of this material is comparable to that of cholesteryl digitonides. These authors hypothesised that the pathogenesis of dikoor may involve the conversion of dietary saponins (first implicated by Henrici[7] when it was found that geeldikkop-associated *Tribulus* contained saponins whereas non-toxic *Tribulus* contained none) to sapogenins by bacteria in the digestive tract or by biotransformation after absorption through injured epithelium. Since sporidesmin appears not to be implicated in the pathogenesis of dikoor[2,3], the saponin hypothesis seems all the more plausible. If dietary saponins were possibly aetiologically involved in the biliary microliths in the Boer goats, either the green oats (possibly as a result of a

Table 1: Comparison of parenchymal (non-birefringent and birefringent) crystalloids and biliary microliths (light microscope)

Component	Non-birefringent crystalloids	Birefringent crystalloids	Biliary microliths	Geeldikkop crystalloids
Calcium	+	+ +	-	-
Free fatty acids	+	+ + +	-	-
Cholesterol and its esters	-	-	-	-
Oxalate	-	-	-	-
Size (length) μm	6-12	12-28	45-252	16-40

trace element deficiency in the soil or fungal damage) or the other pasture components could have been the source.

The sinusoidal reticulin fragmentation and the effects of the parenchymal calcium-free fatty acid crystalloids on vessel walls and hepatocytes discounts the possibility that these crystalloids are artefactual. Non-birefringent hepatic crystalloid material has been noted in dogs, cats and sheep affected by a variety of predominantly toxic conditions as well as sheep with white liver disease, a syndrome associated with cobalt deficiency[6]. Birefringent crystalloids have also been seen in cases of ovine white liver disease and "Stellenbosch photosensitivity"[6].

Similar, if not identical, birefringent parenchymal crystalloids, found incidentally in the liver and adrenals of dogs, have been reported[13]. Using ultrastructural and X-ray microdiffraction techniques, the authors found that the crystalloids contained predominantly calcium stearate, possibly mixed with small quantities of other fatty acid salts.

The presence of calcium-free fatty acid crystalloids within veins and sinusoids possibly results from the supersaturation of fatty acid salts in the hepatic circulation. This may follow the consumption of excessive quantities of dietary fatty acids[13]. Another possibility is that toxic hepatocellular damage may result in inefficient fatty acid metabolism with resultant crystallisation. A third possibility is that nutrient deficiencies (e.g. thiamine[13] or cobalt[6]) may affect lipid metabolism in such a way as to enhance crystalloid formation.

"Swellhead" in sheep and goats grazing green oats has been described in Texas but no details were reported[14]. Cultures of *D. campanulata* dosed to sheep, goats or calves caused dose-dependent liver enzyme elevations, diffuse hepatocellular degeneration with anisonucleosis and single-cell necrosis as well as severe necrotic lesions in the forestomach mucosa[6, 15]. Photosensitivity, however, did not occur, although non-birefringent parenchymal crystalloids and the above liver lesions were detected in the livers of dosed goats which were eating freshly-cut green oats ad lib[6]. The diarrhoea syndrome referred to by Schneider et al[15] may have been caused by *D. campanulata*-infected green oats since one of the main signs seen in experimental drechsleratoxicosis is diarrhoea.

In conclusion, it is possible that the aetiology and pathogenesis of the biliary microliths in the photosensitive goats could be similar to those of the crystalloids in geeldikkop and/or dikoor. The significance of the calcium-free fatty acid crystalloids, which appear unrelated to the biliary microliths, is unknown. Although possibly coincidental, there may be an aetiological relationship between the presence of highly toxic *D. campanulata* isolates on green oats and the hepatopathy with biliary microliths and calcium-free fatty acid crystalloids that is described here. Finally, despite some dissimilarities as outlined above, it is reasonable to conclude that this outbreak in goats probably constitutes a fifth form of crystalloid-associated cholangiohepatopathy.

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A SURVEY OF HELMINTHS IN DOMESTIC CATS IN THE PRETORIA AREA OF TRANSVAAL, REPUBLIC OF SOUTH AFRICA. PART 1: THE PREVALENCE AND COMPARISON OF BURDENS OF HELMINTHS IN ADULT AND JUVENILE CATS

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ABSTRACT

The helminths found in 1 502 necropsied cats were examined. The findings indicated that 65% of the cats were infested. The most prevalent helminths encountered were *Ancylostoma tubaeforme* (41%), *Ancylostoma braziliense* (25%), *Dipylidium caninum* (23%), *Toxocara cati* (11%), *Taenia taeniaeformis* (7,7%), *Ancylostoma caninum* (3,3%), *Joyeuxiella fuhrmanni* (2,5%), *Ancylostoma ceylanicum* (1,4%), and *Physaloptera praeputialis* (1,3%). The following helminths were recorded in fewer than 1% of the cats: *Centrorhynchus* spp., *Pterygodermatites* spp., *Toxocara canis*, *Aleurostrongylus abstrusus* and *Vogeloides* spp.. This is the first record of the lungworms, *A. abstrusus* and *Vogeloides* spp. in cats in the Republic of South Africa.

The helminths in adult and juvenile cats are compared. Of the cestodes, *Joyeuxiella fuhrmanni* (4%) and *Taenia taeniaeformis* (12%) are more prevalent in adult cats. *Dipylidium caninum* is marginally more common in adults (24%), and is the most common helminth in juvenile cats, being present in 21% of cases. More adult cats have *Ancylostoma tubaeforme* (58%), *Ancylostoma braziliense* (36%), *Ancylostoma ceylanicum* (2,4%) and *Physaloptera praeputialis* (1,9%) than do juvenile cats. However, juvenile cats were found to harbour more *Toxocara cati* (41%), *Toxocara canis* (0,5%) and *Ancylostoma caninum* (5,2%). Juvenile cats harbour appreciable numbers of both cestodes and nematodes, thus any treatment should be aimed at the elimination of both, with a broad spectrum anthelmintic.

Key words: felines, adult cats, juvenile cats, helminths, prevalence, comparison, mean burdens, ratios, *Dipylidium caninum*, *Joyeuxiella fuhrmanni*, *Taenia taeniaeformis*, *Toxocara cati*, *Toxocara canis*, *Ancylostoma tubaeforme*, *Ancylostoma braziliense*, *Ancylostoma caninum*, *Ancylostoma ceylanicum*, *Physaloptera praeputialis*, *Pterygodermatites* spp., *Aleurostrongylus abstrusus*, *Vogeloides* spp., *Centrorhynchus* spp., protozoa, *Isospora felis*, *Isospora rivolta*.

Baker M.K.; Lange L; Verster A; van der Plaat S. A survey of helminths in domestic cats in the Pretoria area of Transvaal, Republic of South Africa. Part 1: The prevalence and comparison of burdens of helminths in adult and juvenile cats. *Journal of the South African Veterinary Association* **(1989) 60 No. 3, 139-142 (En) 65 van Heerden Street, 0084 Capital Park, Republic of South Africa.**

INTRODUCTION

No studies have been done previously in the Republic of South Africa on the helminth burdens of domestic cats. A number of studies on the helminths found in cats have been conducted in other parts of the world[1,5,6,11,12,13,15]. This study was undertaken to determine, not only which parasites occur in the Pretoria (Transvaal) area, but also to establish whether helminth burdens (number and species of helminths) differ between male and female cats, adult and juvenile cats and also whether the season of the year plays any significant role in the number and species encountered. This paper is the first of 2 and deals with the prevalence of the worms encountered in 1 502 cats and a comparison of the burdens of the different helminths in adult and juvenile cats.

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MATERIALS AND METHODS

During the period 29/1/80 to 25/2/81, 6 052 cats were euthanased at the Pretoria branch of the Society for the Prevention of Cruelty to Animals. The condition of the cats varied from good to lean. Post mortem examinations were performed on 1 502 of these cats. All the cats were necropsied within 2 h after euthanasia. The selection of cats was entirely on a "first come first served" basis, in that the first 10 cats identified in each category were selected for necropsy. On occasion, 10 cats per category were not available and whatever was avialable, was necropsied. The cats were divided into 4 categories viz. adult male, adult female, juvenile male and juvenile female. The criterion for adult or juvenile was based upon the development of the dentition, the presence of deciduous teeth being the criterion for juvenile classification. Juveniles ranged in age from approximately 2 weeks of age to close on 6 months old, when the permanent teeth were already present.

All cats were examined for any pathology. The oral cavity of each was examined for lesions and the presence of the trematode, *Cllnostomum falsatum*. Where lingual ulcers were encountered, throat swabs for virus isolation were taken and the tongue excised and fixed in 10% formalin for histopathology.

The abdominal cavity was opened and the distal part of the oesophagus ligated. The oesophagus was severed and the whole of the stomach and intestinal tract stripped from the mesenterium. The colon was severed and where available, the faeces in the last 5cm of the rectum removed and preserved in a 2% solution of potassium dichromate for later examination for protozoan parasites. The faeces of 5 cats of similar age and sex were collected in a single bottle for ease of examination. The distal part of the colon was then ligated. The gastro-intestinal tracts of 5 cats from each category were placed into a plastic bag. Each bag was labelled with record numbers to correlate with the age and sex of that category. The labelled bags were placed into an insulated container as soon as possible and then frozen on return to the laboratory. After the removal of the gastro-intestinal tract, the remaining organs were examined and when lesions were encountered, appropriate specimens were taken and preserved in 10% formalin for histopathology. The bladder of every cat was opened and examined for the presence of *Capillaria felis-cati*.

In the laboratory, the gastro-intestinal tracts were allowed to thaw at room temperature, and each opened along their entire length using a pair of bowel scissors. The individual contents were collected separately in plastic trays, and the entire length of the tract stripped of the mucosa using a glass slide. The remaining wall was rinsed off over the tray and then discarded.

Any obviously extraneous material in

the ingesta was rinsed off and discarded. The ingesta and mucosa were washed into a 140 mesh (106 μm aperture) Endecott sieve and thoroughly washed with strong jets of water, until the sieved water was clear. The washed ingesta were collected in numbered plastic jars, 40% formaldehyde added for preservation and stored for later examination.

A few drops of Lugol's iodine were added to each jar immediately before examination. Easily visible helminths were removed and preserved in 10% formalin, the remainder of the ingesta were examined in toto under a stereo microscope and all the parasites collected and preserved for later indentification.

The helminths were identified according to the descriptions given by the authors listed in Table 1.

RESULTS

Helminths were recovered from 978 (65%) of the 1 502 cats examined (Table 2). The most prevalent helminths encountered were *Ancylostoma tubaeforme* followed by Ancylostoma braziliense and Dipylidium caninum.

The mean and highest number of each helminth species from individual cats is recorded in Table 3. Trematodes, *Echinococcus granulosus, Toxascaris leonina* and *Capillaria* spp. were not found in any of the cats examined.

The faeces of the first 415 cats were examined for protozoal parasites. *Isospora felis* (34%) and *Isospora rivolta* (14%) were the predominant protozoal parasites. Some *Eimeria* spp. oocysts were encountered. No *Toxoplasma gondii* oocysts were found.

Of the 1 502 cats necropsied, 890 were adults, of which 685 (77%) were infested with one or more helminth species, and 612 were juveniles, of which 293 (49%) were infested. The prevalence and respective ratios of helminths in adult and juvenile cats are recorded in Table 4.

The mean adult and juvenile helminth burdens, mean burden ratios, prevalence ratios and adjusted ratios are given in Table 5.

The cestodes *Joyeuxiella fuhrmanni* and *Taenia taeniaeformis* were more prevalent in adult cats. *Dipylidium caninum* was slightly more prevalent in adult cats, but juvenile cats tended to have greater mean burdens (Tables 4 & 5).

Of the nematodes, *Ancylostoma caninum, Toxocara cati* and *Toxocara canis* were encountered more often in juvenile cats. The two hookworms, *Ancylostoma tubaeforme* and *Ancylostoma braziliense* were more prevalent in adult cats. *Ancylostoma ceylanicum* and *Physaloptera praeputialis* were slightly more prevalent in adult than in juvenile cats.

DISCUSSION

The relatively high prevalence of parasites collected, indicates that conditions are favourable for helminth development, survival and transmission in the Pretoria area.

Dipylidium caninum is the most important cestode of cats in this area (Table 2). The greatest number of scolices recovered from a cat was 2 010. Of these, a small

Table 1: **The sources of reference for the identification of the helminths from cats.**

Helminth	Author
Dipylidium caninum	Witenberg[18]
Joyeuxiella fuhrmanni	Jones[9]
Taenia taeniaeformis	Verster[17]
Ancylostoma caninum	Burrows[3]
	Okoshi & Murata[14]
Ancylostoma tubaeforme	Burrows[3]
	Okoshi & Murata[14]
Ancylostoma braziliense	Biocca[2]
*Ancylostoma ceylanicum**	Biocca[2]
Toxocara cati	Sprent[16]
	Levine[10]
Toxocara canis	Levine[10]
Physaloptera praeputialis	Levine[10]
*Aleurostrongylus abstrusus**	Gerichter[7]
Vogeloides spp.*	Chitwood & Lichtenfels[4]
Pterygodermatitus spp.*	Gibson[8]
Centrorhynchus spp.*	Gibson[8]

(*) New records in cats in the Republic of South Africa

Table 2: **The prevalence* of helminth species recovered from necropsies performed on 1 502 cats**

Species	Number positive	Percentage positive
Cestoda		
Dipylidium caninum	362	23
Joyeuxiella fuhrmanni	38	2.5
Taenia taeniaeformis	115	7.7
Nematoda		
Ancylostoma tubaeforme	615	41
Ancylostoma caninum	49	3.3
Ancylostoma braziliense	374	25
Ancylostoma ceylanicum	21	1.4
Toxocara cati	161	11
Toxocara canis	3	0.2
Physaloptera praeputialis	20	1.3
Pterygodermatites spp.	7	0.5
Aleurostrongylus abstrusus	1	0.1
Vogeloides spp.	2	0.1
Acanthocephala		
Centrorhynchus spp.	12	0.8

* The term prevalence is used for the number of cats in which a particular species of worm was found

Table 3: **The mean and highest burden* of each helminth species irrespective of age or sex of cats**

Species	Mean burden**	Highest burden**
Cestoda		
Dipylidium caninum	29	2 010
Joyeuxiella fuhrmanni	26	217
Taenia taeniaeformis	2.8	38
Nematoda		
Ancylostoma tubaeforme	9.1	101
Ancylostoma caninum	1.9	6
Ancylstoma braziliense	7.7	136
Ancylostoma ceylanicum	3.7	13
Toxocara cati	3.8	11
Toxocara canis	1.0	1
Physaloptera praeputialis	3.8	28
Pterygodermatites spp.	1.2	3
Acanthocephala		
Centrorhynchus spp.	4.1	18

* Burden is the number of a particular species recovered from an individual cat

** Mean taken over infested cats only

Table 4: **Comparison of helminth prevalence in adult and juvenile cats**

	Adults		Juveniles		Ratio
	Number	%	Number	%	Adults/ Juveniles*
Total number of cats	890	(59)	612	(41)	
Number of cats infested with:					
Cestoda					
Dipylidium caninum	211	24	131	21	1.1
Joyeuxiella fuhrmanni	36	4	2	0.3	12.4
Taenia taeniaeformis	107	12	8	1.3	9.2
Nematoda					
Ancylostoma tubaeforme	517	58	98	16	3.6
Ancylostoma braziliense	321	36	53	8.7	4.2
Ancylostoma ceylanicum	21	2.4	4	0.7	3.6
Ancylostoma caninum	17	1.9	32	5.2	0.4
Toxocara cati	74	8.3	87	14	0.6
Toxocara canis	0	0	3	0.5	0/0.5
Physaloptera praeputialis	17	1.9	3	0.5	3.9
Total infested cats	685	77	293	48	

* Ratio of percentage adult cats infested to percentage juvenile infested cats

Table 5: ***Comparison of helminth burdens in infested adult and juvenile cats***

	Adult	Juveniles	Adults/ Juveniles	Adults/ Juveniles	
	Mean* burden	Mean* burden	Mean* ratio	Adjusted ratio**	Comment
Cestoda					
D. caninum	25.9	38.4	0.7	0.75	= (?)
J. fuhrmanni	27.3	2.0	13.7	157.0	A > > J
T. taeniaeformis	2.9	1.5	1.9	17.5	A > > J
Nematoda					
A. tubaeforme	9,8	5.0	2.0	7.0	A > > J
A. braziliense	8.1	5.3	1.5	6.4	A > > J
A. ceylanicum	2.5	6.0	0.4	1.5	A > J
A. caninum	1.6	2.1	0.8	0.27	J > A
T. cati	2.9	4.6	0.6	0.36	J > A
T. canis	0	1.0	0/1	0/0.5	J > A
P. praeputialis	4.1	1.7	2.4	10.0	A > > J

=(?) no marked difference between age groups
> slight difference between age groups
> > marked difference between age groups
A = adults; J = juveniles
* Ratio of mean burdens (adult to juvenile) taken over infested cases only
** Ratio of mean burdens (adult to juvenile) taken over all cats

percentage had attached and developed segments. The remaining scolices were still in an early stage of development. They had evaginated but the bodies were still very rudimentary. Whether or not all of these scolices would have attached and developed to maturity is a point for debate. No deworming programme aimed at this parasite will be successful unless fleas are controlled at the same time.

Joyeuxiella fuhrmanni was not recorded in any of the other studies cited above[1 5 6 11 12 13 15].

Ancylostoma tubaeforme is by far the most important helminth of cats in this area and should be a major consideration in the treatment of cats for helminths by the veterinarian.

Ancylostoma ceylanicum is difficult to differentiate from *A. braziliense*. In the case of the males, the difference lies in the arrangement of the lateral bursal rays. In *A. braziliense* the medio-lateral and postero-lateral rays are divergent and widely separated, whereas in *A. ceylanicum* the medio-lateral and postero-lateral rays lie closely together in parallel[2]. The females are not easily differentiated. Some female *A. ceylanicum* may thus have been incorrectly identified as *A. braziliense*.

The infestation with *Toxacora canis* probably represents an accidental infection from close contact with dogs. It would appear that cats do not routinely become infested nor harbour this parasite for any length of time.

Physaloptera praeputialis were located in the stomach and in some cases were firmly attached to the mucosa.

A provisional identification of *Pterygodermatites affinis* (Jägerskiold, 1904) was made from the limited number of helminths available. (Gibson, D. I. 1985 British Museum (Natural History) Personal communication).

No species identification of *Centrorhynchus* spp. was possible as all the specimens collected were immature. Gibson (1986, Personal communication) states that raptors are the usual definitive hosts and assumes that the cat is an accidental host which acquires the parasites by feeding upon small mammals, reptiles, etc. which act as paratenic hosts.

Aleurostrongylus abstrusus was recorded only once during this survey. This is the first record of this lungworm in the Republic of South Africa. Subsequent to this study, further cases have been found in Pretoria, Cape Town, the eastern Cape Province and Natal. (Unpublished data).

The lungs of 2 cats contained adults and eggs of *Vogeloidos* spp. This is also a first record of this lungworm in cats in the

Republic of South Africa.

Very little is known about the helminth burdens of adult cats as compared to those of juvenile cats. In surveys elsewhere[1,5,6,11,12,13,15], no differentiation was made between the ages of the cats. One of the intermediate hosts of *Joyeuxiella fuhrmanni* are lizards and of *Taenia taeniaeformis*, rodents. Adult cats will thus probably have a greater burden of these parasites than juveniles. In the case of *D. caninum* the difference between adults and juveniles is negligible. This is to be expected as the cat flea, *Ctenocephalides felis* would be present on kittens from a very early age.

Toxocara cati and the dog helminths *Toxocara canis* and *Ancylostoma caninum* are predominant in juvenile cats. This is possibly due to juveniles being in closer contact with puppies and having less resistance than adults to these parasites.

The hookworms Ancylostoma tubaeforme, Ancylostoma braziliense and to a lesser degree *Ancylostoma ceylanicum* are predominantly adult cat parasites. These findings tend to confirm the theory that transmammary transmission of these parasites does not occur in cats.

Physaloptera praeputialis is also predominantly a parasite of adult cats. Juveniles probably receive sufficient nourishment from their mothers without resorting to eating cockroaches, beetles and crickets.

These findings indicate the importance of using a broad spectrum anthelmintic in both adult and juvenile cats. Clinicians and breeders frequently treat kittens against roundworms and hookworms, and tend to overlook the tapeworms. Any deworming treatment should thus be aimed at both nematodes and cestodes, coupled with advice on flea control.

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THE NON-SIGNIFICANT EFFECT OF FEEDING LEVEL, GROWTH RATE AND AGE ON LIBIDO OF YOUNG AFRIKANER BULLS

C MAREE*, N H CASEY* and I E JACOBI*

ABSTRACT:

Afrikaner bulls (n = 30) between 9,5 and 15 months old, were allocated to 2 dietary treatments fed ad libitum. Diet 1 consisted of 70% concentrate and 30% *Eragrostis curvula* hay (11,4 MJ ME kg^{-1} DM) and Diet 2 of 40% concentrate and 60% *E. curvula* hay (9,3 MJ ME kg^{-1} DM). Five libido tests were conducted per bull between the ages of 16 and 28 months. Bulls were pre-stimulated for 10 min by close contact with previously synchronised oestrous heifers. Three bulls were then simultaneously allowed a period of 15 min with 6 heifers. Manifestations of libido were indexed as follows: 1. smelling heifer and definite signs of interest; 2. attempt to mount without erection; 3. mount with erection but no intromission; 4. successful service. Libido values fluctuated highly both within and between bulls. Libido did not improve over the 16 to 28-month age period nor with experience as successive tests were conducted. Bulls on Diet 1 grew significantly faster during the first 7,5 months of the trial, but demonstrated non-significantly higher libido scores. It was concluded that maturation over the 16 to 28-month age period, learning experience and level of feeding had no effect on libido.

Key words: Libido, Afrikaner bulls, dietary effects, age effects, learning effects

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INTRODUCTION

Libido in bulls is an important component of fertility and its manifestation depends on various factors. The genetic basis of libido is evident from clear similarities between monozygous twin brothers[1 5] and in differences found between crossbred bulls[7].

The serving capacity of bulls is improved significantly by sexual experience[8 9]. No seasonal changes in libido were recorded but the effect of some learning experience was evident from one year to the next. Reviews by Chenoweth[4 5] suggest that the basic pattern of sexual expression in bulls is innate. These reviews also confirm the importance of learning experience and that the rearing method can alter libido. Overfeeding for example has been shown repeatedly to inhibit the libido of young bulls[4 12].

Reports on libido of young Afrikaner bulls are scant, although in comparison, Afrikaner crossbred bulls have achieved the highest scores[7]. In addition, the question arises whether high level feeding (10,79 MJ ME kg^{-1} DM) which is given in Phase C (Director 1988 Animal and Dairy Science Research Institute, Department of Agriculture and Water Supply, Private Bag X2, 1675 Irene, Republic of South Africa) of the official performance testing scheme administrated by the Department of Agriculture and Water Affairs for the RSA, may affect libido in Afrikaner bulls. Consequently a trial was conducted to test the libido of young Afrikaner bulls at 2 levels of feeding.

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MATERIALS AND METHODS

Afrikaner bulls (n = 30) were randomly allocated to high (Diet 1) and low (Diet 2) feeding levels. Bulls on Diet 1 (n = 14) had an initial mass of 241 (201-282) kg and were 11 (9,4-12,6) months old; bulls on Diet 2 (n = 16) had an initial mass of 254 (226-283) kg and were 12,5 (10-15) months old. Diet 1 consisted of 70% concentrate and 30% *Eragrostis curvula* hay (11,44 MJ ME kg^{-1} DM) containing 11,5% crude protein. This diet was comparable to that employed in Phase C of the official performance testing scheme. Diet 2 consisted of 40% concentrate and 60% *E. curvula* hay (9,3 MJ ME kg^{-1} DM) and 9,2% crude protein. The bulls were each weighed weekly from Week 1 to Week 74. Treatment effects on growth were described by logistic functions using the model $Y = A / (1 + B.R.^x)$ where A represents the asymptote (total gain), B is a function of potential growth, R is a function of growth rate and x the timescale in weeks.

Between the ages of 16 and 28 months the bulls were subjected to 5 libido tests, 3 during the first year, in March, May and August with 82 d intervals and another 2 the following year, in March and May. Tests were conducted between 07h30 and 09h30 in a familiar, quiet paddock, with observations being made discreetly.

Bulls were pre-stimulated by allowing them to remain in an enclosure next to heifers in oestrus, synchronised with 2 mg Cloprostenol sodium (Estrumate, Coopers), for a period of 10 min. Three bulls were then simultaneously allowed a period of 15 min with 6 heifers.

Manifestations of libido were recorded and given index values as follows:

1 - Smelling heifer and definite signs of interest
2 - Attempt to mount without erection
3 - Mount with erection but no intromission
4 - Successful service

Three observers per bull were employed *each time and average scores were* recorded.

Data was interpreted by means of a correlation coefficient for each bull between week of trial and libido index to indicate libido changes with increasing age. The effect of level of feeding was determined by the Student t test. Between-bull coefficients of variation (CV) were determined to estimate differences between individuals while the repeatability of individual libido scores was determined to indicate the consistency of scores.

Repeatability was calculated according to Lasley[11]:

$$r = \frac{(MS_B - MS_W) / ni}{(MS_B - MS_W) / ni + MS_W}$$

where MS_B = between-animal mean square

MS_W = within-animal mean square

ni = number of observations per animal

RESULTS

The logistic function provided a good sigmoidal fit of the pooled data of both Diet 1 and Diet 2 (R^2 = 0,94). Growth on Diet 1 was described by $y = 337,85 / (1 + 8,66 \times 0,93^x)$ with an estimated initial weight of 238,3 kg (R^2 = 0,99) and on Diet 2 by $y = 277,34 / (1 + 11,92 \times 0,93^x)$ with an estimated initial weight of 254,5 kg (R^2 = 0,98). The total gain of bulls on Diet 1 exceeded that of bulls on Diet 2 very

significantly. A profile analysis indicated that between Week 1 and Week 30, the growth rates differed highly significantly, but with decreasing significance (Week 1-10 $P<0,0001$; Week 10-20 $P<0,0037$; Week 20-30 $P<0,0045$). After Week 30 the growth rates were similar ($P<0,10$).

The correlation coefficient between week of trial and libido was statistically significant in one bull only ($r = -0,9$; $P = 0,03$). On the strength of values obtained for the remaining 29 bulls, it is concluded that libido did not improve between the ages of 16 and 28 months, nor with experience as successive libido tests were conducted.

Mean libido scores for bulls on Diets 1 and 2 respectively were 15,2 (CV = 43%) and 14,4 (CV = 51%) and repeatabilities $r = 0,24$ and 0,25.

DISCUSSION

In terms of the low repeatability values and the high coefficients of variation obtained for both groups, it is evident that libido values fluctuated both within and between bulls. Further, the difference in the levels of feeding that were employed had no significant influence on libido scores, although the libido scores of the bulls on Diet 2 were slightly lower. Evidently therefore neither maturation, learning experience nor level of feeding could be shown to have any effect on libido values obtained.

Blockey[1,2], working with Hereford bulls, reported that 2-year-old bulls displayed their inherent serving capacity on their virgin test, with no learning experience evident subsequently. In this investigation libido scores within bulls remained relatively stable. Good repeatability of libido scores was also reported by Chenoweth et al.[6] and by Gardiner.[10] Other reports indicate a considerable effect of learning experience on libido scores[4,5,8]. These reports all deal with non-Zebu bulls and stand in sharp contrast to libido values obtained for Afrikaner bulls in this investigation where an inherently low display of maturing activity was recorded with only a few successful matings over the entire period.

Lack of heterosexual experience may play a role but reports are available on the inherent sexual sluggishness of *Bos indicus* bulls[3]. Crichton & Lishman[8] also reported that out of 5 Brahman and 2 Afrikaner bulls, not a single full service was recorded during 5 serving capacity tests while regular services were recorded by bulls of other breeds (Sussex, Hereford, Simmentaler and South Devon).

It is concluded therefore that whilst libido scores of the Afrikaner bulls in this investigation were not affected by level of feeding, further investigation is required to compare mating behaviour of Afrikaner and other Sanga bulls with bulls of other breeds, notably Zebu.

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COMPLICATIONS OF OVARIAN AUTOTRANSPLANTATION IN BITCHES

N.L. DAVIES*

ABSTRACT

Autotransplantation of the ovary to the portal vein drainage area was performed in 1 130 bitches over a period of 5 years. Complications of this procedure occurred in 41 bitches and included gastric ulceration, recurring pro-oestrus and neoplastic transformation of the transplant.

Key words: Ovarian autotransplantation, gastric ulceration, recurring pro-oestrus, neoplastic transformation

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Ovarian autotransplantation into the portal vein drainage area (ATOPA) was proposed as a method of preventing obesity in ovariectomised bitches[2-4]. By maintaining circulating oestradiol levels, it was postulated that ATOPA would abolish oestrus yet prevent the "eunuchoid syndrome" seen after ovariectomy[2-5]. This short article describes some of the complications of ovarian autotransplantation.

Over a period of 60 months a total of 1 130 ATOPA operations were performed on healthy bitches presented for routine sterilisation. In 986 bitches the sliced ovaries were implanted in a subserosal pouch on the greater curvature of the stomach, using described methods[2-4]. In the remainder, the ovaries were implanted in a subcapsular pouch formed on the caudo-ventral surface of the spleen[2]. In all cases the entire uterus was removed.

Post ATOPA complications developed in 41 (3,6%) bitches. Three basic clinical groups emerged:

i) Continuation of cyclical activity and signs of pro-oestrus. Pro-oestrus signs (haemorrhagic vaginal discharge and attractiveness to male dogs) developed in 17 patients. The mean time of onset of signs of pro-oestrus was 31 months after the ATOPA was performed, with a minimum of 2 and a maximum of 108 months. Clinically, this was only obvious to male dogs in the same household, and the signs were usually eliminated by a single injection of depoprogesterone. However, in 10 of the 17 bitches, surgical removal of the implanted ovaries was necessary to prevent the continual nuisance of cyclical pro-oestrus signs. The interval between periods of pro-oestrus was usually 6 months. Three bitches exhibited recurrent pro-oestrus and developed gastric ulceration at a later stage.

(ii) Gastric ulceration. Signs of gastric ulceration developed after a mean period of 70.4 months after the ATOPA surgery, with a minimum of 13 months and a maximum of 102 months. In these cases the implanted ovaries underwent hyperplasia, varying in diameter from 10mm in 90mm. Ulcers varying from 5 to 10mm in diameter developed immediately below the hyperplastic ovaries. Acute, severe anaemia and melaena were consistent signs. Vomiting and haematemesis occurred in only 7 of the 24 bitches which presented with gastric ulceration. Two patients died from the anaemia. Excision of the transplanted ovary and the underlying gastric ulcer, in conjunction with a blood transfusion, was necessary to eliminate the clinical signs.

iii) Signs of severe anaemia, melaena, vomiting and diarrhoea associated with the development of neoplasia within the ovarian transplant occurred in 2 cases. One bitch showing these signs was euthanased 84 months after surgery and post mortem examination revealed a malignant tubular carcinoma of ovarian origin which had caused a large gastric ulcer. Laparotomy in the second bitch, 79 months after the ATOPA operation, revealed an extensive gastric ulcer underlying an infiltrative tumorous ovary. Histopathology revealed a malignant granulosa cell tumour. No signs of metastasis to the liver were seen during the laparotomy. Radical gastric resection was necessary to remove the infiltrative tumour.

The occurrence of "brief, occasional periods of pro-oestrus" was reported in the original article in a "small proportion of ATOPA bitches"[2,3].

The large ulcers seen immediately under the site of the ovary transplant were probably due to pressure necrosis of the underlying gastric bloodvessels and serosa caused by abnormal proliferation of the transplanted ovarian tissue. This increase in size of the graft of up to 10 times the original size, was noted by Le Roux[2]. Histopathological examination of the resected ulcers and associated ovarian tissue, revealed ovarian luteal hyperplasia and necrosis of the stomach wall. The histopathological ovarian changes concur with those described in experimental animals[1]. The development of neoplasia in the transplanted ovaries also followed the same course as that seen in experimental animals, as reported by Mardones et al[6]. Of the 41 cases presented, only 4 had the ovary implanted into the spleen. This was probably due to the fact that far fewer splenic ATOPA operations were performed (144 out of 1130). As the splenic implants were performed 4 years after the gastric implants, complications will probably only manifest in the future.

The number of patients which returned to the practice with complications, are not an accurate estimate of the rate of incidence of complications. An unknown number of the ATOPA recipients are no longer patients of the practice, including 105 Guide Dog bitches which were posted around the country. The rate of complications may thus be greater than recorded here.

In view of the potentially fatal complications of the ATOPA operation, it is advisable to warn clients of these dangers before performing this operation.

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DEVELOPMENTAL KYPHOSCOLIOSIS IN A FOAL

R M Kirberger* and R D Gottschalk*

ABSTRACT:
The clinical, radiological and anatomical changes in a yearling foal with kyphoscoliosis are described. The lesion was due to a primary malformation of the eleventh, twelfth, fourteenth and fifteenth thoracic vertebral bodies resulting in hemivertebrae. Secondary changes occurred in the laminae, pedicles, spinous and articular processes of the affected vertebrae and the adjacent vertebrae. The possible pathogenesis and differential diagnosis are discussed.

Key words: developmental, scoliosis, kyphosis, kyphoscoliosis, hemivertebra, foal

Kirberger R.M.; Gottschalk R.D. Developmental kyphoscoliosis in a foal. *Journal of the South African Veterinary Association* **(1989) 60 No. 3, 146-148 (En.) Department of Surgery, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort, Republic of South Africa**

INTRODUCTION

Developmental abnormalities of the equine spine are rare occurrences. In a survey by Jeffcott[3] comprising 443 horses with thoraco-lumbar complaints, only 15 cases had vertebral malformations and of these only 3 were definitely congenital. Foals with congenital back problems are frequently put down as they are unlikely to be ridden or worked in later life.

CASE REPORT

An American saddle horse mare and 5-month-old foal were purchased in poor condition. Other than emaciation, the foal appeared normal to the owner. After a month, however, it was noticed that the foal had a "camel back" appearance which in time became more obvious.

The foal was dewormed regularly and fed on a balanced ration. At the age of one year, the foal was referred to the Department of Surgery for further investigation.

The animal presented with an obvious kyphosis (Fig. 1) and scoliosis of the thoracolumbar spine. No defects were detectable on neurological examination

The horse had a lower than normal arc to the swing phase of the stride of both hind limbs. This had caused a mild degree of abnormal wear of the hooves of the hind limbs characterised by wearing of the dorsal aspect of the toe.

Ventrodorsal and lateral radiographs of the spine were taken with the foal under general anaesthesia. Owing to the size of the patient and the abnormal shapes of the vertebrae, it was difficult to distinguish individual vertebral abnormalities. Scoliosis and kyphosis were visible extending from the ninth to the seventeenth thoracic vertebrae. On the lateral radiograph, the fifteenth thoracic vertebra could be distinguished as a hemivertebra. A myelogram was performed by injecting 40cc Iopamidol (Jopamiron 300, Berlimed) via the lumbosacral space. The contrast medium was well visualised up to the fourteenth thoracic vertebra where its passage was blocked by the compressed cord (Fig 2).

The horse was euthanased and the vertebral column was macerated for examination.

Macroscopic findings included asymmetry of the vertebrae which started at the seventh thoracic vertebra and ended at the fourth lumbar vertebra. The fifteenth thoracic vertebra had the worst malformation, with a vertebral body length of 15mm on the left side and a normal length of 45mm on the right (Fig. 3). This resulted in only two-thirds of the cranial extremity of the vertebral body being present (Fig. 4) whereas the caudal extremity was complete. The fourteenth thoracic vertebral body was similarly affected, but the cranial extremity of the vertebral body was complete. The eleventh and twelfth thoracic vertebrae had similar changes but the changes were reversed with the right side of the vertebral body being shorter (Fig. 5). Both these vertebrae only had two-thirds of the cranial extremities of the vertebral bodies present (Fig. 4). The abnormal shape of the vertebral bodies resulted in a virtual 90° turn to the left, cranial to the fifteenth thoracic vertebra and another 90° turn to the right, cranial to the thirteenth thoracic vertebra. In addition, the dynamic forces acting on these wedge-shaped vertebral bodies, resulted in dorsal rotation of these vertebrae, leading to kyphosis. The eleventh, twelfth, fourteenth and fifteenth thoracic vertebrae had severely malformed spinous processes, articular facets, laminae and pedicles. The cranial and caudal articular facets were hypoplastic on the convex sides and hyperplastic on the concave sides. These abnormalities were also visible on the adjacent vertebrae but in a decreasing degree the further away the vertebrae were from the primary abnormal vertebrae. The transverse processes of the lumbar vertebrae were also malformed.

DISCUSSION

Spinal malformation may be acquired as a result of trauma, metabolic disturbances, toxic effects, infections or it may be congenital.

Congenital scoliosis, which may be accompanied by kyphosis, has been attributed to several causes:

Congenital synostosis or block vertebrae may result in a mild scoliosis but was excluded radiologically and on the macerated specimen[3].

Congenital torticollis and head scoliosis have been investigated in depth in the foal[7]. These malformations were presumed to result from unfavourable intrauterine positioning. The head and neck were reflected and compressed in the narrow tip of the uterine horn resulting in uneven growth of the 2 sides of the head and neck. Foals that survived the birth process usually recovered completely. In this series, the thoracolumbar area was rarely involved. Uterine malposition is excluded as a possible cause in this case, as the foal was born seemingly normal and became progressively worse only after 5 months.

The horse is prone to new bone proliferation at sites of periosteal damage or trauma[2]. There were no signs of periosteal reactions on the radiographs or on the macerated specimen, thus excluding trauma as a possible cause. There was also no history of post-natal injury. Had this occurred, the extensive and severe degree of deformation would have caused severe paresis, if not total paralysis.

Selenium toxicity, alone or in combination with a manganese deficiency has been implicated as a teratogen causing arthrogryposis and kyphoscoliosis in calves[4]. This has not been described in foals.

The history, clinical signs and macroscopic changes excluded possible infectious agents.

Rooney[5] described 2 cases of scoliosis where hypoplasia of the vertebral articular facets on one side with normal facets on the other side was implicated. This resulted in the capsular ligaments being too 'large' for the hypoplastic joint, making the joint more mobile. He makes no mention of the shape of the vertebral bodies in his series. In this case it is felt that the vertebral body malformation resulted in secondary hypoplasia of the articular facets due to minimal contact of the articular facets.

Lateral hemivertebrae are the most common cause of scoliosis in man[6] and some other species[1]. It is postulated that lateral hemivertebrae were the cause of the kyphoscoliosis in this case. The

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Fig. 1 The year-old foal with signs of kyphosis and scoliosis of the thoracolumbar spine

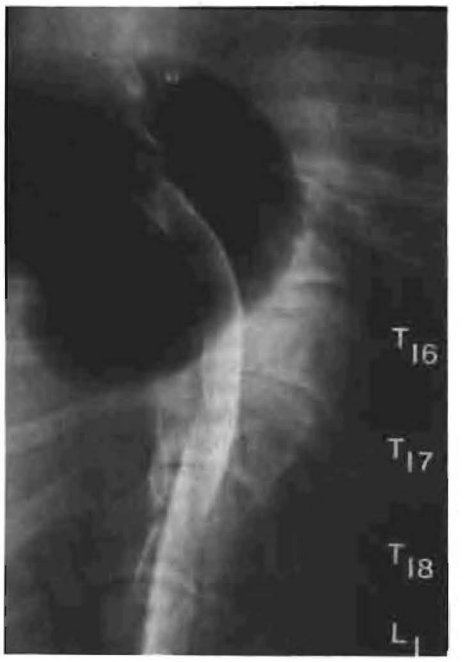

Fig. 2: Ventro-dorsal radiograph of the thoracolumbar vertebrae showing scoliosis and the abrupt termination of contrast medium

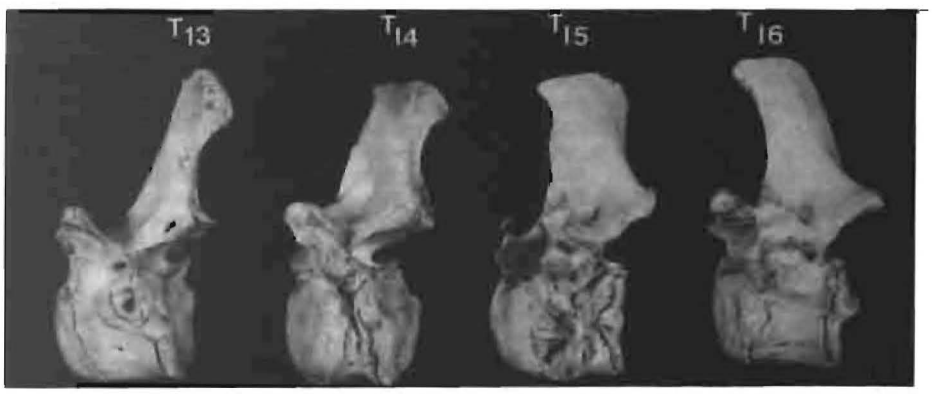

Fig. 3: Lateral view from the left side of T_{13}, T_{14}, T_{15} and T_{16}

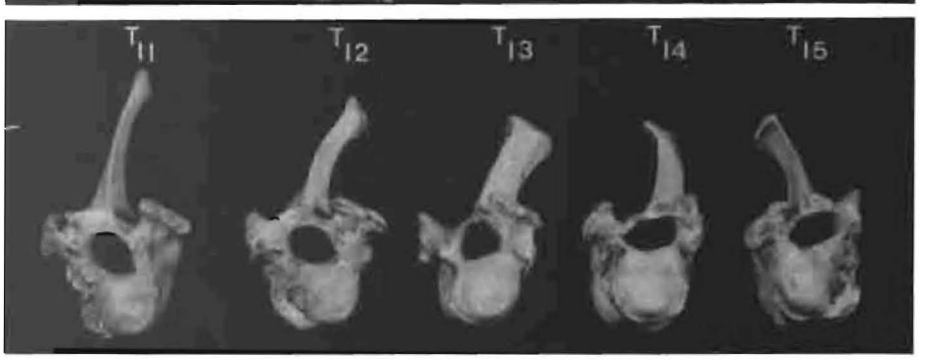

Fig. 4: Cranial view of T_{11}, $T_{12}T_{13}$, T_{14} and T_{15}

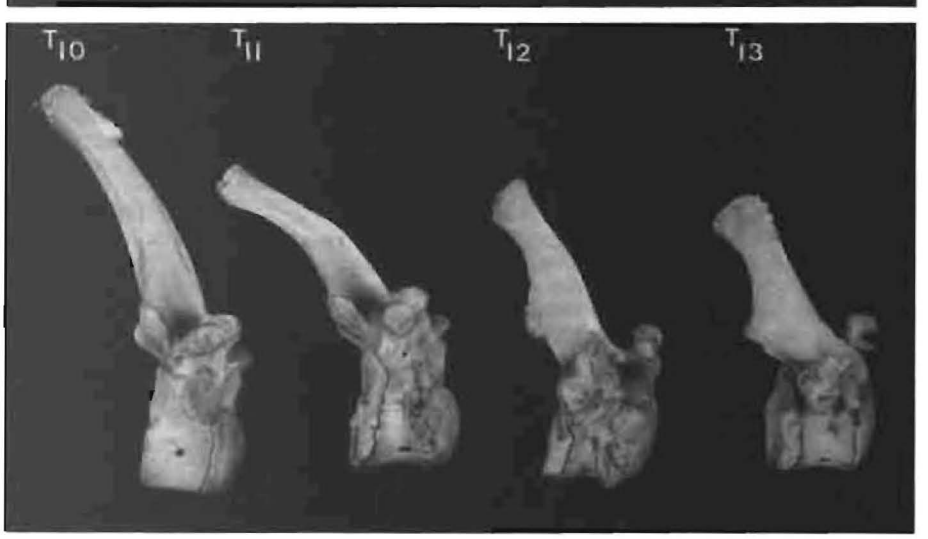

Fig. 5: Lateral view from the right side of T_{10}, T_{11}, T_{12} and T_{13}

pathogenesis of the lateral hemivertebrae may be due to a lack of vascularisation of one half of the vertebral body resulting in a lack of ossification of the cartilaginous vertebral body anlage or from hemimetameric segmental displacement of the primitive somites during transformation into final segments.

It is impossible to say which of these 2 possibilities was the cause of the kyphoscoliosis, as after birth the forces acting on the vertebral column will grossly exacerbate the condition. One or 2 vertebrae may have been involved intially resulting in compensatory abnormalities of the other vertebrae or the 4 most severly affected vertebrae initially may have been hemivertebrae.

A possible genetic background to this case could not be determined.

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ODONTOMA IN AN AFRICAN ELEPHANT *(Loxodonta africana)*

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ABSTRACT

The first known case of an odontoma in an African elephant *(Loxodonta africana)* is described. The tumour was fused with the coronal cementum of the sixth right mandibular molar tooth, thus preventing its eruption.

Key words: African elephant, *Loxodonta africana*, odontoma

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INTRODUCTION

The term "odontoma" by definition alone, refers to any tumour of the dental tissues[4]. Through usage, however, it has come to be employed in a much more restricted sense and refers to a tumour in which induction has resulted in the development of both enamel and dentine[1].

Odontomas represent a hamartomatous malformation rather than a neoplasm[9]. Thus, they are frequently formed in the place of a missing tooth, or if all the teeth are present, an odontoma may represent a malformation of a supernumerary tooth germ[6]. Odontomas are subdivided according to morphological features into complex and compound odontomas. The complex odontoma consists of a mass of irregularly-arranged enamel, dentine, cementum and connective tissue, bearing no morphologic similarity to teeth. In the compound odontoma the enamel, dentine and cementum are laid down in an orderly fashion so that toothlike structures can be identified. In humans the complex type of odontoma is less common than the compound type, although some lesions are a combination of both types[6].

Odontomas have been reported in various animals, including dogs[11], horses[2 3 8] and nonhuman primates[10 12]. This report describes the first known case of an odontoma occurring in an African elephant *(Loxodonta africana)*.

CASE REPORT

A dried mandible of an African elephant, containing a 350 x 250 x 200 mm calcified tumour in the right corpus was submitted to the Department of Oral Pathology for examination and diagnosis. The tumour caused buccal and lingual expansion (Fig. 1) and was partially erupted and functional: the abraded occlusal surface showed haphazardly-arranged cementum, enamel and dentine. The tumour had an irregular surface and was not attached to the surrounding bone which showed features of osteomyelitis. A portion of a molar tooth protruded from the anterior (rostral) surface of the lesion which had a total mass of 7,8 kg (Fig. 2). The associated molar tooth was clearly visible on the sectioned surface and the cementum of the tooth was fused to the tumour, the latter of which was composed of cementum-like tissue surrounding well-formed enamel and dentinal structures (Fig. 3).

Radiographic examination of the distal corpus and ramus of the right mandible failed to exhibit additional developing teeth. The 6th molar tooth on the left was fully developed, erupted and partially abraded.

Microscopic examination of the tumour revealed cellular cementum, dental enamel and regular dentine arranged in an orderly fashion (Fig. 4).

DISCUSSION

Odontomas develop in place of a tooth or, if the normal complement of teeth are present, from a supernumerary tooth germ. They follow the normal growth pattern of a developing tooth and even though quite large dimensions may be attained, the cellular activity of odontomas cease after completion of hard tissue formation.

Unlike humans, elephant have a total of 6 successive developing molar teeth in each quadrant which are abraded and shed throughout the life of the animal. Examination of the left mandible of our specimen showed the 6th molar to be fully developed and erupted and the age of the animal was estimated to be in excess of 35 years. As the chronology of tooth development in the specimen is unknown, the origin of the odontoma suggests two possibilities. The lesion may have originated from the germ of the 5th molar which develops and erupts between the ages of 16 and 43 years[5]. Alternatively, in the presence of a normal complement of teeth, the odontoma could have developed from a supernumerary tooth germ. The occurrence of supernumerary molar teeth in elephant however, has not been described.

The macroscopic and microscopic appearance of the lesion were consistent with a mature compound type odontoma. Enamel, dentine and cementum were arranged in an orderly fashion and the interface between these tissue types resembles that found in a normal tooth. Fusion between the cementum of the odontoma and the associated molar tooth was the result of cementum formation on the enamel surfaces in both structures. The formation of cementum on enamel is a normal phenomenon in many animals[7]. The forces of eruption of the fused molar tooth probably forced the odontoma into occlusion with the opposing maxillary molar tooth, hence the smooth abraded area on the ventral surface thereof. Partial exposure of the odontoma to the oral environment resulted in the development of an osteomyelitis in the bone surrounding the lesion.

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We wish to express our appreciation to Mrs C S Begemann for secretarial services.

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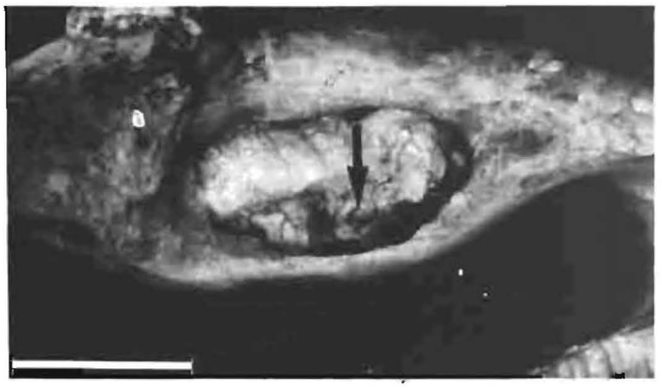

Fig. 1: Right corpus of mandible with partially erupted odontoma. (arrow) and buccal and lingual bone expansion (bar = 10 cm) Note abraded surface

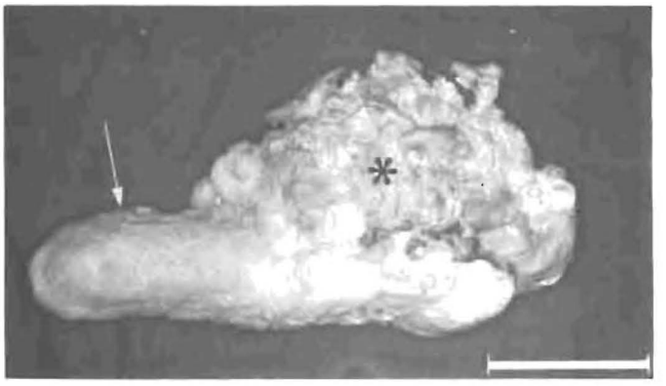

Fig. 2: Molar tooth (arrow) with attached odontoma (asterisk) (bar = 10 cm)

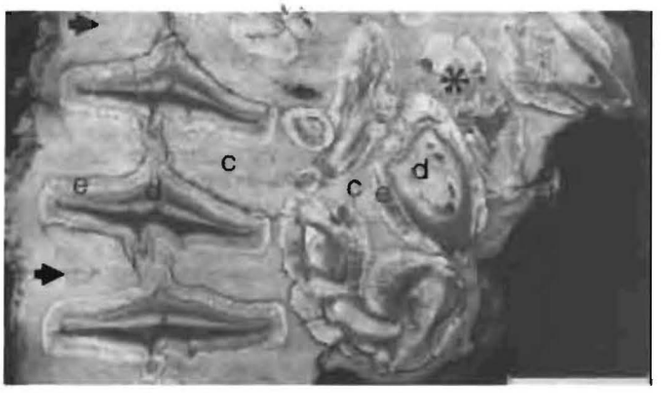

Fig. 3: Length section through odontoma (asterisk) and molar tooth (arrows), showing the enamel (e), dentine (d) and cementum (c) of both structures (bar = 2 cm)

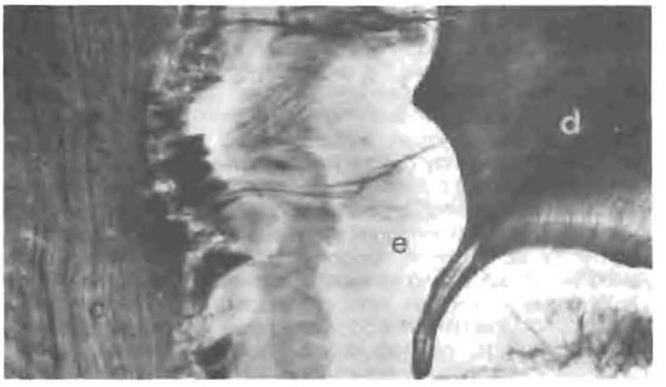

Fig. 4: Micrograph of the orderly-arranged cementum (c), enamel (e) and dentine (d) of the odontoma (Ground section, x 20)

NARCOLEPSY IN A LONG-HAIRED DACHSHUND

J VAN HEERDEN* and G N ECKERSLEY*

ABSTRACT:
Narcolepsy was diagnosed in a three-month old, longhaired Dachshund presented with a history of sudden onset of episodes of complete collapse. Littermates or immediate ancestors were not affected. Numerous daily cataplectic attacks as well as excessive sleepiness were the main clinical features. The dog did not respond to treatment with methylphenidate and imipramine. The clinical signs of the disease disappeared after treatment with dexamphetamine. This report describes a case presentation, findings of cerebrospinal fluid assay for catecholamines after various treatment regimens and the response of the patient to treatment.

Key words: Narcolepsy, canine

Van Heerden J.; Eckersley G.N. Narcolepsy in a long-haired Dachshund. *Journal of the South African Veterinary Association* **(1989) 60 No. 3, 151-153 (En.) Department of Companion Animal Medicine and Surgery, Faculty of Veterinary Science, Medical University of Southern Africa, 0204 Medunsa, Republic of South Africa.**

INTRODUCTION

Narcolepsy is a disease syndrome of ill-defined aetiology which results in a severe disturbance of the sleep cycle. It is an incurable syndrome which in humans is characterised by cataplexy (sudden attacks of flaccid paralysis), excessive day-time sleepiness, sleep paralysis (a sensation of being unable to move when drifting into sleep or just upon awakening) and hypnagogic hallucination (hallucination occuring between sleeping and awakening[2]). The latter two signs are subjective phenomena that cannot be determined in animals. Excessive day-time sleepiness and cataplexy are therefore the main features of the condition in dogs[12].

Normal sleep patterns include nonrapid eye movement (NREM) sleep (which consists of light slow-wave sleep and deep slow-wave sleep) which is followed by rapid eye movement (REM) sleep. During REM sleep the postural reflexes are atonic, occasional fasciculations of distal and facial muscles occur, the eyes may be partially open and rapid eye movements are present[12]. The disturbance in the sleep pattern is characterised by sleep onset, rapid eye movement (SOREM) sleep without the usual initial period of NREM sleep[9].

Neurochemical findings and pharmacological testing have supported a hypothesis of similar mechanisms being involved in normal REM sleep and cataplexy[4]. Decreased concentrations and turnover of serotonin, a decreased turnover of norepinephrine and a decreased concentration of dopamine in the reticular activating system of narcoleptic patients have been suggested by the results of some of these investigations[5]. In another study[6], certain examined areas in the brain of narcoleptic Doberman Pinschers contained significantly higher concentrations of dopamine and its metabolites than those of control normal dogs. Elevated dopamine receptor numbers have been reported in the same animals. The authors claimed these results to be consistent with an hypothesis of a dopamine-release problem in narcoleptic patients. Central cholinergic inhibitory mechanisms may also play a role in the production of cataplexy[4]. An increase in the number of muscarinic cholinergic receptors in the brainstem of narcoleptic Doberman Pinschers was described by Kilduff et al[11] These findings, as well as evidence of difficulties with the release of dopamine, may be indicative of a molecular membrane deficit in narcoleptic patients.

The condition was first reported in the late nineteenth century in man and has since been reported to occur in 5-10 people out of every 100,000. It has also been described in dogs, in a cat as well as in horses and ponies[10]. The development of cataplectic signs early in life is a characteristic feature of the disease in both humans and dogs. The familial occurrence of narcolepsy is an important aspect of the disease in humans. Both a multifactorial mode of inheritance as well as a single dominant gene with a low penetrance have been suggested as possible modes of transmission in humans[2]. In at least 2 (Dobermann Pinchers and Labrador Retrievers) of the 15 different purebred and mixed breeds of dog in which the disease has been diagnosed, a genetic background has been proposed In Dobermann Pinchers, narcolepsy is recessively inherited and in Labradors this mode of transmission is also suggested. In miniature Poodles and Beagles there is only sufficient data to exclude the simplest genetic hypothesis of a fully penetrant autosomal or sex-linked dominant or recessive gene[7].

Diagnosis is usually based solely on typical clinical signs of the disease[8]. Provocative testing by the food-elicited cataplexy test or the physostigmine provocative test, usually greatly amplifies clinical signs. In the food-elicited cataplexy test, 10 items of food, approximately 1 cm in size are placed in a row and spaced 30,5cm apart for small and 50cm apart for larger breeds. The time it takes the dog to finish all the pieces of food and again be capable of voluntary movement, is recorded. Normal dogs do not show any cataplectic attacks and will finish all the food within 45 s[1]. The Multiple Sleep Latency Test is also used to quantify readiness to fall asleep. The use of sophisticated procedures like electromyography and electroencephalography may be of little help in diagnosing narcolepsy in dogs.

Treatment is aimed at the control of cataplexy and excessive sleepiness. The drugs used, which include sympathomimetics and the tricyclic antidepressants may increase the concentrations of biogenic amines in the central nervous system. Tricyclic antidepressants such as protriptyline and imipramine are uptake blockers of, amongst others, norepinephrine. They also have anticholinergic properties.

Dosage levels of drugs in the treatment of narcolepsy are not definable and dosages depend on the reaction of the patient. The complete abolishment of cataplexy is also rarely achieved and the aim of treatment should be acceptable reduction in the frequency and duration of cataplexy[8]. Excessive day-time sleepiness can be controlled by the use of methylphenidate hydrochloride at a dosage of 0,25mg kg^{-1} Control of cataplexy may be attempted by the use of imipramine at a dosage rate of 0,5-1 mg kg^{-1} three times daily[3 10 12].

Narcolepsy in dogs should be differentiated from narcoleptic hypersomnia, myasthenia gravis, epilepsy and episodic syncope.

This report describes what we believe to be the first reported case of narcolepsy in a dog in South Africa.

CASE REPORT

A 3-month old longhaired Dachshund bitch was presented with a history of sudden onset of episodes of varying dura-

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tions of complete collapse. No evidence could be found of similar signs recorded for littermates or in the ancestors of the dog. The dog apparently had a very playful nature and these episodes were first noticed during periods of play with other dogs.

An initial clinical examination of the puppy revealed no other abnormalities apart from a cardiac dysrhythm. Observation of the dog however, revealed frequent attacks of complete loss of motor tone.

Baseline haematology and blood chemistry (inclusive of blood glucose and total calcium concentrations) investigations did not reveal any abnormal findings. Contrast radiography of the oesophagus likewise failed to reveal any abnormalities. Electrocardiographic examination of the patient identified the dysrhythm as a second degree atrio-ventricular block.

The intravenous administration of edrophonium chloride (Tensilon, Roche) failed to eliminate clinical signs. The dog, on the contrary, showed more severe clinical signs over the following 12 h.

The intravenous administration of amitriptyline hydrochloride (Tryptanol, Frosst-MSD) likewise failed to eliminate clinical signs of cataplexy and excessive sleepiness.

The food-elicited-cataplexy test was abandoned after 660 s on the first attempt because of the excessive prodding required to keep the dog awake. On another occasion it took the patient 438 s to complete the test.

Cerebrospinal fluid (CSF) and blood was collected after general anaesthesia with thiopentone sodium (Intraval, Maybaker) and insertion of an endotracheal tube. Initial restraint of the patient for general anaesthesia resulted in considerable excitement and struggling (after subsequent treatment trials the patient was restrained with considerably more ease). Specimens of CSF were preserved with reduced glutathion (4 mmol ℓ^{-1}) immediately after collection prior to storage at -20°C. All specimens were later assayed for adrenaline, noradrenaline and dopamine (electrochemical detection, BAS; HPLC, Varian Vista 5000). CSF specimens taken from 4 mongrel dogs of approximately the same age were collected, handled, stored and subjected to the same analyses. These control dogs were more easily restrained than the patient.

The results of analysis of CSF are presented in Table 1. The concentration of norepinephrine was marginally lower and that of dopamine slightly lower in the patient's CSF than the average of the control dogs.

Observation of the dog over the following 3 months revealed excessive daytime sleepiness and numerous daily cataplectic episodes. Cataplectic episodes were often readily induced by offering the dog food, particularly hard dry pellets and bones which had to be chewed and crushed during eating. Cataplectic episodes could also be induced by excitement and exercise. Normal exercise was impossible as the patient fell asleep approximately every 15 metres. The patient was easily aroused during a cateplectic episode by gentle prodding or by noise. Cataplectic episodes could not be induced by painful stimuli such as attempting venipuncture, scaring the dog or physical punishment such as a rap over the buttocks or a bite from another dog. Feeding the dog very hot food (which was left untouched by other dogs) also resulted in the temporary disappearance of clinical signs.

Vomition was observed infrequently. In general, there was a tendency in the dog to pass relatively small amounts of soft faeces.

Considerable variation occurred from time to time in the number and frequency of attacks, especially with regard to the narcolepsy-eliciting effect of food. The dog often finished her food without a single attack. Her appetite was always good. Episodes of complete collapse (complete cataplectic episodes) were sometimes preceded by weakness of the hindquarters, (partial cataplectic episode) bobbing of the head, general weakness and weak attempts to stand up. The dog slept with the eyelids either closed or open. Rapid eye movements were not always observed and following eye movements could not be demonstrated. Sleep was often accompanied by fasciculations of the distal musculature, vocalisation and weak chewing movements - especially when the attack occurred whilst the dog was chewing or attempting to chew a bone.

accessible places such as the small space between a refrigerator and a wall; hyperactivity and complete refusal of all food. These behavioural patterns were more disturbing to the owners than the frequent cataplectic attacks.

Analysis of cerebrospinal fluid 2 h after administration of dexamphetamine sulphate yielded epinephrine concentrations well in excess of those of control dogs (Table 1).

DISCUSSION

The signalment and clinical signs of the presented patient are typical of those described for narcolepsy in smaller breeds of dogs.

The patient differed from most reported cases in that no noticeable response was obtained by the use of imipramine hydrochloride. Quantification of the patient's response to drug treatment using the food-elicited cataplexy test, was not determined. The use of this test to quantify or monitor the response of this patient to treatment is, in view of the patient's variable repsonse to food intake, probably of doubtful value. Complete elimination of clinical signs of cataplexy were only obtained after administration of a relatively high dosage of dexamphetamine sulphate. Treatment with this drug, however, had to be abandoned as the owners were more distressed by the side-effects of dexamphetamine than by the clinical signs of narcolepsy. Despite the shortcomings of single assays of cerebrospinal fluid in the patient (and comparison with a small number of control dogs) for catecholamine concentrations, the obtained results confirmed clinical observations of a poor response to treatment with imipramine and methylphenidate hydrochloride. Initial baseline concentrations of adrenaline, noradrenaline and dopamine in the cerebrospinal fluid of the patient may have been increased by struggling and considerable excitement during restraint of the patient prior to general anaesthesia.

The observed clinical signs and behaviour of the dog clearly reflect that fear and pain resulted in remission of clinical signs whereas feeding, chewing, excitement and playful behaviour elicited attacks.

Narcolepsy may be confused with narcoleptic hypersomnia, epilepsy, myasthenia gravis and episodic syn-

Table 1: **Concentrations of norepinephrine, epinephrine and dopamine in the cerebrospinal fluid of a dog before and after treatment with methylphenidate hydrochloride and imipramine as well as dexamphetamine sulphate**

	Norepinephrine ng $m\ell^{-1}$	*Epinephrine* ng $m\ell^{-1}$	*Dopamine* ng $m\ell^{-1}$
Patient	1,15	4,60	3,75
Patient after treatment with methylphenhidate hydrochloride and imipramine	0,33	6,90	0,45
Patient after treatment with dexamphetamine sulphate	1,10	8,30	3,00
Control (n=4) ($\bar{x}$;SD)	1,31(± ,06)	4,40(± 0,60)	5,90(± 3,51)

Treatment by the oral administration of methylphenidate hydrochloride (Ritalin, Ciba) at a dosage rate of 0,5 mg kg^{-1} once daily in combination with imipramine hydrochloride (Tofranil, Geigy) (0,8 mg kg^{-1}, three times daily) was attempted for a period of 6 weeks. This treatment regimen did not result in any obvious improvement in clinical signs. Cerebrospinal fluid analysis at the end of this period revealed an increase in the concentration of epinephrine (Table 1).

Treatment with dexamphetamine sulphate (Dexedrine, Smith Kline & French) at a total oral daily dosage of 5 mg, resulted in complete remission of clinical signs of excessive sleepiness and cataplexy. A dose-related effect was however observed in that approximately 4 mg (approximately 3/4 of a dexamphetamine sulphate tablet) or a lesser dosage did not result in disappearance of clinical signs. Although a total dosage of 5 mg effected complete remission of clinical signs, the following untoward side-effects were observed: peculiar behavioural patterns which included excessive and intensive sniffing of the ground surface; a desire to climb into in-

cope[8, 13]. Narcoleptic hypersomnia[1] is characterised by excessive daytime sleepiness and difficulty in arousing an animal from sleep. Sleep attacks are not observed. Epilepsy is often characterised by urinary and faecal incontinence as well as tonic muscle contractions during the seizures. Myasthenia gravis typically presents as progressive weakening of muscles during exercise. Episodic syncope is usually associated with cardiovascular and/or respiratory abnormalities.

The behaviour of the dog, laboratory findings and the response of the patient to treatment, support the hypothesis of altered activity/release of neurotransmitter substances in the central nervous system of patients suffering from narcolepsy.

ACKNOWLEDGEMENT

Prof J Hatting of the SSERTS Laboratory, University of the Witwatersrand, is thanked for the catecholamine assays.

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Book Review/Boekresensie

GAME RANCH MANAGEMENT

J DU P BOTHMA (Editor)

J L van Schaik (Pty)Ltd, Pretoria 1989 pp 672, 27 tables, 136 figures and 8 colour illustrations. Price: R59-50 (ISBN 0 627 01589 1)

This is an English translation of the popular Afrikaans publication **Wildplaasbestuur,** which was reviewed on page 155 of volume 58 of this journal. The editor used this opportunity to rectify some errors which appeared in the original publication; in table 12 (p 190) the toxin of vermeerbos *Geigeria* sp, previously stated to be unknown, is given as sesquiterpene lactones, while a questionable treatment procedure for capture myopathy has been deleted. The index has been vastly expanded, doing justice to the wealth of information in the book.

References are not quoted, but listed at the end of chapters which hampers verification of stated facts. Some errors have slipped through, which is probably inevitable in a book of these dimensions. The primary food of the grey rhebok is said to be grass (table 5, p 113); although this antelope species often frequents grassland, separate studies in the Natal Drakensberg and south-eastern Orange Free State have shown that forbs make up 80-90% of its diet. In table 8 (p 121) the interfoaling period of Cape mountain zebras is said to be 18 months, while the 2 listed references to this subspecies clearly give means of 22 and 25 months. In the same table the number of mares per stallion in mountain zebra breeding herds is said to be 4, while the average is closer to 2.

With the growing demand for buffalo, Corridor disease has become a focus of attention. On p 182 the brown ear tick is stated to be the vector involved, while *Rhipicephalus zambesiensis,* which is also regarded as an important vector, is omitted. The statement that none of the theilerioses responds to treatment is incorrect. Various drugs are quite effective against *Theileria* species, but registration is not allowed in the RSA as their use could lead to cattle recovering from Corridor disease and thus becoming reservoirs of *Theileria parva lawrencei.*

These are really minor points of criticism. This book is highly recommended, not only to game ranchers and wildlife veterinarians, but to everyone interested in wildlife conservation and utilisation. It should find a ready market not only locally, but also far beyond our shores.

B L PENZHORN

VETERINARY REPRODUCTION AND OBSTETRICS

G H ARTHUR, D E NOAKES and H PEARSON

6th Edn. Bailliere Tindall, London. 1989 pp ix and 641, illustrations 344 and 33 tables, Price not available (ISBN 0-7020-1288-2)

This is the 6th edition of the well-known text first published in 1938 as Veterinary Obstetrics by F. Benesch. The 4th edition appeared in 1975 under the current title with G.H. Arthur as the sole author. In the 5th (1982) and latest edition, the assistance of D.E. Noakes and H. Pearson has been enlisted as co-authors.

The new edition follows the same trend as the previous one, being divided into various sections which in turn consist of 1-12 chapters, each containing its own list of references. Throughout the entire text, new information has been incorporated. An entirely new section dealing with embryo transfer has been added as well as a chapter on reproduction in the buffalo and an appendix containing a list of hormones and vaccines available in the UK (many of which are also available in South Africa) for use in the control of reproduction. The sections dealing with cats have been totally revised and/or new sections added.

The book is well presented, clear and concise with ample reference lists. Some of the illustrations have been simplified and the line-drawings of postural abnormalities and obstetric manipulations remain good. Personally I find the quality of reproduction of some photographs relatively poor in comparison to those in the previous edition.

The various sections are devoted to:-

1. Normal reproductive functions, with a single chapter on the oestrus cycle and its control in all domestic species. Noteworthy is the inclusion of new knowledge regarding endocrine control of reproduction and the use of eCG (equine chorionic gonadotropin) rather than PMSG (pregnant mare serum gonadotropin).
2. Pregnancy and parturition, consisting of chapters on embryonal and foetal development, pregnancy diagnosis including sonar scanning, teratology, vaginal prolapse, parturition and puerperium and care of the newborn.
3. Dystocia and other disorders associated with parturition with chapters on general principles, the approach to obstetric cases, treatment of maternal dystochia, aetiology and incidence of foetal dystocia, obstetric manipulation, foetal oversize, postural defects, defects of position and presentation, twins and monstrosities, parturient injuries and diseases, retained foetal membranes and uterine prolapse.
4. Operative interventions which are restricted to caesarian operation and genital surgery in dogs and cats.
5. Infertility with chapters on infertility in cows, small stock, mares, pigs, dogs and cats, as well as on the veterinary control of herd fertility.
6. The male animal, consisting of chapters on the normal sexual apparatus, reproductive abnormalities and artificial insemination.
7. Exotic species with chapters on reproduction in camels and domestic buffalo.
8. Embryo transfer in large domestic animals including cattle, small stock, pigs and horses. Reference is also made to cryopreservation and manipulation of embryos.

This book is primarily aimed at veterinary students and "practising veterinarians who wish to keep abreast of continuing professional development". As such it is highly recommended as an adjunct to other available texts.

H M Terblanche

0038-2809 Tydskr. S. Afr. vet. Ver. (1989)60(3): 153-154

LYME DISEASE - A NEW DISEASE IN SOUTHERN AFRICA?

B H FIVAZ* and T N PETNEY**

ABSTRACT:
Lyme disease is a recently-described zoonotic disease occurring widely in the U.S.A., Europe and Asia. The causative organism, *Borrelia burgdorferi*, is transmitted predominantly by ticks of the genus *Ixodes* and infects a wide host range.
The infection in humans causes the human disease syndrome erythema chronicum migrans resulting in arthritis, neurological symptoms and/or cardiac abnormalities. Similar clinical signs have been described in domestic animals.
The status of Lyme disease in southern Africa is presently unknown but preliminary evidence indicates that the disease may occur in humans in the Republic of South Africa. The abundance of hosts and tick vectors would favour the establishment of the infection in Africa.

Key words: Erythema chronicum migrans, *Borrelia burgdorferi*, domestic animals, humans, antibiotic therapy, tick vector

Fivaz B.H.; Petney T.N. Lyme disease - a new disease in southern Africa? *Journal of the South African Veterinary Association.* (1989) 60 No. 3, 155-158 (En.) Tick Research Unit, Department of Zoology and Entomology, Rhodes University, P.O. Box 94, 6140 Grahamstown, Republic of South Africa.

INTRODUCTION

Lyme disease, named after a community in Connecticut, U.S.A., also known as Lyme borreliosis or erythema chronicum migrans (ECM) was originally regarded as a specific human disease characterised by the appearance of an expanding annular erythematous lesion originating from a tick bite. Joint and neurological symptoms may occur during the chronic phase. However, the discovery of the spirochete *Borrelia burgdorferi*, as the aetiological agent has led to renewed interest in a disease which was recognised in 1909[15].

Current serological and clinical evidence indicates that Lyme disease has a worldwide distribution in humans, domestic and wild mammals and birds[10 11 13 14 22 39]. The recent serological evidence that Lyme borreliosis may occur in humans in the Republic of South Africa (P.L. Botha 1988. Department of Microbiology, Faculty of Medicine, University of the Orange Free State, R.S.A.)[52], raises the possibility that this disease may be a potential problem in southern Africa and may assume medical and veterinary significance in future.

AETIOLOGY

Borrelia burgdorferi is a motile, gram negative spirochete which stains with Giemsa. The spiral organism ranges in length from 4 to 30 μm and in diameter from 0,18 to 0,25 μm[10]. The structure and biochemical characteristics of *B. burgdorferi* are similar to the other members of the genus[8]. Serological studies have indicated that antigenic surface proteins of organisms from various geographical locations show a degree of antigenic heterogeneity[6]. This antigenic variation may explain why subclinical infections occur despite humoral host responses[14]. Antigenic heterogeneity may influence the sensitivity and specificity of serological assays currently used in epidemiological studies[28].

The organism is readily passaged in hamsters and cultured in Barbour-Stoenner-Kelly medium at 30°C[7].

VECTORS

No information is available on vectors in South Africa. Ticks of the genus *Ixodes* are the dominant vectors in Europe, (*I. ricinus*[1]) in the United States and Asia, (*I. dammini, I. pacifus* and *I. scapularis*)[11 14].

All 3 American vectors belong to the *I. ricinus* complex of ticks[11]. Although these ticks are considered to be the dominant vectors in their respective areas, *B. burgdorferi* has been found in members of the genera *Amblyomma, Haemaphysalis* and *Dermacentor* in the United States[36 49].

The infection rate of ticks is variable. It has been reported that 60-70% of adult *I. dammini* contained *B. burgdorferi*[10], although lower percentages seem more common[14]. The variation in infection rates appears to be related to geographical variability and to the tick species[14].

Insects have also been found to be infected with *B. burgdorferi*. These include fleas and batflies[2], deer flies, horse flies and mosquitos[38]. The significance of these insects in the epidemiology of infection is currently unknown.

VECTOR-ORGANISM INTERACTION

B. burgdorferi is most commonly found in the digestive tract of its tick vector[14]. It has also been found in the haemolymph[12] and saliva[47].

Once a tick has been infected with *B. burgdorferi* both transovarial and transstadial transmission occur and thus any life history stage of the tick may be infective[35]. There is no evidence that *B. burgdorferi* is pathogenic for the vector.

HOSTS

B. burgdorferi has been isolated from a relatively wide variety of avian and mammalian hosts[2 3 4]. Avian dispersal may explain the cosmopolitan distribution of *B. burgdorferi*[4]. Nevertheless, small mammals, especially rodents, are considered to be the dominant hosts[1 2 4 36] of which the white-footed mouse, (*Peromyscus leucopus*) plays a major role in the United States[2 3 36 37].

Large ruminants are also known to host *B. burgdorferi* infections[34] and are common hosts for the adult stages of the tick vectors[40 52 56]. In a study conducted in northern California, 27% of native Black-tailed deer (*Odocoileus hemionus*) were infected while 50% and 56% respectively of introduced Axis (*Cervus axis*) and Fallow deer (*Cervus dama*) were infected[15]. Anderson and his colleagues were able to show that neither white-footed mice nor meadow voles (*Microtus pensylvanicus*) were infected by *B. burgdorferi* on islands not inhabited by white-tailed deer (*Odocoileus virginianus*) while on islands on which the deer occurred, 35/51 rodents were infected[5]. *Ixodes dammini* were also absent on islands without deer. These findings emphasised the important role of white-tailed deer as hosts of adult *I. dammini* and as potential reservoirs of *B. burgdorferi*[34].

TRANSMISSION

The initial observation that infection of the tick vector was predominantly limited to the mid-gut, suggested that transmission took place by regurgitation of infected gut contents during feeding or by deposition on the skin of tick faeces contaminated with *B. burgdorferi*[27]. However, the spread of infection to the salivary glands which occurs during tick feeding, suggests that the salivary route is more likely[47]. Chance of transmission by the vector is dependent on the duration of tick attachment with maximum transmission occurring after about 72 h[46]. This corresponds with the time needed for the *B. burgdorferi* to become detectable in the saliva[47]. The likelihood of contracting an infection appears to depend on the seasonal activity patterns of the tick vectors[46].

Contact infection is also possible. When uninfected White-footed mice and Deer mice were housed with infected individuals of the same species, the infection was transferred[16]. Such infection may

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THE ROLE OF ANAEROBIC BACTERIA IN BOVINE MASTITIS: A REVIEW

J H DU PREEZ*

ABSTRACT:
Routine bacteriological diagnosis of bovine mastitis does not provide an index to the obligate anaerobic flora involved. No anaerobic bacteria were recovered from cows with normal quarters or those with latent facultative anaerobic or aerobic udder infections as diagnosed according to the criteria of the International Dairy Federation. Simultaneous isolation of anaerobic bacteria from udder quarter abscesses and mastitic milk from the same quarter occur in some lactating dairy cows. In most dairy herds there are cows with anaerobic udder infections. Anaerobic bacteria have been isolated from lactating as well as from dry cows.

Most anaerobic bacteria were isolated concurrently with facultative anaerobic bacteria except in aseptic mastitis cases. The polimicrobial nature of udder infections shows that multiple anaerobic as well as facultative anaerobic species colonise and act together. In spite of antibiotic therapy, anaerobic bacteria were still isolated from mastitic quarters. Gram-positve anaerobic bacteria were mostly sensitive to pencillin-G but most of the Gram-negative anaerobic bacteria were resistant and some *Bacteroides fragilis* strains produced betalactamase. Nearly all the aminoglycosides display consistently poor activity against anaerobic bacteria. A high degree of resistance against tetracyclines was also demonstrated. Anaerobic Gram-positive cocci and rods were sensitive to common antibiotics. The pathogenicity of several pure cultures of anaerobic bacteria has been demonstrated by their ability to induce clinical mastitis in healthy lactating udders. Anaerobic bacteria may act as mastitis pathogens probably in a primary and/or secondary role because clinical mastitis could be induced in normal quarters under experimental conditions. Their high prevalence of anaerobic bacteria in mastitic quarters and the ability of some strains to produce heparinase also suggests a pathogenic role. The handling of udder abscesses and the therapy of mastitis where anaerobic bacteria may be involved, is fully discussed.

Key words: Anaerobic bacteria, bovine mastitis, mastitis therapy.

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INTRODUCTION

International literature on bovine mastitis refers almost exclusively to the disease in association with aerobic micro-organisms; it shows in principle a predominance of only 3 main states of udder health, namely (i) mortality, (ii) non-specific secretory disturbances (aseptic mastitis) and (iii)mastitis related to aerobic as well as facultative anaerobic bacteria. This has led to a situation where mastitis caused by anaerobic bacteria apparently is considered an oddity rather than a problem, possibly more prevalent than generally expected under present circumstances.

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The main reasons for the limited work conducted in the past on anaerobic mastitis pathogenic bacteria have presumably been difficult culturing conditions. However, recent advances in laboratory techniques have alleviated such methodological limitations. Culturing of anaerobic bacteria has become practical. The application of such modern techniques to milk samples has provided insight into previously undisclosed aspects of bovine udder health. More extensive further research could well show that the present preoccupation with aerobic mastitis pathogenic bacteria may be too subjective and not necessarily conducive to the correct understanding of epidemiological, prophylactic and therapeutic aspects of sub-clinical and clinical mastitis during lactation, non-lactation and, particularly, their transitional periods at the beginning and end of the lactation cycle.

In the light of the aforediscussed and personal research on anaerobic mastitis pathogenic bacteria, it seems desirable to put into perspective present data relevant to udder health problems with anaerobic bacteria. This review therefore has the aim to compile and collate applicable data, focus attention on anaerobic bacteria in bovine udder health, identify problems requiring particular attention and propose necessary improvements.

Whether this aim will be attained, depends ultimately on concise understanding of the general aetiology of bovine mastitis. It therefore should be noted that mastitis develops when micro-organisms challenge the udder, succeed in invading it at times of lowered resistance and/or increased susceptibility and commence under further adverse conditions affecting the cow, to infect and damage the udder tissue at levels which provoke an inflammatory reaction. Concerning the development of mastitis related to anaerobic bacteria, it seems desirable to assess certain elements of the above-mentioned epidemiological chain of events involved in the development of mastitis. Aspects of particular interest are: growth requirements and cultural isolation of anaerobic bacteria, their presence in humans and the environment, possible routes of intramammary challenge and invasion, mammary defence and factors predisposing to mastitis, evidence on natural udder infections with anaerobic bacteria, their prevalence in milk samples, certain types of natural mastitis, and pathology of artificially-induced mastitis.

Growth requirements of anaerobes

Oxygen sensitivity of bacteria

"The world arose anaerobically, and much of it still remains so in terms of the numbers of living things rather than their size"[35].

Billions of years ago our planet was totally anaerobic. Live organisms originated under anaerobic conditions whilst free atmospheric oxygen, as we know it today, did not exist.

All molecular oxygen was bound to water, carbon-dioxide, carbonates and sulphates. Man has an inescapable and absolute need for free oxygen and this possibly accounts for the tendency to overlook the importance of the anaerobic world which forms part of our ecology[35].

The composition of atmospheric air is as follows:[45] Oxygen (O_2) 20,95%; Carbon dioxide (CO_2) 0,03%; Nitrogen (N_2) 78,08%; Other gasses 0,93%.

Obligate anaerobic bacteria are anoxybiontic, anaerophylic organisms which have the capacity to generate energy

and to synthesise their nutrients without recourse to molecular oxygen and are particularly sensitive to oxygen, which makes it impossible for them to grow in air at one atmosphere[49].

Several reasons are postulated, viz:

- the inability to produce catalase as this enzyme breaks down the hydrogen peroxide which is formed when the organisms are exposed to oxygen;
- that oxygen bound to chemical components in the medium is inhibitory for bacterial growth. This appears to be particularly true for some species of clostridia and for many of the more oxygen-sensitive members of the normal flora;
- that a superoxide free radical (a biologically produced intermediate in the bacteria resulting from the univalent reduction of molecular oxygen) is more toxic to organisms than is hydrogen peroxide. Strict anaerobes do not produce superoxide dismutase, an enzyme which acts to form hydrogen peroxide and oxygen from two superoxide radicals and hydrogen. In this view, neither oxygen nor peroxide is a danger to the organisms as is the free radical superoxide[35].

In the past, oxygen sensitivity of anaerobic organisms was the main reason to place their role in infectious conditions into perspective. The culturing of anaerobic bacteria has given a new dimension to the field of infectious diseases[35].

Special techniques are required for sampling and isolation of anaerobic bacteria. Among different anaerobic species, varying degrees of oxygen and peroxide tolerance occur as well as different requirements for oxidation-reduction potential (Eh) which influences and determines the growth of anaerobic bacteria[17]. A scale of "uncertainty" exists as to where bacteria may be cultured with difficulty or with ease. Facultative anaerobic bacteria have the ability to grow in the presence or absence of molecular oxygen[35]. Micro-aerophilic bacteria are classified under obligate anaerobic bacteria and require a reduced oxygen tension (below 0,2 atm of air) for their growth needs, although they grow on solid media with 10% carbon dioxide. A number of the bacteria are capnophilic rather than micro-aerophilic, which means that they require an increased carbon dioxide tension rather than a reduced oxygen tension for growth[17 39 68]. *Clostridium perfringens* is relatively oxygen-tolerant and grows in most so-called "anaerobic" systems. Other anaerobic bacteria such as *Eubacterium* are more demanding and have to be cultured in the absence of oxygen[68].

DISCREPANCIES OF ROUTINE MASTITIS DIAGNOSIS

The routine laboratory methods of collecting milk samples and examining them bacteriologically for the diagnosis of bovine mastitis do not provide for the isolation of obligate anaerobic flora. Anaerobic bacteria are frequently overlooked as a cause of bacterial infection when they are present in mixed cultures. The culturing of facultative anaerobic organisms from a mixed sample, especially if they show the same morphology and reaction to the Gram-staining method as anaerobic bacteria (eg. Gram-negative rods), may create the false impression that no anaerobic bacteria are present. If no aerobic or facultative anaerobic bacteria can be cultured, but can nevertheless be demonstrated microscopically by means of Gram-staining, there is good reason to suspect that anaerobic bacteria are present in the material.

ANAEROBIC SAMPLING TECHNIQUES

Because the normal routine foremilk sampling of dairy cows for the determination of the health status of the bovine quarters/udders according to the bacterio-cytological criteria of the International Dairy Federation[34 73] does not make provision or take into account the possibility of existing anaerobic mastitogenic bacteria, a proved anaerobic sampling routine and isolation procedure will be described.

After thorough udder washing with clean running water, drying with a disposable paper towel, disinfecting the teat tip with 70% alcohol and cotton wool and discarding the initial three jets of foremilk, a quarter milk sample must be aseptically and anaerobically collected via the teat canal from the teat and/or gland cistern of the udder. This is accomplished by means of a 150mm x 1,0mm catheter attached to a 10ml disposable syringe. The syringe and catheter must have been preflushed with oxygen-free CO_2 to remove atmospheric oxygen from the system.

Pus from udder abscesses were aspirated under similar conditions by means of a 19-gauge needle and 10ml disposable syringe after disinfection of the abscess skin area with 70% alcohol and cotton wool. Milk or pus samples must immediately be injected into 100 x 30mm vaccine type bottles, equipped with crimped butyl rubber sealers and containing only an atmosphere of oxygen-free CO_2. Samples must be transported on ice, or at 4°C, and analysis must be initiated within 6 hours[12 13 28].

ISOLATION PROCEDURES: OBLIGATE ANAEROBIC BACTERIA

Obligate anaerobic bacteria can be isolated on supplemented brain heart (BH) agar (Difco Laboratories, Detroit, USA) by streaking a loopful of the sample onto rotating rolltubes under an atmosphere of oxygen-free CO_2[31]. All media used for the propagation of obligate anaerobes must be pre-reduced with oxygen-free CO_2 and anaerobically sterilised (PRAS) according to the methods of Holdeman et. al[31]. The rolltubes must be sealed anaerobically and incubated for up to 7 d at 37°C. Sub-culturing and isolation of single colonies must be accomplished on rolltubes or PRAS blood agar plates, using standard anaerobic jar methods. The analysis of acids and alcohol products for generic identification must be accomplished by gas chromatography. Ether extracts and methyl derivatives from culture products grown in both chopped meat carbohydrates (CMC) medium (Difco Laboratories, Detroit, USA.) and peptone yeast extract glucose (PYG) broth* must be prepared according to Holdeman, et. al.[31]. Analysis can be performed on a Pye Unicam model 242 gaschromatograph equipped with 200 cm 0,6 cm glass columns. The columns must be packed with 5% free fatty acid phase Carbowax 20M on 80/100 mesh chromosorb-G and operated for thermal conductivity at 150°C. Helium must be used as carrier gas at a flow speed of 120 ml/min. Speciation of anaerobes can be done according to Holderman et al.[31] and Sutter et al.[71].

Isolation procedures: Facultative anaerobic and micro-aerophylic bacteria which may occur together with anaerobic bacteria.

Facultative anaerobic and micro-aerophylic bacteria can be isolated by streaking a loopful of the same sample as mentioned above on each of 2 blood agar plates. Both plates must be incubated for 48 h at 37°C, one anaerobically and the other under micro-aerophylic conditions. Characterisation of species can be done according to the method of Carter[4] and Cowan & Steel[6].

ANAEROBIC BACTERIA IN HUMANS AND ENVIRONMENT

Human disease

Recent developments and improvements in the techniques for isolating strict obligate anaerobic bacteria have made it possible to determine their importance as infectious agents[1 17]. In more than 60% of purulent infections in humans, either pure cultures of anaerobic bacteria or anaerobic bacteria together with facultative anaerobic bacteria are isolated[49].

Natural occurrence

Anaerobic bacteria occur in soil as saprophytes and form the bulk of the commensal flora of man and animal as well as of certain insects[6 35]. In the human, anaerobic bacteria are found on the mucous membranes, especially those of the oral cavity and lower sections of the digestive tract. In the oral cavity, the number of anaerobic bacteria is approximately the same as the facultative anaerobic bacteria[35].

The ratio of anaerobic bacteria to facultative anaerobic bacteria increases in the distal parts of the digestive tract, amounting to ca. 1000:1 in the colon. The concentration of anaerobic bacteria in faeces varies from 2 to 4 hundred million organisms per gram dry faecal material. Of the wet weight of human faeces, 20%-30% is a solid mass of live bacteria, almost all of which are anaerobic organisms[35].

Anaerobic bacteria are also the predominant organisms inhabiting the human skin and occurring primarily in the fat glands[35]. *Propionibacterium acnes* occurs extensively on the hair and skin of adults as well as in the oropharynx and digestive tract[78]. *Peptococcus asaccharolyticus* forms part of the normal bacterial flora of the nasopharynx as well as the digestive tract and female genital tract[78].

Most anaerobic bacteria involved in pathological processes in the human, also occur as commensals[16]. Holdeman et al.[31] indicate that anaerobic bacteria are the dominant organism in samples of deep tissue infections in humans where they occur as frequently or even more fre-

quently than facultative anaerobic bacteria. The same pattern is observed in abscesses in the human. In intra-abdominal sepsis, anaerobic infections are caused by a mixture of anaerobic spp. of bacteria; aerobic and facultative anaerobic bacteria may also be present[16]. *Bacteriodes fragilis* is by far the most prevalent pathogen in human infections. Contrary to the other anaerobic spp. it is generally resistant to penicillin-G[35].

Hughes[32] refers to the bacterial load of anaerobic bacteria in the digestive tract of animals. The dominant anaerobic bacteria occurring in the digestive tract are lactobacilli and bacteroides. *Propionibacterium* inhabits inter alia the human body and that of certain animals and also occurs in dairy products[43]. *Bacteroides* is a strict anaerobic bacterium and many species are obligate parasites of humans and animals and do not normally occur outside the body[78]. *Fusobacterium necrophorum* inhabits the digestive tract of animals[68] and it is also a recognised pathogen of the human and a variety of animals[3 63]. Faecal contamination of the environment implies that many anaerobic bacteria may be present. *C. perfringens* occurs widely in nature: in soil, water and offal as well as in the digestive tract of humans and animals. The natural habitat of *Clostridium botulinum* is the soil. *Clostridium tetani* occurs especially in cultivated soil[78].

Dairy environment

The above-mentioned particulars serve as an indication of the numbers of bacteria to which the dairy cow's udder may be exposed, particularly if one considers the unhygienic conditions under which milk-producing herds are kept during the rainy season. Giesecke & Van den Heever[20] pointed to the economic losses in the Republic of South Africa as a result of mastisis. This investigation was based on the assumption that the septic forms of mastitis are responsible for milk losses as a result of aerobic and facultative anaerobic bacteria.

Giesecke & Van den Heever[21] and Giesecke et al.[23] revealed that teat canal infections (TCI) occur much more generally than is assumed. TCI may lead to udder infections but the prevalence of mastitis is reduced markedly if TCI is controlled by regular teat dipping[55 56]. *Staphylococcus aureus* colonises in the teat canal[10] and in the parenchyma of the udder[60]. Since many aerobic and facultative anaerobic bacteria are reductive[35] they can pave the way for possible anaerobic bacterial colonisation in the teat canal.

Du Preez & Van den Heever[11] showed that therapy of teat canals colonised by staphylococci or streptococci with small amounts of antibiotics is fairly successful in eliminating the bacteria concerned. One could therefore expect that teat canal therapy could also readily eliminate any bacteria which may be present.

Various forms of mastitis occur[34 73]. A distinction should therefore be made particularly between septic and aseptic mastitis. The latter is generally assumed to be the situation where either symptoms or clinical and subclinical mastitis are present, but where attempt to culture the causative organism from the secretion, produce negative results.

ANAEROBIC BACTERIA IN DAIRY COWS AND RELATED INFORMATION

Routes of udder infection

Giesecke[22] described the routes by which pathogenic bacteria reach the udder parenchyma, namely via the teat canal (galactogenous being the most important route), the blood supply (haematogenous) and via open lesions on the skin of the udder and teats (traumatogenous). According to Schalm[60] and Schalm et al.[61] the most common mode of pathogenesis of bovine mastitis is the penetration of pathogenic bacteria through the teat canal into the gland cistern of the udder if the internal environment of the gland is favourable for multiplication of the bacteria, then metabolites are produced which damage the udder tissue and provoke an inflammatory reaction. Heidrich & Renk[29] described the route by which organisms invade the udder to initiate spontaneous mastitis as follows: The responsible organisms reach the udder parenchyma through the teat canal, the teat cistern, gland cistern and mammary ducts (galactogenous infection) or via defects in the skin of the teat or udder (lesion infection) or via the bloodstream of an infection focus situated elsewhere in the body (haematogenous infection). The postulates by all the above-mentioned authors regarding the routes by which organisms invade the udder and the pathogenesis of udder infections and mastitis relate to the aerobic, facultative anaerobic and micro-aerophilic bacteria. At the present stage there is little clarity and no description regarding the precise pathogenesis of cow mastitis in which anaerobic bacteria are involved. By means of induction it was proved experimentally that certain anaerobes are virulent[13 14]; the precise pathogenic effect for mastitis still needs to be researched.

Anaerobic bacteria in the human

The anaerobic bacterial flora of the human are harmless in situ but become virulent when they override the mucosal barrier[35]. Normally the microflora will not harm the host unless preceding pathology creates an opportunity, although interaction between the host and microflora continue throughout the lifetime[35]. Skin and soft-tissue infections with anaerobic bacteria may develop in damaged tissue such as post-surgical sites, wounds and following ischaemia of the extremities. The probable causative organism is determined by the specific local tissue circumstances. For instance, infections of the lower half of the human body are generally caused by contaminative faecal flora such as *B. fragilis* and *Peptostreptococcus* spp. *S aureus* and *Peptostreptococcus* spp. work synergistically to cause necrosis and ulceration of the skin and subcutaneous tissue[17]. Brain abscesses may follow anaerobic bacterial pulmonary infection. Two to 10% of cases of bacteraemia as a result of anaerobic bacteria were reported[17]. The non-sporeforming Gram-negative rods (also referred to as "Bacteroides") are the anaerobic bacteria generally isolated from blood cultures[17]. Anaerobic bacteria are involved in a wide variety of infections of the female genital system[17]. Most organisms found in anaerobic infections in the human are of endogenous origin, i.e. they form part of the normal bacterial flora of the infected host which have overcome the normal mucomembranous barrier in some way or another[35].

ANAEROBIC BACTERIA

Peptococcus indolicus is a Gram-positive anaerobic micrococcus which is isolated regularly with *Corynebacterium pyogenes* in cases of summer mastitis[66 70 75]. Sorensen[67] was also able to isolate *P. indolicus* and *C. pyogenes* from apparently healthy cow udders. Stuart et al[70] and Sorensen[65] proved experimentally that *P. indolicus* in a mixed culture with *C. pyogenes* can cause mastitis by means of induction in healthy non-lactating heifers. Recently Shinjo et al[62] reported that they were able to culture obligate anaerobic bacteria *Peptococcaceae*, *Bacteroides* spp. and *F. necrophorum* in mastitis outbreaks and from the healthy udders of non-lactating heifers. Heidrich & Renk[29] were able to isolate *C. welchii* from cows with gangrenous mastitis, and *F. necrophorum* from sporadic cases of cows with necrotising mastitis. Giesecke et al.[23] reported on bovine mastitis caused by *C. perfringens* type A and *Bacillus cereus*.

Seen against the fact that both clinical and subclinical mastitis are frequently regarded as aseptic since routine diagnostic procedures fail to demonstrate any bacterial cause, and in view of the fact that special and refined bacteriological procedures have succeeded in establishing an entirely different concept with regard to the role of strict obligate anaerobic bacteria in tissue infections of humans and animals, it is probable that anaerobic bacteria could also be responsible for the initiation of mastitis -either as a secondary cause or in conjunction with primary udder pathogenic organisms (micro-aerophylic or facultative anaerobic bacteria).

Du Preez et al.[12] investigated the role of obligate anaerobic bacteria in the aetiology of mastitis of lactating dairy cows. They found that anaerobes were isolated from 12% of lactating mastitic cows, which was representative of 50% of the 10 dairy herds examined (Table 2). *B. fragilis* was the most frequently isolated organism (50%), followed by *Peptococcus indolicus* (33%), *Eubacterium lentum* (33%), *E. aerofaciens* (17%), *Propionibacterium granulosum* (17%) and an anaerobic *Streptococcus* sp. (17%). These obligate anaerobes were always isolated together with organisms primarily involved in mastitis. All *B. fragilis* strains were resistant to penicillin-G and tetracycline. In addition, one strain was also resistant to ampicillin, cephalothin and amoxicillin. Anaerobic Gram-positive cocci and bacilli were sensitive to most antibiotics. These finding imply an important role for anaerobes in the aetiology of mastitis.

Du Preez[13] reported on the role of obligate anaerobic bacteria in the aetiology of udder infection in 101 lactating and one dry cow in 16 herds. Anaerobic and aerobic quarter milk specimens were taken aseptically from healthy quarters and from those with septic clinical mastitis (CM), septic subclinical mastitis (SCM) and "aseptic" mastitis (ASM) for the purpose of isolating

anaerobic and facultative anaerobic bacteria. Pus from udder abscesses of 4 lactating and one dry cow was also examined.

Fourteen anaerobic species were isolated from foremilk specimens from 13,9% of the lactating cows, representing 9,0% of the quarters which showed CM, SCM, and ASM. Anaerobic bacteria were isolated from 10 (62,5%) of the 16 herds investigated. No anaerobic bacteria were isolated from quarter milk samples of cows with either healthy udders or TCH.

Du Preez[13] isolated the following anaerobic bacteria from the udders of lactating cows with CM, SCM and ASM: *Peptococcus indolicus* (37,5%); *Bacteroides fragilis* (12,5%); *Eubacterium combesii* (12,5%); *E. lentum* (8,3%); *Fusobacterium necrophorum* (8,3%); **E.** *aerofaciens* (4,2%); *Propionibacterium granulosum* (4,2%); *P. acnes* (4,2%) and an anaerobic *Streptococcus* sp. 4,2%. Two anaerobes *P. indolicus* and *C. sporogenes* plus *Corynebacterium pyogenes* were cultured from a dry cow with mastitis. These obligate anaerobes, with one exception (that of ASM), were always associated with primary facultative anaerobic bacteria isolated from mastitic bovine milk. Although no attempt is usually made to culture anaerobes in the routine bacteriological examination of foremilk for mastitis, Du Preez[13] cultured in one case only the anaerobes *P. indolicus*, which was apparently responsible for the so-called ASM. Du Preez[13] also isolated both anaerobic and facultative anaerobic bacteria from udder abscesses. The following anaerobic bacteria were isolated *B. fragilis; B. eggerthii; E. combessi; P. indolicus; P. acnes* and *B. fragilis; E. aerofaciens; E. lentum* and *P. indolicus* were recovered from abscesses at the base of the teats. Pure cultures of *B. fragilis, P. indolicus, E. lentum, E. aerofaciens, P. granulosum,* and an anaerobic *Streptococcus* spp., introduced via the teat canal of lactating quarters, caused a clinical mastitis within 24-48 h and the corresponding bacteria were recovered[12 13].

In spite of antibiotic treatment, Du Preez[12 13] isolated bacteria from the milk of cows with mastitis and udder abscesses, and also from the pus of one cow with an udder escutcheon abscess. The pus from all the abscesses, except one which was situated at the base of the teat, had a characteristic putrid odour. All the *B. fragilis* isolates produced beta-lactamase[50] and were resistant to penicillin-G and tetracycline, while some strains were resistant to ampicillin, cephalothin and amoxicillin[77]. Anaerobic Gram-positive cocci and rods were sensitive to the common antibiotics.

The relation of anaerobic bacteria in milk from quarters with CM to healthy quarters and ASM to healthy quarters, differs statistically on a 5% level of significance[13]. The prevalence of facultative anaerobic bacteria in SCM, CM and ASM in relation to anaerobic bacteria in milk from quarters with the same status, mostly differ statistically[18 70] on a 0,1% or 5% level of significance[13].

The gross and microscopic pathology of the udders of lactating cows after experimental infection with pure cultures of *P. granulosum, P. indolicus, E. aerofaciens* and *E. lentum* were studied by Du Preez et al.[14]. They presented evidence that these asporogenous obligate anaerobic bacteria are capable pathogens of the bovine udder.

Greeff, et al[27] reported on the prevalence of strictly anaerobic bacteria in the secretions from untreated cases of mastitis in lactating dairy cows (Table 1). The study involved 147 Friesland cows in 12 highveld herds. *Peptococcus* spp. was associated with *C. pyogenes* and *Bacteroides* spp. with *S. areus* in 10% anaerobic udder infections. Gram-positive anaerobic bacterial species were mostly sensitive to penicillin-G but all the Gram-negative anaerobic bacteria were resistant. In addition, all *B. fragilis* strains produced beta-lactamase. The ability to produce heparinase was demonstrated in one strain of *P. indolicus* and a *Peptostreptococcus* sp. isolated from mastitis quarters. The polymicrobial nature of udder infections however often involves multiple anaerobic as well as facultative anaerobic bacteria together.

More recently du Preez et al[15] reported on the effect on lincomycin-neomycin treatment of experimental anaerobic bacterial bovine mastitis. They experimentally infected 3 healthy lactating quarters of a Friesland cow with a pure culture of a strain of either *B. fragilis, E. lentum* or a *Peptostreptococcus* spp. respectively. The onset and progression of the condition to clinical mastitis was monitored for 12 h by examination for clinical signs of inflammation, bacterial culture, somatic cell counts and with a strip cup. All infected quarters developed clinical mastitis within 24 h after installation of the bacteria in the udder cistern via the teat canal. The quarters infected with *B. fragilis* and *E. lentum* respectively were treated 4 times consecutively at 12 h intervals, commencing at 24 h after the onset of clinical mastitis by intramammary installation of 10ml of a mixture containing 200mg lincomycin hydrochloride, 200mg neomycin sulphate and 5mg methylprednisolone (Lincocin Forte, Upjohn).

Both quarters became clinically normal and no bacteria could be detected in the secretions 12 h after the first treatment. After 36 h the strip cup became negative. The somatic cell count dropped to 500 x 10^3, 72 h after the initial treatment. The quarter infected with a *Peptostreptococcus* sp. was unable to overcome the infection by means of its natural defence mechanism when intramammary treatment was delayed for the first 36 h after the onset of clinical mastitis. Subsequent treatment of this quarter gave results similar to those in quarters treated earlier.

Recently Greeff & du Preez[28] isolated a variety of non-sporulating anaerobic bacterial species from abscesses in 10 lactating dairy cows. Fifty percent of the abscesses yielded multiple anaerobic bacterial species. The anaerobic bacteria, however, were always accompanied by classical facultative anaerobic mastitogenic bacteria. In 4 of the 5 cows afflicted with mastitis, anaerobic and facultative anaerobic bacteria in the quarters with abscesses and in the mastitic quarters themselves were identical. *P. indolicus* was the most commonly isolated organism followed by *Eubacterium* and *Bacteroides* spp. *B. fragilis* was resistant to penicillin, ampicillin and tetracycline.

Defence mechanisms

Mastitis milk shows an increased bacteriostatic to bacteriocidal activity compared with milk from healthy udders[37]. This characteristic is mainly due to phagocytosing cells and specific antibodies emanating from the bloodstream or which are synthetised locally in the udder itself. Other endogenic antimicrobial systems in milk are non-cellular, non-antibody factors such as xanthine oxidase, lactoferrin, lysozome and the lactoperoxidase-thiocyanate-hydrogen peroxiae (LP/SCN/H_2O_2) system. The LP/SCN/H_2O_2 system plays a potential role in resistance of the udder to mastitis[37].

Factors predisposing to infections

Greeff[25] points to the pathogenic and predisposing factors which may contribute to the establishment of anaerobic infections. All predisposing factors are to a greater or lesser extent involved in reducing the Eh of the tissue[25]. Predisposing factors which are of cardinal importance and which may play a role in the pathogenesis of cow mastitis caused by anaerobic bacteria as well as udder abscesses are:

Blood supply:

- Arterial bovine blood contains approximately 200 mℓ O_2 per litre of blood and the venous blood 150 mℓ O_2 per litre of blood. These levels of oxygen are much higher than the minimum of 0,1% oxygen, which is still toxic for obligate anaerobic bacteria[25].
- The collateral arterial blood supply of the cow udder supplies approximately 7ℓ to 10ℓ blood per min. This provides oxygen and nutrients to the ca. 2 400 000 000 alveoli of udders producing ca. 20 kg milk per d[22]. Youmans et al[79] point out that any obstruction of capillary blood flow leads to the reduced Eh, which predisposes to anaerobic bacterial infection.
- Severe disturbances in the blood supply of the udder are necessary to create a favourable redox potential for the growth of anaerobic bacteria. Tissue damage in the udder causes a disturbance in the Eh of normal udder tissue. One of the body's main defence mechanisms against anaerobic infections is its normal Eh of ca. + 120 mV.[25 31]. Under anaerobic conditions, the body's granulocyte-phagocyte-bacteriocidal system does not function effectively[17].
- Tustin[79] indicated the result of necrosis and tissue destruction in peracute mastitis. Schalm et al.[22] explained that the alpha-toxin of *S. aureus* causes prolonged vasoconstriction and subsequent ischaemia. Heidrich & Renk[29] discuss various forms of blood flow obstruction in the udder. A significant degree of obstruction of the oxygen-rich arterial blood supply of the cow udder would have to arise in order to reduce the Eh to such an extent that anaerobic bacteria could multiply.
- Oxidation reduction potential (Eh):

Any matter has the tendency to oxidase, thereby, making electrons available for donation. The Eh value is an expression of the oxidation-reduction potential of a system or the

possibility of a system liberating electrons[35]. The oxidation-reduction potential[79] is expressed by a positive or negative electronic potential over a calomel half-cell (Hg-compound) and may be determined in vivo or in vitro. The oxidation-reduction potential in any part of the body is mostly (but not always) related either to the distance from the site where oxygen-carrying red blood cells are situated or to the integrity of the vascular capillary network[79]. Reduction of blood supply to any tissue may lead to local necrosis and/or anaerobic metabolism by the cells of such tissue. This reduces the Eh, thereby bringing about an anaerobic condition[35].

Injuries causing obstructions of the blood supply such as trauma, surgery and ischaemic necrosis lead to a reduced Eh and predispose anaerobic bacterial growth. The Eh of blood depends on the degree of saturation of the haemoglobin in the red blood cells with oxygen and to a lesser extent on the oxygen solubility in the serum. The Eh of most body tissues ranges between +126 Millivolt (mV) and 246 mV, depending on the blood supply and whether the reading has been close to high arterial or low venous oxygen-saturated sites[79]. Secondary infections in which non-sporeforming obligate anaerobic bacteria are involved, arise as a result of predisposing factors related to reduction of the Eh of the tissue, which favours the growth and multiplying of the anaerobic bacteria. Parts of the body which are far removed from the active capillary perfusion such as the lumen of the colon and vagina, normally have large populations of anaerobic bacteria. Other parts of the body where anaerobic bacteria occur in large concentrations are the nasal passage, tonsillary crypts and other parts of the body where they are protected against high oxygen concentrations[79]. The indirectly determined Eh for milk is given as -218 mV during normal lactation to about -250 mV after milking has been suspended for 7 d[42].

Necrosis in the cow udder: Ischaemic necrosis leads to obstruction of the blood supply and a consequent decrease in the Eh of the tissue: this predisposes to anaerobic bacterial growth[79]. The necrotoxins of staphylococci and streptococci as well as the Gram-negative coliform bacteria with endotoxins in their cell-walls[8] may lead to necrosis of tissue. The alpha-haemolysin of *S. aureus*[5] produces vasoconstriction which may lead to ischaemic necrosis. The logical conclusion is that the facultative anaerobic bacteria which more particularly cause coagulative necrosis of the udder in bovine mastitis, predispose to a possible multiplication of anaerobic bacteria. Premature regression[42] may play a role in anaerobic udder infections.

Surgery of the cow udder: Surgical trauma obstructs the blood supply and reduces the Eh of the tissue, thereby predisposing to anaerobic bacterial growth[79]. Keusch & Weinstein[35] point out that post-operative infections are an important consequence of abdominal surgery. Bacteraemia or wound infection, or both, may arise over and above abscessation. An infection figure of 5% to 10% may occur following uterine and vaginal surgery in which *B. fragilis* may be one of the causative organisms[79]. The role anaerobic bacteria may play in udder infections following surgical intervention, cannot be ignored.

- The use of aminoglycocidal antibiotics:
  The aminoglycocide group includes some of the following antibiotics: gentamycin, canamycin, neomycin and streptomycin. Some of these possess a weak degree of activity against anaerobic bacteria[35]. Clinical laboratories make use of the above-mentioned property of aminoglycocides by for instance including canamycin in selective media for culturing anaerobic bacteria[35]. According to Greeff[25] some infections treated with aminoglycocidal antibiotics may indirectly favour anaerobic bacteria by possibly hampering the colonisation of aerobic and micro-aerophilic bacteria.
- Facultative anaerobic and obligate anaerobic bacterial growth in the udder: Many kinds of bacteria, both facultative anaerobic and obligate anaerobic bacteria, reduce the environment in which they grow. The reducing capacity of micro-organisms probably plays a role in their capacity to provoke infection in the tissue and this partly explains why so many anaerobic infections are mixed or polimicrobial[35]. In all the cases where anaerobic bacteria were isolated from the mastitis milk or abscess pus of 18 cows (except in one case of ASM), facultative anaerobic and micro-aerophilic bacteria were also isolated[13].

POSSIBLE PATHOGENIC ROLE OF ANAEROBIC BACTERIA IN BOVINE MASTITIS

The pathogenicity of anaerobic bacteria in bovine mastitis has not been fully established as yet[12 13]. Stuart et al.[70], Sorensen[66] and Weber et al.[75] consistently isolate *P. indolicus*, a Gram-positive anaerobic micrococcus, together with *C. pyogenes* from the udders of cows with summer mastitis. However, Sorensen[67] was also able to isolate the above-mentioned 2 bacteria from apparently healthy cattle. Shinjo et al.[62], on the other hand, simultaneously isolated the classic facultative anaerobic bacteria normally involved in mastitis, as well as obligate anaerobic bacteria (*Peptococcaceae, Bacteroides* spp. and *F. nucleatum*) during outbreaks of mastitis as well as from the healthy udders of non-lactating heifers. Du Preez[13] isolated an anaerobe (*P. indolicus*) from the milk of a case of so-called ASM from an udder quarter without also being able to culture facultative anaerobic and micro-aerophilic bacteria from it. In all the other cases where anaerobic bacteria were isolated from the milk of an udder quarter or uader abscess, the facultative anaerobic bacteria usually associated with bovine mastitis, were also isolated[12].

HISTOPATHOLOGY OF ARTIFICIAL ANAEROBIC MASTITIS

Although it was possible in the study conducted by Du Preez[13] to induce clinical mastitis by means of several pathogenic anaerobic bacteria under experimental conditions, their capacity to act as primary pathogens in nature remains unclear. The cases of mastitis induced with anaerobic bacteria, were acute but with varying degrees of pathological deviation[13 14]. The histopathology of the alveoli of the above-mentioned infected udder quarters parenchyma throughout, shows a lesser or greater degree of neutrophilia which is an indication of an acute purulent mastitis. The presence of eosinophiles, lymphocytes and plasma cells in the udder parenchyma possibly points to a hyper-sensitivity reaction amongst other things.

ASEPTIC MASTITIS AND ANAEROBIC BACTERIA

It is not clear whether anaerobic bacteria are in themselves as a rule, pathogenic to the udder parenchyma[13]. Since the role of the primary mastitis pathogenic bacteria in all forms of mastitis is so predominant (except in the case of so-called ASM where routine facultative anaerobic bacterial culturing methods produced no bacteria) it is to be expected that in most forms of mastitis from which anaerobic bacteria are isolated, primary mastitis pathogens will also be present[13]. Statistical analysis of ASM cases compared with healthy cases, reveals significantly more anaerobes in the milk of quarters with ASM than in healthy quarters[13]. Anaerobic bacteria quite possibly play an important role in ASM whereas in the past it was assumed that no bacteria whatsoever were present, although no specific investigation for anaerobes was conducted. Because the sophisticated techniques required for strict anaerobic bacteria isolation have been lacking until now, the cause of ASM was conveniently ascribed to physical-chemical factors. This now appears not to be necessarily so.

THE ROLE OF *P. INDOLICUS*

The role of *P. indolicus* as a possible cause of bovine mastitis and udder abscessation is important; these anaerobic bacteria occur more frequently than any other in dairy herds, udder quarters with mastitis and udder abscesses[12 13 28]. Sorensen[65] succeeded in inducing typical summer mastitis with a mixed culture of *C. pyogenes* and *P. indolicus* in 2 non-pregnant heifers. Smith & Jones[64] describe summer mastitis as an acute purulent mastitis with copious amounts of pus and abscesses which erupt through the skin and also cause necrosis and flaking away of the udder tissue. In a cow which showed approximately these clinical signs, *C. pyogenes* and *P. indolicus* were isolated together with other anaerobic bacteria[13]. This brings the pathogenic role of anaerobes sharply into focus. The exudate of mastitis caused by *C. pyogenes* and *P. indolicus* was puslike, greenish to chocolate brown in colour and had an unpleasant odour[13]. It is not quite clear whether the anaerobic bacterium *P. indolicus*, played a primary role in the above-mentioned cases. *P. indolicus* occurs widely amongst so-called healthy cattle and is found with

C. pyogenes as a cohabitant[65]. According to Lovell[38] the preceding udder trauma and initial udder infection by certain pathogenic facultative and anaerobic bacteria, play an important role in summer mastitis. The above-mentioned fact confirms that optimal predisposing conditions must prevail in order for *P. indolicus* to play its role in the pathogenesis of mastitis and udder abscessation. *P. indolicus* may be cultured from insects such as *Simulium* and *Culicoides* and *C. pyogenes* from *Hydroteae* and *Simulium*[66]. Whether this was the case in the investigation conducted by Du Preez[13] into the insects harboured in the herd in which *P. indolicus* and *C. pyogenes* were isolated, was not established. Since these insects occur so widely in the areas in which the dairy herds are kept, there is a possibility that this might be the case, but it will still have to be proved by research.

Lack of the necessary sophisticated techniques for the culturing and identification of strict anaerobic bacteria until quite recently was the reason why the role of anaerobes in bovine mastitis was overlooked. As yet, these techniques are not fully utilised in veterinary bacteriological procedures for the culturing and identification of anaerobic bacteria in bovine mastitis[13].

B. FRAGILIS, BETA-LACTAMASE AND *F. NECROPHORUM*

Most *B. fragilis* isolates produce at least small quantities of beta-lactamase and the degree of resistance to benzilpenicillin was found to be directly proportional to the degree of beta-lactamase production[53]. Du Preez[13] indicates that 3 strains of *B. fragilis* produce beta-lactamase. Very few *B. fragilis* strains are resistant to clindamycin[9 33 44].

Certain reports indicate that *B. fragilis* is the most antimicrobic-resistant anaerobic isolate cultured from clinical material[71 72 78]. More than 40% of clinical isolates of *B. fragilis* were found by Kislak[3] and Martin et al.[44] to be resistant to tetracyline. In the investigation conducted by Du Preez[13], 3 of the *B. fragilis* strains were resistant to ampicillin, cephalotine and amoxycillin. It therefore appears to be desirable that where antimicrobial therapy is used or recommended for cases of mastitis, it should also be effective against anaerobic components of the infection. Infection by *C. welchii* and *F. necrophorum* leads to severe udder pathology[62]. What the specific role is of the *F. necrophorum* isolated by Du Preez[13] from udder quarters with SCM, is not very clear but it could play a contributory role in the SCM. *B. fragilis* is isolated regularly from purulent lesions in the human[17] and this was the organism which was second most prevalent in the [14]anaerobe-infected lactating cow udders of 10 different dairy herds[13]. *B. fragilis* also shows the widest spectrum of resistance to various antimicrobial agents[72]. This is the only beta-lactamase-producing anaerobic bacteria isolated by Du Preez[13]. The virulence of *B. fragilis* was studied in animal models[51 76]. The capacity of certain strains from abscesses is associated with the presence of a capsular material containing polisaccande[52].

B. fragilis as well as all the other anaerobic bacteria, except *P. indolicus* which was isolated from the case of ASM, were isolated simultaneously with primary mastitis pathogens from udder quarters with different forms of mastitis[13]. No anaerobic bacteria could be demonstrated from the milk of healthy udder quarters of cows[12 13].

Mixed bacterial infections are sometimes found in human lesions from which anaerobic bacteria have been isolated[2 59]. Secondary infections are involved which occur after the effect of favourable predisposing factors which reduce the Eh of the tissue to the level where the multiplication of anaerobic bacteria can take place. Primary infections by aerobic, micro-aerophilic and facultative anaerobic bacteria may lead to a reduced blood supply as a result of tissue necrosis and abscess and gas forming factor which all combine to produce a low redox potential[17]. The culturing of identical bacteria from the milk and the contents of an udder abscess of the same cows[13 28] suggest at least the secondary role for the anaerobic bacterial isolates. It appears that the anaerobic bacteria mostly perform their pathogenic role when they occur together with the classic mastitogenic bacteria and the latter influence the parenchyma tissue's Eh to the extent of favouring the growth of the anaerobic bacteria.

Apart from the 3 anaerobic bacteria already discussed, namely *B. fragilis, P. indlicus* and *F. necrophorum*, the other 7 anaerobic species isolated from cow udders by Du Preez[13] were recorded and their prevalences set out. The role of those 7 anaerobic species in the different forms of mastitis is probably secondary; they were always isolated together with the mastitogenic bacteria.

In the light of the induction of mastitis by installation of pure cultures of certain anaerobic bacterial species via the teat canal[13] and proof from available literature[7 26 43 78], it is difficult to reason away the role or contributory role played by anaerobic bacteria in the pathogenesis of mastitis.

THERAPEUTIC ASPECTS

Antimicrobial therapy of antibiotic infections generally requires high doses and prolonged treatment due to the tissue necrosis and tendency towards flaring up which accompany the anaerobic bacterial infection of tissue[17]. Henke[30] points out that anaerobic bacterial infections present problems for researchers and clinicians in that these types of infections sometimes require immediate therapy. Isolation, identification and establisment of antibiotic sensitivity generally take some time and hence valuable time is lost before the results are available, the specific cause of the particular disease is established and the patient can be treated. Consequently the treatment of anaerobic infections has to be empirical and based on the appearance of the micro-organisms which are usually associated with a large proportion of such cases and the antimicrobial sensitivity of such bacteria as established earlier. Keusch & Weinstein[35] point out that anaerobic bacteria are, with a few exceptions, fairly sensitive to antibiotics and that it is possible for the clinician to select a recommended drug for treatment based on a preliminary identification of the organism.

Henke[30] points out that the bulk of information gathered in connection with anaerobic infections is based on human data. The animal clinican is therefore obliged to use these data as a guideline for treating suspected anaerobic infections. The general data collected in connection with anaerobic infections in animals largely correspond with data on human cases[13].

As in the human, a drug for anaerobic infections is selected on the basis of pharmacological properties[72], especially the capacity to penetrate into a vascularly infected tissue as well as into purulent conditions. Keusch & Weinstein[35] state that parenteral antibiotics should only be used in septicaemia cases or where a primary wound expands despite thorough drainage. In many infections in which anaerobic bacteria are found, both anaerobic and facultative anaerobic bacteria are involved[13 17]. The nature of the non-aerobic organisms will invariably influence the choice of antimicrobic therapeutic drugs[17].

Surgical intervention, where possible, is most important in anaerobic infections. Abscesses should be drained and nectoric tissue removed. Repeated drainage of abscesses is sometimes required. Hyperbaric oxygen is useful in certain selected cases of gas gangrene in humans. Local treatment with hydrogen peroxide or zinc peroxide sometimes produces a dramatic improvement in certain persistent anaerobic infections[17].

Penicillin-G is the drug of choice in all anearobic infections, except where resistant *B. fragilis* and certain strains of *Fusobacterium* are involved. Pathogenic strains of *B. fragilis* can produce penicillinase[17 35] or otherwise large doses of penicillin are required to obtain suppression. The activity of penicillin is decreased only slightly in milk[80]. Penicillin is a weak organic acid with pKa 2,7 and it is therefore largely ionised in plasma. Because of this, milk levels after systemic therapy, will always be lower than plasma levels[57]. However, penicillin does penetrate mammary tissue relatively well in both normal and mastitic glands, except where large areas of necrosis have occurred[19]. Moreover, penicillin concentrations are higher in the milk from mastitic udders than from normal udders[19]. With due regard to the above-mentioned data concerning penicillin for therapy of mastitis caused by facultative anaerobic bacteria, some of these principles may be incorporated in the therapy of mastitis where anaerobic bacteria play a role. The sensitivity of anaerobic mastitis-causing bacteria to penethamate hydriodide will achieve a concentration in the milk 5-7 times the concentration achieved in plasma following intramuscular injection[56]. This drug could be used with good effect against sensitive anaerobic bacteria that cause mastitis. Certain strains of *B. fragilis* which produce penicillinase, will destroy penicillin. In certain cases even large doses of penicillin appear to be clinically non-effective against anaerobic bacterial infections[17]. McDiarmid[40] and Ziv[82] recommended a penicillin dosage of 16 500 iu/kg against mastitis caused by facultative anaerobic bacteria to maintain a therapeutic milk level for a 24-h period. Most strains of *B. fragilis* are resistant to cephalosporin and some strains produce cephalosporinase[17].

Cephalotin possesses a good activity against anaerobic bacteria[35]. McDiarmid[41] reported that cephoxazole is bacteriocidal and resistant to destruction by staphylococcal penicillinase and has a mutually potentiating effect with penicillin. The use of penicillin together with a cephalosporin therefore appears to be a logical step in mastitis therapy caused by anaerobes. Chloramphenicol may at present be regarded as the drug of choice for serious *B. fragilis* infections and certain other serious anaerobic infections[17]. Injectable chloramphenicol possesses limited intramuscular bioavailability properties and should therefore be administered intravenously[83]. Doses intramuscularly of about 50 mg kg^{-1} chloramphenicol would have to be given for mastitis cases caused by facultative anaerobic bacteria (*S. aureus*) in order to maintain therapeutic milk levels of 22,8 mca $m\ell^{-1}$ at 12 h and 12,2 mcg $m\ell$ at 24 h[24 40].

Considering the lack of knowledge regarding the chloramphenicol dosage and therapeutic milk levels in respect of mastitis cases caused by anaerobic bacteria, it is recommended that the above-mentioned data be used until such time as data in respect of anaerobes becomes available. Certain strains of *Clostridium* are resistant to chloramphenicol[35].

Clindamycin possesses a good activity against most anaerobes, including *B. fragilis*. However, there are reports indicating that certain fusobacteria, streptococci and some clostridia are resistant to clindamycin[35]. Clindamycin produced very good results in the therapy of a variety of serious anaerobic infections[17]. Metronidazole is the only drug which shows consistently good bactericidal activity against *B. fragilis*. To date encouraging results have been achieved with metronidazole against anaerobic infection[17]. At present only the intravenous form of metronidazole is available for mastitis therapy and large quantities have to be administered parenterally, which appears to be impractical at this stage. As a result of bacterial resistance, especially by *B. fragilis*, tetracycline is no longer strongly recommended for the treatment of anaerobic infections[17 35]. Oxytetracycline and chlortetracycline are both partially inactivated in milk by chelation with magnesium and calcium ions and by binding with casein[46]. Both are irritant following local infusion, absorption of both is very poor and both are very unevenly distributed even in normal udder tissue[81].

Oxytetracycline by intramuscular injection will not produce therapeutic levels in the milk. Slow absorption (bioavailability) from the injection site is responsible. However, large doses, in the order of 10 mg kg^{-1} administered intravenously, will maintain milk concentrations of oxytetracycline above 1 mg $m\ell^{-1}$ for 24 h[24 83].

The use of tetracycline for mastitis caused by anaerobic bacteria which are in most cases also infected with facultative anaerobic bacteria[13] is not recommended unless the anaerobes are sensitive and then it must be administered intravenously.

The aminoglycosides as a group have a consistently poor record of activity against anaerobic bacteria[35]. Aminoglycosides have an extremely low lipid-solubility which makes them unsuitable for systemic treatment of mastitis[41]. Ziv[82] suggested that dehydrostreptomycin (DHS) in doses of 10-20 mg kg^{-1} every 6-12 h may be suitable for treatment of Gram-positive infections. Following intramammary infusion the absorption rate of all the aminoglycosides is extremely poor (6,0-8,0 c.f urea). DHS has a very uneven distribution inside the udder, taking up to 8 h to become widely distributed[81]. The use of aminoglycosides against mastitis where anaerobic bacteria play a role, is not recommended.

The great majority of udder quarters from which anaerobic bacteria have been isolated, have previously been treated with some antibiotic or another[13]. From the above fact and the above-mentioned references from literature in connection with antibiotic resistance of anaerobic bacteria, it is clear that where anaerobic bacteria are found in certain cases of bovine mastitis, a high degree of antibiotic resistance is sometimes encountered. In all the cases of mastitis, except for a few cases of aseptic mastitis, from which anaerobes were isolated, facultative anaerobes and microaerophiles were also isolated and this should be borne in mind in mastitis therapy[13]. The nature of the non-aerobic bacteria will consequently influence the choice of antimicrobial agents. This implies that mastitis therapy cannot in all cases be applied successfully without the results of an antibiogram.

ABSCESSES

From a study of literature and research results[12 13 15] in respect of anaerobic bacteria, the following therapy is recommended for udder absceses where anaerobes are or may be present:

Udder abscesses

i) Drain the abscess. Repeat if necessary.
ii) Cut away the dead necrotic tissue if present to try and normalise the Eh.
iii) Flush with 30% hydrogen peroxide or zinc peroxide as frequently as necessary.

THERAPY OF ACUTE MASTITIS WHERE ANAEROBES ARE OR MAY BE INVOLVED

i) Parenteral administration of large doses of long-acting antibiotics after sensitivity tests have been carried out. A good preliminary choice for antibiotic therapy, i.e. before the results of an antibiogram are available, would be: chloramphenicol, clindamycin or penicilin-G, in this order according to availability and price. Parenteral therapy of chloramphenicol has a potentially good distribution throughout the udder[83] although high minimal inhibitory concentrations (MICs) and the requirement for enormous doses make chloramphenical quite unsuitable for the systematic treatment of mastitis[41]. Parenteral formulations of chloramphenicol also lack good intramuscular bioavailability[49 80]. Whereas chloramphenicol will penetrate well into the mammary gland, its very low blood levels after intramuscular injection and its rapid disappearance from the plasma would mean that doses of about 50 mg kg^{-1} would have to be given in order to maintain therapeutic milk levels for 24 h[40]. After intravenous injection of chloramphenicol base which possesses a high bioavailability[83] or the intramuscular administration of the watersoluble and more bioavailable chloramphenicol sodium succinate ester, free drug concentrations are reached that are considered effective against moderately sensitive isolates[83].

Despite certain pharmacokinetic disadvantages of chloramphenicol, this is still the drug of choice for mastitis where susceptible anaerobic bacteria, especially *B. fragilis*, play a role.

Metronidazole is the only agent that shows consistently good bacteriological activity against *B. fragilis*[17]. There is no intramammary preparation to date that contains metronidazole for mastitis therapy and parenteral mastitis therapy with metronidazole requires further research. Clindamycin is active against anaerobic Gram-negative bacilli such as *Bacteroides*, anaerobic Gram-positive non-sporeforming bacilli such as *Eubacterium* as well as Gram-positive cocci such as *Peptococcus*[13 17]. Large doses of penicillin-G are also a drug of choice for mastitis where susceptible anaerobic bacteria play a role except those due to *B. fragilis* and some strains of *Fusobacterium*[13 17]. In general, antimicrobial therapy for anaerobic infections requires large doses and prolonged treatment because of tissue necrosis and tendency towards relapse.

ii) Intramammary therapy with chloramphenicol or penicillin which are non-irritating to sensitive anaerobic bacteria[13]. Drugs that are distributed throughout the udder and are quickly absorbed into the general circulation such as ampicillin or lincomycin[83] and to which anaerobes are sensitive, should be preferred[13].
iii) For acute and peracute mastitic cases where anaerobes are also involved, supportive therapy is recommended, such as isotonic saline, glucose, concentrated multivitamins, corticosteroids and drugs that help maintain cardiac output, reduce pain and are anti-inflammatory.
iv) Oxytocin therapy and frequent milking at 1 to 2 h are recommended to remove toxins, debris, bacteria and other inflammatory products and to maintain milk duct patency[54].
v) Treatment should commence as soon as possible after the first appearance of mastitis - if possible within hours -since the prognosis depends on this[13].
vi) Cases of mastitis caused by anaerobic bacteria should be treated in the classic manner as long as overlapping or antagonism with the above-mentioned anaerobic therapy does not arise[13].
vii) Cases of subacute clinical and subclinical mastitis where anaerobes are involved should only be treated by intramammary infusion.

CONCLUSIONS

Anaerobic bacteria do play a role in the

Table 1: **Anaerobic and facultative anaerobic bacteria isolated concurrently from various forms of mastitis †**

Cow No	Herd No	Anaerobes	Facultative
Clinical mastitis			
1	A	*P. saccharolyticus*	*C. pyogenes*
2	A	*Bacteroides melaninogenicus*	*S. aureus*
3	B	*P. indolicus* •	*C. pyogenes*
		Eubacterium combesii	*S. aureus*
		P. acnes	*Streptococcus* spp.
4	B	*P. indolicus*	*S. aureus*
		E. combesii	
5	C	*Veillonella parvula*	*E. coli*
		Megasphaera elsdeni	
6	D	*P. indolicus*	*C. pyogenes*
		F. necrophorus	
7	E	*P. indolicus*	*C. pyogenes*
		Clostridium sporogenes	
8	F	Bacteroides fragilis	S. aureus
			S. agalactiae
9	G	*B. fragilis*	*S. aureus*
			S. agalactiae
10	H	*B. fragilis*	*S. agalactiae*
Subclinical mastitis			
11	C	*B. fragilis*	*S. aureus*
12	I	*P. indolicus*	*S. aureus*
13	J	*E. lentum*	*S. aureus*
13	K	*Peptostreptococcus* spp. •	*S. aureus*
"Aseptic" mastitis			
15	L	*P. indolicus*	absent

• Produced heparinase
† From Greeff, et al.[27]

Table 2: **Concurrence of anaerobic bacteria and recognised mastitogenic bacteria isolated from lactating mastitic udders°**

Animal	Sample	Anaerobic bacteria isolated	Facultative anaerobic bacteria isolated
Cow 1	Milk	*E. aerofaciens*	*S. aureus*
		E. lentum	*C. pyogenes*
			S. agalactiae
		P. indolicus	
Cow 1	Udder abscess	*E. aerofaciens*	*S. aureus*
		E. lentum	*S. agalactiae*
		B. fragilis	*C. pyogenes*
		P. indolicus	
Cow 2	Milk	*P. indolicus*	*S. aureus*
Cow 3	Milk	*B. fragilis*	*S. aureus*
Cow 4	Milk	*E. lentum*	*S. aureus*
Cow 5	Milk	*B. fragilis*	*S. agalactiae*
		P. granulosum	*S. aureus*
Cow 6	Milk	*Peptostreptococcus* spp.	*S. aureus*

°From du Preez, et al.[12]

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bovine mastitis complex. Although it is evident from this review that various pathogenic anaerobic bacteria may induce clinical mastitis under experimental conditions, their propensity to act as primary pathogens in nature and their precise pathogenesis is still unclear. The polymicrobial nature of udder infections however often involves both multiple anaerobic as well as facultative species[27]. Many anaerobic species, notably *B. fragilis,* are resistant to commonly employed antibiotics. These factors therefore may very well influence the efficacy of a single drug being used in the field. Much more research must be done to obtain clarity on the precise role of anaerobic bacteria in bovine mastitis.

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'N HISTORIESE PERSPEKTIEF OP MENS-DIER-INTERAKSIES AS STUDIEVELD

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ABSTRACT:
A chronological review is given of the most important people, publications, scientific gatherings and societies which have contributed to the development and recognition of human-animal interactions as a field of study. Lorenz is recognised as the founder of this new field of study. His contribution was to emphasise the role of humans who have to have a thorough knowledge of animal behaviour and he also pointed out the fact that modern man needs to keep contact with nature. Other pioneers were Levinson who pointed out the therapeutic benefits of companion animals and the Corsons who used animals in hospital settings. The explotion of knowledge in this particular field took place during the 1980's. The first world congress on the subject was held in 1980 in London. Societies involved in this new study field, were established in the United Kingdom, Europe, United States of America, Australia, Canada and South Africa. In South Africa the interest in this field emerged during the 1980's with articles published in veterinary periodicals. It has since developed into a multi-disciplinary approach. A new chair was furthermore inaugurated to teach human-animal-interactions as part of the veterinary curriculum. There is no doubt that the new study field has important implications for the veterinary profession.

Key words: History, human-animal interactions, field of study

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INLEIDING

Gedurende die afgelope 2 tot 3 dekades word aandag wêreldwyd op die rol van geselskapsdiere in die moderne samelewing toegespits. Die aanhou van geselskapsdiere as sodanig is geen nuwe neiging nie, maar die bestudering van hierdie verskynsel op multidissiplinêre vlak met behulp van moderne metodiek, is wel nuut.

In die lig van fragmentariese inligting beskikbaar oor die ontwikkeling van die studieveld in verskeie dele van die wêreld, is die doel van hierdie artikel om perspektief op die globale vordering te plaas deur na leidende persone, publikasies, belangrike byeenkomste en verenigings wat bygedra het tot die erkenning van die studieveld te verwys.

PERSONE EN PUBLIKASIES

Konrad Z Lorenz, 'n Oostenrykse Nobelpryswenner word vandag erken as die vader van die moderne etologie. Hy word ook aanvaar as die eerste persoon wat die verhouding tussen mens en geselskapsdier as 'n studieveld bekend gestel het en is in 1983 op 'n wêreldkongres as sodanig vereer.

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TYDPERK 1950-1959

Lorenz se studiebenadering was uniek in die sin dat hy diere nie op 'n afstand bestudeer het nie, maar dat hy daaraan geglo het om die dier so intiem moontlik in sy natuurlike staat te leer ken. Toe Lorenz met sy studies besig was, was daar twee skole wat dieregedrag bestudeer het. In Europa is die navorsers etoloë genoem. Hulle het gekonsentreer op diere se instinktiewe gedrag deur waarneming van diere in hul natuurlike omgewing. In die Verenigde State van Amerika is wetenskaplikes wat dieregedrag bestuur het, dieresielkundiges genoem. Hulle was meer geïnteresseerd in wat hulle van dieregedrag kon leer onder onnatuurlike laboratoriumtoestande. In sulke studies was die mens sover as moontlik, 'n onbetrokke waarnemer.

Lorenz se boeke wat in die 1950's[19 20] die lig gesien het, het onlangs nuwe betekenis gekry en dui die pad aan vanwaar vandag se studies ontwikkel het. Die navorser se uitgangspunt is steeds dieregedrag, maar die geselskapsdiere word in noue verband met die mens en die omgewing bespreek.

Lorenz beskou homself as deel van die natuur, maar verkondig terselfdertyd die onderskeid tussen mens en dier. So word sy besondere persoonlikheid van integrale belang in sy studies. Hy kan homself ewe goed met natuur en mensdom vereenselwig. Hierdie studies, anders as laboratoriumwerk, kan nie deur enige persoon herhaal word nie. Sy waarnemings mag dus subjektief wees en dit is dan ook die vernaamste kritiek teen sy metodes. Dit doen egter nie afbreuk aan die feit dat hy die besondere verhouding tussen mens en dier geïdentifiseer en soms raak beskryf het nie.

King Solomon's Ring[19] se subtitel, "New light on animal ways", gee 'n aanduiding van Lorenz se nuwe benadering. Hy begin die boek deur na die negatiewe kant van huisdiere te verwys. Hy maan dat die mate van bereidwilligheid wat 'n mens openbaar om die donker kant van diere te verdra, 'n aanduiding is van 'n mens se liefde vir diere. Hy glo dat die gedrag van "hoër en verstandelik aktiewer diere" slegs geken kan word as hulle sonder beperkinge in 'n huis (menslike omgewing) kan rondbeweeg. Die mens moet dan bereid wees om die gevolge van sodanige vryheid te verdra.

Volgens Lorenz[19] het die verstedelikingsproses die mens van die natuur vervreem, en daarom kan die aanhou van geselskapsdiere al hoe belangriker word.

Sover vasgestel kon word, is Lorenz[19] die eerste persoon wat oor die "band" tussen mens en geselskapsdiere geskryf het. (Vergelyk dit met die "human-animal bond" waarna daar vandag dikwels verwys word). Hy sê dan ook dat hierdie band soms sterker kan wees as bande wat tussen mense bestaan. Daar moet egter besef word dat 'n suksesvolle band tussen mens en dier nie bloot enige mens plus enige dier impliseer nie. Daar moet 'n passing tussen die mens en dier plaasvind. Die sukses van 'n band tussen 'n spesifieke persoon en 'n spesifieke dier, bly egter nog 'n raaisel.

In Lorenz[20] se tweede boek skets hy 'n spekulatiewe tablo van hoe hy dink dat die hond mak gemaak is. Hy stel homself voor dat die mens bloot op 'n "impuls" gereageer het om die dier naby hom te hê. Die hond het sy band met die mens gevorm soos jong wilde honde dit met die moederdier vorm. Troplojaliteit onder honde en die volg van 'n leier kan ook tot die band bydra. Lorenz voel sterk oor spesie-identiteit. Hy glo dat die mens 'n dier moet behandel soos die dier in die natuur deur ander lede van sy eie spesie behandel sou word. Om dit in 'n mens-dier-verhouding toe te pas, is 'n deeglike kennis van dieregedrag nodig. 'n Voorvereiste van sulke kennis word dan vandag as 'n algemene beginsel aanvaar waar die mens met diere kontak maak.

Aansluitend hierby, spreek Lorenz[20] hom duidelik uit teen 'n antropomorfistiese benadering teenoor diere. Dit verhoed hom nie om ware menslike eienskappe

gaan diere toe te dig nie, soos bv. jaloesie, leuens vertel, misleiding, 'n gewete, met vroulike grasie beweeg en die verstaan van gesprekke tussen mense - nie as gevolg van die klanke nie, maar werklike begrip van woorde. Hierdie eienskappe word egter net verklarend en vir interpretasie gebruik en beteken nie dat die diere soos mense behandel word nie. Lorenz stel dan ook 'n positiewe verhouding tussen mens en mens steeds as die ideale en primêre verhouding, teenoor dié met diere. Liefde vir die medemens behoort die oorsprong te wees van die liefde vir diere. Mense wat teleurgesteld is in ander mense en dáárom hul liefde van mense onttrek en dit aan diere gee, pleeg "sosiale perversie". Diere is nie beter as mense nie en haat teenoor mense en liefde vir diere maak 'n slegte kombinasie. Dit beteken nie dat diere se substituut-funksie hiermee uitgesluit is nie, maar net dat haat nie die rede moet wees waarom geselskapsdiere aangehou word nie.

Lorenz[20] beweer dat slegs 2 spesies menslike huishoudings vrywillig betree het, naamlik die hond en die kat. Ander mak-gemaakte spesies is gevange geneem en gedwonge diensbaar aan die mens gemaak. Die betrokkenheid van geselskapsdiere by die vorming van die band tussen mens en dier dui dus op 'n aktiewe rol aan die kant van die diere; dit het dus nie net aan die mens se kant ontstaan nie. 'n Mens kan dalk hieruit aflei dat die hond en kat, wat vandag nog nie die grootste aantal geselskapsdiere uitmaak, nie net as die tradisionele nie, maar ook as die oorspronklike geselskapsdiere beskou moet word.

Uit 'n wetenskaplike oogpunt kan Lorenz se werk as te emosioneel en te subjektief beskou word. Die styl waarin dit geskryf is, is ook te poëties. Aan die ander kant is dit soms moeilik om die gevoelens van diere-eienaars op 'n intelligente manier te beskryf, weens die persoonlike en emosionele aard daarvan. Die waarde van sulke inligting vir die studieveld moet dus nie onderskat word nie. Waarnemers wat totaal buite so 'n noue band tussen mens en dier staan, kan kwalik begryp waaroor dit alles gaan, as hierdie reaksie nie in berekening gebring word nie. Lorenz se emosionele eerlikheid bring juis insig in die mens-tot-dier-verhouding. Hy spreek die leser nie vanaf 'n hoorsê-platform aan nie, maar praat van eie ondervinding. As hy dit nie so gedoen het nie, kon sy werk 'n mate van geloofwaardigheid verloor het. Hierdie openhartigheid het dan ook latere studies beïnvloed. Hy het besef dat sy studies mank sal gaan aan volledigheid indien die emosionele sy daarvan ontken word. Vandag word erken dat hierdie studierigting met ander parameters as met proefbuise en laboratoriumdiere geëvalueer moet word.

Die fondamente wat Lorenz vir die ontwikkeling van die mens-tot-dier-verhouding as studieveld gelê het, kan soos volg opgesom word:

(i) die identifisering van die belang van die mens-tot-dier-verhouding
(ii) die herwaardering van die rol van geselskapsdiere in die stedelike omgewing
(iii) die metode om dieregedrag in 'n omgewing wat so natuurlik en vry moontlik is, waar te neem
(iv) kennis van dieregedrag wat 'n basis vir kontak tussen mens en dier moet vorm
(v) die erkenning van die emosionele sy van die mens-geselskapsdier-verhouding
(vi) 'n realistiese beskouing van die las en moontlike nadele wat die aanhou van diere kan meebring
(vii) die erkenning dat een van die behoeftes aan diere verband hou met die mense se verwydering van die natuur
(viii) die gebruik van die term "band" tussen mens en dier
(ix) riglyne oor die tipe verhoudings tussen mens en dier (die plek van die mens en die plek van die dier) en
(x) die wedersydse voordele wat die verhouding tussen mens en dier vir albei inhou.

TYDPERK 1860-1969

In die 1960's het Boris Levinson, 'n psigiater van New York, as nuwe pionier na vore getree. Hy was die eerste persoon wat hierdie studieveld in 'n toegepaste rigting begin stuur het. Levinson het begin met wat bekend geword het as psigoterapie wat deur middel van diere ondersteun word ("animal-facilitated psychoterapy").

In 1962 publiseer Levinson[11,12,13] artikels waarin hy konstateer dat die belangrikheid van die geselskapsdier vir die mens eerder psigologies as prakties is. Hy het sy eie hond reeds in die vyftigerjare as "mede-terapeut" in sy spreekkamer gebruik. Hy glo veral aan die voordeel wat die gebruik van die hond vir kinders inhou omdat die hond as katalisator dien om die kind met self-aanvaarding, selfontdekking en emosionele selfgenesing te help. Levinson voorspel reeds op daardie vroeë stadium dat honde as geselskapsdiere 'n al-hoe-belangriker rol as psigoterapeutiese hulpmiddels gaan speel.

Levinson meen dat die dood van 'n geselskapsdier dalk die kind kan voorberei op die afsterwe van 'n persoon. Die geselskapsdier kan egter aan die ander kant as 'n tydelike kruk dien waarop die kind kan leun as 'n ouer te sterwe sou kom, totdat aanvaarding plaasvind. Omdat daar nie altyd besef word dat kinders 'n behoefte het om voorwerpe soos diere wat sag en warm is, lief te hê nie, word geselskapsdiere in koshuise verbied[16]. In Levinson se eerste boek[18] word die rol van sy hond as "mede-terapeut" volledig bespreek.

Levinson[17] bespreek ook die voordele van geselskapsdiere vir bejaardes. Bejaardes toon dikwels, nes kinders, swakker verdedigingstrukture. Sulke persone kan ondersteuning vind in 'n geselskapsdier, aangesien sosiale status of ouderdom nie vir die dier van belang is nie.

Levinson[14] het ook 'n bydrae gelewer wat spesifiek vir die veearts van belang is. Die verhouding tussen mens en geselskapsdier het oor die eeue verander deurdat dit meer intens geword het, en die veearts behoort sy rol dienooreenkomstig aan te pas. Hoe meer geselskapsdiere die behoeftes van die mens bevredig, hoe meer sal die veearts betrokke moet raak by die kliënt se behoeftes.

Levinson het in die 1970's en 1980's op hierdie temas uitgebrei. Hy het veral klem gelê op die sielkundige voordele wat die interaksie met geselskapsdiere vir die mens kan inhou. Hy het voortgebou op die fondamente wat Lorenz gelê het en die aandag van al hoe meer ander navorsers op die studieveld gevestig. Die nuwe rigting het begin om aansien te verwerf. In die 1960's is Levinson se publikasies egter nog nie algemeen deur sy kollegas aanvaar nie. Puriste was skepties oor sy waarnemeings en het selfs die spot daarmee gedryf. Vandag word daar anders oor Levinson se werk geoordeel. Hy kan in sy besondere rigting, psigoterapie, as 'n baanbreker beskou word. Wat veral vir die veterinêre professie van belang is, is sy insig oor die deurslaggewende rol wat geselskapsdierveeartse kan speel. Levinson het moontlik met hierdie uitsprake die weg gebaan vir multi-dissiplinêre samewerking wat op hierdie gebied sou volg.

'n Privaat veearts, Jacob Antelyes, het ook al gedurendie die 1960's na die belangrikheid van die verhouding tussen veearts, kliënt en pasiënt verwys. Omdat veeartse so deeglik opgelei is om na diere om te sien, het hy klem gelê op wat beskryf word as "die menslike aspekte van veeartspraktyk".

Antelyes[2] meen dat eienaarskap van 'n geselskapsdier diep psigologiese implikasies het. Die veearts wat die eienaar se emosionele betrokkenheid by sy dier verstaan, sal in staat wees om na die geesteswelsyn van die eienaar om te sien, sowel as na die fisiese welsyn van die geselskapsdier. Antelyes[3] meen ook dat 'n besoek aan die veearts se spreekkamer altyd in die lig van 'n terapeutiese interaksie tussen pasiënt, kliënt en veearts beskou moet word. Hieruit is dit duidelik dat redes vir die aanhou van diere ook van dieselfde belang is as redes vir besoeke aan die veearts en dat die dier nooit los van sy eienaar in die spreekkamer hanteer mag word nie. Antelyes[1] sê dan ook reguit dat die veearts tot 'n sekere mate 'n sielkundige ondersteuningsrol moet speel in die hantering van die verhouding tussen mense en hul geselskapsdiere, want die dier dien dikwels as 'n emosionele uitlaatklep vir die eienaar. Die veearts moet voorsien dat sulke kliënte soms onredelik en irrasioneel sal optree en behoort daarom nie morele oordele oor sy kliënte te fel nie. In die 1970's en 1980's word die bydraes van Antelyes[4,5] eers werklik erken.

TYDPERK 1970-1979

Samuel en Elizabeth Corson het die eerste studies oor die gebruik van geselskapsdiere in 'n psigiatriese inrigting in Ohio, VSA gedoen. Alhoewel daar lank tevore reeds geselskapsdiere in inrigtings gebruik is, is daardie pogings nooit as wetenskaplike studies aangebied nie. Die Corsons se artikels het deurbrake op hierdie gebied ingelui.

'n Kongres, met die tema "mens-dier-verhoudings" wat in Engeland op 'n multidissiplinêre basis gereël is, het tot die publikasie van die 14 referate wat gelewer is, gelei. Die referate het groot klem gelê op die rol en verspreiding van geselskapsdiere in moderne gemeen-

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skappe. Onderwerpe wat aangespreek is, was die ekologie van geselskapsdiere in stede, eienaarskap, bevolkingstendense, betrokkenheid van plaaslike owerhede, rondloperhonde, soönoses, diere as hulpmiddels in terapieë en 'n voorskou vir die jaar 2000. Hierdie byeenkoms het 'n goeie basis vir toekomstige internasionale byeenkomste gelê.

TYDPERK 1980-1988

Gedurende die 1980's, het die verwagte kennisontploffing in die studieveld gevolg. Wetenskaplikes uit 'n verskeidenheid dissiplines het die verskynsel van mens-tot-dier-verhoudings begin navors en artikels daaroor begin publiseer. Op dié wyse het die vele fasette wat die studieveld bied, na vore gekom.

Michael Fox[8] van die VSA het die klem verskuif na die erkenning van die dier in eie reg, teenoor slegs 'n voorwerp wat vir die mens tot nut kan wees. Roger Mugford[22] van Engeland is 'n dieregedragsanalis wat hom by hierdie studieveld gevoeg het deur klem te lê op gedragsprobleme van geselskapsdiere, asook die behandeling van sulke probleme.

Sedert 1983 het die getal wetenskaplikes wat bydraes tot die studieveld begin lewer het, so toegeneem dat dit nie meer moontlik is om enkele persone uit te sonder nie. Hierdie navorsers is nou ook in al die moderne gemeenskappe van die wêreld versprei en verteenwoordig natuur- sowel as geesteswetenskaplikes.

Alhoewel bydraers, vóór en ná Lorenz en Levinson, elkeen 'n rol gespeel het om die nuwe studieveld te ontwikkel, kan Lorenz en Levinson as die balanseerpunt beskou word vanwaar die skaal in die guns van 'n nuwe erkende studieveld geswaai het. Die onlangse toename in navorsers, projekte, publikasies, byeenkomste, dissiplines, akademiese en openbare belangstelling het die balans verder ten gunste van die studieveld laat swaai. Die veterinêre professie het wel nie in groot getalle nie, maar tog op 'n beslissende wyse ook sy rol in hierdie ontwikkelings sedert die 1980's begin speel.

WETENSKAPLIKE BYEENKOMSTE

Nadat die studieveld multidissiplinêre erkenning begin verwerf het, was wetenskaplike byeenkomste 'n logiese gevolg.

TYDPERK 1970-1979

Die eerste internasionale byeenkoms is in 1974 deur die British Small Animal Veterinary Association in London gereël. Kleiner simposiums is in die vroeë 1970's deur die Canadian Veterinary Medical Association en die Canadian Federation of Humane Societies aangebied.

TYDPERK 1980-1988

Die British Small Animal Veterinary Association het in 1980 'n verdere internasionale simposium geborg, met die tema "The human-companion animal bond". Hierdie byeenkoms het die studieveld spesifiek as 'n multidissiplinêre veld erken en staan bekend as die eerste wêreldkongres oor die studieveld.

In 1981 word die eerste internasionale byeenkoms in die VSA (en tweede wêreldkongres) by die Universiteit van Pennsylvania gehou. Die tema was "New perspectives on our lives with companion animals".

Die derde wêreldkongres is in 1983 te Wenen, Oostenryk gehou. Die tema was "The human-pet relationship". Die kongres is deur die Instituut vir Interdissiplinêre Navorsing oor Mens-dier-verhoudings gereël, onder beskerming van die Society for Companion Animal Studies (Verenigde Koninkryk), die Delta Society (VSA) en die Joint Advisory Committee on Pets in Society (Australië).

In 1986 is die grootste byeenkoms (vierde wêreldkongres) dusver, deur die Delta Society in Boston, aangebied. Die tema was 'Living together: People, Animals and Environment". Sedert 1983 reël die Delta Society ook jaarlikse byeenkomste in Noord-Amerika, wat internasionale status verkry het omdat sprekers van ander wêrelddele gereeld daar optree.

Die vyfde wêreldkongres word in 1989 in Monaco gehou.

WETENSKAPLIKE VERENIGINGS

Die dryfkrag agter enige studierigting is gewoonlik lewenskragtige wetenskaplike verenigings. So het die verenigings van hierdie studieveld sedert 1980 'n groot rol gespeel om die moderne gemeenskappe bewus te maak van die belang van geselskapsdiere.

TYDPERK 1980-1988

In 1979 het 'n paar professionele persone, bekend as die Group for the study of the human-animal bond, vir die eerste kongres in London informeel bymekaar gekom. In 1981 het hulle 'n vereniging gestig en die naam verander na Society for Companion Animal Studies. Die lede was van die begin af multi-dissiplinêr verteenwoordig, sowel as internasionaal. Die meeste lede is egter van die Verenigde Koninkryk en Europa. Die vereniging se vernaamste doelstelling is om die band tussen mens en geselskapsdier te bestudeer, sowel as die rol van geselskapsdiere in 'n breër konteks[21].

Nie lank hierna nie, is die French Association for Information and Research on Companion Animals, gestig. Hulle werk nou saam met die Society for Companion Animal Studies en is heelwaarskynlik in die lewe geroep weens die taalverskil.

In 1980 is die Joint Advisory Committee on Pets in Society, kortweg bekend as Jacopis, in Australië gestig. Hierdie groep se ontstaan was gebaseer op 'n komitee met dieselfde naam wat in 1974 in die Verenigde Koninkryk in die lewe geroep is. Jacopis Australië dien as 'n sambreelorganisasie vir alle individue en groepe wat dieselfde doel nastreef, naamlik om 'n eenstemmige mening oor geselskapsdiere in die gemeenskap uit te bring. Die vereniging raak betrokke by probleme wat met diere in die gemeenskap in verband staan, voorsien advies aan owerhede en bevorder verantwoordelike eienaarskap. Die Australiese Veterinêre Vereniging dien ook op die bestuur van Jacopis[9].

In 1981 is die Delta Society soos dit vandag bekend is, tydens die tweede wêreldkongres in Pennsylvania gestig. Hierdie vereniging is 'n uitvloeisel van die Delta Foundation wat in 1976 gestig is. Die lede van hierdie vereniging is ook multi-dissiplinêr en internasionaal. In die Delta Society se doelstellings word groot klem gelê op die wetenskaplike navorsing oor mens-tot-dier-interaksies, asook die effek van die band tussen mens en geselskapsdier op die mens se emosionele en fisiese welsyn. Die Delta Society is die grootste en aktiefste vereniging in hierdie vakgebied[10]. In 1987 is die Human/Animal Bond Association in Kanada gestig.

Al bogenoemde verenigings skakel onderling met mekaar en neem saam deel aan die ontwikkeling van die studieveld. Hulle reël gesamentlike byeenkomste, koördineer navorsing en kennis word deur middel van die verenigings se publikasies uitgeruil.

ONTWIKKELINGS IN SUID-AFRIKA

Belangstelling in die nuwe studieveld het in Suid-Afrika gou gevolg nadat dit in ander moderne gemeenskappe posgevat het.

TYDEPRK 1980-1988

Die eerste publikasie in hierdie verband in Suid-Afrika, het in 1980 verskyn[30]. In 1981 word 'n artikel[23] gepubliseer waarin na die rol van die geselskapsdier in die gemeenskap verwys word en hoe dit die veearts ten opsigte van veearts-kliënt-verhoudings kan raak. Die eerste bydrae uit Suid-Afrika wat oorsee gepubliseeer is, het klem gelê op die bydrae van die geselskapsdierveearts in die moderne gemeenskap[24].

In ander artikels word op die moderne toepassing van geselskapsdiere in rehabilitasie gewys[25], terwyl die houding van die veearts teenoor kliënt, pasiënt sowel as teenoor homself tydens die verlies van 'n geselskapsdier ook bespreek word[26]. In 198.7 volg 'n bespreking van die veearts en diereregte[27] en 'n herevaluering van eienaar-hond-verhoudings[28].

Suid-Afrika is op die derde en vierde wêreldkongresse verteenwoordig en by laasgenoemde is 'n referaat oor die mens-dier-verhouding in die veterinêre spreekkamer gelewer[29]. Suid-Afrika is ook in die reëlingskomitee van die vyfde wêreldkongres verteenwoordig.

Op 14 Februarie 1984 is die Studiegroep van die Mens/Dier-Kontak in Bloemfontein gestig. Die doelstellings is om kennis oor die mens-tot-dier-interaksies te versamel, kennis daaroor te versprei en om die kennis in praktiese projekte toe te pas.

In 1986 is die Epol-leerstoel in Geselskapsdiersoötegnologie en -voeding aan die Fakulteit Veeartsenykunde, Universiteit van Pretoria, ingestel. Die opdrag is om die etologie van geselskapsdiere en mens/dier-verhoudings vanuit 'n veterinêre oogpunt te bestudeer.

GEVOLGTREKKING

Dit is duidelik dat die studierigting finaal sy beslag in die moderne gemeenskappe regoor die wêreld gekry het. Hierdie nuwe rigting is dalk een van die mees revolusionêre bewegings van ons tyd. Die multi-dissiplinêre aard daarvan bied groot moontlikhede op 'n tydstip waar hiper-spesialisasie dreig om navorsers hul perspektief te laat verloor. Die studies was van die begin af internasionaal en holisties, terwyl nuwe dissiplines steeds tot die studieveld toetree. Daar bestaan

geen twyfel dat die studieveld belangrike implikasie vir die veterinêre professie inhou nie en dit is feitlik vanselfsprekend dat die veearts toenemend betrokke sal wees.

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