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VOORDRAG BY WINTERPROMOSIEPLEGTIGHEID VAN DIE FAKULTEITE VEEARTSENYKUNDE, GENEESKUNDE EN TANDHEELKUNDE, UNIVERSITEIT VAN PRETORIA, 19 JUNIE 1987

Een van my eerste gewaarwordings toe ek gevra is om hierdie rede te voer was skok eerder as trots, want om by so 'n belangrike geleentheid op te tree is nie net 'n eer nie, dit is ook 'n besonder verantwoordelike taak. My gedagtes is dadelik teruggevoer na 'n soortgelyke gebeurtenis meer as 30 jaar gelede toe ek in u skoene gestaan het, en die verskillende emosies wat daarmee gepaard gegaan het: 'n Mens se eie gevoel van prestasie en antisipasie enersyds, getemper deur 'n tikkie onsekerheid; die besonder trotse, maar ook waarskynlik effens verligte ouers; al die ander betrokkenes, en hulle gevoelens; geeneen wat werklik beswaard is nie, al word daar soms, om goeie redes, 'n traan weggevee.

Maar wie die geleentheidspreker was en waaroor hy gepraat het, moet u my asseblief nie vra nie. Dit het my laat besef dat die persoon en sy tema miskien nie so belangrik is nie, solank daartoe bygedra word om "Gradedag" 'n hoogtepunt vir al die betrokkenes te maak.

Daarom wil ek begin deur u, die graduandi, van harte geluk te wens en vir almal wat u gehelp het om u doelwit te bereik, hetsy ouers, wederhelftes, ander familie en geliefdes, ook te felisiteer.

Die tema vir my praatjie is die belangrikheid van navorsing vir die beroepe waaraan u van vandag af behoort. Ek wil dit motiveer deur te beweer dat die sukses van elkeen van u se loopbaan daarvan sal afhang in watter mate u in die toekoms van nuwe navorsingsresultate gebruik gaan maak. Elkeen van u gaan self tot 'n meerdere of mindere mate navorsingsresultate genereer, want iemand wat so talentvol is, en dit so vêr gebring het, gaan nie met die huidige stand van kennis oor die vakgebiede wat u gaan betree, tevrede wees nie.

Volgens Nobelpryswenner Peter Medawar is navorsing "the art of the soluble". Min wetenskaplikes is myns insiens inderdaad beter toegerus of gemotiveer om met spoed oplossings vir probleme te vind as juis diegene met 'n kliniese opleiding.

'n Vraag wat by mens opkom is: Tot watter mate is die medies-georienteerde beroepe tans by navorsing betrokke?

Wat tandheelkunde betref was dit vir my insiggewend om te ontdek dat ongeveer 250 navorsingsprojekte in die sg. Nasionale Register verskyn. Hierdie syfer is waarskynlik konserwatief want die veeartsenykundige projekte wat daarin opgeneem is, is nie volledig nie. Die 4 grotes op hierdie gebied, naamlik Onderstepoort se Fakulteit, Onderstepoort se Navorsingsinstituut, Medunsa se Veterinêre Fakulteit en Roodeplaat-Navorsingstasie spog met 'n totaal van ongeveer 350 projekte, en daar is nog ander veterinêre instansies wat navorsing doen. Wat geneeskundige navorsing betref lys die Register waarna ek flussies verwys het meer as 1 800 projekte, wat sekerlik ook 'n onderskatting is.

Daar is 2 biologiese navorsingsrigtings wat na my mening in die afsienbare toekoms die meeste aandag gaan geniet. Hulle is: biotegnologie en omgewingswetenskappe.

Ek gaan my vandag by biotegnologie bepaal omdat ek meen dat die grootste wetenskaplike deurbrake in hierdie rigting sal plaasvind.

Ek gaan nie die omvattende woord "biotegnologie" probeer omskryf nie, maar eerder deur praktiese voorbeelde illustreer wat ek daarmee bedoel.

Om die belangrikheid daarvan te beklemtoon kan mens praat van 'n biotegnologiese rewolusie wat tans besig is om wêreldwyd plaas te vind.

Eerstens wil ek "rekombinante DNA-tegnologie" noem, wat tans die belangrikste vorm van genetiese manipulering of "genetic engineering" is. Dit behels die inbou van stukkies DNA wat vir 'n bepaalde substans van 'n organisme of sel kodeer in die genoom van ander organismes, soos bakterieë, gisselle, virusse en eukariotiese selle wat dan ekspressie aan die geen gee, en inderdaad as klein fabriekies vir die produk dien.

Hierdie tegnologie word dan ook toegepas in die ontwikkeling van nuwe entstowwe wat beter gedefinieer, veiliger en meer stabiel (beide geneties en fisies) as bestaande entstowwe sal wees. Hulle bestaan uit subeenhede van organismes, d.w.s. proteïene of selfs polipeptiede wat immunogenies is, i.p.v. die hele organisme. Daar is reeds 'n subeenheidentstof teen Hepatitis B ontwikkel wat deur gisselle vervaardig word. Ook is vêr gevorder met die ontwikkeling van subeenheidentstowwe teen verskeie ander belangrike siektes, wat bek-en-klouseer, malaria, influensa en bloutong van skape insluit. Die tegniek hou groot belofte in vir die ontwikkeling van entstowwe teen mikro-organismes en parasiete wat nie op die gewone wyses gekweek kan word nie. Wat parasiete betref is sistiserkose, babesiose en anaplasmose van beeste primêre kandidate. Laasgenoemde 2 geniet reeds aandag, beide in ons land en oorsee.

Ek merk in die literatuur op dat daar ook gepoog word om deur rekombinante DNA-tegnologie, 'n entstof teen *Streptococcus mutans*, die hoofoorsaak van tandkaries, te vervaardig.

Nommerpas, sintetiese peptiedentstowwe verteenwoordig die uiteindelike biotegnologiese mikpunt.

Menslike groeihormoon, insulien en interferon word reeds in die praktyk m.b.v. rekombinante DNA-tegnologie deur bakterieë vervaardig. USFDA (United States Food & Drug Administration) goedkeuring vir die gebruik van beesgroeihormoon, wat deur bakterieë geproduseer word, word in 1988 verwag. Hierdie hormoon stimuleer nie slegs spiergroei in beeste sowel as varke en hoenders nie, maar ook melkproduksie by koeie.

Sogenaamde DNA-peilers vir die diagnose van verskeie moeilik diagnoseerbare siektes, of die identifikasie van laegraadse besmettings, neem steeds toe in belangrikheid. Peilers word m.b.v. rekombinante DNA-tegnieke op groot skaal in bakterieë geproduseer, en met radio-isotope gemerk. Hulle werk op die beginsel dat die opspoorbare radio-aktiewe peiler met 'n nukleïensuur wat chemies net soos hy lyk, hibridiseer en deur outoradiografie opgewys word.

In die geval van hartwater sal bv. 'n peiler om besmette bontbosluise te identifiseer van groot waarde vir epidemiologiese studies en die toepassing van beheermaatreëls wees.

Sekelselanemie en ander oorerflike gebreke kan bv. reeds in ongebore babas m.b.v. amnionsentese deur peilers gediagnoseer word. 'n Peiler word selfs reeds vir vaderskapbepalings in die geregtelike geneeskunde gebruik.

Tweedens wil ek iets sê oor "monoklonale teenliggame". Monoklonale teenliggame het 2 belangrike eienskappe:

Hulle word in vitro, in die spreekwoordelike proefbuis, i.p.v. in vivo, soos die teenliggame wat ons ken, vervaardig.

Hulle bestaan uit groot getalle identiese teenliggaammolekules en het dus volkome spesifisiteit.

Monoklonale teenliggame het die tydvak van kitsdiagnostiek ingelui in die spreekkamer, tuis of langs die drukgang op die plaas d.m.v. die ensiematiese buisie of "dipstick" tegnieke. Stel u voor dat 'n spesifieke virusinfeksie binne 30 minute gediagnoseer kan word. Die boer in Europa kan reeds 95 dragtigheidstoetse binne 35 minute op melkmonsters doen met 'n monoklonale teenliggaam teen progesteroon.

Monoklonale teenliggame met radioaktiewe of ander "plofkoppe" bewapen sal moontlik vir die bestryding van gewasse ingespan kan word.

Monoklonale teenliggame wat teen *E. coli* se fimbriae gerig is, word reeds vir passiewe immunisering van kalwers aangewend. Hulle word ook vir die bestudering van die samestelling van periodontale weefsels gebruik, en is pragtige werktuie vir molekulêr-biologiese, mikrobiologiese, parasitologiese en ander navorsing.

"Embrio-oorplasing", wat reeds in mens en dier gebruik word, is 'n derde voorbeeld van die aanwending van biotegnologie. Dit strek egter in die laboratorium veel verder as bloot die in vitro bevrugting en oorplasing van hooggeteelde embrio's van beeste en skape in surrogaat moeders, wie se enigste funksie dit is om hierdie "superkinders" in die wêreld te bring en groot te maak.

Die verwekking van sg. transgeniese diere is tot op datum die grootste enkele deurbraak met genetiese manipulasie

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van soogdiere. Dit is moontlik gemaak deur tegnieke wat ontwikkel is om DNA direk in die bevrugte eisel in te spuit. 'n Groeihormoongeen is bv. uit mensweefsel geïsoleer en in die genoom van 'n bakteriese plasmied ingebou en vermenigvuldig. Daarna is ongeveer 600 kopieë van die geen met restriksie-ensieme uitgesny en in die manlike pronukleus van bevrugte eiselle van muise ingespuit. Die resultaat was reuse muise, ongeveer 2 keer so swaar as hulle kontrole maats. Die reuse müise het selfs die geen aan hulle nageslag oorgedra.

Die laaste voorbeeld van biotegnologie waarby ek wil stilstaan is in vitro-kweking van weefsels en — ek sê dit met ontsag — organe. Die in vitro-kweking van liggaamselle is seker al so oud soos ekself. Ons kan organe wat vir oorplantingsdoeleindes benodig word reeds taamlik lank aan die lewe hou.

Wat 'n wonderlike deurbraak sou dit nie wees nie as ons weefsels en selfs organe vir oorplantingsdoeleindes in vitro kan kweek vanaf die selle of weefsels van die ontvanger?

Daar is reeds vordering in hierdie rigting. Velweefsel van pasiënte met brandwonde word reeds in vitro vermeerder vir outo-transplantasie doeleindes. Wat van die moontlikheid van orale mukosa vir mond-kaak sjirurgiese doeleindes? 'n Nuwe stel tande vir 'n haasbek, of 'n weelderige haardos vir 'n pankop, om nie van niere, lewers en harte te praat nie? Dit klink vergesog, maar so ook was 'n man op die maan slegs 'n paar dekades gelede.

Ons weet lank reeds dat elke gedifferensiëerde liggaamsel die genetiese inligting bevat om enige ander orgaansel te wees. Alhoewel ons kennis van die stelsels wat die besonder komplekse soogdiersel beheer snel vorder, weet ons van die meganismes wat seldifferensiasie beheer nog bloedweinig. Een van die mikpunte van biotegnoloë moet sekerlik wees om die kennis te genereer om "onderdele" te kweek.

Al doen u tydens u loopbaan nooit iets anders as kliniese werk nie, sal u van vandag af u doelbewus en sistematies moet inspan om op die hoogte te bly van die kennisontploffing wat gaan plaasvind. Voortgesette opleiding is van meet af aan u voorland as dit u oogmerk is om 'n doeltreffende diens, hetsy as klinikus, akademikus, navorser of selfs as bestuurder, te lewer.

Ek wil afsluit met 'n aanhaling uit 'n toespraak wat Sir Arnold Theiler, stigter van Onderstepoort en eerste Dekaan van die Fakulteit Veeartsenykunde, in 1920 by 'n soortgelyke geleentheid gelewer het.

"I appeal to the young South African as the future veterinarian who above everything else has the welfare of the country at heart; to the young man who has the altruistic desire to be useful to his fellow citizens; and to the man who loves science for science's sake, who will be satisfied with a decent living, and will labour to elucidate the problems which are as yet unsolved."

Hierdie woorde is natuurlik nie slegs van toepassing op die manlike geslag en veeartse nie, maar ook op die dames en die ander twee beroepe wat deur u hier verteenwoordig word. Mag hierdie siening ook in u loopbaan uiting vind.

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THE PATTERN OF VENOUS DRAINAGE OF THE EQUINE ILEOCAECAL JUNCTION

ANET H KOTZÉ*

ABSTRACT

The veins draining the ileocaecal junctions of horses (n = 19), donkeys (n = 3) and a plains zebra, *(Equus burchelli antiquorum)* were injected with latex via the ileocolic vein, and dissected. In all specimens the ileocaecal papilla was drained by 2 major papillary veins: one cranial and one caudal to the papilla. A smaller dorsal vein drained either into the cranial or into the caudal vein. The submucosal veins seemed to increase in number in the ileocaecal junction to form a venous plexus. This plexus, together with veins from the caecum and the distal ileum immediately bordering the ileocaecal junction, drained either into the cranial or the caudal veins of the papilla. In 14 specimens both the cranial and caudal veins drained into a common vein, which opened into the *V. caecalis lateralis* in 10 specimens or into the *R. ilei mesenterialis* in 4 specimens. In 3 specimens the 2 veins opened separately into the *V. caecalis lateralis*. In 2 specimens the cranial vein opened into the *V. caecalis lateralis*, while the caudal vein drained into the *R. ilei mesenterialis*.

Key words: Equine, ileocaecal junction, venous drainage, venous plexus, veins

Kotzé Sanet H. The pattern of venous drainage of the equine ileocaecal junction. *Journal of the South African Veterinary Association* (1988) 59 No. 3, 131-133 (En.) Department of Anatomy, Faculty of Veterinary Science, University of Pretoria, P/Bag X04, 0110 Onderstepoort, Republic of South Africa.

INTRODUCTION

The ileocaecal junction of the human has been well described[2 3]. Ferraz de Carvalho et al.[2] described a submucosal venous plexus in the area of the ileocaecal papilla in man. This plexus, after engorgement, was thought to complement the action of the sphincter muscles in this area, aiding closure of the ileocaecal opening.

A similar plexus exists in the ileocaecal junction of the horse and has been described briefly by Schummer[5]. During in vivo endoscopic examinations of the base of the caecum in the horse, Dyce & Hartman[1] noted a purple discolouration and swelling of the ileocaecal papilla, which they thought to be due to the presence of a venous plexus. However, no detailed description of the structure or extent of the plexus was given.

In the present study, 23 specimens of equine ileocaecal junctions were examined after injection of the venous system with blue latex to confirm the presence of the plexus, and to establish the pattern of the venous drainage of this plexus.

MATERIALS AND METHODS

The present study was carried out on the ileocaecal junctions of horses (n = 19), donkeys (n = 3) and a plains zebra, *(Equus burchelli antiquorum)*. The specimens were taken from clinically healthy animals of both sexes whose ages ranged from 7 months to 12 years. The animals were anaesthetised with 20% chloral hydrate, the left common carotid artery was catheterised and the animals were exsanguinated. The zebra specimen was obtained from an animal which had been shot in the Kruger National Park. Immediately after death, the ileocaecal junction of each animal was removed to include the terminal ileum, ileocaecal papilla and the surrounding caecum and colon together with associated blood vessels. The specimens were stored at a temperature of −12° C for periods ranging from 2 weeks to 10 months, after which they were thawed at room temperature. Sixteen specimens taken from horses were then injected with blue latex via the ileocolic vein. These specimens were fixed by immersion in 10% formalin for a period of 7 d. The veins draining the plexus were dissected and the venous plexus was exposed by removal of the mucosa of the ileocaecal area using a sharp scalpel. The ileocaecal papilla, and the bordering distal ileum and caecum of the specimens were cut longitudinally, halfway between the dorsal mesentery and the ileocaecal fold. The specimens were photographed and schematic drawings of the different patterns of venous drainage of the ileocaecal area were made. This procedure was repeated on the specimens taken from the donkeys and the plains zebra.

An additional 3 specimens taken from horses were injected in the same manner using a mixture of latex and barium sulphate (Baritop 100 suspension, Noristan). Radiographs of these specimens were taken and then photographed, after which the specimens were dissected in the same way as described above.

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RESULTS

(a) Papillary venous plexus

Upon removal of the mucosa in the ileocaecal junction and on longitudinal sections of the papilla, the submucosal veins of the junction were seen to form a dense venous plexus. This plexus was apparent in the last 10mm of the distal ileum. From here, the submucosal plexus extended into the caecum immediately surrounding the papilla (Fig. 1 & 2). The injected veins caused the papilla to protrude and the mucosa took on a blueish colour due to the presence of the blue latex in the submucosal plexus.

(b) Venous drainage of the papilla

In all the specimens the venous plexus of the ileocaecal papilla was drained by 2 major papillary veins: one cranial and one caudal to the papilla. A smaller dorsal vein drained either into the cranial or into the caudal vein (Fig. 3a-d). The latter two veins were arranged in a circular fashion around the base of the papilla (Fig. 5 a & b), where they lay within the papillary muscle layers, before emerging on the serosal aspect of the ileocaecal junction. The cranial, caudal and dorsal papillary veins drained the cranial, caudal and dorsal aspects of the ileocaecal venous plexus respectively. Submucosal veins draining the surrounding caecum as well as branches draining the distal ileum, joined the cranial and caudal veins at regular intervals. In 14 specimens (73,4%), the cranial and caudal veins anastomosed to form a common vein that joined the *V. caecalis lateralis* in 10 specimens (52,6%) (Fig. 3c) and the *R. ilei mesenterialis* of the ileocolic vein in 4 specimens (21,1%) (Fig. 3d). In 3 specimens (15,8%) (Fig. 3a & 4), both the cranial and caudal veins drained separately into the *V. caecalis lateralis*. In 2 specimens (10,5%) (Fig. 3b) the cranial vein opened into the *V. caecalis lateralis*, while the caudal vein joined the *R. ilei mesenterialis* of the ileocolic vein. In the zebra specimen the cranial and dorsal veins opened into the *V. caecalis lateralis* and the caudal into the *R ilei mesenterialis*. In the donkey specimens the cranial and caudal veins both drained separately into the *V. caecalis lateralis* while the dorsal vein drained into either the cranial or caudal veins.

DISCUSSION

The presence of a submucosal venous plexus in the ileocaecal area in humans has been described[2 3]. The increase in the number of veins in the ileocaecal area is confirmed numerically by Ferraz de Carvalho et al.[3] They describe the submucosal venous distribution as polygonal meshes which condense in the ileocaecal papilla. These authors postulate that the junction functions as an angiomuscular system, rather than a purely muscular sphincter. These findings coincide with the "compressable venous cushion" that Stieve describes in the human ileocaecal junction[6].

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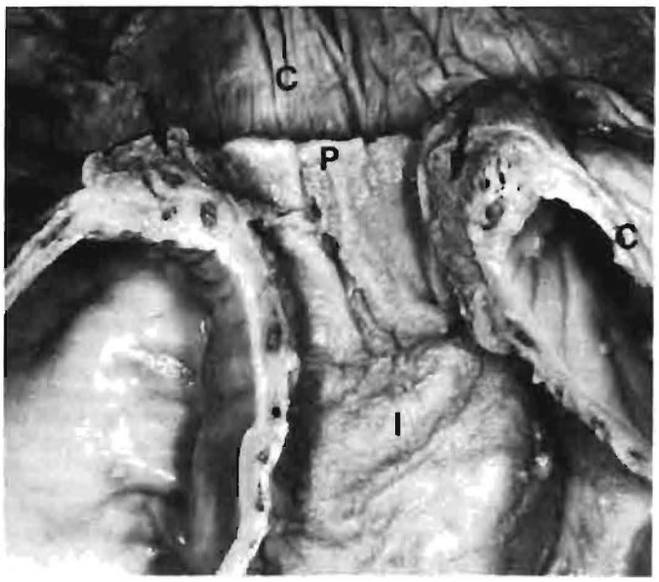

Fig.1: The ileum (i), the ileocaecal papilla (p) and the adjoining caecum (c), are sectioned longitudinally halfway between the ileocaecal fold and the dorsal mesentery in a specimen injected with latex. The submucosal venous plexus in the ileocaecal pipilla is indicated with arrows. The plexus is visible in the last 10 mm of the distal ileum and extends into the tip of the papilla to reach the adjacent caecum

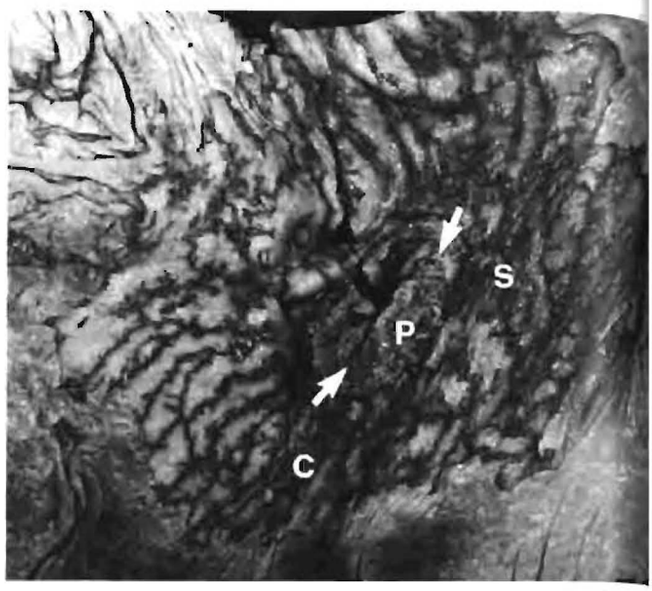

Fig. 2: The ileocaecal papilla (p), photographed from the lumen of the caecum (c), with the caecal and papillary mucosae removed. The network of submucosal caecal veins surrounding the papilla is marked (s), and the dense, fine venous plexus in the papilla is indicated with arrows

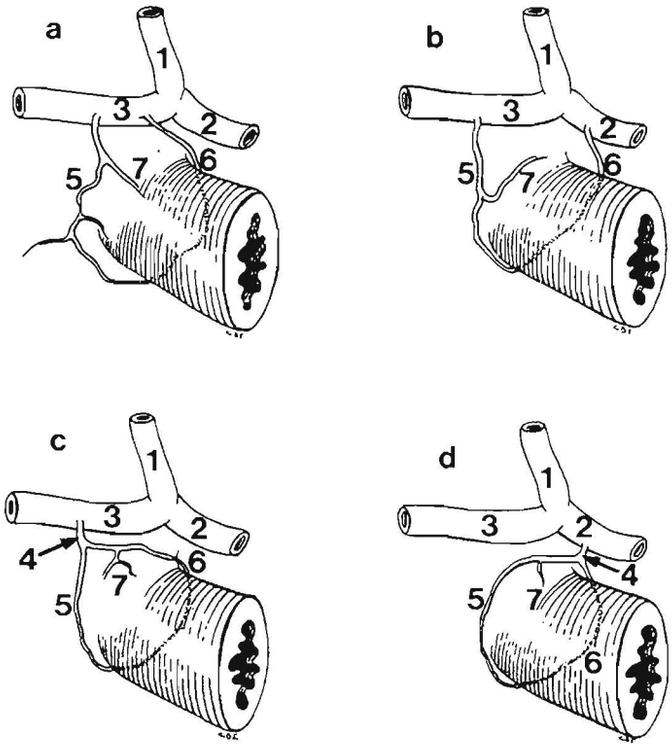

Fig. 3 (a-d): This schematic drawing demonstrates the variations of the venous drainage of the ileocaecal junction in the horse. The following structures are shown: the *V. ileocolica* (1), the *R. ilei mesenterialis* of the *V. ileocolica* (2), the *V. caecalis lateralis* (3), the common vein draining the ileocaecal papilla (4), the cranial vein of the ileocaecal papilla (5), the caudal vein of the ileocaecal papilla (6), and the dorsal vein of the ileocaecal papilla (7)

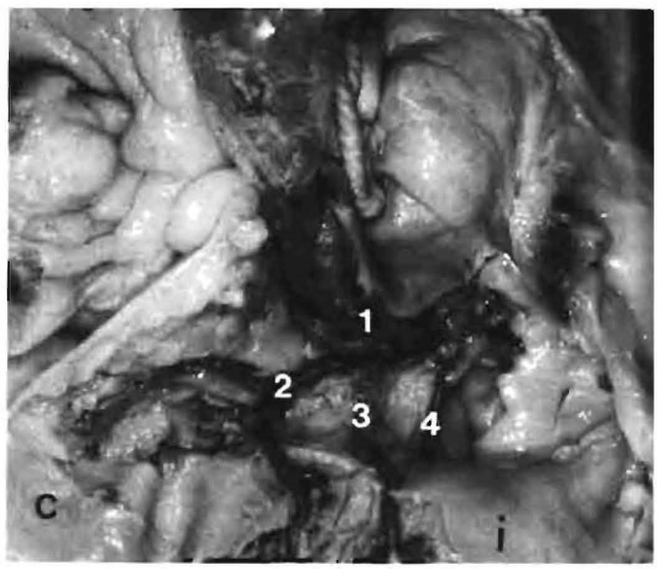

Fig. 4: This specimen, dissected from the serosal aspect of the ileocaecal junction shows the veins draining this area. The ileum is marked (i), and the caecum is marked (c). The lateral caecal vein is marked (1), the cranial vein of the papilla (2), the dorsal vein (3), and the caudal vein (4). This specimen is an example of where the cranial and caudal veins drain separately into the lateral caecal vein

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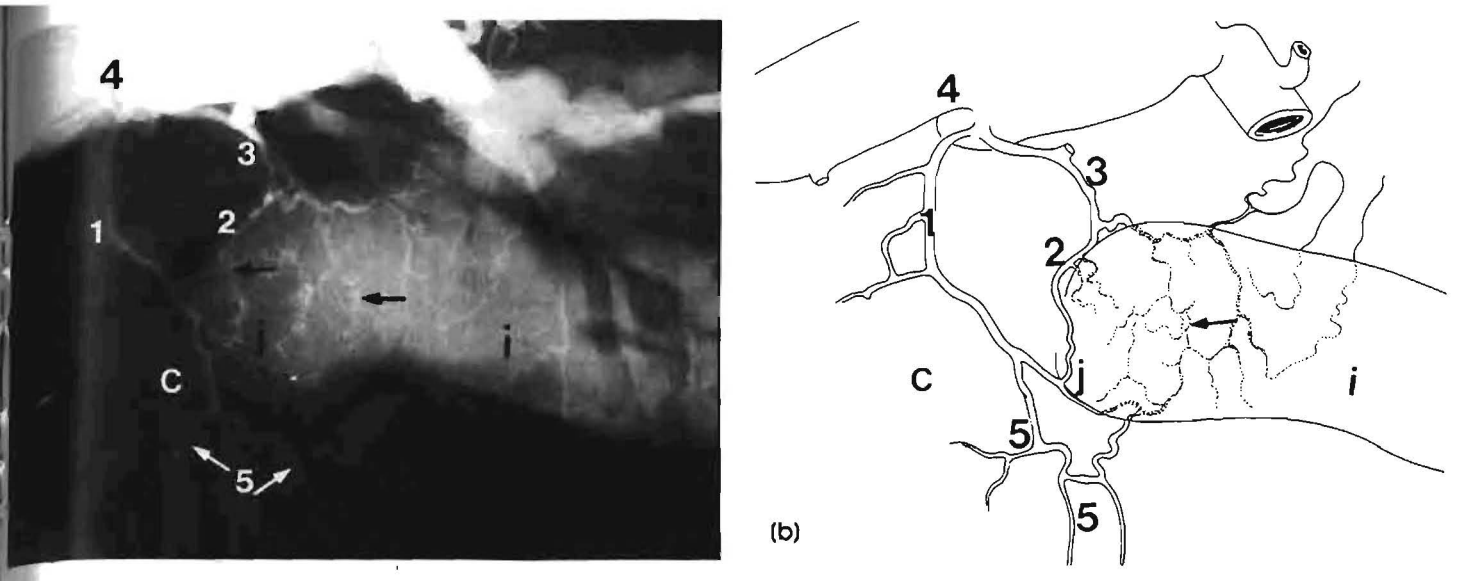

Fig. 5 (a-b): These are a radiograph (a) and a traced illustration (b) of a specimen showing the venous drainage of the ileocaecal papilla, where the ileocolic vein has been injected with a mixture of latex and barium sulphate. The cranial and caudal veins are arranged in a circular fashion around the base of the papilla. The ileum (i), enters the caecum (c), at the ileocaecal junction (j). The cranial vein of the papilla is marked (1), the dorsal vein (2), the caudal vein (3), the lateral caecal vein (4). Venous branches draining the caecum around the junction, and joining the cranial and caudal veins are marked (5). The venous plexus in the ileocaecal papilla is indicated with arrows.

Dyce & Hartman[1] describe a swelling and purple discolouration on endoscopic examinations of the ileocaecal papilla of the horse. Engorgement of a venous plexus at the ileocaecal orifice should narrow the opening by the subsequent increase in volume of the submucosal tissue. However the mechanism by which the plexus engorges and empties is not certain. Dyce & Hartman[1] discuss the possibility that all the veins in the plexus might not be filled simultaneously and that blood may be shunted from one area to another. The cranial and caudal veins which are mainly responsible for drainage of the plexus, lie within the muscle layers at the base of the papilla. At the base of the papilla, the muscle layers contributing to the papilla are best developed[4]. However, the major part of the plexus is situated in the tip of the papilla, where the contributing muscle layers are attenuated[4]. Owing to the thinner walls of veins in general, contraction of the ileal musculature at the base of the papilla may compress the larger veins draining the plexus while allowing the mucosal venous plexus to fill. The arteries which supply the papilla should not be affected to the same extent by muscle contraction, due to their thicker walls.

Verminous arteritis of the ileocaecocolic artery is a common cause of equine colic[7]. This condition impairs the blood supply to different areas of the equine intestine and may affect the blood supply to the plexus in the ileocaecal area. Subsequently the engorgement of the plexus and its role in closure of the orifice may be affected.

In a review article on the ileocaecal sphincter, Quigley & Phillips[8] discuss its nervous and hormonal control and its function in mediating the gastroileal and gastrocolonic reflexes. The exact mechanism by which the ileocaecal junction functions seems to be uncertain.

The space-occupying aneurisms in the cranial mesenteric artery, associated with verminous arteritis, may have an affect on the cranial mesenteric plexus, which may in turn affect the function of the venous plexus. This matter needs further investigation.

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A COMPARISON OF HEALTH PARAMETERS IN TWO DIFFERENT CANINE POPULATIONS. PART II: CHEMICAL PATHOLOGY DATA

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ABSTRACT

Blood samples were collected on a random basis from 2 canine populations. A selection of serum enzymes, serum electrolytes, serum protein fractions and serum concentrations of iron, creatinine and urea were investigated in a population of kennelled dogs and a population of dogs from a rural township in a developing country. Significant differences between the 2 populations were found for 16 of the 26 constituents evaluated, although differences in variance and distribution made comparisons difficult for some of the tests.

The dogs from the rural township had a low mean serum iron, a very low mean serum albumin and a very high mean serum gammaglobulin concentration compared to that of the kennelled dogs. It is postulated that these differences were caused by environmental factors of a biological, physical and social nature.

Key words: Dogs, health parameters, chemical pathology, developing country

Rautenbach G.H., Joubert H.F. A comparison of health parameters in two different canine populations. Part II: Chemical pathology data. *Journal of the South African Veterinary Association* **(1988) 59 No. 3 (En.), 135-138 Department of Production Animal Medicine, Faculty of Veterinary Science, Medical University of Southern Africa, 0204 Medunsa, Republic of South Africa.**

INTRODUCTION

The Faculty of Veterinary Science of the Medical University of Southern Africa recently established a clinic in the town of Maboloka in the district of Odi, Bophuthatswana. Maboloka is a rural town with a human population of 26 000 and an estimated canine population of 2 300. The housing is of a moderate to low standard with tin shanty houses being generally present. The average annual income per household is estimated to be under R3 000,00 (approximately 1 470 USA dollars). There are no formal sanitary services in the town.

The occurrence or pattern of diseases observed in this area appeared to be different from that found in more affluent societies[11]. It became apparent that the planning of a health care service to control and prevent disease was a high priority, but no previous data on mortality, morbidity or disease occurrence existed for the area. As these data are needed for the planning of preventive programmes, it became necessary to collect new information in the form of a cross-sectional survey.

A battery of chemical pathology tests was conducted as part of this cross-sectional survey. The aim was to establish laboratory reference values for a population of dogs whose health was relatively unaffected by veterinary intervention as well as to elucidate the prevalence of certain conditions in the population.

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MATERIALS AND METHODS

Two groups of dogs were used. Group I consisted of dogs (n=220) of the town of Maboloka, Bophuthatswana while Group II consisted of dogs (n=101) belonging to the South African Police Dog School. Dogs in Group I were randomly selected while no selection was done for Group II other than to exclude dogs with a record of chronic disease. Dogs under 3 months of age and those with a mass of less than 2,5 kg were excluded from the study, as it was considered deleterious to their general health to collect the amount of blood required for the various tests.

Group I dogs were all individually-owned pets or watchdogs and all the animals in this group were mongrels of a mixed genetic background. Group II animals were all kept in an intensive kennel situation and were composed of 57% German Shepherd dogs, 13% Dobermanns, 12% Bloodhounds, 9% Border Collies and 9% Dobermann-Rottweiler crossbred dogs.

The mean estimated age of Group I was 30,8 months with a range of 3 to 96 months. The mean age of Group II was 21 months.

Of the 220 dogs sampled in Group I, 124 (56%) were male and 96 (43,6%) female. None of the females sampled had been sterilised and only 4 male dogs had been castrated. Group II was composed of 55 male animals (54,5%) and 46 females (45,6%).

The serum samples were collected over a period of 106 d from the beginning of February to the end of May 1985. Approximately 15 ml blood was collected from each animal.

In all cases blood was collected from the *vena cephalica antibrachii* or the *vena jugularis externa;* the first being used in the case of the larger dogs and the latter in that of the smaller dogs. Blood specimens were collected in evacuated tubes and were transported to the laboratory in a coolbag. After the samples had clotted, they were centrifuged and serum was drawn off. The serum was then stored in capped plastic test tubes, frozen for batch testing and kept at approximately −22°C. Storage time varied from 7 to 121 d.

The activities of serum enzymes listed in Table 3 were measured at 30°C with a Flexigem centrifugal analyser (Electro-Nucleonics Inc. Fairfield, New Jersey, USA) and all the reagent kits used for these determinations were Gemini™ manufactured for Electro-Nucleonics Inc. by E. Merck, Darmstadt, F.R. Germany. The methods used were those described by Van Heerden et al.[16]

A Technicon SMA II™ continuous flow analyser (Technicon Instruments Corp., Tarrytown, New York, USA) was used for the following tests:

Sodium (NA^+) and potassium (K^+) levels were determined simultaneously with the Technicon flame photometer using lithium sulphate as an internal reference standard.

Chloride ($C\ell^-$) levels were determined after addition of acidic mercury thiocyanate and ferric perchlorate reagents.

Phosphorus (inorganic) levels were determined using ammonium molybdate in a hydrochloride acid medium. Phosphomolybdic acid which formed was in turn reduced by stannous chloride hydrazine sulphate with the formation of molybdenum blue.

Iron (Fe^{3+}) levels were determined after liberation of iron from its carrier protein by hydrochloric acid. The liberated iron was then complexed with FerroZine in a sodium acetate buffer solution. In order to reduce protein and copper interferences, sodium chloride and neucuproine hydrochloride were added.

Urea levels were determined using the diacetylmonoxime and thiosemicarbazide method.

Creatinine levels were determined using the Jaffe method.

Total serum protein (TP) was determined by using the Biuret reagent. A blank channel for the purpose of correcting all intrinsic interferences was run simultaneously.

Albumin (ALB) levels were determined by using the Bromcresol Green (BCG) dye binding technique.

Except for Na^+, and K^+, colorimetric methods were used for the determination of all the above-mentioned constituents.

Serum protein electrophoresis (SPE): SPE was conducted at buffer pH 8,6 using equipment, cellulose acetate membranes and reagents of Helena Laboratories (Beaumont, Texas). The membranes were stained with Ponceau S and scanned with a Beckmann Model R-115 scanning densitometer (Beckmann Instruments Inc., La Brea, California).

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Total serum calcium (t-Ca) and total serum magnesium (t-Mg) levels were determined with a Perkin-Elmer 5000 ICP atomic absorption spectrophotometer (Perkin-Elmer Corp., Analytical Instruments, Norwalk, USA) using an air-acetylene flame (oxidising) and a 0,16% (v/w) lanthanum oxide solution as diluent. The different wavelengths used for t-Ca and t-Mg determinations were 422,7 nm and 285,2 nm, respectively.

The mean ($\bar{x}$), median (me), standard deviation (S), standard error of the mean (SE), variance (var) and the actual range were determined for each of the measured parameters.

Student's *t*-test was applied in order to compare the mean values obtained between the two groups. Variance was tested as an integral part of this test to assure that the assumptions of the *t*-test were met. Where it was found that the variance between groups was too great to satisfy the assumptions of the *t*-test, log normal transformation was carried out and the *t*-test recalculated on the transformed data. Results were compared between groups and also with recorded reference values.

RESULTS

The values of the different parametes are presented in Tables 1 to 3.

The means of 16 of the 26 parameters evaluated were found to be significantly different ($p < 0,02$) between groups. Six of the 7 enzymes tested differed too much in variance and distribution between groups for Student's *t*-test to be reliable. In these (ALP, AST, LD, CK, ALT and AMS) the p value was calculated after log normal transformation of the data. There was a positive correlation ($r = 0,41$) between urea and creatinine concentrations in Group I.

DISCUSSION

TP and albumin ranges as well as the albumin globulin ratio (A/G) in Group II were similar or close to those reported in the literature[1,4,6,7,13] but those of Group I differed substantially from what was expected of a randomly selected, individually owned dog population. TP and albumin concentrations in Group I were very low and a hypoalbuminaemia and hypergammaglobulinaemia were evident. A significant difference occurred in the mean concentration of TP and albumin in the 2 groups.

The low albumin concentrations in a large percentage of the dogs in Group I are probably a reflection of the degree of malnutrition and parasitism that this group has to contend with; it is unlikely that such a large percentage of the population suffers from liver disease, intestinal malabsorption, renal disease or other rarer causes of hypoalbuminaemia.

The serum protein electrophoretic values clearly resolved in 3 alpha globulin fractions, 2 beta globulin fractions and one gamma globulin fraction in most cases in both groups. Keay[7] found that both alpha 1 and alpha 2-globulins separated into two subfractions while the beta I-globulin separated into 3 subfractions with beta 2-globulin forming a separate fraction. Jordan[5] found that the alpha globulins separated into 2 fractions and the beta globulins into 3 fractions while Lumsden et al.[8] could distinguish 2 alpha and 2 beta fractions only. These differences in interpretation make it difficult to compare results.

The beta 2-globulin values of Group I were substantially higher than those of Group II. It is known that an appreciable amount of immunoglobulins are found in this fraction[1] and it is postulated that this rise in beta 2 globulin fraction is part of a general hyperimmunoglobulinaemia.

The mean gammaglobulin values of the two groups differed significantly. The mean value of Group II corresponded to some degree with that of reference values in the literature[5,7], while that of Group I differed markedly from the expected value of a randomly selected, individually owned canine population. All proteins in the gammaglobulin fraction are known to be immunoglobulins although not all immunoglobulins are found in this region of the electrophoretic strip[1]. A diffusely raised gammaglobulin

Table 1: **Total serum protein and serum albumin concentrations as well as concentrations of different fractions of globulins in 220 dogs from a rural township (Group I) and a kennelled control group (Group II)**

Test	Group	N	$\bar{X}$	ME	SD	S E	Var.	Range	P
TP	I	220	70,48	69,5	16,72	1,13	279,6	24 – 114	0,00004
g ℓ^{-1}	II	70	62,01	61,0	5,88	0,70	34,6	48 – 75	
S-Albumin	I	220	20,19	20,0	4,77	0,32	22,7	7 – 32	<0,01
g ℓ^{-1}	II	70	32,17	31,0	6,15	0,74	37,8	24 – 59	
Alpha I	I	220	2,24	2,18	0,57	0,04	0,323	0,40 – 4,05	0,024
g ℓ^{-1}	II	69	2,07	2,05	0,33	0,04	0,109	1,35 – 3,02	
Alpha II	I	190	3,69	3,52	1,44	0,10	2,066	0,90 – 8,58	0,904
g ℓ^{-1}	II	31	3,73	3,25	1,94	0,35	3,780	1,65 – 12,43	
Alpha III	I	188	5,70	5,67	1,45	0,11	2,117	2,44 – 9,80	0,0004
g ℓ^{-1}	II	31	6,69	6,84	1,09	0,20	1,187	4,76 – 8,63	
Beta I	I	147	7,37	6,94	2,71	0,22	7,375	2,31 – 17,34	0,0298
g ℓ^{-1}	II	49	6,44	5,62	2,42	0,35	5,835	3,42 – 16,88	
Beta II	I	144	9,00	8,80	2,30	0,19	5,284	3,12 – 14,80	< 0,01
g ℓ^{-1}	II	56	6,06	6,67	2,63	0,35	6,928	4,02 – 10,50	
Gamma glob	I	216	22,85	19,04	16,43	1,12	269,8	2,25 – 85,20	< 0,01
g ℓ^{-1}	II	69	5,49	5,10	2,25	0,27	5,065	7 – 15,77	
Alpha II and Alpha III	I	30	7,25	2,36	0,43	5,55	832,5	4,06 – 10,4	0,00109
g ℓ^{-1}	II	38	9,01	9,15	1,89	0,31	3,587	6,72 – 11,98	
Beta I and Beta II	I	70	15,74	15,58	6,40	0,76	40,94	4,21 – 47,9	0,102
g ℓ^{-1}	II	20	13,32	13,06	2,29	0,51	5,252	10,11 – 16,82	

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Table 2: **Serum sodium, potassium, chloride, phosphate, urea, creatinine, total calcium, magnesium and iron concentrations in 220 dogs from a rural township (Group 1) and a kennelled control group (Group II)**

Test	Group	N	$\bar{X}$	ME	SD	S E	Var.	Range	P
Sodium	I	220	144,1	144,0	3,63	0,24	13,2	114 – 154	<0,01
mmol ℓ^{-1}	II	70	147,2	147,0	2,92	0,35	8,5	142 – 154	
Potassium	I	220	4,90	4,9	0,45	0,03	0,2	3,7 – 6,2	0,64
mmol ℓ^{-1}	II	70	4,88	4,9	0,35	0,04	0,12	4,1 – 5,5	
Chloride	I	220	113,4	113,0	3,44	0,23	11,8	105 – 128	0,56
mmol ℓ^{-1}	II	70	113,1	114,0	5,01	0,50	25,1	98 – 124	
Phosphorus	I	214	1,695	1,62	0,48	0,03	0,23	0,82 – 3,20	0,65
mmol ℓ^{-1}	II	70	1,67	1,55	0,39	0,05	0,15	1,15 – 2,69	
Urea	I	220	6,06	5,3	4,61	0,31	21,2	1,3 – 49,0	0,61
mmol ℓ^{-1}	II	70	6,35	6,3	1,71	0,20	2,9	2,4 – 10,8	
Creatinine	I	220	79,33	75,0	35,69	2,41	1273	24 – 496	0,0065
μmol ℓ^{-1}	II	70	91,27	90,0	12,97	1,55	168,3	63 – 127	
Calcium	I	220	2,438	2,45	0,22	0,01	4,8	1,80 – 3,16	<0,01
mmol ℓ^{-1}	II	70	2,623	2,64	0,138	0,02	1,92	2,2 – 2,90	
Magnesium	I	220	0,756	0,75	0,089	0,006	0,008	0,53 – 1,03	0,012
mmol ℓ^{-1}	II	70	0,786	0,79	0,074	0,008	0,005	0,62 – 0,96	
Iron	I	217	18,90	16,3	10,10	0,69	102	1,3 – 71,2	<0,01
μmol ℓ^{-1}	II	70	28,72	27,5	9,62	1,15	92,6	10,1 – 48,5	

Table 3: **Serum concentrations of various enzymes in 220 dogs from a rural township (Group I) and a kennelled control group (Group II)**

Test	Group	N	$\bar{X}$	ME	SD	S E	Var.	Range	P
Alkaline phosphatase	I	220	204,1	145,5	178,6	12,04	31884	20 – 1456	<0,01°
U ℓ^{-1}	II	70	120,8	72,5	283,2	33,8	80206	23 – 2413	
Aspartate transaminase	I	220	21,99	20,0	12,48	0,84	155,8	10 – 167	<0,01°
U ℓ^{-1}	II	70	30,59	25,5	26,29	3,14	691,1	15 – 231	
Lactate dehydrogenase	I	220	314,78	240,5	275,4	18,6	75845	34 – 2452	0,345°
U ℓ^{-1}	II	70	246,7	207,0	135,5	16,2	18348	81 – 750	
Creatine kinase	I	220	91,25	77,5	66,30	4,47	4396	14 – 652	<0,01°
U ℓ^{-1}	II	70	142,89	94,5	109,5	13,1	11993	48 – 599	
Alanine transaminase	I	220	31,59	24,50	32,93	2,22	1084	5 – 422	0,00002°
U ℓ^{-1}	II	70	53,54	33,0	137,6	16,45	18929	12 – 1180	
Gammaglutamyl-transferase	I	220	2,63	2,0	2,66	0,18	7,1	1 – 28	0,035
U ℓ^{-1}	II	70	3,41	3,00	2,82	0,38	7,96	0 – 16	
Amylase	I	219	837,6	806,0	289,7	19,6	83929	166 – 2685	<0,01°
U ℓ^{-1}	II	69	633,4	612,0	168,5	20,3	28392	10 – 1088	

° P calculated on log normal values.

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fraction occurs generally in chronic infectious conditions and, relatively rarely, in auto-immune disease. Van Heerden[15] stated that hypergammaglobulinaemia was one of the clinical pathological findings in canine ehrlichiosis. Polson & Malherbe[10] found an absolute increase in the total globulin fraction in what they called rickettsiosis caused by *Rickettsia canis* (later identified as *Ehrlichia canis*).

It is postulated that the high mean gammaglobulin values in Group I are a reflection of the general state of ill-health of the canine population of Maboloka. Further investigation is needed to determine the incidence of ehrlichiosis in the area.

The mean urea values in the present study were substantially higher than those reported in the literature[1 4 14]. The difference is probably due to laboratory procedure.

There was an unusually broad range of blood urea values in Group I. An increase in serum urea concentration may be caused by factors that are not directly related to the urinary system[9]. It is possible that dehydration played a role in some of the cases in Group I with increased blood urea levels. At the other end of the scale a low protein intake was probably responsible for some of the low values recorded in Group I.

The serum creatinine values of Group II were very similar to those reported previously[1 4] but those of Group I had a substantially broader range. The means of the two groups differed significantly.

Creatinine is produced primarily in muscle tissue by the degradation of creatine. This takes place at a relatively constant rate in individual animals[3]. It is probable that the suspected lesser muscle mass per kg live-mass of the dogs in Group I, together with the lower glomerular filtration rate due to dehydration in some of the dogs of this group, are responsible for the broad range of creatinine values found in this group.

The serum electrolyte values of the present study differed to some extent from previously reported data[1 4 13]. The sodium and calcium values of Group I were appreciably lower than those of Group II. The chloride and phosphorous values of both these groups were appreciably higher than reference values[1 4 12]. Significant differences between the means of Group I and II were found in sodium, calcium and magnesium values, but not in the anion values.

The serum sodium concentration may be used as an index of total body sodium if the extent of hydration is known[4].

Group I generally showed signs of dehydration judged on skin-fold[11]. It is therefore obvious that the subnormal serum sodium values are an indication of depleted total body sodium. The common causes of depleted total body sodium such as vomiting, diarrhoea, and renal disease probably played a part in the results obtained in Group I, but dietary deficiency of salt may have been a contributing factor.

Serum total calcium concentrations are influenced directly by plasma albumin[4]. The mild hypocalcaemia found in Group I was therefore to be expected as an appreciable number of dogs in this group were clearly hypoalbuminaemic.

Serum iron values reported here were appreciably lower than previously reported reference values[6] and a broad range of values were found, especially in Group I. Iron deficiency was expected in Group I as these dogs were generally heavily infested with external and internal parasites[11]. No correlation was found between the number of ova of internal parasites in the faeces and the iron values ($r=0,046$) or the number of ticks collected from the animals in Group I and serum iron values ($r=-0,13$)[11].

Enzyme chemistry results vary widely between laboratories depending on the methods used. This was confirmed with the mean values of some of our tests deviating substantially from published reference values[1 2 4], especially in the case of alkaline phosphatase and lactate dehydrogenase. No specific trends were apparent on analysis of the serum enzyme activity or by comparing group means with one another and with published reference values.

There were 3 obvious deviations in the Group I results: hypoalbuminaemia, hypergammaglobulinaemia and low blood iron levels. It is unlikely that genetic background played a role in these group differences and it is therefore postulated that these differences were caused mainly by environmental factors. External and internal parasites were rife in the Group I population[11], probably playing a role in the hypoalbuminaemia and low iron levels. A high prevalence of chronic infectious conditions was probably the cause of the hypergammaglobulinaemia; poor nutrition presumably played a role in all 3 deviations. Group II had to contend with intensive kennel conditions, but received a balanced diet while scientific management procedures were applied. The chemical pathology test results of animals in this group conformed largely to previously reported reference data and it seems that the negative effects of the intensive kennel conditions were neutralised by good management procedures.

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TRICHOMONIASIS AND CAMPYLOBACTERIOSIS IN BULLS IN THE REPUBLIC OF TRANSKEI

S M PEFANIS*, S HERR*, CATHARINA G VENTER*, L P KRUGER**, CRISTINA C QUEIROGA*** and L AMARAL***

ABSTRACT

Trichomonas foetus was demonstrated in 23/87 (26,4%) and *Campylobacter fetus* in 25/87 (28,7%) of bulls tested in Transkei. A total of 16/87 (18,39%) of bulls tested were positive for both *Trichomonas foetus* and *Campylobacter fetus*. Bulls from 14 sites in Transkei were tested and *Trichomonas foetus* was isolated at 9 of these sites. *Campylobacter fetus* was isolated at 10 of the 14 sites. The results indicate that both *Trichomonas foetus* and *Campylobacter fetus* are widespread throughout the cattle population in the Republic of Transkei and may account for infertility problems.

Key words: *Trichomonas foetus*, *Campylobacter fetus*, Transkei, bulls, infertility

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INTRODUCTION

Campylobacter fetus (originally known as *Vibrio fetus*) was first identified in South Africa in 1931[9] and *Trichomonas foetus* in 1937[6]. By 1954 "*Vibrio foetus*" had been established as a cause of infertility in all 4 provinces of the then Union of South Africa[10]. These early reports give no detail of the cattle breeds or the districts involved, and no work on either disease has since been published in South Africa. There are no previously published reports on the prevalence of these 2 diseases in the Republic of Transkei. This study was undertaken in response to a request from the Department of Agriculture and Forestry of the Republic of Transkei to assist with an investigation into the occurrence and incidence of these two diseases in the area as infertility amongst cattle had been widely experienced (Beserati 1987 Director of Veterinary Services, Transkei, unpublished data).

MATERIALS AND METHODS

Animals

Bulls (n=87), selected from 14 different sites in the Republic of Transkei, were used in this study. The object of selection was to determine whether these diseases occurred in the Republic of Transkei, and no attempt was made to perform an epidemiological survey. A number of criteria were used to select the animals to ensure the best chance of isolating these organisms. These included using areas where poor conception rates had been reported and often where a communal grazing system was used. Districts and sites had to be within easy travelling distance of the Umtata Veterinary Laboratory to ensure the survival of the organisms. Twelve out of the 27 districts in the Republic of Transkei were selected. Wherever possible, specimens were collected from bulls older than 4 years as these were the animals most likely to be infected[1]. Although an attempt was made to select only bulls older than 4 years, they ranged in age from 1,5 to 10 years. The bulls were aged by examining the teeth as well as by obtaining the approximate age from the owner. The average age of the bulls was 5-6 years. The highest number of bulls sampled in any single district was 14 with the number for other districts ranging between 3 and 8. The breeds of bulls varied and included Nguni, Afrikaner, Jersey and Simmentaler crossbred types.

Sampling

Preputial samples were collected from each bull by infusing 50 mℓ of phosphate buffered saline (PBS; pH 7,2) into the prepuce of the bull. The prepuce was then held closed at the anterior orifice and massaged vigorously 100 times. The PBS was then collected again and poured into a 50 mℓ bottle. Bottles were packed on ice at 4°C, and transported in a container to protect them from sunlight, to reach the laboratory within 6 to 8 h of collection[5].

Culture techniques

Specimens were centrifuged at 1 200 g for 10 min as soon as possible after receipt. Five mℓ of the supernatant was immediately drawn up into a sterile plastic syringe and the remaining supernatant was decanted into a sterile 100 mℓ centrifuge tube with a cap. The sediment was resuspended in the final few drops. Three drops of this suspension were inoculated into each of the *Trichomonas* media (see below). The cultures were incubated at 32°C and examined after 48, 96 and 144 h. A fourth drop was subjected to direct observation by examination under a cover slip using phase contrast microscopy. A 0,65 μ millipore filter[8] in a Swinney adaptor was attached to the syringe containing the supernatant. The first 4 mℓ of filtrate was discarded and 3 drops of the final 1 mℓ filtrate were dropped on to each of 5 agar plates (see below). The drops were spread using a sterile glass spreader. These agar plates were then incubated at 37°C in an atmosphere containing 5% O_2, 10% CO_2 and 85% N_2[1,3]. The plates were examined at 48 and 96 h for *Campylobacter* colonies. The remainder of the supernatant in the capped centrifuge tube, was centrifuged at 5 000 g for 30 min. This sediment was used for a fluorescent antibody test (FAT) using a conjugate prepared at the Section of Reproduction of the Veterinary Research Institute (VRI), Onderstepoort, from a *Campylobacter fetus* subsp. *venerealis* biotype *intermedius* strain known to give good results in the FAT.

Media and typing

Trichomonas: Two media were used for the culture of *Trichomonas foetus*. The first, a modification of Plastridge's medium[4], was a nutrient broth containing 10% D+ glucose, 20% horse serum and 0,07% agar. The second medium was a modification of Stenton's medium[3] also made up in a nutrient broth but containing 0,075% ascorbic acid, 10% inactivated horse serum and 0,1% agar with a layer of liquid paraffin on top. No attempt was made to serotype the *Trichomonas* isolates.

Campylobacter: Five nutrient agar plates were used consisting of 1 tryptose agar with 10% horse blood without any antibiotics (BTA), 2 BTA plates each containing 1 μg brilliant green ml^{-1} and 5 μg novobiocin ml^{-1} and lastly 2 BTA plates with vancomycin 20 μg ml^{-1}, trimethoprim 10 μg ml^{-1}, polymixin B sulphate 5 IU ml^{-1} and actidione 100 μg ml^{-1}. All isolates of catalase-positive *Campylobacter* were typed according to the biochemical tests and methods described[7].

RESULTS

The distribution of *Trichomonas* and *Campylobacter* positive samples in relation to the different regions tested in the Republic of Transkei is shown in Table 1. *Trichomonas foetus* was found in 9 out of the 14 sites tested, *Campylobacter fetus* was found in 10 of the 14 sites tested.

Of the bulls tested 23/87 (26,4%) were positive for *Trichomonas*, 25/87 (28,7%) were positive for *Campylobacter* and 16/87 (18,39%) were positive for both *Trichomonas* and *Campylobacter*.

The age distribution of the positive *Trichomonas* and *Campylobacter* bulls is shown in Table 2.

All isolates of *Campylobacter* were typed as *Campylobacter fetus*

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Table 1: **The distribution of *Trichomonas* and *Campylobacter* infection in bulls according to the sites tested in the Republic of Transkei**

Site	Number of bulls tested	Positive for Trichomonas	Campylobacter Culture	Campylobacter FAT
Mjanyana (Engcobo)	8	0	0	0
Elliotdale	7	0	0	0
Mqanduli	8	0	0	1
Engcobo	5	1	1	1
Qumbu	3	1	1	2
Tsolo	5	1	1	1
Mt Frere	8	5	3	6
Umtata	6	1	0	0
Port St Johns	5	0	2	3
Lusikisiki	7	3	0	3
Idutywa	14	7	4	6
Butterworth	6	2	1	1
Nqamakwe	3	0	0	0
Tsolo	2	2	0	1
Total	87	23	13	25

Table 2: **The age distribution of bulls possitive for *Trichomonas foetus* and *Campylobacter fetus* in the Republic of Transkei**

Age distribution in years	No. of bulls tested	Positives Trichomonas	Campylobacter Culture	Campylobacter FAT
0-3	11	0	0	2 (18%)
4-8	68	17 (25,0%)	11 (17%)	20 (29,4%)
8 years	8	6 (75%)	2 (25%)	3 (37%)
Total	87	23	13	25

subspecies *venerealis* biotype *intermedius* except for one culture from Butterworth which typed as *Campylobacter fetus* subspecies *fetus*.

DISCUSSION

The small number of animals tested in the 0-3 year old range is due to specifically older bulls having been selected for this investigation. It is, however, very interesting to note that there were no positive *Trichomonas* isolates from these young bulls and that only 2 out of the 11 bulls tested were positive on the FAT test for *Campylobacter*. This is in agreement with the findings of other authors that trichomoniasis is not usually a problem in younger bulls[1]. These results show that bulls of 4 years and older are likely to be the carriers of infection in the herd and this is in accordance with other reports[1,3]. It is also reasonable to assume that, due to the communal grazing system at these sites, both diseases may be more widespread than revealed by this investigation. It is, however, almost impossible to determine the impact of these two diseases on reproduction in the Republic of Transkei as there is no effective way of evaluating these findings in relation to the number of cows which could possibly already be immune to these diseases. It is very obvious though that if one assumes that the diagnosis of one positive bull for either of these diseases in a herd indicates a positive herd[1], then 71% of the herds tested are positive for at least one of these two diseases. BonDurant[1] suggests that the precise number of bulls infected may not be as important as the number of herds infected. This would indicate that trichomoniasis and campylobacteriosis may play a significant role in infertility in the Republic of Transkei.

The control of campylobacteriosis does not really present much of a problem in this situation and as there appears to be no fixed breeding season for most of these herds, vaccination of all breeding animals twice a year has been suggested. The control of trichomoniasis does, however, present a few practical problems as treatment, closed herds, culling of infected bulls and artificial insemination are totally impracticable. The people of Transkei attach exceptional status value to their cattle and would not easily be induced to cull bulls under any circumstances. Even though many assurances were given, these cattle owners viewed the collection of samples with great suspicion and in some areas even refused to bring the bulls in to be tested. The suggestion given below for the control of trichomoniasis was based on this resistance to culling and the Australians' encouraging results[2] in controlling trichomoniasis by culling older bulls and using two-year old bulls[2]. It was suggested that a bull breeding station be opened for the breeding of good quality bulls. Older bulls should then be exchanged for two-year old bulls from the breeding station and the older bulls fattened and slaughtered to defray costs. The introduction of good quality two-year old bulls should assist in the control of trichomoniasis as well as in a gradual upgrading of cattle over a period of years.

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THE USE OF GAMMA LINOLENIC ACID, LINOLEIC ACID AND NATURAL VITAMIN E FOR THE TREATMENT OF MULTICENTRIC LYMPHOMA IN TWO DOGS

JUNE H WILLIAMS*

ABSTRACT

The use of Gamma linolenic acid (GLA) and other essential fatty acid (EFA) metabolites in malignant cancer treatment in vitro and in vivo is briefly reviewed. Treatment of two dogs with multicentric lymphoma with large empirical daily doses of a combination of 40 mg gamma linolenic acid, 350 mg linoleic acid and 10 mg natural vitamin E per capsule resulted, after approximately one week, in slight to marked reduction in size of enlarged peripheral lymph nodes, spleen, skin nodules and tonsils. Both animals showed transient improved habitus and appetite, but deteriorated due to complications, apparently unrelated to therapy.

Key words: Essential fatty acids, gamma linolenic acid, vitamin E, reversability of cancer, lymphoblastic lymphosarcoma, dogs.

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INTRODUCTION

Horrobin[16] in 1980 hypothesised that it should be possible to "normalise" malignant cancer cells and thus reverse cancer growth by restoration of normal prostaglandin E1 (PGE1) synthesis. He suggested that this could be done by providing gamma linolenic acid (GLA) or dihomogammalinolenic acid (DGLA) which then bypasses the enzyme delta 6 desaturase[11 18], which converts the essential fatty acid (EFA) linoleic acid (LA) to GLA. Delta 6 desaturase in man apparently does not function effectively after puberty and is also inhibited by several dietary and environmental factors including trans-fatty acids, saturated animal fats, excessive glucose, deficient protein, alcohol, glucocorticoids (stress), zinc deficiency, starvation, ageing, oncogenic viruses and ionising radiation[8 18]. GLA and DGLA proceed to form the one-eicosanoid series of metabolites including PGE_1, which together with thromboxane A2 has effects which make it able to reverse the metabolic abnormalities common to most malignant cancer cells. This would imply a simple, non-toxic approach to cancer treatment, and possibly even prevention of malignant cancer by simple dietary supplementation of GLA or DGLA[16].

It has since become evident in various in vitro trials[2 3 4 7 10 12 13 19] and in trials in laboratory animals[6 9 14] that GLA and DGLA plus some other fatty acids and eicosanoid products can inhibit proliferation of various types of malignant cancer cells. Linolenic acid, apart from being converted normally to gamma linolenic acid which has a suppressive effect, has also been shown on its own to suppress proliferation of human osteogenic sarcoma cells in tissue culture[4]. Since vitamin E is stored in the body and is rarely deficient, the effect in vivo of supplementation in small amounts, as in the capsules used in this report, is unsure. Booyens[5 8] has proposed that the problem lies in a chronic arachidonic acid:eicosanoid imbalance and that this is a common feature in important diseases prevalent in the world today, amongst others coronary artery disease, hypercholesterolaemia, cancer, chronic inflammatory and auto-immune disorders, allergic eczema and premature ageing. The imbalance arises from the body's inability to utilise linolenic and linoleic acids in the trans form (as it occurs in heat-treated products) for eicosanoid synthesis. Trans-form fatty acids may be used for energy-production only, whilst cis-form EFA may be converted to eicosanoids. Arachidonic acid is plentiful in meat, dairy products and plants, but linolenic and linoleic acids are derived from plants only.

Provision of GLA enables increased PG1 series synthesis, and PGE1 is known to stimulate T-cell and suppressor-T-cell function, both of which may play a role in the body's own immunity to tumour cells[15 17 20]. Since zinc, nicotinic acid, pyridoxine and ascorbic acid are co-factors in the formation of PGE_1 from polyunsaturates, they could be supplemented and possibly enhance the effect of GLA in the treatment of malignant tumours[15].

Trials in human cancer patients with various malignancies are in progress using GLA in high doses (eg. 36 capsules daily) and have had variable results: some showed slight improvement before relapse and death, some show promising improvement and a few have apparently gone into remission to date[21 22]. Two of the main advantages have been the lack of toxic side-effects in terminal patients and general improvement of patient-habitus (C F van der Merwe 1987 Personal communication, Department of Internal Medicine, Faculty of Medicine, Medical University of Southern Africa).

GLA occurs in quantity in human breast milk and some plants including the oil-of-the-evening primrose. "Efamol G" capsules (Efamol Ltd, 40 Warton Road, London E152JU, UK) contain approximately 40 mg GLA, 350 mg linoleic acid and 10 mg natural Vit E in a gelatin and glycerine capsule of ½ cm^3 volume.

This paper reports on the use of Efamol G capsules alone as treatment in 2 adult dogs over 5 years of age of different breed and size, both with lymphoblastic lymphosarcoma.

CASE 1

History and clinical signs

A 7 year-old male dog of nondescript large breed, was presented with marked enlargement of all superficial lymph nodes, tonsils and the spleen (Table 1). The dog was thin (with a body mass of 41 kg) and appeared moderately depressed on presentation.

Diagnosis, clinical pathology and treatment

The right mandibular lymph node was removed under general anaesthesia before treatment started and histological examination confirmed a diagnosis of lymphoblastic lymphosarcoma. The blood urea (BU) and creatinine levels of the dog were elevated before treatment began (Table 2).

Efamol G was dosed over a period of 39 d (Table 3) starting at 20 capsules per day (10 capsules twice daily) and after 6 d the lymph nodes showed marked reduction in size (the left mandibular lymph node was half the original size) and they were palpably softer (Table 1). The tonsils were sufficiently reduced in size to allow the dog to start eating and its habitus improved.

On Day 21, examination of the left mandibular lymph node (after its surgical removal) showed marked reduction in size and histologically there was poor cortico-medullary differentiation and prominent necrosis of neoplastic cells. On palpation, the spleen appeared to be reduced to approximately normal size (Table 1) and the tonsils appeared normal.

On Day 24, the dog was discharged with a body weight of 33 kg (he would only eat chunks of raw lean meat but refused commercial dog food) and its habitus was good. On Day 38, the patient was readmitted in a state of depression and dehydration and exhibiting signs of uraemia (Table 2). Euthanasia was performed on request of the owner.

Pathology

The post mortem investigation revealed uraemic changes and multicentric lymphoma involving the lymph nodes, spleen and portal areas of the liver (Table 1). The liver was moderately enlarged. On

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Table 1: **Measurements of peripheral lymph nodes, tonsils and spleen of Case 1 before and after treatment (all measurements made via palpation through the skin except those at post *mortem*)**

	Before treatment	Day 6	Day 7	Day 10	Day 21 Surgery	Day 31	Day 38	Post mortem
Mandibular lymph nodes	L 5x3 cm R 5x3 cm (removed)	L 2,5 cm long	Not measured	Not measured	L 3x1 cm (removed)			
Prescapular lymph nodes	L 8x3 R 5x2,5	L 5x3 R *normal size*, soft	L 4 cm long	L 3 cm long	L 2,5 cm	L 2,5 cm	L 2,5 cm	L 4x2.5x1.5 R 3,5x2,5x2
Politeal lymph nodes	L 4 cm long R 4 cm long	Just palpable Just palpable	Not measured	Not measured	L 2x2 cm R 2x2 cm	L 3x2 cm R 3x2 cm	L 3x2 cm R 3x2 cm	L 4,5x2,5x1,3 R 3x2,5x1,1
Tonsils	4x1,5-2 white bulging	Not examined	Not examined	Not examined	Not enlarged, flat	Not enlarged	Not examined	Not enlarged
Spleen	Midway up right flank, firm, smooth	Not measured	Not measured	Only just palpable behind left costal arch	Not enlarged	20x9 cm	Not measured	30 cm long, focal disseminated soft nodules ,5-1,5 cm white pulp more prominent in nodules

Table 2: **Haematological and blood chemical parameters before and during treatment of Case 1**

	Before treatment	Day 7	Day 13	Day 20	Day 32	Day 39
Red cell count $1x10^{12}$ ℓ^{-1}	6,3	4,7	4,4	4,2	4,7	4,5
Haematocrit	,44	,33	,31	,31	,36	,32
White cell count $1x10^{9}$ ℓ^{-1}	10,3	6,7	8,0	6,1	21,0	12,5
Polymorphonuclear cells %	75	88	82	75	85	81
Stab. Band cells %	2	2	3			
Lymphocytes %	19	7	13	19	5	12
Monocytes %	1	3	1		1	
Eosinophils %	3		1	6	2	7
Anisocytosis	+	+	+	+	+	+
Platelets $1x10^{9}$ ℓ^{-1}	77 000	87 000	144 000	303 000	493 000	182,000
Urea mmol ℓ^{-1}	32,7		23,0	32,5	25,9	28,3
Creatinine μmol ℓ^{-1}	357		312	489	486	523

Table 3: **Treatment of Case 1 with Efamol G**

	Day 1	Day 18	Day 19	Day 24	Day 30	Day 38	Day 39	Day 42
1. Efamol G	10 caps bid	15 caps divided bid	6 bid	5 bid	8 bid	8 caps once daily	Efamol stopped	Euthanased

2. Doxycycline 10 mg kg^{-1} from Day 10 to Day 20 (due to possibility of concurrent *Ehrlichia canis* infection causing drop in WCC and low platelets although no morulae were seen)
3. Mebendazole 100 mg d^{-1} for 5 d from Day 24 as broad-spectrum anthelmintic (routine deworming)

Table 4: **Measurements of lymphoid tissue (Case 2) (all measurements made via palpations through the skin)**

	Before treatment	Day 6	Day 14	Day 17
Skin	Nodules 2x1 cm	Disappeared	Disappeared	Disappeared
Mandibular lymph nodes	Enlarged	Smaller	Smooth, fairly firm 4x3 cm	R 3x2 L 2x1
Prescapular lymph nodes	L 10x4	L 8x3	R 4x3	L 4x3 R 3x3
Popliteal lymph nodes	Both enlarged	Smaller	2,5 cm (both)	R 3x2 L 3x2
Spleen	Not enlarged		5 cm to right of midline	Still enlarged
Eyes	White sediment anterior chambers	Virtually clear (both)	White sediment again present in anterior chambers	

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histological section, the lymphnodes showed absence of cortico-medullary differentiation. The mitotix index was low. There was scattered necrosis of lymphoblastic cells through all the neoplastic tissue. The kidneys showed radial calcification in the medulla, tubular hyaline droplet degeneration, moderate focal disseminate periglomerular fibrosis and glomerular calcification plus scattered cortical areas of plasma cell and lymphocyte accumulation. There was lower mucosal calcification of the stomach.

CASE 2

History, clinical signs and treatment

A spayed 8-year old Beagle bitch was admitted to the hospital showing two slightly ulcerated skin nodules on the mid-dorsal aspect of the cranium, white sediment ventrally in the anterior chambers of both eyes, marked enlargement of the politeal, mandibular and prescapular lymph nodes, and enlarged tonsils. The spleen was not enlarged (Table 4).

Histopathological examination of a skin nodule and part of the left prescapular lymph node showed a very active lymphoblastic lymphosarcoma.

Treatment with Efamol G (7 capsules b i d) was started as well as a 7-day course of procaine penicillin as post-surgical treatment. From Day 5 after treatment began, the dose of Efamol G was increased to 10 capsules twice daily per os. A reduction in size of affected peripheral lymph nodes and skin nodules occurred until euthanasia was performed at the owner's request on Day 18 after admission (Table 4). At this latter stage the dog had suddenly developed a head tilt to the right and had no facial tone on the right hand side. The white sediment (which had cleared for a while) had returned to the eyes.

Macroscopic pathology

On post mortem examination, the skin nodules on the head were observed to be hard and indurated and one nodule on the left thoracic wall was 1 cm in diameter and poorly-defined. The bone marrow showed red metaplasia with a number of irregular white areas. The spleen was mildly enlarged and showed only prominent lymphoid hyperplasia but not neoplasia. The liver was swollen, light in colour, with prominent lobulation and showed indistinct light mottling throughout. There were also isolated, well-defined but non-encapsulated nodules throughout the liver parenchyma of 1-1,5 cm in diameter. There was white precipitate in the anterior chamber of the right eye and prominent retrobulbar haemorrhage along the right optic nerve. The cause of the head tilt and loss of facial tone was not established. The lungs showed mild emphysema and congestion.

Histopathology

Examination of tissue sections showed the ciliary body and iris of both eyes to be infiltrated with neoplastic (lymphoblastic lymphosarcoma) cells, plus neoplastic cells floating free in the anterior chambers. There was a homogenous population of large lymphoblastic cells in the lymph nodes (and their capsules) with focal disseminate areas of neoplastic cells especially around blood vessels.

The liver nodules consisted of diffuse but particularly periportal infiltration of neoplastic cells, and loose areolar connective tissue showed perivascular infiltrates. The bone marrow appeared hyperplastic but not neoplastic. A section of skin showed a small focus of viable neoplastic cells and the small intestine showed focal subserosal infiltrates. There was a large splenic infarct associated with thrombosis.

The pathological anatomical diagnosis was multifocal lymphoblastic lymphosarcoma. Prominent necrosis of neoplastic aggregates was observed in sections of the lung and liver.

DISCUSSION

Cases 1 and 2 showed definite and obvious regression of cancerous tissue due to necrosis of neoplastic cells and an apparent decrease in proliferation. Although there were still tumour cells present in the lymph node removed after Day 20 of treatment in Case 1, the node was showing fairly recognisable structure with peripheral lymphoid follicles and there were many normal lymphocytes: features which were not apparent in the pre-treatment node. There were also areas of necrotic dark-staining spindle-shaped cell remnants.

This response to GLA and various other essential fatty acids, eicosanoids and vitamins has been seen in vitro (see introduction) in several malignant tumour cell lines[2 3 4 7 10 12 13], as well as in laboratory animal trials[6 9 14] and also in cultured tumour cells from biopsy material from a few canine patients (J W Williams 1987 Unpublished data) and human patients (N Dippenaar 1987 Personal communication, Department Physiology, Medical University of Southern Africa). In vivo, there may be a combination effect — the biochemistry of the malignant cells depending on a lack of the specific substance/s used (in this case GLA, vitamin E and linoleic acid), and/or a stimulation of the animal's immune system to cope with the tumour.

It might be speculated that the necrosis of neoplastic cells seen in the liver and lung sections of Case 2 may have been due to high doses of fatty acids (GLA & LA) reaching those 2 organs via the portal circulation but insufficient doses reaching the tissues more peripheral in the circulation. That the neoplasia appeared to relapse was possibly due to ineffective dosages of GLA & LA being maintained. Similar findings of tumour regrowth have been found in some human patients (C F van der Merwe 1987, Personal Communication, Faculty of Medicine, Medical University of Southern Africa) and now high doses are being used and maintained indefinitely. In humans, it appears that certain tumours react more favourably than others. Whether this is tumour-related, dose-related or associated with variable circulatory access to tumour cells is not yet known. Another possibility is that some tumours develop resistance to certain chemotherapeutic agents possibly by changing their biochemistry and thus response is only transient.

It seems clear that these 2 cases suggest that further research is warranted in the use of GLA, other fatty acids and vitamins as treatment for malignant cancers in man and animals. One of the main advantages in the use of GLA is the fact that it is an essential fatty acid derivative and therefore a normal physiological supplement for cellular biochemistry, and there are no serious adverse side-effects. Most humans and animals treated so far have shown improved habitus and general health during the treatment.

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EIENAAR-HOND-VERHOUDINGS — 'N DEKADE LATER

J S J ODENDAAL* en D R OSTERHOFF*

ABSTRACT

The first cynological study in South Africa was done in 1976. To establish possible new tendencies in dog ownership, the study was repeated a decade later. The questionnaire included questions on breed, nutrition, care, reproduction, behaviour, training and replacement, and the information was gathered by door to door visits. External factors such as the need for specific breeds for specific situations (security, economics) in the country, availability of reading matter about dogs, the convenience of commercial dog foods, the growing tendency for mothers to enter into fixed employment, campaigns against undesired breeding, improved veterinary services, reports on canine heroism in the media, could all have influenced the patterns of dog ownership. The awareness of these tendencies could be of importance to the companion animal veterinarian.

Key words: Owner-dog relationship, cynological survey

Odendaal J.S.J., Osterhoff D.R. Owner-dog relationships — a decade later. *Journal of the South African Veterinary Association* **(1988) 59 No. 3 145-148 (Afrik) Department of Zootechnology, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort, Republic of South Africa.**

INLEIDING

Gedurende 1976 het Osterhoff[1] die eerste uitgebreide kinologiese ondersoek in Suid-Afrika uitgevoer. Die 46 vrae van die ondersoek het gepoog om insig te gee in die verhouding tussen eienaars en hulle honde. Die houding van honde-eienaars teenoor hulle diere bly egter nie noodwendig dieselfde nie. Eksterne faktore kan byvoorbeeld die patroon van honde-eienaarskap beïnvloed.

Hierdie studie is derhalwe onderneem om moontlike veranderings in die verhouding tussen honde en hul eienaars oor die afgelope 10 jaar vas te stel.

MATERIAAL EN METODE

Vraelyste (met geringe wysigings) is opgestel in ooreenstemming met die ondersoek van Osterhoff[1]. Wysigings het die volgende ingesluit:

(a) By Vrae 9 en 14 is aanpassings (met inagneming van die inflasiekoers) gemaak ten opsigte van die geldwaardes.

(b) Vrae 9, 27, 35, 40 en 41 in die 1976-vraelys[1] is in die 1986-vraelys uitgelaat om oorvleueling te verminder. Vraag 20 van 1976 is saamgevoeg met vraag 19 in 1986.

(c) By Vrae 26, 30 en 31 is meer relevante moontlikhede vir antwoorde bygevoeg. Vraag 25 se moontlikhede is uitgebrei met "Operasie", Vraag 30 met nege gedragsmoontlikhede ná "Loop rond" en Vraag 31 met "Veearts".

(d) By Vrae 21, 34 en 36 is enkele moontlikhede by die antwoorde weggelaat. Vraag 21 se moontlikhede is verkort deur "Honde-toilette" uit te laat, Vraag 34 verkort deurdat die moontlikhede van die antwoord verander is na "Ja" en "Nee" en Vraag 36 verkort deurdat "Toertjies" uitgelaat is.

Gedurende 1986 is aan die tweedejaar studente aan die Fakulteit Veeartsenykunde, Universiteit van Pretoria, opgedra om die vraelyste deur honde-eienaars te laat voltooi, deur middel van deur-tot-deur besoeke. Die inligting wat aldus versamel is, is verkry vanaf honde-eienaars dwarsdeur die land. Die vraelyste is gekodeer en op 'n rekenaar verwerk.

Die antwoorde verkry met die huidige ondersoek is afgerond tot die naaste persentasie-punt en is, waar van toepassing, vergelyk met dié van die vorige ondersoek[1].

RESULTATE

Van die 500 vraelyste wat uitgestuur is, is 408 terug ontvang.

Vraag 1: Hoeveel honde besit u

Aantal honde aangehou	% Eienaars	
	1986	1976
Een	45	54
Twee	34	29
Drie	15	9
Vier en meer	6	8

Vraag 2: Watter ras is u hond(e)?

Honderas	Persentasie	
	1986	1976
Kruisras	30	28
Malteser	10	3
Duitse Herdershond	8	5
Dobermann	5	2
Labrador	5	5
Rottweiler	5	(0-<2)
Fox Terrier	4	3
Franse Poedel	4	8
Dachshund	3	4
Staffordshire Bull Terrier	3	(0-<2)
Spaniel	2	3
Corgi	2	3
Engelse Bulhond	2	2
St Bernard	2	(0-<2)
Toy Pom	2	6
Border Kollie	2	(0-<2)
Schipperke	(0-<2)	3
Boxer	(0-<2)	3
Keeshond	(0-<2)	2
Ander	11	21

Vraag 3: Wat is die geslag van u hond?

Geslag van hond	Persentasies	
	1986	1976
Manlik	52	51
Vroulik	48	49

Vraag 4: Van wie het u die hond gekry?

Oorsprong van hond	Persentasies	
	1986	1976
Private Persoon	43	38
Teler	27	22
Geskenk	11	12
Welsynsorganisasie	7	11
Optelhond	4	2
Troeteldierwinkel	3	6
Eie teling	4	7
Ander	1	2

Vraag 5: Lees u enige boeke oor honde?

Onderwerpe vir lees oor honde	Persentasies	
	1986	1976
Algemeen	36	45
Siektes	7	3
Afrigting	12	5
Teling	6	4
Geen	39	43

Vraag 6: Hoe lank besit u u hond?

Tydperk van honde besit	Persentasies	
	1986	1976
Onder een jaar	7	7
Een jaar	9	8
Twee jaar	12	12
Drie jaar	14	14
Vier jaar	11	11
Vyf jaar	10	9
Ses jaar	7	8
Sewe jaar	5	7
Agt jaar	8	7
Nege jaar	4	5
Tien jaar en langer	13	12

Vraag 7: Sien u kans om sonder 'n hond te lewe?

Kan die eienaar sonder 'n hond lewe?	Persentasies	
	1986	1976
Nee	73	85
Ja	16	12
Onseker	11	3

Vraag 8: Waarom hou u 'n hond aan? (Moontlike antwoorde voorsien).

Redes vir eienaarskap van honde	Persentasies	
	1986	1976
Plesier	33	29
Beskerming	27	16
Gehegdheid	16	21
Het altyd honde gehad	9	11
Ander	15	23

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Vraag 9: Dui u maandelikse inkomste aan.

Maandelikse inkomste in Rand	Persentasie 1986	1976	
Minder as R500	4	3%	(R250)
R 500-R1 500	12	14%	(R250-500)
R1 500-R2 500	18	19%	(R500-750)
R2 500-R3 500	17	23%	(R750-1 000)
Meer as R3 500	17	24%	(R1 000>)
Wil nie sê nie	32	17%	

Vraag 10: Hoe dikwels voer u u hond per dag?

Aantal voedings per hond per dag	Persentasies 1986	1976
Een keer	52	63
Twee keer	42	34
Meer dikwels	6	3

Vraag 11: Wat voer u u hond hoofsaaklik?

Bron van hoofvoedsel	Persentasies 1986	1976
Hondekorrels	52	34
Vars vleis	9	22
Tafel oorskiet	17	18
Blikkieskos	12	16
Afval vir honde	7	7
Ander (vis)	3	3

Vraag 12: Gee u u hond bene?

Hoe dikwels kry die hond bene	Persentasies 1986	1976
Elke dag	13	19
Een keer per week	29	24
Meer as een keer per week	37	35
Selde	15	13
Nooit	6	9

Vraag 13: Gee u u hond lekkergoed?

Kry die hond lekkergoed?	Persentasies 1986	1976
Nooit	60	57
Soms	34	39
Dikwels	6	4

Vraag 14: Hoeveel geld spandeer u jaarliks op hondekos?

Geld in rand spandeer op hondekos per jaar	1986	1976	
Minder as R100	17	40%	R 50)
R100-R150	23	35%	(R100-150)
R150-R200	16	10%	(R100-150)
R200-R250	14	5%	(R150-200)
Meer as R250	23	1%	(R200>)
Weet nie	7	9%	

Vraag 15: Wie versorg meesal die hond?

Hoofversorger van die hond	Persentasies 1986	1976
Moeder	47	56
Vader	23	16
Kinders	22	21
Huishulp	8	7

Vraag 16: Hoeveel tyd word aan die hond per dag bestee?

Tyd deur eienaar aan hond bestee	Persentasies 1986	1976
Minder as 'n halfuur	18	16
Een uur	34	43
Twee ure	15	14
Meer as twee ure	31	26
Geen tyd	2	1

Vraag 17: Hoe dikwels word die hond gebad?

Badpatrone van honde	Persentasies 1986	1976
Een keer per week	13	13
Een keer per maand	36	42
Soms	37	33
Nooit	14	12

Vraag 18: Aan wie is die hond die meeste geheg?

Persoon aan wie die hond die meeste geheg is	Persentasies 1986	1976
Moeder	44	42
Vader	23	27
Kind oor 15 jaar	18	16
Kind onder 15 jaar	9	14
Huishulp	1	1
Onseker	5	–

Vraag 19: Aan wie is die hond die meeste gehoorsaam?

Persoon aan wie die hond die meeste gehoorsaam is	Persentasies 1986	1976
Moeder	31	42
Vader	44	36
Kind oor 15 jaar	18	15
Kind onder 15 jaar	5	6
Huishulp	2	1
Onseker	5	–

Vraag 20: Wie pas die hond vakansies op?

Hondeversorging tydens vakansies	Persentasies 1986	1976
Dierehotel	21	30
Bure/vriende	37	30
Neem saam	15	18
Huishulp	27	22

Vraag 21: Wat van ontlasting op straat?

Opruim van honde-uitskeidings	Persentasies 1986	1976
Eienaar se verantwoordelikheid	72	68
Voorsien spesiale areas	9	17
Hou honde uit sekere areas	6	7
Onseker	13	8

Vraag 22: Hoe dikwels besoek u 'n veearts?

Honde-eienaars se besoeke aan veearts	Persentasies 1986	1976
Net wanneer nodig	73	87
Een keer per jaar	14	1
Gereeld	9	11
Nooit	4	1

Vraag 23: Hoe gereeld teel u met u teef?

Teelfrekwensie met tewe	Persentasies 1986	1976
Nooit	73	44
Een keer	14	37
Twee keer	7	8
So dikwels moontlik	6	11

Vraag 24: Sal u 'n gesteriliseerde teef verkies?

Keuse oor sterilisasie van 'n teef	Persentasies 1986	1976
Ja	84	69
Nee	16	31

Vraag 25: Sal u 'n gekastreerde reun verkies?

Keuse oor kastrasie van 'n reunhond	Persentasies 1986	1976
Ja	37	16
Nee	63	84

Vraag 26: Hoe verhoed u dragtigheid in 'n teef?

Geboortebeperking in 'n teef	Persentasies 1986	1976
Operasie	64	(<37)
Toesluit	13	27
Hondehotel	5	19
Tablette	6	17
Ander	12	37

Vraag 27: Noem 3 uit elf gegewe eienskappe in u hond wat die belangrikste is.

3 Belangrikste eienskappe van hond vir eienaar
Goeie temperament
Getrouheid
Intelligensie

Vraag 28: Laat u die hond toe:

Vryhede aan hond toegelaat deur eienaar	Persentasies 1986	1976*
In die huis	50	75
In die eetkamer	23	37
In die motor	8	36
Op stoele	3	31
Om kos tydens etes te vra	1	14
Om teen besoekers te spring	2	16
Niks van bogenoemde	13	–

* Meer as een kombinasie is toegelaat

Vraag 29: Waar slaap die hond?

Slaapplek van hond	Persentasies 1986	1976
In die kombuis	22	30
Hondehok	39	26
Slaapkamer	17	19
Buite	8	11
Op bed	5	11
Ander	9	3

Vraag 30: Enige eienaardige gedrag?

Eienaardige gedrag van hond	Persentasies 1986	1976
Nee	27	42
Bang vir onweer	17	–
Jaloesie	12	–
Urienmerking (in die huis)	8	3
Baklei	7	–
Rondloop	5	3
Te veel blaf	5	10
Verniel tuin	–	10
Kou skoene/meubels	–	7
Lek	–	5
Jaag motors	–	5
Hap na denkbeeldige voorwerpe	–	4
Ander	19	11

Vraag 31: Is u hond antagonisties teenoor:

Antagonisme van hond teenoor:	Persentasies 1986	1976
Veeartse	4	–
Mans	1	–
Vrouens	1	–

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Kinders	2	–
Katte	20	21
Wit mense	3	5
Swart mense	40	34
Ander diere	–	8
Ander honde	13	20
Glad nie	16	12

Vraag 32: Het u hond al iemand ernstig gebyt?

	Persentasies	
Ernstige byte deur hond toegedien	1986	1976
Nee	81	80
Ja	19	20

Vraag 33: Hoeveel oefening kry u hond?

Hoeveelheid oefening	Persentasies	
wat hond kry	1986	1976
Aktiewe afrigting	5	5
Stap met eienaar	26	28
Op die erf	50	62
Op die plaas	13	1
Niks spesifiek	6	4

Vraag 34: Laat u u hond op straat toe?

	Persentasies	
Hond op straat toegelaat	1986	1976
Ja	12	10
Nee	88	90

Vraag 35: Het u hond enige formele afrigting gehad?

	Persentasies	
Afrigting van honde	1986	1976
Afrigting tuis	31	34
Vir skoue	3	3
Vir verdediging	4	3
Ander	2	3
Geen	60	57

Vraag 36: Neem u hond aan skoue deel?

	Persentasies	
Honde se deelname aan skoue	1986	1976
Nooit	92	88
Soms	5	7
Gereeld	3	5

Vraag 37: Hoe straf u u hond?

	Persentasies	
Strafmetodes van honde-eienaars	1986	1976
Raas	44	49
Ignoreer die hond	3	2
Klap	24	21
Slaan met voorwerp	8	9
Sluit toe	2	2
Trek die oor	2	–
Negatiewe houding	14	–
Geen	3	2
Kombinasies	–	15

Vraag 37: As u hond sou doodgaan, sal u weer een aanskaf?

	Persentasies	
Vervanging na 'n hond se dood	1986	1976
Ja	85	84
Nee	3	6
Onseker	12	10

Vraag 39: Sal u weer dieselfde ras kies?

Vervanging van honde ten opsigte van ras	Persentasies	
	1986	1976
Dieselfde	58	72
Enige ander ras	25	19
Spesifieke ander ras	17	7
Onseker	–	2

BESPREKING

Daar was 'n neiging onder hondeeienaars om meer as een hond per huis aan te hou. Kruisrasse maak steeds die grootste groep van honde uit. Die Malteser wat as 'n tipiese skoot- of troetelhond beskryf kan word, is egter nou die mees algemene erkende ras, teenoor die Franse Poedel 'n dekade gelede. Tipiese "waghonde" soos die Duitse Herdershond, Doberman, Rottweiler en Labrador neem egter die volgende plekke in, en hierdie vier rasse alleen verteenwoordig nou 23% van alle honde teenoor die 12% as 'n groep in 1976. Ander "waghond"-rasse het natuurlik ook voorgekom en van die kruisrasse sal ook as "waghonde" kwalifiseer. Rasse wat hulle plek op die gewildheidsleer die afgelope tien jaar verkry het, was behalwe die Rottweiler, ook die Staffordshire Bull Terrier, St Bernard en die Border Collie. Die neigings na spesifieke rasse verteenwoordig 'n toename in geselskapsdiere, wat spesifiek vir spanningontlading gebruik kan word (Malteser), asook rasse spesifiek vir beskerming (waghonde). Dit reflekteer moontlik die veranderde veiligheidsituasie van die afgelope dekade in Suid-Afrika.

Honde-eienaars hou ongeveer dieselfde aantal reuns en tewe aan. In meer gevalle is honde van private persone en erkende telers verkry, as wat hulle as geskenke ontvang is of van welsynsorganisasies of troeteldierwinkels bekom is. Dit is 'n positiewe verskynsel as eienaars eerder hul honde van bekende bronne verkry. Dit lyk of honde-eienaars meer lees oor hulle diere en ook meer oor spesifieke onderwerpe soos siektes, afrigting en teling. Die verspreiding van die ouderdomme van honde wat in eienaars se besit is, het merkwaardig min verander. Die meeste eienaars se honde is tussen 2 en 5 jaar oud, wat 47% verteenwoordig.

Meer mense as in 1976 sien egter kans om sonder hul honde te lewe en dit kan wees omdat waghonde wat toegeneem het, 'n losser band met hul eienaars vorm. Die 73% wat nie kans sien om sonder 'n hond te lewe nie en die 11% wat onseker is oor hierdie aspek, is nogtans besonder hoog. In die redes waarom eienaars honde aanhou, het "beskerming" soos verwag, 'n belangriker plek (11% hoër) ingeneem as tien jaar gelede. Nogtans het "plesier" steeds die belangrikste rede gebly, waarom honde aangehou word. Honde-eienaars se inkomstes is soos in ander studies,[1 2 3 4] redelik eweredig tussen inkomste-groepe versprei.

Voeding van honde geskied nou eerder twee of meer keer per dag, as een keer per dag, terwyl hondekorrels as hoofvoedsel, sterk (18%) toegeneem het. Die twee neigings kan dalk met mekaar in verband staan, aangesien mense geneig is om die maklik toedienbare korrels meer gereeld aan te vul. Die verhoogde besteding aan hondekos kan ook aan die groter verbruik van kommersiële kosse toegeskryf word.

In huishoudings is die vader meer betrokke by die versorging van die hond, moontlik omdat moeders meer dikwels self 'n beroep beoefen, en die huiswerk gedeel moet word.

Honde is steeds meer geheg aan die moeder in die huis, maar is meer gehoorsaam aan die vader. Dit kan ook op 'n groter betrokkenheid van die vader by die huishouding dui. Gedurende vakansies maak mense meer gebruik van bure/vriende of 'n huishulp om honde te versorg, as dierehotelle. Die redes hiervoor kan die kostes verbonde aan dierelosies wees, of weens die feit dat die hond as 'n sekuriteitsmaatreël by die huis gelaat word.

Die opruim van honde-ontlasting op straat word steeds meer as die eienaar se verantwoordelikheid gesien en eienaars voel ietwat sterker oor hierdie aspek.

Vir die veearts is daar voordelige, sowel as nadelige tendense. Minder eienaars sien die veearts net wanneer nodig en jaarlikse besoeke het toegeneem. Nogtans het sowat 73% van die eienaars steeds net die veearts besoek wanneer nodig.

Daar bestaan 'n sterk tendens weg van doellose teling met tewe. Die gewildheid van sterilisasie-operasies het dan ook drasties toegeneem (minstens 30%) teenoor minder akkurate metodes van voorbehoeding. Daar is verder 'n toename in mense wat gesteriliseerde tewe verkies en daar is selfs 'n toename in mense wat gekastreerde reuns verkies. Die "kastrasievrees" is egter steeds herkenbaar deurdat meer as dubbeld soveel eienaars sterilisasie in 'n teef verkies as kastrasie in 'n reun.

Die voorkeur-eienskappe in 'n hond het dieselfde gebly, terwyl honde heelwat minder "intimiteite", in en om die huis toegelaat word. Dit het waarskynlik te make met meer buite-waghonde, kleiner wooneenhede en meer uithuisigheid van die eienaar. Daar word egter meer hondehokke aan honde voorsien ten koste van slaapplek in die kombuis.

Eienaars is toenemend bewus van hul honde se "eienaardige" gedrag en dit kan wees omdat hulle meer objektief is, of meer ingelig is oor hondegedrag. Vrees vir onweer is as die grootste probleem uitgewys (17%). Veeartse sal moet kennis neem van die 4% eienaars wat hulle honde as antagonisties teenoor veeartse beskou. Die antagonisme teen swart mense is volgens die hondeeienaars steeds die hoogste (40%). Die persentasie van ernstige bytwonde het egter nie veel verander nie.

Menings oor oefening vir honde en honde op straat, het feitlik dieselfde gebly. Formele afrigting kon dalk ietwat afgeneem het en so ook deelname aan skoue. Omdat sulke aktiwiteite ekonomiese implikasies het, is dit te verstane.

Die houding teenoor die vervanging van 'n hond het nie veel verander nie, maar minder mense is geneig om dieselfde ras hond weer aan te skaf. Die hoë persentasie kruisrasse mag 'n invloed op hierdie tendens hê.

Vir die veearts is kennisname van moontlike tendense, sowel as die bevindings in die 1986-ondersoek van belang. Veral vir die geselskapsdierveearts bied die inligting insig in die verhoudings tussen kliënt en hond. Dit help dus om die veearts voor te berei vir enige effek wat dié resultate op sy praktyk mag uitoefen, soos byvoorbeeld die toename in waghond-tipes, 'n toenemende vraag na sterilisasie-operasies en die gewildheid van sekere rasse wat deur die media bekendgestel word. Sulke mode-tendense onder rasse het dikwels 'n nadelige invloed op die teelprogramme van die betrokke rasse.

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BLOOD BIOCHEMICAL PARAMETERS AND MEAT pH OF FEEDLOT CATTLE SLAUGHTERED ON ARRIVAL OR AFTER OVERNIGHT REST AT AN ABATTOIR

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ABSTRACT

The effect of different pre-slaughter rest periods at the abattoir before slaughter on the blood biochemical parameters and meat pH of a number of cattle were studied on 2 occasions, once in summer and once in winter. In the first trial, 42 out of a consignment of 50 and in the second trial, 45 out of a consignment of 54 feedlot-finished oxen were randomly selected and divided into 3 groups each. One group was slaughtered on arrival at the abattoir, a second group 3 h after arrival and the third group was rested in the lairage prior to slaughter the following day. Carcasses were subjected to low voltage electrical stimulation at slaughter.

Significant differences between the values obtained at the feedlot and those at the abattoir were found in respect of haematocrit, total plasma protein concentration, plasma glucose, plasma creatine kinase, plasma lipid and cortisol concentration in both the trials. In general, however, no great differences were found between the 3 groups in either of the trials. If any, results in respect of some of the blood parameters were in favour of the groups slaughtered as soon as possible after arrival at the abattoir.

No significant differences were found in the initial (35-45 min) and ultimate (24 h) pH of the meat between the 6 groups of slaughtered animals in the 2 trials. Exsanguination appeared to be satisfactory in all groups and no differences between groups were found.

It was concluded that feedlot cattle transported to an abattoir over a relatively short distance, need not necessarily be rested in the lairage overnight before slaughter.

Key words: slaughter cattle, stress, rest period before slaughter, meat pH

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INTRODUCTION

Regulation 1.(3) of Part V of the Standing Regulations under the Animal Slaughter, Meat and Animal Products Hygiene Act, 1967 (Act no. 87 of 1967)[2] stipulates that no animal shall be slaughtered without having been rested overnight in the abattoir lairage. A large cattle feedlot organisation, slaughtering the bulk of its animals at the company's own abattoir, applied for exemption of this regulation on the grounds that the animals were derived from a feedlot situated relatively close to the abattoir.

According to Thornton & Gracey[9], it is necessary to rest an animal for a minimum of 24 h in a lairage before slaughter. An animal slaughtered without an adequate period of rest may show a reduction in keeping quality of the flesh due to incomplete development of acidity of the muscles and also due to early invasion of the system by putrefactive bacteria from the intestinal tract. They emphasise that although cattle subjected to stress for a short period may recover rapidly, those subjected to stress for a long period, may take several days to regain physiological normality.

Laurie[4] quotes Mitchell & Hamilton who showed that exhausting exercise immediately prior to slaughter, could cause a high ultimate pH in the muscles of cattle. Depletion of glycogen reserves in cattle also occurred if enforced exercise took place immediately after train travel. Temperament may also affect the ultimate pH of the meat of cattle. Glycogen reserves are depleted by isometric tension in the muscles of excitable cattle. Fear is considered an important factor during train travel and can be responsible for the depletion of glycogen reserves and a consequent high ultimate pH of the meat. Schönberg[7] states that the meat of healthy rested animals attains a pH of 5,2 to 6,0 within 24 h whereas exhausted or otherwise abnormal animals produce meat which never reaches the desired acidity.

McClean[5] found that the ultimate pH of meat of young cattle transported by rail or road over distances of less than 350 km, ranged from 5,0 to 6,1 regardless of whether or not they had been rested prior to slaughter. For adult cattle the corresponding range was 5,2 to 6,0. After travelling for distances of 1 550 to 1 733 km, the ultimate pH of the meat of unrested calves ranged from 5,25 to 5,65 whereas that of rested calves ranged from 5,5 to 6,1. In the case of adult cattle transported over 1 550 to 1 733 km, the ultimate pH of the meat ranged from 5,4 to 6,1 regardless of whether they were rested or not.

According to Section II B2 of Schedule 6 of the Standing Regulations under Act 87 of 1967[2] ultimate pH readings above 6,3 for cattle are considered as evidence of inferior quality and require the Veterinary Meat Inspector to re-evaluate the carcass.

In an attempt to determine whether the compulsory rest period was in fact necessary, 2 trials, one during the winter and the other during summer, were performed on groups of feedlot oxen intended for slaughter. Possible biochemical indicators of stress in the animals as well as changes in meat pH were studied in rested and unrested groups of oxen. It was expected that the reaction to transportation and change of environment would be more pronounced in nervous than in tame animals. Highly excitable animals were therefore chosen for the purpose.

MATERIALS AND METHODS

Trial A (Winter)

Three groups (A1, A2, A3) of eartagged Brahman crossbred oxen (n=42) were randomly selected at a feedlot from a batch of 50 which were ready for slaughter. Heparinised jugular blood samples were collected in evacuated tubes at the feedlot the day before they were dispatched to the abattoir. For the determination of plasma glucose and lactate the blood was collected in tubes containing Anderson's anti-coagulant solution. Early the next morning the 50 animals were all loaded on the same truck and transported to the abattoir over a distance of approximately 200 km. They arrived at the abbatoir just before 10h00, were off-loaded and moved to appropriate lairage pens with available drinking water. The 14 oxen of the first group (Group A1) were restrained in a crushpen and bled within 0,5 h after arrival and slaughtered thereafter. The oxen of Group A2 were bled and

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slaughtered approximately 3 h after arrival and those of Group A3 the following morning, approximately 23 h after arrival.

The carcasses were subjected to equal periods of low voltage electrical stimulation immediately after throat-cutting. Approximately 35 - 45 min later, the pH of the *M. triceps brachii* as exposed by the incision prescribed for examination for cysticercosis, was determined by means of a spear-pointed glass electrode connected to a properly calibrated (pH 4,0 and 7,0) temperature compensated mains operated pH-meter. The mean of three separate readings on both halves of the carcass was recorded as the reading for each carcass. These pH-readings were repeated similarly on each chilled carcass within 24 h after slaughter. The carcasses and organs were subjected to routine prescribed veterinary inspection and were all passed as fit for human consumption.

Each blood sample was split into suitable aliquots for the different analyses immediately after collection. The plasma aliquots were obtained by centrifugation within 0,5 h after collection and then stored at −20°C for later analysis.

The parameters mentioned below were determined on each blood sample:

Haematocrit: The haematocrits of the blood samples were determined by centrifugation in microhaematocrit tubes[3].

Total plasma proteins: These were determined by means of the biuret method as described[11].

Blood urea: The enzymatic UV-spectrophotometric method as described[8] was employed.

Plasma creatine kinase (CK): Enzyme-activity was determined by using a Boehringer-Mannheim kit based on an enzymatic UV-spectrophotometric assay[1]. The results are expressed as the geometric mean ± SEM[10].

Plasma lactate: The assays were performed by using a Boehringer Mannheim kit based on enzymatic spectrophotometry[6].

Plasma glucose: The GOD-Perid colorimetric method (Boehringer Mannheim) was used for the determinations.

Total plasma lipids: The assays were performed by means of a colorimetric method as described[12].

Cortisol: The plasma samples were subjected to a double antibody radioimmuno assay [(^{125}I) Cortisol R.A. Premix Kit, Diagnostic Products Corporation].

Reliability control: Known quality control sera were used throughout to ensure reliable results.

Trial B (Summer)

The same procedure as in Trial A was followed with the following exceptions: Brahman crossbred oxen (n=45) finished in the same feedlot as those used in Trial A, were randomly selected for each of 3 treatment groups (B1, B2 & B3). Group B1 was slaughtered on arrival at the abattoir, group B2 approximately 3 h after arrival and group B3 the following morning. Due to the fact that the procedure of blood collection at the abattoir appeared to be a source of additional stress to the animals, the blood samples were collected from the jugular vein during exsanguination. (The differences between blood samples collected by venipuncture just prior to slaughter and those collected during exsanguination have previously been determined by the authors (1983, unpublished data.).

An additional test for plasma creatinine was included in Trial B. The method employed was the enzymatic UV-method of Boehringer Mannheim.

At the time of the pH readings the carcasses of 2 other consignments of 18 oxen each, originating from the same feedlot, were available. These animals had been rested overnight before slaughter and had not been subjected to the stress associated with the collection of blood samples before dispatch. They were therefore regarded as a valuable control group in respect of the pH changes in the meat and were divided into groups X and Y.

Statistical analysis: The results of the blood analyses were statistically compared by means of 2 methods: the values obtained from each ox at the feedlot and at the abattoir, were compared by means of a paired *t*-test. An analysis of variance was applied to compare the mean differences between the 3 groups in each trial.

The statistical differences between the pH-values of the meat of the different groups were determined by means of an analysis of variance.

RESULTS

The mean values obtained from the analyses of the blood samples of each of the 6 groups of oxen are presented in Tables 1 & 2.

The pH measurements, approximately 35 to 45 min after electrical stimulation of the carcasses during the slaughtering process and again 24 h later, gave the readings presented in Table 3. Ultimate values of below pH 6,3 are desirable.

DISCUSSION

Haematological parameters: There was a highly significant increase in the haematocrit of all 3 groups (Trial A) from the feedlot to the abattoir. (In the subsequent trial haematocrits could unfortunately not be determined.) This rise continued at the abattoir and a highly significant difference was also evident between the haematocrits of group A3 and the other two groups. The initial rise was probably the result of stress-induced splenic contraction and the further increase due to dehydration. The feedlot values all fell within the normal range but the mean value of 52,8% encountered in group A3 after standing at the abattoir overnight, must be regarded as abnormally high.

Total plasma proteins: In Trial A the plasma proteins increased highly significantly by about 10% from the feedlot to the abattoir and a further 4% while standing at the abattoir overnight. The increase in trial B was not as high, but the group rested overnight again showed the greatest increase, possibly indicating a degree of dehydration.

Plasma glucose: In both trials the plasma glucose concentration decreased from the feedlot to the abattoir. In Trial A it decreased further during the period of rest (highly significantly, $p<0,001$). However, in Trial B, the mean concentration in groups B2 and B3 returned to the feedlot values. In a previous study by the authors (1983 unpublished data) it was found that the plasma glucose concen-

Table 1: **The mean values obtained from the analyses of blood samples collected both at the feedlot and prior to slaughter and the differences between them (trial A)**

	Group A1			Group A2			Group A3		
	Feedlot	Abattoir	Diff	Feedlot	Abattoir	Diff	Feedlot	Abattoir	Diff
Haematocrit (%)	38,2	43,8	5,6 **	38,0	47,0	9,0 **	38,8	52,8	14,0 ***
Blood glucose (mmol ℓ^{-1})	3,3	3,2	−0,1*	3,7	3,0	−0,7**	3,6	2,6	−1,0 **
Plasma proteins (g ℓ^{-1})	71	78	7 **	68	75	7 **	70	80	10 **
Plasma lipids (g ℓ^{-1})	6,3	7,1	0,8*	6,4	6,9	0,5*	6,9	7,0	0,1 *
Plasma lactate (mmol ℓ^{-1})	6,3	6,3	0	4,9	5,3	0,4	4,9	3,5	−1,4
Plasma urea (mmol ℓ^{-1})	4,63	4,53	−0,10	4,77	4,13	−0,64**	4,63	4,53	−0,10
Plasma creatine kinase (U ℓ^{-1})	224	372	148	100	513	413	513	112	−401
Plasma cortisol (nmol ℓ^{-1})	236	203	−33	237	128	−109	257	85	−172

* Significant at the 5% level
** Significant at the 1% level
*** Significant at the 0,1% level

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Table 2: **The mean values obtained from the analyses of blood samples collected at both the feedlot and during exsanguination and the differences between them (trial B)**

	Group B1			Group B2			Group B3		
	Feedlot	Abattoir	Diff	Feedlot	Abattoir	Diff	Feedlot	Abattoir	Diff
Plasma proteins (g ℓ^{-1})	73,4	76,6	3,2	76,3	78,2	1,9	76,0	80,4	4,4*
Blood glucose (mmol ℓ^{-1})	3,9	3,5	−0,4	3,2	3,2	0	3,4	3,4	0
Plasma lipids (g ℓ^{-1})	8,4	9,0	0,6	8,5	8,6	0,1	8,1	8,1	0
Plasma lactate (mmol ℓ^{-1})	3,5	6,8	3,3***	5,0	8,3	3,3 *	5,1	8,7	3,6 *
Plasma urea (mmol ℓ^{-1})	6,66	9,21	2,59***	7,51	9,2	1,69	5,77	7,47	1,7 **
Plasma creatinine (μmol ℓ^{-1})	136,6	139,7	3,1	133,3	138,0	4,7	138,9	155,9	17,0 *
Plasma creatine kinase (U ℓ^{-1})	100	275	175***	107	575	468***	100	302	202***
Plasma cortisol (nmol ℓ^{-1})	167	201	34	193	182	−11	177	155	−22

* Significant at the 5% level
** Significant at the 1% level
*** Significant at the 0,1% level

Table 3: **The mean pH (and the range within each group) of the meat 35-45 min and again 24 h after slaughter**

		Time after slaughter	
Group	Rest period	35-45 min	24 h
A1	0	6,04 (5,85-6,43)	5,86 (5,73-6,23)
A2	3h	5,90 (5,64-6,22)	5,76 (5,68-5,84)
A3	24 h	5,84 (5,63-6,42)	5,75 (5,65-5,99)
B1	0	5,65 (5,45-5,87)	5,72 (5,46-6,08)
B2	3 h	5,65 (5,48-5,89)	5,76 (5,53-5,86)
B3	24 h	5,74 (5,40-5,89)	5,79 (5,54-5,95)
X	24 h	–	6,65 (5,51-5,85)
Y	24 h	–	5,85 (5,60-6,48)

tration in blood collected during exsanguination, was 32% higher than in blood collected by venipuncture just prior to slaughter. It is, therefore, safe to assume that had the animals been bled by venipuncture, the glucose concentration in Groups B2 and B3 would have been lower than at the feedlot. The plasma glucose values at the feedlot were, however, higher than the normal range (2,3 -2,8 mmol ℓ^{-1}) usually encountered in this type of animal and could be ascribed to the nervousness of the animals.

Plasma lipids: In both trials the concentration of total plasma lipids increased from the feedlot to the abattoir. In Group A3 and B3 it decreased and returned to the feedlot values after the overnight resting period.

Plasma lactate: No significant differences were found in the concentration of plasma lactate in the animals between the feedlot and the abattoir in the first trial. In the second trial, however, the concentration of lactate in the plasma at the time of slaughter was significantly higher than at the feedlot. The way in which the blood samples were collected is probably to be blamed for this discrepancy. In a previous study by the authors (1983, unpublished data) it was found that blood collected during exsanguination contained an average of 80% more lactate than the samples collected by venipuncture from the same animals just prior to slaughter.

The conclusion drawn is, therefore, that there was no significant change in the concentration of plasma lactate between the feedlot and the abattoir, nor between the groups in each trial.

Plasma urea: The feedlot (pre-treatment) values of plasma urea differed greatly in the 2 trials. The reason for this is unknown. In Trial B (with the higher pre-treatment values), there was a highly significant increase in the concentration of the plasma urea above the feedlot values in groups B1 and B3 at slaughter. In Trial A the opposite, a slight decrease, was observed. No explanation for these discrepancies can be given.

Plasma creatinine: Due to the inconclusive results of the urea determinations in Trial A, it was decided to look at creatinine as well in Trial B. The normal concentration of creatinine in plasma of beef cattle is usually less than 133 μmol ℓ^{-1} (unpublished data). In the experimental animals, values from 66 to 235 μmol ℓ^{-1} were found. The higher levels were probably indicative of decreased glomerular filtration in the kidneys due to dehydration. The greatest increase was seen in group B3 (significantly higher at slaughter than at feedlot), but the differences between the 3 groups were not significant.

Plasma creatine kinase: With the exception of group A3, the concentration of creatine kinase in the plasma of all other groups was higher at the abattoir than at the feedlot. In Trial B these differences were highly significant but not significant between groups. As with lactate, creatine kinase is also a plasma constituent that rises during the slaughtering process. The method of blood collection may therefore be partially responsible for the higher levels at the abattoir in Trial B. The mean values were within the expected limits but great variation (251 to 1 023 U ℓ^{-1}) was found between individual animals. This may be ascribed to different degrees of traumatic bruising during handling and transportation.

Plasma cortisol: In Trial A the mean concentrations of cortisol in the plasma of all 3 groups were conspicuously lower at the abattoir than at the feedlot. Even so, the mean values at the abattoir were still higher than those normally found in beef cattle. In the second trial the drop in plasma cortisol concentrations was less marked. Group B1 was the exception. Its mean value was higher at slaughter than it was at the feedlot.

Meat pH: In the first trial, the pH of the meat decreased most in group A3 and least in Group A1. The difference between the groups after 45 min was statistically significant at the 5% level. After 24 h the pH of 2 carcasses in Group A1 had not yet dropped to below pH 6 but all the carcasses were below the limit of pH 6,3. In the second trial, the pH decreased as desired in all animals and no statistical difference could be shown

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between the 3 groups and one of the control groups (group X). In the other control group the pH readings, 24 h after slaughter, were significantly higher ($p < 0,05$) than in the experimental groups and the pH of one carcass did not drop to below the 6,3 limit. No organic disease or abnormality was present to explain the result in this latter case.

The findings of McClean[5] indicate that satisfactory ultimate pH-readings were obtained regardless of whether the cattle had been rested overnight or not. It would therefore appear that muscle glycogen levels are not as readily depleted as is supposed and that this would support the satisfactory findings with regard to immediate slaughter of cattle transported from a nearby feedlot.

CONCLUSIONS

Although some differences were found between the unrested groups and those rested for 24 h at the abattoir before slaughter (in the case of haematocrit, total plasma proteins, plasma glucose and plasma creatinine, less favourable in the groups rested for 24 h), the overall conclusion is that there were no important differences between animals not rested, rested for 3 h or rested for 24 h before slaughter. Furthermore, no statistical differences could be shown in the ultimate pH of the meat which might have been caused by the different methods of treatment.

It should, however, be kept in mind that the experimental animals were accustomed to intensive conditions and were only transported over a relatively short distance of approximately 200 km.

No marked differences could be detected between animals transported to the abattoir during summer or winter.

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BLOOD LEAD CONCENTRATIONS IN HIPPOPOTAMI *(HIPPOPOTAMUS AMPHIBIUS)* IN THE KRUGER NATIONAL PARK

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ABSTRACT

Venous whole-blood samples for the determination of lead concentrations were obtained from hippopotami *(Hippopotamus amphibius)* (n=26) during a population control programme on the banks of the Sabie River. A mean blood lead concentration of 26,3 $\mu g\ \ell^{-1}$ was lower than the mean concentration of 34,0 $\mu g\ \ell^{-1}$ cited for a group of young people living in a remote area where lead pollution was considered to be negligible. These concentrations indicate that lead pollution in this area of the Kruger National Park is at present low.

Key words: Hippopotamus, *Hippopotamus amphibius*, whole-blood lead concentration, pollution

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INTRODUCTION

Mielke et al.[7] demonstrated in 1983 that vegetable garden soils collected within a 50 km radius of Baltimore city centre had a median lead concentration of 100 ppm. These authors also found that roadside soil lead levels were directly related to emissions from vehicular traffic using leaded petrol.

An organic lead compound (tetraethyl lead) is used in motor fuels as an "anti-knock" additive throughout the world but in different quantities. Disler et al.[4] stated that in South Africa petrol with the highest lead concentration in the world is used (0,836 g ℓ^{-1} vs 0,29 g ℓ^{-1} in the USA, 0,31 in Japan and 0,04 in France) but there are indications that these concentrations will gradually be reduced in future.

In order to study the possible influence of lead from vehicle exhaust fumes on the blood lead concentrations of animals living adjacent to a busy tourist route in the Kruger National Park, whole-blood lead levels obtained from hippopotami *(Hippopotamus amphibius)* in the Sabie River area as well as lead levels in two water samples collected from the river were determined.

MATERIALS AND METHODS

Hippopotami (n=26) of different ages were immobilised during a population control programme. Blood samples obtained from the vena jugularis within one hour of immobilisation were collected in heparinised specimen tubes (L.H. 3 200 Vacutainers™, Becton-Dickinson, Rutherford, New Jersey 07070) and stored at 4°C until analysed in a batch.

Water samples of the Sabie River, the habitat of the hippopotami under study, were also collected at 2 different spots and delivered into suitable evacuated tubes (Vac-u-test, Radem Laboratory Equipment, Wijnberg) for lead analyses.

Whole-blood lead concentrations were determined with the micromethod described by Fernandez & Hilligoss[5]. This method utilises an atomic absorption spectrophotometer (Model 5500 Perkin-Elmer Corp., Norwalk, Conn. USA 06856) equipped with a graphite furnace (Model HGA 500, Perkin-Elmer) and automatic sampler (Model AS-40, Perkin-Elmer). The spectrophotometer was accurately calibrated using lead standards prepared in ammonium dihydrogen phosphate in an acid medium.

RESULTS

The mean whole-blood lead concentration of the hippopotami was 26,3 $\mu g\ \ell^{-1}$ (± 9,6 SD) and the range 6 - 44 $\mu g\ \ell^{-1}$. Two young animals had blood lead concentrations of 6 $\mu g\ \ell^{-1}$ while one old bull had a concentration of 44 $\mu g\ \ell^{-1}$. The water samples from the Sabie River contained no lead. Figure 1 shows the frequency distribution of the whole-blood lead levels in the animals.

DISCUSSION

Lead is a hazardous and non-essential metal in the nutrition of animals and humans. Despite this, goats, cows, young calves and horses abroad[2] have normal blood lead concentrations of 0,05 - 0,25 ppm (50–250 $\mu g\ \ell^{-1}$)[2]. Lead is usually present in plants, and in soil it is found in an insoluble form. However, in acid soils the solubility increases and lead may then become toxic to plants[1].

Most airborne lead originates from human activities with the major source believed to be the combustion of leaded petrol. In Britain[1] rural air contains approximately 0,1 μg lead per cubic metre while on motorways this value may be as high as 12 μ lead per cubic metre. Lead levels in vehicle exhaust fumes vary

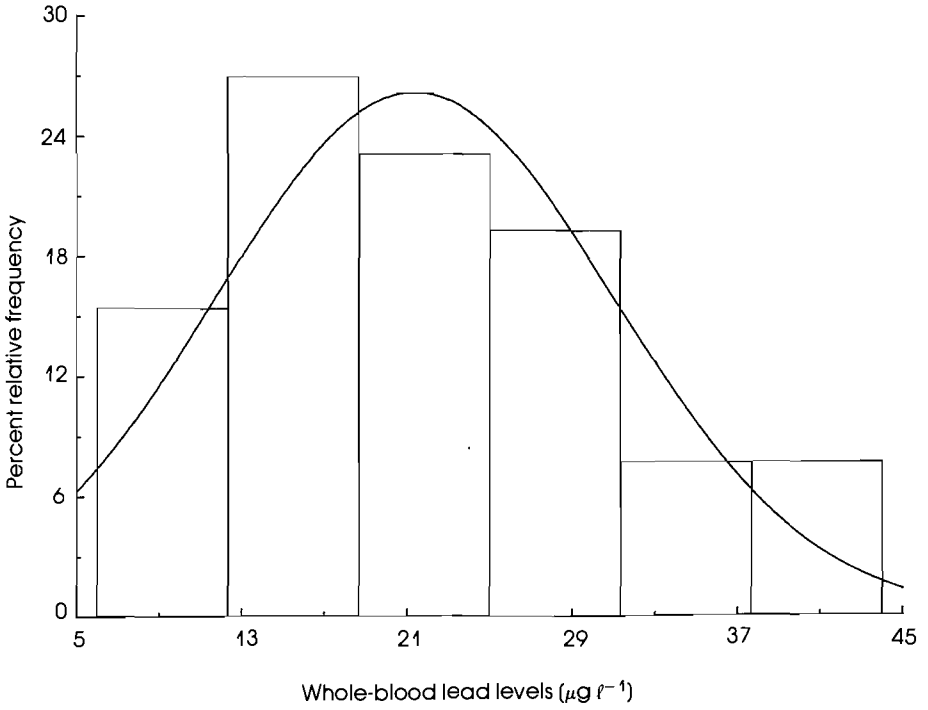

Fig. 1: The frequency distribution of whole-blood lead ($\mu g\ \ell^{-1}$) in 26 hippopotami

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between 2 000 and 10 000 μg per cubic metre and these levels correlate significantly with the mode of driving: fast acceleration increases lead emission[6].

In a previous study, the mean whole-blood lead concentration of 15 μg ℓ^{-1} was found in elephant from a remote area in the Kruger National Park[3]. Grobler et al.[6] using the same methodology to quantitate whole-blood lead concentrations, found a mean concentration of 34 μg ℓ^{-1} in humans living in a remote rural area in the north-western Cape Province, while in an urban control group in the Peninsula and in motor mechanics, the concentrations were 97 μg ℓ^{-1} and 284 μg ℓ^{-1} respectively[6]. The water of the Sabie River contained no lead and therefore the only other source of the lead found in the hippopotami, was apparently the vegetation ingested. Also of interest was the low concentrations of 6 μg ℓ^{-1} in young animals compared to concentrations of 30 μg ℓ^{-1} and 44 μg ℓ^{-1} in some adults. This may be indicative of accumulation of ingested lead over long periods by the older animals. It has been shown in humans that approximately 10% of ingested lead is absorbed and then transported by the red blood cells mainly to bone ($\sim$ 95%) with the remainder entering a rapidly exchangeable pool in blood and soft tissues[4]. Furthermore, absorption of lead usually exceeds excretion unless consumption is low[4]. Lead binds strongly to amino acids and other organic compounds such as enzymes, causing inhibition of enzyme activities which may lead to a wide variety of biochemical and clinical abnormalities. Overt symptoms of lead poisoning in humans[6] have been reported at levels of 700 to 800 μg ℓ^{-1} while in a series of cases of lead poisoning in cattle, lead levels of 350 to 2 360 μg ℓ^{-1} were found.[2] The whole-blood lead concentrations found in the hippopotami in this study were therefore well below toxic levels.

The determination of whole-blood lead concentration is the most meaningful index of assessing exposure to lead, utilising a reference method where meticulous attention is paid to detail[4]. We suggest that the levels of lead be monitored at least annually in different animal species living in areas where motor vehicle exhaust emissions and other forms of lead pollution may invade the atmosphere and subsequently pollute the animals' available sources of food.

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CORRIDOR DISEASE IN SOUTH AFRICA: A REVIEW OF THE CURRENT STATUS*

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ABSTRACT

The African buffalo *(Syncerus caffer)* is a reservoir host of *Theileria parva lawrencei* the causative organism of Corridor or buffalo disease of cattle. This organism can apparently transform its behaviour when tick-passaged through cattle to resemble that of *Theileria parva parva*, causing classic East Coast fever (ECF). These are major considerations for the strict quarantine measures imposed on buffaloes from Corridor disease endemic areas in South Africa. Results of continuing studies on diagnosis, chemotherapy, transmission and attempts to transform the behaviour of *T.p. lawrencei* in the laboratory, are discussed.

Potgieter F.T.; Stoltsz W.H.; Blouin E.F.; Roos J.A. Corridor disease in South Africa: A review of the current status. *Journal of the South African Veterinary Association* (1988) 59 No. 3, 155-160 (En). Department of Protozoology, Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.

INTRODUCTION

In South Africa the Kruger National Park and two of the Zululand nature reserves, Hluhluwe and Umfolozi, are currently regarded as endemic areas for buffalo or Corridor disease of cattle, described by Neitz[23].

As far as is known, the African buffalo *(Syncerus caffer)* is the only wild ruminant species that is a carrier of the causative organism *Theileria parva lawrencei*. Waterbuck are suspected of being susceptible to *T. p. lawrencei* infection and circumstantial evidence indicates that they could play a role in outbreaks of bovine theileriosis in Kenya[2]. Although Neitz[24] reported that 2 captive buffalo calves died as a result of natural *T. p. lawrencei* infections, experimental infection of a fully susceptible year-old buffalo calf by means of *Rhipicephalus appendiculatus* resulted in an extremely mild reaction. It is generally believed that buffalo calves are resistant to infection, but in cattle a highly fatal disease results. Corridor disease is a controlled animal disease.

Buffaloes outside the endemic areas, particularly those in the Addo Elephant National Park, are thought to be free of *T. p. lawrencei* infections on the basis of limited serological evidence and no clinical disease reported in cattle associated with these buffaloes[7]. The absence of the principal vector, *R. appendiculatus*, in this nature reserve (I.G. Horak 1985 Faculty of Veterinary Science, University of Pretoria, personal communication) and presumably of *T. p. lawrencei* carriers are possible reasons why this buffalo herd is free of Corridor disease. This herd forms a nucleus of *T. p. lawrencei*-free buffaloes from which animals have in the past been relocated to nature reserves in other areas of the country.

With an increasing demand for private ownership of buffaloes and limited *T. p. lawrencei*-free stock, buyers have resorted to the importation of buffaloes from other countries. These imports are controlled by the Department of Veterinary Services.

The need has arisen to start a buffalo register to keep track of the numbers, origins and movements of the buffaloes in this country. Attempts are being made to monitor the buffalo population outside endemic areas (V. de Vos 1985 unpublished information, presented at the Buffalo Symposium of the Wildlife Group, South African Veterinary Association, National Zoological Gardens, Pretoria).

It is the responsibility of scientists, nature conservationists and farmers to maintain and improve the quality of life for both wildlife and domestic stock. We must aim at maintaining the stable and peaceful co-existence that we have enjoyed for the past 30 years, especially as far as East Coast fever (ECF) is concerned. It has been policy that cattle and buffaloes should be kept apart in areas were Corridor disease is endemic, firstly to prevent Corridor disease outbreaks, that normally have mortality rates of > 90%[26], and for which no therapeutic drugs are available on the local market. Secondly it has been shown in Kenya that tick-passage of this parasite through cattle, changes its behaviour to that resembling *Theileria parva parva*, the causative organism of classic ECF[3,34].

An epidemic of ECF followed the introduction of a highly pathogenic strain of *T. p. parva* into South Africa in 1902. It was responsible for an estimated 5,5 million cattle deaths which cost the country R100 million[1]. Through tick control, quarantine and slaughter of exposed cattle, the disease was finally eradicated.

Theileriosis caused by the so-called *T. parva* — group of organisms is a complex of different disease syndromes occurring in many parts of Africa and poses a major constraint on expansion and improvement of cattle farming. At present ECF is controlled primarily through acaricidal tick control, and even though therapeutic drugs are available, treatment is expensive and the diagnosis must be made early for treatment to be most effective. Thus, after decades of intensive research, there is still an urgent need for alternative methods of ECF control. The African buffalo seems to play a key role in the epidemiology of ECF. We are currently using *T. p. lawrencei* isolates to study the chemotherapy, transmission and diagnosis of typical Corridor disease. Attempts have also been made to transform *T. p. lawrencei* to *T. p. parva* through tick-passages in cattle.

DIAGNOSIS

As far as is known, all outbreaks of Corridor disease in this country have been associated with the presence of buffaloes from endemic areas. Diagnosis is normally confirmed by the demonstration of macroschizonts in the lymphocytes. Clinical signs and pathology are well defined[24].

Serology

An indirect fluorescent antibody (IFA) test is used in the laboratory to detect antibody levels to *Theileria* spp. in sera of infected cattle and buffaloes. The technique employed is essentially that used by Gray and De Vos for *Babesia spp.*[13]. The low level of piroplasm parasitaemia found in animals recovered from *T. p. lawrencei* infections necessitates the use of *T. p. parva* piroplasm antigen. The serological cross- reactivity between *T. p. lawrencei* and *T. p. parva* is well recognised[16,19,30]. Low antibody titres, mixed infections and serological cross-reactivity between species such as *Theileria taurotragi* and *T. p. lawrencei*, often produce inconclusive results[8].

Attempts have been made to use *T. p. lawrencei* infected *Rhipicephalus zambeziensis* salivary glands as sporozoite antigen in the IFA test. To identify infected ticks, both salivary glands are dissected from each of the adult ticks collected from an infected bovine on Day 4 post attachment. One gland is placed in a well of a teflonised glass slide for use in the IFA test. The other gland is placed on a separate glass slide, stained with Methyl-Green/Pyronin[32] and screened for infected acini with a light microscope. If this salivary gland is infected, the corresponding salivary gland is fixed in cold acetone and used as antigen in the IFA test with anti-bovine IgG conjugate, as described above. Known *T. p. lawrencei* positive bovine serum is used as a control.

The sera of 6 buffaloes were tested using both piroplasms and sporozoites as antigen. The results indicated that sporozoite antigen may be more sensitive (Table 1).

The *T. p. lawrencei* salivary gland/sporozoite antigen is thus far our only source of pure antigen of this parasite and it may have application once monoclonal antibodies are available to differentiate be-

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Table 1: **Results of the IFA test using *Theileria parva parva* piroplasm and *Theileria parva lawrencei* sporozoite antigens to test buffalo sera for *T. p. lawrencei* antibodies**

Sera	Antigen	
(1/80 dilution)	**Piroplasm**	**Sporozoite**
Positive Control	+	+
Negative Control	−	−
Buffalo Tombi	+	+
Buffalo No. 2	−	−
Buffalo No. 9	−	+
Buffalo No. 11	+	+
Buffalo No. 14	−	+
Buffalo Ben	−	−

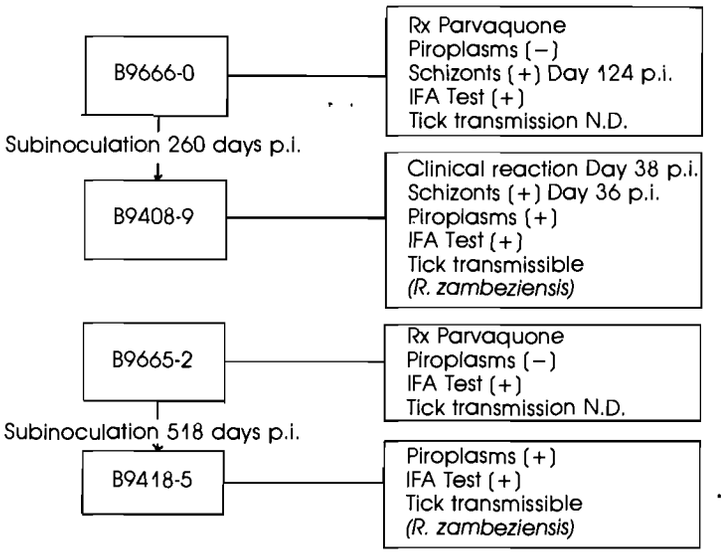

Fig. 1: Schematic representation of attempts to diagnose tick-transmitted *Theileria parva lawrencei* in 2 intact bovine carriers by subinoculation of blood (1000 ml i.v.) into susceptible splenectomised cattle. (p.i. = post infection; N.D. = not done)

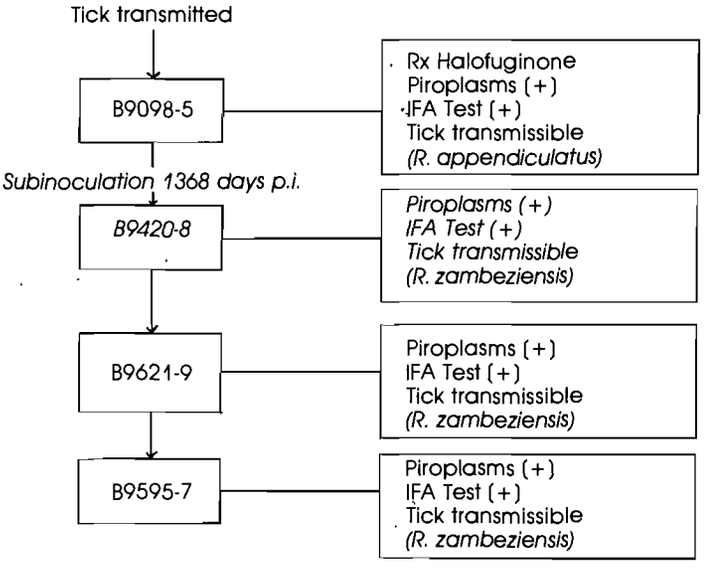

Fig. 2: Schematic representation of the results obtained with the serial subinoculation of *Theileria parva lawrencei* infected blood (1000 ml i.v.) into splenectomised cattle. (p.i. = post infection)

tween various *Theileria* isolates. Attempts to establish *Theileria*-infected cell lines from buffaloes and cattle have thus far failed.

Subinoculation of blood

Subinoculation of unstated volumes of blood from a *T. p. lawrencei*-infected buffalo resulted in piroplasm parasitaemias after about 4 weeks in 2 splenectomised calves, from which Corridor disease was transmitted with *R. appendiculatus* (Neitz, cited by De Vos[7]). Two attempts to isolate *T. p. lawrencei* from 4 buffaloes from Hluhluwe by subinoculating 100 and 50 ml of pooled blood respectively into 2 splenectomised cattle, resulted in the transmission of *Theileria mutans* infections only[7]. Subinoculation of approximately 500 ml of blood from each of 2 buffaloes in the Loskopdam Nature Reserve into splenectomised cattle, also resulted in the transmission of *T. mutans* infections to both recipients (Stoltsz 1985 Unpublished observations).

Mixed theilerial infections harboured by free-ranging wild animals make this method of isolating pure *Theileria* infections undesirable. However, in the laboratory we have recently shown that *T. p. lawrencei* can be transmitted between cattle by transfusing 1 ℓ of blood intravenously. This was made possible by the use of effective chemotherapy in order to establish piroplasm parasitaemias in splenectomised as well as intact, recovered animals (Fig. 1 & 2).

One of the recipient animals, B9408-9 (Fig. 1), showed a schizont parasitaemia and developed severe clinical signs of Corridor disease. It is assumed that this animal received a sublethal dose of infected lymphocytes during the blood transfusion. Histocompatability may have played a role in that the donor and recipient animals could have had matched bovine lymphocyte antigen (BoLA) systems[10]. Unfortunately the BoLA types were not identified to confirm this. The animal recovered without treatment.

The IFA test was also used to test the recipient cattle for possible sero-conversion, after having received blood from *T. p. lawrencei* carriers (Fig. 1 & 2). This indirect method of identifying possible *T. p. lawrencei* carriers may prove to be of some value especially if the test animals have low specific antibody titres and harbour mixed infections.

Nymphal ticks were fed on the recipient cattle showing piroplasm parasitaemias and in all 6 cases typical Corridor disease was transmitted transstadially as indicated in Fig. 1 & 2.

Xenodiagnosis

The technique of feeding vector ticks on an infected animal to isolate parasites is well known. However, feeding ticks on wild animals, especially buffaloes is not always practical and indirect methods whereby ticks can be infected with piroplasms in the laboratory are currently being investigated. This procedure has been successfully employed to isolate different field strains of *Theileria* spp. in Kenya[4,5,15], and is currently being followed in an attempt to isolate *T. p. lawrencei* from a captive Zululand (Umfolozi) buffalo. Blood from the buffalo was injected into splenectomised rabbits while *R. zambeziensis* nymphs were engorging on their ears. The ensuing adult ticks were fed on

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a susceptible bovine with negative results. This work will be repeated.

Mixed infections also present a problem with xenodiagnosis, especially in the case of Corridor disease, because *R. zambezienis*, like *R. appendiculatus*, transmits *T. taurotragi* and *T. p. lawrencei*[18]. Our first attempt to infect *R. zambeziensis* with *T. taurotragi* (Vaalwater)[8] failed. At this stage it is not clear whether buffaloes are indeed susceptible to other mild theilerial infections transmitted by either of these tick species. Present studies are concentrating on the comparative abilities of *R. appendiculatus* and *R. zambeziensis* to pick up infections from carriers of *T. p. lawrencei* and *T. taurotragi*. Results may indicate that with low parasitaemias, this technique has some application in the differentiation between these 2 *Theileria* spp. in the laboratory, as *R. appendiculatus* has been shown to maintain high infection rates of *T. taurotragi*[12].

Chemotherapy

Early work reviewed by Neitz[24] indicates that the drug aureomycin is effective against *T. p. lawrencei*, provided treatment commences during the incubation period. Neitz[24] concluded at the time that the treatment regimens had no practical therapeutic value. However, many years later, application was found for the infection-and-treatment method of immunising cattle against ECF as reported in a recent study where long-acting oxytetracycline was compared with parvaquone in the treatment of cattle infected with *T. p. parva* tick stabilates[9].

Effective chemotherapeutic agents against pathogenic *Theileria* spp. were only discovered during the past 10 years. Naphtoquinone (menoctone)[20] and halofuginone lactate[11 29] have been shown to be active against theilerial infections. Another naphtoquinone, parvaquone (Clexon, Wellcome), was soon identified as the first specifically active remedy for ECF[21], and currently buparvaquone, an improved formulation, has actually been shown to have therapeutic value, as well as prophylactic potential[22].

One of our main interests in effective anti-theilerial drugs concerns their use in establishing experimental *T. p. lawrencei* carriers in the laboratory to study the phenomenon of transformation of *T. p. lawrencei* to *T. p. parva* (See below). Attempts have also been made to sterilise *T. p. lawrencei* infections with parvaquone both in intact and splenectomised cattle. The results are summarised in Table 2 & 3.

Intact and splenectomised recovered animals were shown to have microscopically detectable piroplasm parasitaemias at one stage or another after treatment. Piroplasms were only demonstrated in intact animals after splenectomy or by the subinoculation of blood into susceptible cattle.

Chemical sterilisation of the carrier state of *T. p. lawrencei* in buffaloes and cattle would be advantageous in our present disease control strategy. Apart from the therapeutic value, herds suffering outbreaks could be treated to sterilise the infections to prevent the possibility of the infection from spreading amongst cattle. It would also contribute to the possible release of buffaloes from Corridor disease endemic areas. However, there are no accurate methods available to prove that animals are in fact sterilised. Even xenodiagnosis can fail. Until we have more specific and sensitive tests with which to diagnose *T. p. lawrencei* infections, all buffaloes from endemic areas must be regarded as being infective to ticks.

Therapeutic treatment of *T. p. lawrencei* infections in cattle is not recommended in this country, because recovered intact animals may develop a carrier status and could infect ticks[28]. Earlier observations showed that recovered splenectomised animals could serve as reservoirs of the infection[25]. We have no proof that naturally infected recovered cattle have played any role in outbreaks of Corridor disease in this country. Nevertheless it is suggested that the old belief of Corridor disease being a self-limiting infection in cattle[24], be reconsidered.

Transmission

R. appendiculatus has always been regarded as the principal vector of *T. p. lawrencei*[26]. A "new" tick species resembling *R. appendiculatus* especially in the adult stage, *Rhipicephalus zambeziensis*, was described in 1981[33]. This tick has been found to exist sympatrically with *R. appendiculatus* but interspecific mating seldom occurs. It occurs in hotter and drier climates than does *R. appendiculatus* and was first identified in 1982 in this country in the Transvaal. The tick has been collected from a wide variety of domestic and wild animals including cattle and buffaloes[27].

R. zambeziensis can transmit *T. p. lawrencei* and it was observed that infection rates were relatively high[18]. *Rhipicephalus zambeziensis* adults were collected from cattle in the northern Transvaal and established as a laboratory strain. It was decided to compare the vector potential of *R. zambeziensis* (Killkenny) and *R. appendiculatus* (Rietvlei) for *T. p. lawrencei* (Hluhluwe 3). Nymphs of both species were fed concurrently on the same two *T. p. lawrencei*-carrier cattle. The ensuing adult ticks were fed on rabbits, removed after 4 days, dissected and the salivary glands removed and stained with Methyl-Green/Pyronin and examined microscopically for the presence of infected acini[32]. The results are presented in Table 4.

In the laboratory, *R. zambeziensis* has thus far proved to be a more efficient vector of *T. p. lawrencei* than *R. appendiculatus*. It is also believed that this tick plays a major role in the transmission of Corridor disease in certain areas in Zimbabwe[18].

Transformation

The possibility of *T. p. lawrencei* transforming to *T. p. parva* under natural conditions in South Africa, has always been a highly contentious topic as it places the move-

Table 2: **Chemotherapy of tick-transmitted *Theileria parva lawrencei* infections in intact cattle using parvaquone at a dosage rate of 10 mg kg^{-1} i m**

Animal No.	1st Schizonts Day	1st Schizonts Level	1st and 2nd Temperature reactions Temp °C	Day	Max temp °C	Day	Parvaquone treatment (days p.i.)	1st Piroplasms (day p.i.)	Schizonts (day p.i.)	Outcome
9523-3	10	VR	39,9	9	42	14	Untreated control	–	–	Died day 17
9641-0*	11	VR	40,1	10	40,3	11	11,13	207	–	Piroplasms appeared after splenectomy
9665-2	10	R	40,7	10	41,0	11	11,13	–	–	Subinoculation: piroplasms (+)
			40,4	19	41,4	20	20,22			
9666.0	10	R	40,4	10	41,0	11	11,13	–	124	Subinoculation: piroplasms (+)
9667-8	8	VR	39,7	9	41,2	12	11,13	–	–	Blood smears (–)
			40,4	19	41,3	20	20,22			
9668-6	10	VR	40,0	10	40,5	14	14,6	–	–	Blood smears (–)
			40,6	22	40,6	22	22,24			

* = Infected using a tick-derived stabilate
VR = Very Rare, 1 to 3 parasites during 3 to 5 min examination
R = Rare < 1 parasite per 15 microscopic fields

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Table 3: **Chemotherapy of tick-transmitted** *Theileria parva lawrencei* **infections in splenectomised cattle using parvaquone at a dosage rate of 10 mg kg^{-1} i m**

Animal No.	1st Schizonts Day	1st Schizonts Level	1st and 2nd Temperature reactions Temp °C	Day	Max temp °C	Day	Parvaquone treatment (days p.i.)	1st Piroplasms (day p.i.)	Schizonts (day p.i.)	Outcome
9088-9	11	VR-R	39,5	12	41,4	16+17	Untreated control	–	–	Died Day 28
9406-3	11	VR	40,4	13	41,9	14	"	–	–	Died Day 19
9236-8	11	NR	39,8	12	41,5	14	14+16	36	–	Recovered Piroplasms frequently seen
9434-5	11	NR	40,2 39,2	10 21	41,0 40,4	13 22	11+13	–	–	Recovered Piroplasms rarely seen
9504-9*	8	VR	39,6 40,6	10 20	41,3 41,2	12 21	12+14 25,27+29	23	108	Recovered Piroplasms reappeared Day 188
9512-9	8	VR	40,0 40,0	10 22	41,1 41,0	11 24	11+13 26,28,30	26	56	Recovered Piroplasms reappeared Day 160
9514-5	11	NR	39,6 40,9	11 22	41,7 41,6	13 24	13+15	28	–	Died Day 37
9516-0	8	R-NR	39,5 39,7	9 32	41,3	12	12,14,16, 18,20+22	30	–	Recovered Piroplasms reappeared Day 286

* = Combined treatment of parvaquone and primaquine (1 mg kg^{-1} i m
VR = Very Rare, 1 to 3 parasites during 3 to 5 min examination
R = Rare < 1 parasite per 15 microscopic fields
NR = > 1 parasite per 15 fields, but < 1 parasite per field

Table 4. **Comparative infection rates of** *Theileria parva lawrencei* **in adult** *Rhipicephalus zambeziensis* **and** *Rhipicephalus appendiculatus* **fed concurrently as nymphae on the same two** *T. p. lawrencei* **carriers**

Ticks	Nymphae: engorging parasitaemia	No adult ticks recovered*	Adult ticks infected (%)	No. infected acini per tick
R. zambeziensis	<0,1% in both animals	62/200	50	18,7
		41/200	98,7	28
R. appendiculatus		35/200	0	0
		72/200	4	3,3

* Adult ticks removed 4 days post infestation

ment of buffaloes under severe constraints.

In 1966 it was reported from Kenya that the repeated experimental passage of *T. p. lawrencei* through cattle, using ticks, resulted in some profound changes in the behaviour of the parasite[3]. These changes rendered *T. p. lawrencei* microscopically indistinguishable from *T. p. parva*. Barnett, according to De Vos[7], obtained the same results using a South African isolate of the parasite when he repeated the work. Similar observations have also been made with an isolate from Serengeti[34] and it was concluded that *T. p. parva* and *T. p. lawrencei* were merely different behavioural forms of the same parasite in different hosts and that wherever strains of the "lawrencei-type" occur, classic ECF may well re-emerge spontaneously[3].

Despite the potential lability of *T. p. lawrencei*, the parasite has failed to revert to ECF under natural conditions in South Africa in spite of opportunities to do so[7].

With the discovery of effective therapeutic drugs against the pathogenic *Theileria* spp., cattle experimentally infected with *T. p. lawrencei* could be treated in the laboratory to become carriers of the infection. Several attempts to transform *T. p. lawrencei* (Hluhluwe isolates, particularly No. 3) have failed[7]. Repeated attempts over a number of years have enabled us to reach a 4th generation of parasites through tick-passage in cattle, but we have so far failed to show any behavioural change. The results of the attempts to transform this parasite are briefly outlined in Fig. 3.

Similar attempts at transformation of *Theileria parva bovis*, the causative organism of January disease (Zimbabwean theileriosis), thought to be a modified strain of *T. p. parva*, have also failed[17,31].

The mechanism by which *T. p. lawrencei* alters its behaviour to resemble *T. p. parva* remains unclear. Parasites isolated from buffalo may stem from a heterogenous population of which a subpopulation of parasites capable of completing their cycle in cattle may be selected during the tick-passages in cattle[6]. Alternatively, the transformation of the buffalo parasite may be the result of its adaptation to the cattle host[14].

CONCLUSION

Efforts should be made to overcome the problems encountered with the serological diagnosis of mixed *Theileria* infections. The *Theileria* parasites specifically of game species and their vectors should be studied and their roles defined in disease syndromes. It is also advisable that other isolates of *T. p. lawrencei*, including ones from the Kruger National Park, should be studied.

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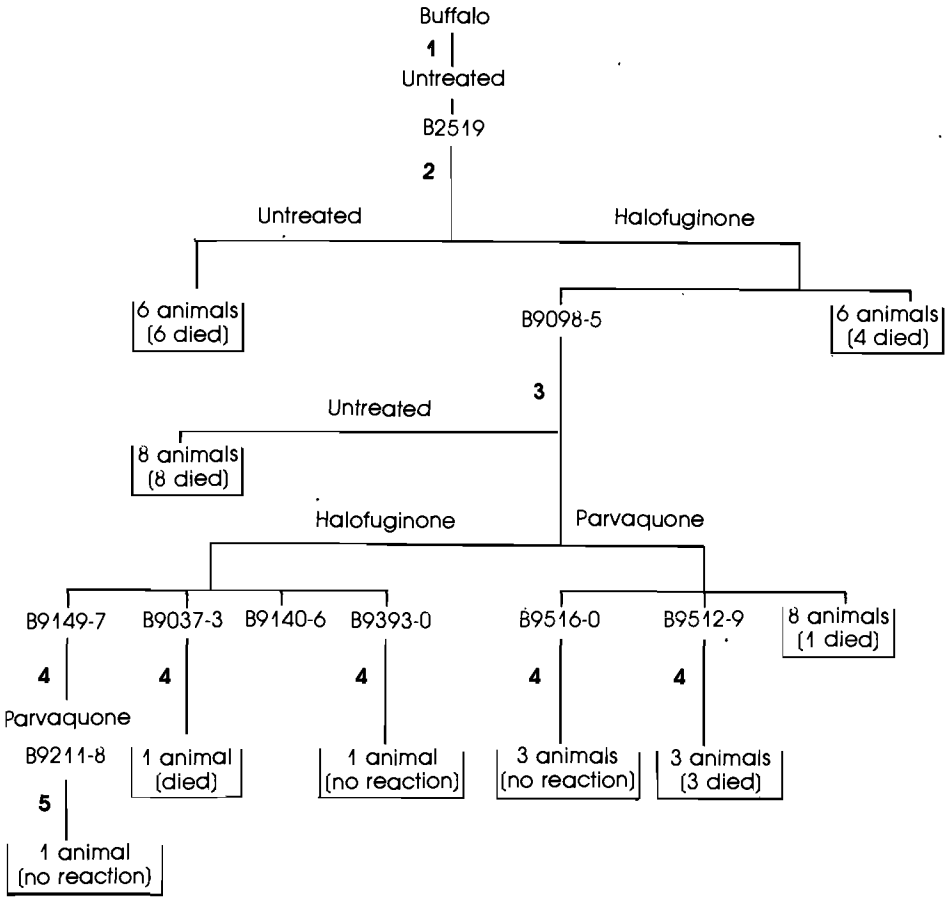

Fig. 3: Schematic representation of the transmission of *Theileria parva lawrencei* buffalo isolate (Hluhluwe 3) from cattle to cattle with *Rhipicephalus appendiculatus*. Each tick-passage is indicated by numerals 1 to 5

It is now at least possible to obtain piroplasm parasitaemias and seroconversion in susceptible splenectomised cattle following subinoculation of large volumes of blood from infected recovered cattle.

The vector potential of other tick species that may be involved in the transmission of *Theileria* infections, should be studied for xenodiagnostic application. More attention should be given to indirect methods using laboratory animals to obtain pure *T. p. lawrencei* isolates from buffaloes, as wild buffaloes are extremely difficult to work with.

Indications are that *T. p. lawrencei* infections cannot easily be sterilised and probably not with any of the existing anti-theilerial drugs. However, the effective therapeutic drugs used here have provided splenectomised and intact carrier cattle needed for the transformation studies.

Tick-transmission studies in the laboratory should concentrate on the use of *R. zambeziensis*. It is also recommended that more information regarding the biology and distribution of this tick be gathered.

Attempts to study possible behavioural changes of *T. p. lawrencei* under conditions of tick-passage in cattle will probably continue with *T. p. lawrencei* isolates from buffaloes in the Kruger National Park. More use of intact cattle for this purpose is envisaged. Having shown that recovered intact cattle can infect ticks, it is emphasised that chemotherapy of infected cattle involved in a Corridor disease outbreak is not recommended as it may create carriers.

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TREATMENT OF VARIOUS FORMS OF BOVINE MASTITIS WITH CONSIDERATION OF UDDER PATHOLOGY AND THE PHARMACOKINETICS OF APPROPRIATE DRUGS: A REVIEW

J H DU PREEZ*

ABSTRACT

Various forms of clinical and subclinical mastitis occur in the bovine, and intramammary therapy alone or in combination with parenteral therapy must be implemented with due consideration of udder pathology and the pharmacokinetic properties of drugs used for the treatment of mastitis. Supportive therapy for cows with acute or peracute mastitis and optimal animal husbandry are of the utmost importance for a relatively good prognosis.

The withholding period of milk for human consumption after the last intramammary antibiotic treatment as well as minimum intervals between antibiotic dry cow treatment and calving are given. The main reasons for mastitis therapy failure are udder pathology caused by the inflammatory process, the pharmacokinetic properties of mastitis drugs under those conditions, inadequate supportive therapy and poor animal husbandry.

Du Preez J.H. Treatment of various forms of mastitis with consideration of udder pathology and the pharmacokinetics of appropriate drugs: a review. *Journal of the South African Veterinary Association* **(1988) 59 No. 3, 161-167 (En). Department of Veterinary Public Health, Faculty of Veterinary Science, University of Pretoria, Private Bag X04 0110 Onderstepoort, Republic of South Africa.**

DEFINITION AND CLASSIFICATION OF MASTITIS

"Mastitis" means inflammation of the milk gland with physical, chemical and microbiological changes characterised by an increase in somatic cells, especially leucocytes, in the milk and by pathological changes in the mammary tissue. In clinical mastitis all 5 cardinal signs of udder inflammation are present to a greater or lesser degree, in contrast to subclinical mastitis where the clinical signs are absent and the udder secretion, which reflects the status of the udder parenchyma, is used to determine the health status of the milk gland. It is important to classify the various forms of mastitis as therapy must be applied accordingly.

Bovine mastitis is dynamic and therefore the threshold values used in the diagnosis thereof can only be selected more or less arbitrarily. More than one objective parameter must always be used in the classification of mastitis[23]. In the undermentioned classification of the various forms of mastitis only aerobic, facultative anaerobic and microaerophylic micro-organisms have been taken into account and not obligate anaerobic pathogenic micro-organisms as isolated from mastitis cases by Du Preez & Greeff[11].

Normal quarter and secretion (Table 1): The quarter shows no outward signs of a pathological condition and the milk it produces is free from pathogenic organisms and has a normal somatic cell count[21 49].

Latent infection (Table 1): Mastitis pathogenic micro-organisms exist within the mammary gland without evidence of subclinical or clinical mastitis and the milk has a normal somatic cell count[21 49].

Teat canal infection (TCI): Micro-organisms invade and proliferate in the teat canal tissue causing local, cellular damage with a normal or sometimes increased somatic cell count[8 9].

Aseptic or non-specific mastitis (Table 1): There is no recognisable udder/quarter infection and the clinical signs may be subclinical or clinical[21 49].

True subclinical mastitis (Table 1): Where pathogens are detected in milk samples collected aseptically with inflammatory changes in the secretion that can be detected by laboratory methods, but without macroscopic changes in either the secretion or the udder tissue.

Examination of milk reveals udder infection due to the presence of products typical of inflammation, such as leucocytes and epithelial cells as seen in the increased somatic cell count, fibrin clots, serum etc and by changes in the clinical composition of the milk due to suppression of secretion, with the transfer of sodium chloride and bicarbonate from blood to milk usually bringing about a shift of pH to a more alkaline level[21 49].

In all these definitions of the various forms of subclinical udder infections/inflammation, the somatic cell count of the milk is most important. The threshold[49] value of normal foremilk is recognised as being no higher than 500 000 cells ml^{-1}.

The clinical forms of mastitis classified according to severity and duration can be divided into peracute, acute, subacute and chronic mastitis[21 23 44 49].

Peracute mastitis: It is usually characterised by a sudden onset of inflammation. All 5 cardinal signs of udder inflammation are readily apparent in the peracute form of mastitis and in addition there are accompanying systemic signs of fever, depression, shivering, anorexia, rapid loss of weight, rapid weak pulse, dehydration (e.g. sunken eyes), and weakness. Other systemic signs can also occur depending on the causative pathogenic mastitis micro-organism and its virulence. The milk is macroscopically abnormal.

Acute mastitis: Characterised to a lesser extent by all 5 gross cardinal signs of udder inflammation with an accompanying slight to mild fever and mild depression. The milk is macroscopically abnormal.

Subacute mastitis: The cardinal signs of mastitis are subdued and not accompanied by systemic effects. Changes in the udder secretion are less marked.

Chronic mastitis: An inflammatory process that persists over many months or from one lactation period to the next. The quarter fails to respond to treatment over a period of time and may atrophy. There is a progressive development of fibrous tissue and consequently a morphological disturbance with induration detectable

Table 1: **The classification of subclinical bovine mastitis (udder infections/inflammation) according to the International Dairy Federation (IDF) criteria[21 49]**

Somatic cell count ml^{-1} milk	Pathogenic micro-organisms	
	Not isolated	**Isolated**
<500 000	Normal secretion	Latent udder infection (LUI) and teat canal infection or colonisation (TCI)*
>500 000	Non-specific mastitis or aseptic mastitis (ASM)	Mastitis (true sub-clinical mastitis)

* TCI is classified here according to Du Preez[8 10]. According to the IDF criteria no such condition as TCI exists, nor can it be identified.

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on palpation and enlargement and asymmetry of the gland in shape and size. The affected quarter/s may show these abnormal clinical changes for the rest of the animal's life. Chronic mastitis manifests for the most part in a subclinical form with periodic flare-ups or recurrent udder inflammation producing subacute or acute clinical signs which usually subside shortly thereafter and revert to the subclinical form.

MASTITIS THERAPY

For antibacterial therapy of mastitis to be successful, the active drug must reach the bacteria at the focus of infection in concentrations exceeding the minimal inhibitory concentration (MIC), and these levels must be maintained for the appropriate time necessary to break the production and toxin-producing cycle of the causative pathogen[26].

Intramammary treatment is accepted as the route of choice for subclinical, chronic or mild clinical mastitis[15, 35]. In acute clinical mastitis, however, frequent failures in intramammary antibiotic therapy are considered to be due to poor or uneven distribution of the drug throughout the intensely swollen udder parenchyma, the milk duct system being either compressed or blocked by inflammatory products[34 58]. It has been suggested that parenteral antibiotic therapy is indicated under these circumstances[15 34 58]. A decision must be made as to whether intramammary, systemic, or a combination of systemic and intramammary antibiotic therapy should be used in treating mastitis[29].

LOCAL OR INTRAMAMMARY MASTITIS THERAPY

The administration of drugs by intramammary infusion is by far the most common method of treating bovine mastitis[26 58]. For streptococcal infections where the bacteria are not tissue-invasive and for early cases of staphylococcal mastitis, intramammary drug infusion generally gives satisfactory results[26]. For intramammary treatment, drugs that are distributed throughout the udder and are quickly absorbed into the general blood circulation (Table 2) should be the choice of treatment. In peracute or acute clinical mastitis, cases where systemic signs always occur, intramammary infusion with antibiotics alone or in combination with corticosteroids is recommended[58]. The withholding period of milk for human consumption after the last intramammary antibiotic treatment, is indicated in Table 3.

Any teat canal or intramammary treatment must be administered after milking. After oxytocin administration, thorough stripping of the affected quarter prior to antibiotic infusion, is a useful procedure in the treatment of acute mastitis[53]. If there is no clinical improvement within 24 - 48 h after initiation of intramammary and/or parenteral therapy, it is advisable to change to a different antibiotic. The undermentioned therapy for teat canal infection or mastitis is recommended according to existing literature[8 9 20 25 26 29 35 42 44 53 59 60 61]

The protein binding of antibiotics in bovine serum varies considerably. The higher the proportion of antibiotics bound to serum proteins, the less effective is the drug in destroying or eliminating the pathogen[1 65].

Table 2: **Classification of antibacterial drugs according to their potential distribution throughout the udder after parenteral and intramammary administration***

Parenteral**	Intramammary***
Good distribution:	Good distribution:
Sulphanilamide	Quinolines
Erythromycin	Sulphanilamide
Oleandomycin	Other sulphonamides
Tylosin	Dapsone
Spiramycin	Nitrofurans
Lincomycin	Erythromycin
Clindamycin	Oleandomycin
Penethamate	Tylosin
Chloramphenicol	Spiramycin
Trimethoprim	Lincomycin
Tiamulin	Clindamycin
Limited distribution:	Penethamate
Other sulphonamides	Ampicillin
Penicillin G	Amoxycillin
Cloxacillin	Hetacillin
Ampicillin	Cephalexin
Amoxycillin	Chloramphenicol
Cephalosporins	Trimethoprim
Tetracyclines	Novobiocin
Novobiocin	Rifamycin SV
Rifamycins	Limited distribution:
Fusidic acid	Penicillin G
Poor distribution:	Cloxacillin
(Dihydro) streptomycin	Oxacillin
Neomycin	Cephoxazole
Kanamycin	Cephalonium
Aminosidine	Cephapirin
Spectinomycin	Cephacetrile
Gentamycin	Tetracyclines
Polymyxins	Poor distribution:
Vancomycin	Bacitracin
	Tyrothricin
	(Dihydro) streptomycin
	Neomycin
	Kanamycin
	Aminosidine
	Gentamicin
	Polymyxins

* From Ziv[61]
** As determined experimentally from the milk/serum drug concentration ratios
*** As determined experimentally from the rate of drug absorption from the udder or from physicochemical properties of the drug

Table 3: **Lactating cow intramammary preparations and resulting milk withholding period for human consumption after the last treatment***

Preparation	After the last treatment milk for human consumption may be take from: h	Milkings
Cefuroxime 250 mg (Spectrazol Milking Cow)	60	5
Cloxacillin 200 mg, ampicillin 75 mg (Ampiclox Lactating Cow)	60	5
Nifuroquine 10 mg ml^{-1} (Abimasten)	84	7
Oxytetracycline HCl 30 mg g^{-1} x 15 g (Terramycin Intramammary Solution)	72	6
Sodium benzylpenicillin 180 mg, dihydrostreptomycin sulphate 100 mg, sodium nafcillin 100 mg (Nafpenzal Milking Cow)	84	7
Sodium cloxacillin 200 mg (Orbenin Lactating Cow)	60	5

* From Booth[4]

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Table 4: **Binding of antibiotics and sulphonamides in bovine serum**

Drug	Percentage bound to serum protein
Oxytetracycline	20
Metacyline	Extensively, 80-90
Doxycycline	Extensively, 80-90
Minicycline	Extensively, 80-90
Penicillins	
Cloxacillin	Highly bound, about 75
Penethicillin	Moderately bound, 35-65
Phenoxymethylpenicillin	Moderately bound, 35-65
Benzylpenicillin	Moderately bound, 35-65
Ampicillin	18
Cephalexin	Concentration-dependent
Chloramphenicol	Bound between 25-50
Macrolides	
Erithrimycin	Bound between 25-50
Lincomycin	Bound between 25-50
Tylosin	Bound between 25-50
Trimethoprim	Bound between 25-50
Aminoglycosides	Occur predominantly free in blood plasma
Streptomycin	Less than 13% bound
Gentamycin	Less than 13% bound
Kanamycin	Less than 13% bound
Spectinomycin	Less than 13% bound
Sulphonamides	Varies with the compound
Sulphadimethoxine	83
Sulphadimidine	65
Sulphadiazine	24

* From Ziv and Sulman[65]

PARENTERAL MASTITIS THERAPY

Successful intravenous or intramuscular mastitis therapy depends on effective passage of the drug from the blood into the milk near the foci of infection. The extent to which a drug gains access into milk from the circulation is largely governed by 3 properties of the drug molecule, namely: lipid solubility, degree of ionisation [dependent on the dissociation constant (pKa)] and the extent of protein-binding with serum[47] (Table 4). Passage of drugs across the blood-to-milk barrier takes place by passive diffusion[27]. Similar to all biological membranes, this barrier is breached readily only by the non-ionised, non-protein bound, lipid-soluble fraction of the drug[37 40].

Plasma-protein binding may influence a drug's therapeutic efficacy since only the unbound or free drug is available to distribute out of the vascular system and to exert pharmacological or antimicrobial activity[1].

The blood circulation[17] through the udder of a cow producing 20 kg milk day^{-1} is approximately 10 000 ℓ blood day^{-1} or 7–10 ℓ min^{-1}. Systemic administration of rationally selected antibacterials to a mastitic bovine udder with such a good blood supply may be used as the sole treatment in acute mastitis. Even when marked udder changes are present, systemic administration of drugs can give very even distribution throughout udder tissue[26].

An ideal antibiotic intended for parenteral mastitis therapy should have the following characteristics[61]:

(a) low MIC against the majority of udder pathogens;
(b) high bio-availability from the intramuscular injection site;
(c) chemically, a weak base or otherwise highly non-ionised in serum;
(d) sufficient lipid solubility;
(e) low degree of serum-protein binding
(f) long half-life in the body;
(g) clearance from body organs and tissues similar to the clearance of the drug from the blood, i.e. no drug accumulation in specific organs such as the udder.

To the best of present knowledge, no single antibiotic meets all these requirements. Following systemic administration, basic drugs [if sufficiently lipid soluble] tend to concentrate in milk. The non-ionised fraction diffuses readily across the blood-milk barrier, reaching an equal concentration on each side[26].

Other preparations which can contaminate the milk include injectable sulphonamides, pessaries, uterine infusions, and approximately 100 other preparations, including corticosteroids, whose data sheets contain information on the withholding periods necessary to avoid the contamination of milk for human consumption[4]. The specific public health implications of residues in milk are not discussed in this review.

SPECIFIC THERAPY ACCORDING TO THE VARIOUS FORMS OF MASTITIS

It is always ideal to treat any teat canal, intramammary infection or mastitis according to the antimicrobial drug sensitivity pattern of the pathogens. As it takes time to do sensitivity determinations, broad spectrum antibiotics must be given initially for practical reasons. In general, narrow spectrum antibiotics are bactericidal and those having a broad spectrum are bacteriostatic[53]. If no sensitivity determinations can be done, broad-spectrum antibiotics effective against Gram-positive and Gram-negative bacteria must be the choice, based on the knowledge of the pharmacokinetic properties of the drugs and their formulation[8 26 29 59 60 61]. Clinical mastitis should be treated as soon as it is diagnosed[53].

Aseptic milk samples should be collected from each clinical case of mastitis before treatment and stored in a refrigerator or deep-freeze. These samples can then be examined bacteriologically if there is no response to therapy[53]. If no success is achieved on treating mastitis during lactation, the affected quarter may be treated with great success at drying-off[53].

TCI: Teat canal therapy with small quantities of antibiotic [1 to 2 droplets] introduced 3 times at 12-hourly intervals to lactating cows, effectively eliminates teat canal infection[8 9].

Subclinical mastitis: The treatment of subclinical udder infections is really only useful if appropriate measures of hygiene have first been taken to avoid a rapid return to the original situation[35] which predisposed the infection. Intramammary antibiotic therapy only is usually advocated. Three infusions of intramammary antibiotic preparations at 12-hourly intervals are recommended. Exceptionally, parenteral antibiotic therapy can be given where extremely virulent pathogenic bacteria are involved or where very valuable dairy cows are affected[29 42 44].

Subacute clinical mastitis: This is the most prevalent form of clinical mastitis in dairy herds. Infusion of intramammary antibiotic preparations at 12-hourly intervals for 2 to 5 d is recommended, depending on the causative pathogen and clinical response. Initially a double intramammary antibiotic preparation may be infused into the affected quarter in high-yielding cows. Routine parenteral antibiotic therapy is not advocated in subacute clinical mastitis but it may be applied in circumstances as mentioned for subclinical mastitis[17 29 41 42 43 44].

Acute clinical mastitis: The same antibiotic or antibiotics with the same antibacterial action or with synergistic action or which are compatible must be given parenterally as well as locally for 3 to 5 d depending on clinical and bacteriological cure. For parenteral therapy the intravenous route must be used to achieve maximum parenchymal diffusion[26 61]. Initially, but only once, infusion with a double intramammary antibiotic formulation into the affected quarters and a single intramammary antibiotic formulation into each of the healthy quarters is recommended to prevent new intramammary infection. After the initial infusion of antibiotic preparations into the affected quarter, intramammary antibiotic preparations, as recommended by the manufacturer, should preferably be infused at 12-hourly intervals and continued for 3 to 5 d. For increased bio-availability of the drug it is preferable to administer a large volume of antibiotic intramuscularly at 2 sites[20 25 26 29 42 43 44 53 61]. For supportive therapy and optimal animal husbandry see Table 5.

Peracute clinical mastitis: In principle, treatment is the same as for acute mastitis but should be more aggressive and special attention should be given to shock[20 29 42 43 44 51 53].

When the response to antibiotic and supportive therapy for acute and peracute mastitic cases is insufficient, additional steps may be required to prevent death or to treat a severely affected quarter. When the affected quarter is gangrenous or severely damaged, intramammary treatment will be ineffective and teat amputation may help drainage[42]. Tying off the mammary veins

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Table 5. **Supportive therapy for cows with acute/peracute mastitis**

	Therapeutic	Dosage	Route of administration	Interval between doses	Comments
1a.	Isotonic saline	5-10 ℓ	iv	4-8 h	Essential to restore circulating volume quickly with endotoxic/hypotensive shock*
1b.	Isotonic saline	25 ℓ	oral	24 h	Possible alternative to above
2.	Oxytocin	20-30 iu	iv	12 h	Facilitates stripping
	Frequent milking			every 1-4 h	Keeps milk ducts patent, removes toxins, bacteria and inflammatory products
3.	20% calcium borogluconate	400-800 ml	iv	–	If indicated (coliform mastitis), give slowly and diluted in saline
4.	Glucose	2-5 ℓ	iv	12h	Reverses hypoglycaemia and for glycogen depletion
5.	Etamiphylline camsylate or theophylline	1400 mg	iv or im	8 h	Helps cardiac output, increases the stroke volume of the heart and improves pulmonary function
6.	Aspirin	30 g	oral	8 h	Reduces pain, temperature and inflammation and restores appetite
7.	Concentrated multivitamins especially B-complex group	30 ml	iv or im	24 h	May help liver, supplement enzyme systems
8.	Corticosteroids (Dexamethasone)	1-3 mg kg^{-1}	iv or im	–	Cost may be prohibitive, impairs defence systems?
9.	Immunoglobulin (γ globulin, 75,0% m/v of total protein)	20-50 ml	iv	6 h	Neutralise toxins, stimulate and improve phagocytosis
10.	Antihistamins		iv or im	12 h	Anti-inflammatory, counteract shock

* Supportive therapy with fluids is often more important than the antibiotic selected; this is certainly true in endotoxic coliform mastitis.
Compiled from Giesecke[17], Jackson and Bramley[20], Report of panel[41] and Robinson[42]

Animal husbandry
a. Water: Fresh, cool drinking water must be freely available.
b. Food: Soft digestable and nutritious food must be available ad lib.
c. Rest: Enough rest without disturbance is of the utmost importance for rapid recovery.
d. Protection against exogenous (e.g. sun, wind, noise etc) and endogenous (decreased drug-induced defence mechanism etc stress factors as far as possible.

has also been advocated in severe cases of mastitis to reduce the invasion of toxins into the bloodstream[42]. Antitoxins may also be used. Severely affected cows that have survived the peracute stage may not recover completely. There may be a persistent pyrexia and evidence of damage to vital organs such as the liver. The prognosis is poor in these cases[41 42].

Chronic mastitis: The ideal is to cull the cow or to eliminate or destroy the affected quarter/s by means of an infusion of 25 to 40 ml of concentrated ether into the quarter to eliminate an important potential source of bacterial infection in healthy quarters. Parenteral and intramammary antibiotic treatment[56] for 3 to 5 d according to the sensitivity pattern of the pathogen, as well as anti-inflammatory therapy may be used, but the prognosis remains poor. Infusion of 100 to 250 ml of a 5% or 10% dextrose solution in combination with above-mentioned antibiotics on 2 or 3 occasions, with 12-hourly intervals, may be beneficial[41 42 44 56].

THE PHARMACOKINETIC PROPERTIES OF COMMONLY AVAILABLE MASTITIS DRUGS

Penicillins

The majority of isolates of haemolytic staphylococci from bovine milk samples are resistant to penicillin whereas virtually all streptococci are sensitive to the drug[13 26]. The activity of penicillin is decreased only slightly in milk[36]. Penicillin is a weak organic acid with pKa 2,7 and it is therefore largely ionised in plasma; milk levels will therefore always be lower than plasma levels[37 39]. While absorption rate is moderate, penicillin is well distributed throughout the udder and diffuses relatively well into mammary tissue in both normal and mastitic glands, except where there are large areas of necrosis[15 50 57]. Such diffusion probably occurs because penicillin is moderately lipid-soluble[56]. Moreover, penicillin concentrations are higher in the milk from mastitic udders than from normal quarters[15]. Penicillin is non-irritating following local infusion, has a low degree of protein binding and is moderately lipid-soluble. Moore & Heider[29] recommended an intramammary penicillin dose of 11 000 IU kg^{-1} twice daily, continued for 3 to 5 d for mastitis therapy.

Sodium benzylpenicillin: The majority of isolates from mastitic cases caused by *Staphylococcus aureus* are resistant whereas virtually all streptococci are sensitive[25 26].

Cloxacillin: This is a narrow-spectrum semi-synthetic penicillin, highly lipid-soluble and resistant to staphylococcal penicillinase. In lactating cows, infusion of cloxacillin is as effective as benzylpenicillin against streptococcal mastitic infections[24 25 26].

Ampicillin: This is a semi-synthetic penicillin with a broad spectrum of activity which diffuses into the udder slightly better than benzylpenicillin[62]. Dosage necessary to maintain milk levels that are effective against micro-organisms (i.e. with MICs in the range of 0,5 to 1,0 mcg ml^{-1} for 24 h is approximately 10-20 mg kg^{-1} [56]. This dosage is several times higher than that usually recommended[25]. Moore & Heider[29] recommended an intramuscular ampicillin dose of 10 mg kg^{-1} twice daily, and an intramammary dose of 62,5 mg twice daily, continued for 3 to 5 d.

Benzathine penicillin: Parenteral doses as high as 9 million units per cow are unlikely to produce bacteriostatic levels in milk[45], which reflects the low blood levels obtainable with benzathine penicillin[25].

Dihydrostreptomycin

Dihydrostreptomycin, one of the aminoglycosides, is a base with pKa 8,0 and a low lipid-solubility and is therefore unsuitable for systemic treatment of mastitis[26]. Dihydrostreptomycin is bacteriocidal at concentrations approximately 3-4 times higher than the MIC[55]. However, the activity of dihydrostreptomycin is markedly decreased in the presence of milk[55], possibly because of extensive binding to milk protein[64]. Ziv[56] suggests that dihydrostreptomycin in doses of 10-20 mg kg^{-1} every 6-12 h may be suitable for treatment of Gram-negative udder infections. Dihydrostreptomycin has a very uneven distribution within the udder, taking up to 8 h to become widely distributed throughout the udder parenchyma[57]. The aminoglycosides have fairly low MICs for staphylococci and for some Gram-negative mastitis pathogens, but their activity against streptococci is low[30]. It is unlikely that dihydrostreptomycin, even in very high doses, will reach therapeutic levels in milk[63]. Dihydrostreptomycin is seldom used alone for intramammary treatment but more often in combination with penicillin[24].

Tetracyclines

Oxytetracycline and chlortetracycline are partially inactivated in milk by chelation with magnesium and calcium ions and by combination with casein[28]. Injectable oxytetracycline possesses limited intramuscular bio-availability properties[1 60] and should therefore be administered intravenously[60]. Oxytetracycline administered by intramuscular injection will not produce therapeutic levels in the

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milk. However, large doses in the order of 10 mg kg^{-1} administered intravenously will maintain milk concentrations of oxytetracycline above 1,0 mcg ml^{-1} for 24 h. The average MIC of oxytetracycline for mastitis pathogenic staphylococci and streptococci is about 1,0 mcg ml^{-1} Gram-negative organisms require higher concentrations, 2,44 mcg ml^{-1} being the mean MIC for sensitive *E. coli*[33]. Oxytetracycline is irritant following local udder infusion, absorption is very poor and it is very unevenly distributed in normal udder tissue[57]. Moore & Heider[29] suggest an intravenous oxytetracycline dose of 20 mg kg^{-1} daily for mastitis therapy.

Neomycin

Ziv & Sulman[65] showed that neomycin passed poorly into milk in both normal and mastitic mammary glands, and suggested that its limited penetration was due mainly to poor lipid-solubility. This would curtail its potential usefulness in the parenteral treatment of mastitis. Milk markedly decreases the activity of neomycin, the MICs being up to 500 times higher when tested in milk[36]. Neomycin had been used as the chief ingredient in combination drugs for intramammary mastitis therapy because of its wide antimicrobial spectrum[43].

Erythromycin

Erythromycin as well as tylosin, lincomycin and spiromycin are macrolide antibiotics. Effective passage of a drug from the blood into the udder is best achieved with the macrolide antibiotics, but the antibacterial spectra of these drugs are limited to Gram-positive pathogens. The macrolide antibiotics are the logical choices when attempting to eliminate persistent Gram-positive udder infections[61]. For the treatment of acute mastitis due to Gram-positive udder pathogens, combined parenteral and intramammary application of the macrolide antibiotics appears to be logical and rational on bacteriologic and pharmacokinetic grounds[61]. Erythromycin is a highly lipid-soluble base with pKa 8,8[56]. After systemic administration, it normally reaches levels in milk 4 - 5 times higher than those present in plasma, but with rising pH, as occurs in mastitis, levels in milk are decreased[37 39]. MICs of erythromycin for common mastitis-causing bacteria range from 0,025 to 0,05 mcg ml^{-1} for streptococci, and from 0,2 to 0,39 mcg ml^{-1} for sensitive strains of *Staphylococcus aureus*. Erythromycin at a dosage of 12,5 mg kg^{-1} repeated at 24 h intervals was shown to maintain milk levels of more than 11 mcg ml^{-1} over the dosage interval. Peak levels ranging from 6,5 mcg ml^{-1} were reported[2]. Moore & Heider[29] recommended an intramuscular dose of 5 mg kg^{-1} erythromycin twice daily, and an intramammary dose of 300 mg, twice daily, for 3 to 5 d.

Chloramphenicol

Chloramphenicol possesses limited intramuscular bio-availability properties[1 61], and should therefore be administered intravenously[61]. MICs of chloramphenicol for sensitive strains of *S. aureus* are between 3,2 and 8,2 mcg ml^{-1}, and the activity of chloramphenicol against these organisms is not significantly affected in the presence of milk[36]. When administered at a dosage rate of 50 mg kg^{-1}, chloramphenicol has been reported to produce milk levels of 22,8 mcg ml^{-1} 12 h and 12,2 mcg ml^{-1} 24 h after injection[18]. Concentrations of chloramphenicol in secretions from acutely inflamed glands are slightly higher than in milk from normal quarters[62].

Sulphonamides and trimethoprim

The average range of MICs of sulphonamides for sensitive streptococci is between 2 and 16 mcg ml^{-1}, and for sensitive staphylococci this lies between 8 and 64 mcg ml^{-1} [16]. Sulphonamide concentrations of 50 to 150 mcg ml^{-1} are considered therapeutic[5 26]. After intravenous injection, the sulphonamides are distributed evenly throughout the mammary gland, in both normal and indurated udder tissue[38]. Sulphadimidine, when given intravenously at a dose of 200 mg kg^{-1}, will maintain milk levels of more than 50 mcg ml^{-1} for 20 h[45]. Moore & Heider[29] recommended an initial dose of sulphamethazine of 100 mg kg^{-1} intravenously and then 50 mg kg^{-1} daily intravenously for 3 to 5 d. Trimethoprim has a rather short half-life in cattle which varies between 50 and 100 min and this limits the usefulness of the drug, necessitating very high doses[6 26]. The suspension of trimethoprim in sulphadiazine (Tribrissen 48%), at a dosage rate of 48 mg kg^{-1}, is absorbed more slowly and might be expected to provide effective milk levels for 12 h[52].

Cephalosporins

The cephalosporins, which possess broad spectrum activity against many Gram-negative udder pathogens and beta-lactamase producing staphylococci, may provide replacement for antibiotic combinations[46]. Cephalosporins have a limited distribution in the udder after parenteral and intramammary mastitis therapy[61]. Cephoxazole is bacteriocidal and resistant to destruction by staphylococcal penicillinase, and by binding to penicillinase produced by Gram-negative bacteria, allows penicillin to act on these otherwise insensitive pathogens. Cephoxazole and penicillin have a mutually potentiating effect[19].

REASONS FOR MASTITIS THERAPY FAILURES

1. Tissue invaders: Tissue-invading bacteria such as staphylococci become walled off in the udder parenchyma by thick, fibrous scar tissue[26] so that the antibiotic cannot reach the pathogen. Therefore bacteriological failures may occur even when the organisms are sensitive to the antibiotics used[34]. *Staphyloccoccus aureus* udder infections promote development of localised scar tissue which does not have blood vessels, so that intramuscular and intravenous injections probably provide little benefit. Therapy may kill the bacteria that are not walled off, but at a later date the bacteria within the scar tissue can break out, multiply, cause additional damage to the udder secretory tissue and promote further formation of scar tissue[32]. *S. aureus* and other pathogens can survive, in some instances, within leucocytes. When antibiotic treatment is administered, such pathogens may not come into contact with the drug and are therefore not killed[32].
2. Milk duct obstruction: In all cases of mastitis, oedema and inflammatory products to a certain extent, obstruct the diffusion of antibiotics by compression or blockage of the milk duct system. The diffusion of antibiotic solutions throughout the gland is impaired and for this reason it is often very difficult to bring antibiotics into contact with mastitis-causing bacteria, especially with intramammary therapy. Many cases of mastitis are thus resistant to treatment even when the mastitis pathogenic bacteria are fully sensitive to the antibiotic used[26 51 61]. Frequent milking at 1 to 2 h intervals is recommended to remove toxins, debris, bacteria and other inflammatory products and to maintain milk duct patency[7 41].
3. Clinical cure: In streptococcal mastitis, and in some staphylococcal mastitis, therapy usually results in a clinical cure, but the bacteriological cure rate is low[35 43]. Improper treatment procedures such as treating for too short a period may result only in a clinical but not a bacteriological cure. Systemic treatment as an adjunct to intramammary treatment has been advocated as a means of overcoming these problems[48].
4. Low bio-availability: Injectable oxytetracycline and chloramphenicol possess limited intramuscular bio-availability properties, and should therefore be administered intravenously[61]. Table 2 lists the drugs which are slowly absorbed and poorly distributed throughout the udder after intramammary and/or systemic treatment.
5. Weak passage of drugs across the blood-milk barrier: The degree of transfer of drugs from blood into milk is directly proportional to the concentration gradient across the membrane and inversely proportional to the extent to which the drug is ionised, since ionised molecules pass through biological membranes only with difficulty[25 26]. Effective passage of drug from blood into the udder is best achieved with the macrolide antibiotics (erythromycin, lincomycin, spiromycin and tylosin), but the antibacterial spectra of these drugs are limited to Gram-positive pathogens[61].
6. Drug resistance: Selecting the wrong and ineffective antimicrobial agent[61], e.g. penicillin to treat beta-lactamase-producing *S. aureus* and *Bacteroides fragilis*. This enzyme destroys penicillin, so that treatment with penicillin would most likely be ineffective. Some *B. fragilis* mastitogenic strains are resistant to ampicillin, cephalotine and amoxycillin[10]. Some strains of *B. fragilis* produce cephalosporinase which make them resistant to cephalosporin[14]. While bacterial resistance receives most attention, more practical problems, e.g. development of localised scar tissue in the udder, blockage of the milk ducts etc., probably have a greater effect[32]. Biochemical resistance of bacteria to antimicrobial agents may occur through mutation, natural selection, transformation, transduction or conjugation[54]. Although the bacteria were initially sensitive to the antimicrobial

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agent, they may become resistant and a different anti-microbial agent must then be used.

7. Aminoglycocidal antibiotics: Some of the aminoglycocidal group of antibiotics possess a weak degree of activity against anaerobic mastitogenic bacteria[10 22]. Aminoglycocides are all basic antibiotics and their poor lipid-solubility makes them unsuitable for systemic treatment of mastitis. With insufficient lipid-solubility the drug does not diffuse readily through cell membranes[26 61].
8. Udder tissue necrosis: Mastitis which causes udder tissue necrosis leading to a poor blood supply of the affected areas and consequently a decreased redox potential that favours anaerobic mastitogenic bacteria[14 17]. There is no effective passage of drugs into necrotic avascular udder tissue.
9. Local tissue concentration: The inability of attaining and maintaining therapeutic concentrations of the drug at the focus of infection in the udder tissue[61]. The success of systemic therapy against mastitis depends to a large extent on the concentration of antibacterial drugs achieved at foci of infection[25]. The MIC of the drug, and the physicochemical properties of the antibiotic are major determinants in the duration of effective drug concentrations in the udder[25 61].
10. Side effects: Expressed side effects with therapy minimise the use of such drugs[61]. Large intramuscular doses of certain tetracylines lead to severe swelling and oedema at the site of injection and consequently to a poor bio-availability.
11. Inadequate supportive treatment: No or minimal non-antimicrobial supportive treatment when advocated[61], e.g. if no shock treatment is given in peracute coliform mastitis.
12. Delayed initial treatment: Treatment should be commenced as soon as possible after the first appearance of mastitis — if possible within hours — since the prognosis depends on this.
13. Inaccurate diagnosis: Wrong clinical diagnosis of the aetiology of mastitis, e.g. if bacterial mastitis is diagnosed instead of a fungal or non-specific/aseptic kind, inappropriate therapy will be administered.
14. High degree of serum protein binding: Both oxytetracycline and chlortetracycline are partially inactivated in milk by chelation with magnesium and calcium ions and by combination with casein[28].
15. Half-life of the drug: The short plasma half-life of trimethoprim in cattle (50 -100 min) limits the usefulness of this drug. Trimethoprim in solution is eliminated too rapidly to make this product useful for mastitis therapy[6].
16. "L" form of bacteria: Many antibiotics such as penicillins and cephalosporins kill bacteria by preventing synthesis of new cell walls as new bacterial cells are being formed. Sometimes certain bacteria develop an "L" form. They survive without a cell wall and are enclosed in their cell membrane only. The "L" form is not susceptible to antibiotics that attack the cell wall. The "L" form can change back into normal bacteria[32 54].
17. Superinfection: Introduction of a second pathogen (e.g. fungi, yeasts) when for example a contaminated cannula is inserted into the teat[50] or with intramammary drug infusion where the teat tips are not thoroughly cleaned and sanitised before treatment[31].
18. Trauma: Tissues lining the teat duct are very delicate and any unnatural manipulation of this structure, such as cannula insertion, may jeopardise its antibacterial function[31]. Although a mastitic quarter is treated with antibiotics, trauma predisposes the quarter to infection or reinfection.
19. Insertion of infusion cannula: In quarters infected at drying off, treatment efficacy with partial insertion of infusion cannula into the teat canal is higher compared to full insertion of infusion cannula into the teat cistern[3].
20. TCI: Standard methods of antibiotic administration into a mastitic quarter or for dry cow therapy do not necessarily eliminate TCI. TCI serves as a potential source of bacteria for infection of the udder parenchyma[8 9 12]. After antibiotic treatment the existing TCI may cause mastitis.

CONCLUSIONS

The pathology of the udder tissue caused by mastitis and the consequent effect thereof on the pharmacokinetic properties of mastitis drugs, rather than widespread antibiotic resistance, seem to be the major reasons for therapy failures. Despite the widespread use of various antibiotics and other chemotherapeutic agents, antibacterial treatment of mastitis has generally been less effective than desired[58]. The answer to the mastitis problem lies in the prevention of new intramammary and teat canal infections which may cause mastitis, rather than the treatment of existing mastitic infections. The success rate in the treatment of mastitis is lower in lactating dairy cows than in dry cows, especially in the case of staphylococcal mastitis.

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PRACTICAL SMALL ANIMAL DERMATOLOGY III: BACTERIAL DISEASE

O M BRIGGS*

ABSTRACT

Bacterial infection is often overlooked as an aetiological factor in small animal dermatology. The misuse of glucocorticoids may result in immunosuppression in the patient. Pyoderma may then become refractory to all forms of therapy.

The impression smear is a simple technique to confirm a pyoderma. Empirical selection of antimicrobial agents is acceptable for initial therapy of uncomplicated pyoderma. Appropriate antibiotics must be used at therapeutic doses for extended periods. Primary pyoderma is caused by pathogenic bacteria alone. The underlying cause in secondary pyodermas must be eliminated in order to prevent recurrence.

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INTRODUCTION

Pyderma is the term used to denote pustular skin infection. Purulent material ("pus") is however, often absent. Surface pyodermas may appear moist and glistening, while superficial pyodermas may present as circular scaling (epidermal collarette)[11]. Furthermore, pustules may be found in diseases such as the pemphigus group, dermatophytosis and demodicosis which are not of bacterial origin[11]. Pustules, being fragile, are often not found on routine clinical examination[7]. Careful examination of the whole integument, including the ventrum and axillae, is required in order to find these less apparent lesions[2].

Pruritus in dogs is not always allergic in origin[11 12]. Bacterial infection is often present and when treated with an appropriate antibiotic, the pruritus diminishes. Impression smears of pustules provide a valuable screening test and are simple to perform[2 7]. As a rule, if neutrophils and engulfed coccoid organisms are seen[2], an antibiotic effective against Gram-positive *Staphylococci* should be administered for at least 3 weeks[13]. If pruritus persists, examine for other causes.

Bacterial disease of surface and superficial layers of the skin is less common in cats than in dogs[14]. Infection secondary to flea bites, and abscesses following fight wounds are relatively more common in cats.

The pet with pyoderma may be in a healthy condition, or it may be apparently healthy, but immunologically incompetent. The latter condition may lead to recurrent and severe pyoderma, is often seen in certain families of dogs, and is particularly difficult to treat.

CLASSIFICATION

Pyodermas may be primary or secondary (Table 1). Primary pyoderma occurs when an animal is exposed to a particularly pathogenic bacteria. In dogs, bacterial skin disease is often secondary to allergies, seborrhoea, parasitic infestation, or abnormal keratinisation. Therapeutic success depends on recognising the distinction between primary and secondary pyoderma.

Bacterial disease is divided into 3 types according to the depth of infection (Table 2). Surface pyodermas occur when pathogenic bacteria colonise areas of inflammation in the superficial layers of the skin. Superficial pyodermas are usually restricted to the epidermis only, whereas deep pyodermas include infections of the dermis and subcutis.

AETIOPATHOGENESIS

The organisms isolated from healthy skin are known as residents. These are present in a micro-environment dependent on normal glandular secretions, normal epidermal surface and absence of irritants. Once this microclimate is disturbed, pathogens become established causing dermatitis. The most frequently isolated residents and pathogenic bacteria on the skin of the dog are listed in Table 3. Previously, coagulase-positive *Staphylococcus aureus* was regarded as the primary skin pathogen in dogs[5 6]. However this role is now considered to be played by *S. intermedius*[7 13]. It is believed that most pathogenic *Staphylococci* previously identified as *S. aureus* were, in reality, *S. intermedius*[13].

Since cat abscesses usually result from fights, the bacteria most frequently isolated are those of the oral cavity such as *Pasteurella multocida*, β-haemolytic *Streptococci*, and *Fusiform* spp. A study of subcutaneous abscesses in cats revealed that they contained a mixture of 72% obligate anaerobes and 28% facultative anaerobes[10].

Bacteroides and *Fusobacterium* were the most prevalent obligate, and *P. multocida* the most prevalent facultative

Table 2: **Classification of canine pyoderma[57]**

Surface Pyoderma
Acute moist dermatitis ("hot spot")
Intertrigo (skin fold pyoderma)
facial
lip
obesity
vulva
tail
Superficial Pyoderma
Superficial pustular dermatitis ("impetigo")
Superficial folliculitis
Pruritic superficial pyoderma
short-haired dog pyoderma
bacterial hypersensitivity
inflammatory pyoderma
Deep Pyoderma
Folliculitis and furuncolosis
muzzle folliculitis ("acne")
nasal pyoderma
pressure-point pyoderma
pododermatitis
generalised folliculitis and furunculosis
Cellulitis
Subcutaneous abscesses

Table 3: **Canine cutaneous bacteriology[11 13]**

Resident organisms
Coagulase-negative *Staphylococcus* spp.
Micrococcus spp.
alpha-haemolytic *Streptococcus* spp.
Acinetobacter spp.
Corynebacterium spp.
Pathogenic organisms
Coagulase-positive *Staphylococcus* spp.
Proteus mirabilis
Pseudomonas aeruginosa

Table 1: **Characteristics of primary and secondary pyodermas**

Primary	Secondary
Occurs in otherwise healthy skin	Occurs in diseased skin
No apparent underlying cause	Underlying cause
One species of organism isolated	More than one species isolated
Characteristic disease pattern	Less characteristic disease pattern
Cured using appropriate therapy	Therapy of underlying disease critical

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anaerobes[3] [10]. Feline folliculitis yields β-haemolytic *Streptococci* and *Staphylococci*[12]. Dogs are infected with anaerobes from bite wounds, traumatic wounds and foreign body-associated infections[3].

CANINE PYODERMA

Surface pyoderma

Surface pyodermas include acute moist dermatitis and skin fold dermatitis (Table 2). Acute moist dermatitis ("hot spot") is common in southern Africa. These start with a small focal area of erythema and pruritus. The skin surface becomes moist and the hair mats over the lesion aggravating the condition. Predisposing factors include hot weather, humidity, sand, salt water, irritant chemicals and dips, patient compliance (is it a licker?) and a matted, unkempt hair-coat. The inciting cause is usually flea bites[11], but the presence of other parasites should not be ignored. Tick bites can mimic flea bites and the resultant self-trauma removes the tick prior to the owner noticing it. *Demodex* must be examined for, if hot spots recur in the same area. Other parasites incriminated include *Sarcoptes, Cheyletiella* and lice.

Aggravating factors in acute moist dermatitis include sand, sea water, greasy medications that mat the hair, and self-trauma[11]. These factors assist in the rapid spread of infection typical of this condition. Early and vigorous therapy is required, and yet only medications sympathetic to inflamed skin may be used. In mild, early, single hot spots, gentle cleansing with a medicated health shampoo (such as Gill, Adcock-Ingram) and the application of a soothing glucocorticoid containing ointment (such as Panalog, Squibb) may suffice. However, most lesions have progressed to the matted hair stage and require this to be removed either with a pair of scissors or under general anaesthesia. Since the condition may be painful and spread rapidly out of control, early general anaesthesia and wide shaving is advisable[11]. Haemorrhage can be controlled with an astiptic containing topical solution (for example, Lotogen, Byk Gulden). Glucocorticoid and antibiotic containing solutions (for example, Curatex, Kruger-Med) may then be applied. Certain hot spots (especially facial) require aggressive systemic antibiotic therapy as for deep pyodermas. These are now classified as both surface and deep pyodermas[7]. Elizabethan collars assist in reducing self-trauma. Whenever hot spots are encountered, it is advisable to review the ectoparasite control regimen.

Fold pyodermas often occur in purebreds for which the offending fold is a breed characteristic. Treatment as for acute moist dermatitis may provide temporary relief. However, the facial folds and "screw" tails of Bulldogs and the sagging jowls of the Bloodhounds and Spaniels may have to be removed surgically[11]. A surgical procedure is used to elevate a recessed vulva, and obesity folds may benefit from long term dietary management.

Superficial pyoderma

Superficial pyodermas involve deeper layers and often the hair follicles as well[12]. Pustular dermatitis usually involves the progression through a few characteristic stages; an erythematous papule; a pustule; a flattened crust which, when removed, leaves a moist, erythemic area. This condition which is likened to impetigo in man, invariably yields coagulase-positive *Staphylococcus* in dogs and *P. multocida* and β-haemolytic *Streptococci* in cats[10]. Primary causes must be examined for. Skin cleansing and general hygiene may provide adequate control in mild cases. In severe cases, a systemic antibiotic, effective against coagulase-positive *Staphylococci* (Table 4) is required.

In superficial folliculitis, the early lesion presents as a pustule with a hair protruding from the centre. It is important to differentiate this from superficial pustular dermatitis as folliculitis requires more intensive therapy. Systemic therapy with antibiotics effective against coagulase-positive *Staphylococci* is essential at therapeutic dosages for at least 14 d.

Pruritic superficial folliculitis must be ruled out in the pruritic dog[11]. The presence of pustules is often overlooked and the condition is misdiagnosed as an allergy. Coalescent pustules form a patch which heals from the centre resulting in a pealing rim of keratin (epidermal collarette) surrounding a hyperpigmented, alopecic centre. The administration of glucocorticoids at this stage results in immunosuppression and an ever-increasing set of complications. Another misdiagnosis is the so-called milk mange, puppy mange or mother mange which is often not mange, but impetigo (superficial pustular dermatitis). If skin scrapings are negative in these young dogs, mild cases may be left untreated while severe cases may require systemic antibiotics.

In bacterial hypersensitivity, the pustules may be erythematous[11]. In severe cases haemorrhagic bullae and focal alopecia may be present[11]. Systemic antibiotic therapy at therapeutic dosages should be continued up to 14 d after all lesions have disappeared. An antihistamine such as trimeprazine tartrate (Vallergan, Maybaker) may assist in controlling the pruritus. Some cases (especially those that have been on glucocorticoids) respond initially, only to relapse on termination of the antibiotic therapy. In these immunoincompetent patients, culture and autogenous vaccine production and administration is often the only alternative to life-long prophylactic use of antibiotics.

Deep pyoderma

Hair follicles are invariably involved in deep pyodermas. The resultant follicular damage may result in rupture of the follicle and perifollicular abscess formation (furunculosis)[12]. Pressure points such as the lateral stifle, lateral elbow, the chin and the dorsum of the nose are frequently affected. Interdigital cellulitis is characterised by bullae, abscesses and fistulas between one or more digits. These

Table 4: **Examples of antimicrobial agents useful in canine pyoderma[5 8 13]**

Group	Generic name	Trade names	Dosage	Administration	Action	Spectrum
penicillins	cloxacillin	Orbenin	10mg kg^{-1} TD	per os	bactericidal	Gram-positive
	amoxycillin and clavulanic acid	Synulox	12,5mg kg^{-1} BD	per os	bactericidal	broad spectrum
macrolides	erythromycin	EMU-V	15 mg kg^{-1} TD	per os	bacteriostatic	Gram-positive
lincosamides	lincomycin	Lincocin	20 mg kg^{-1} BD	per os	bacteriostatic	Gram-positive
	clindamycin	Dalacin-C	10 mg kg^{-1} BD	per os	bacteriostatic	Gram-positive
cephalosporins	cephalexin	Ceporex	15 mg kg^{-1} BD	per os	bactericidal	broad spectrum
aminoglycosides	gentamycin	Genta 20	2 mg kg^{-1} BD	subcutaneously	bactericidal	broad spectrum
	kanamycin	Kanamyn Ka-mycin "100" Kanamycin 10%	7 mg kg^{-1} TD	subcutaneously	bactericidal	broad spectrum
	amikacin	Amikin	5 mg kg^{-1} TD	subcutaneously	bactericidal	broad spectrum
	tobramycin	Nebcin	1 mg kg^{-1} BD	subcutaneously	bactericidal	broad spectrum
chloramphenicol	chloramphenicol	Chloramphenicol	50 mg kg^{-1} TD	per os	bacteriostatic	broad spectrum
potentiated sulphonamides	trimethoprim and sulphadiazine	Tribrissen Sulmethotrim	15 mg kg^{-1} BD	per os	bactericidal	broad spectrum

BD = repeated every 12 h; TD = repeated every 8 h.

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may progress to form deep, draining, painful tracts which cause acute lameness in dogs. Grass-seeds, thorns and other foreign bodies must be examined for under general anaesthesia. Pododemodicosis (*Demodex* of the paws) may occur in the absence of demodicosis lesions elsewhere on the body. Since it is possible that canine generalised demodectic mange is a manifestation of a T-cell immunosuppression due to *Staphylococci*, it is advisable to treat for pyoderma until the demodicosis has been cured.

Multiple perianal fistulas occur in German Shepherd dogs and their crosses[16] and are perianal pyodermas. If an appropriate antibiotic is initiated immediately and maintained at therapeutic dosages for at least 30 d, the condition can often be cured. Advanced and non-responsive cases require surgery, electrosurgery, cryosurgery, or even tail amputation[16].

FELINE PYODERMA

Surface and superficial pyoderma

Flea bite dermatitis frequently results in secondary pyoderma with pruritus, crusting, and even regional lymphadenopathy. Feline folliculitis occurs frequently on the chin[15] and is known as "feline acne". This can be obstinate and resistant to therapy. Immunomodulation may be of assistance in this immunodeficiency syndrome. Draining interdigital fistulas and abscesses may occur even years after declawing[15]. A pustular eruption[14] occurs on the neck of kittens and may spread to involve the head, ventral chest and abdomen.

Deep pyoderma

Abscesses associated with fight wounds appear most frequently on the cheeks and the tail root areas of especially intact male cats[12]. Because of the looseness of the integument in cats, large areas of cellulitis and subcutaneous abscessation can occur. These require drainage and regular flushing with solutions containing antiseptics such as povidone-iodine (Betadine, Adcock-Ingram)[4]. Debridement of necrotic areas can be followed by "tacking" the skin edges together with large mattress sutures, allowing gaps for drainage. Crater-like wounds can heal without residual scarring.

AVIAN PYODERMA

Infectious pododermatitis ("bumble foot")[1] is seen in raptors, waterfowl, and caged birds[9]. The aetiology involves abrasive surfaces, lack of cage hygiene, and sharp objects. *S. aureus* and *Escherichia coli* are frequently isolated. Cage management, curettage, and systemic antibiotic therapy are of assistance[9]. A paediatric oral suspension of chloramphenicol (Chloromycetin palmitate, Parke-Davis) can be administered into the crop through a canula at 50 mg kg^{-1} 3 times daily. Periorbital abscesses are often associated with respiratory infections[9]. Curettage and topical antiseptics are indicated[9]. Abscesses of the uropygial glands of budgerigars and canaries require curettage[1].

SYSTEMIC ANTIMICROBIAL THERAPY

Table 5 lists the characteristics of antimicrobial agents useful in pyoderma. Since coagulase-positive *Staphylococcus* is invariably the pathogen involved, empirical selection of antibiotics is acceptable for initial therapy of uncomplicated pyoderma[5 6 13]. Always select one with known efficacy against this organism.

Table 5: **Characteristics of antimicrobial agents used to treat pyoderma[5 6 7 8 13]**

Group		Characteristics
Penicillins	1.	Bactericidal, but not all are β-lactamase resistant.
	2.	Procaine penicillin G is not sufficiently effective to be of assistance in pyoderma. Neither this nor the broad-spectrum penicillins such as ampicillin and amoxycillin are β-lactamase resistant. The last two may be useful against secondary invaders such as *E. coli* and *Proteus*.
	3.	Oxacillin, cloxacillin, nafcillin, flucloxacillin and methicillin are β-lactamase-resistant and are effective against coagulase-positive *Staphylococci*. Should be given one hour before or after meals. These are expensive.
	4.	Potentiated amoxycillin (amoxycillin and clavulanate) is β-lactamase resistant, bactericidal, and broad spectrum, although relatively expensive.
Macrolides	1.	Erythromycin is bacteriostatic with a narrow spectrum of efficacy against Gram-positive bacteria.
	2.	It is effective against coagulase-positive, β-lactamase-producing *Staphylococci* and relatively inexpensive.
	3.	May cause vomition and should be given with a light meal.
	4.	Must be administered 3 times daily.
	5.	Cross-resistance with lincosamides.
Lincosamides	1.	Bacteriostatic or bactericidal depending on the concentration.
	2.	Cross-resistance with macrolides.
	3.	Has become relatively expensive and may cause diarrhoea.
	4.	Effective against coagulase-positive, β-lactamase-producing *Staphylococci*.
Cephalosporins	1.	Bactericidal, broad spectrum and relatively inexpensive.
	2.	Effective against coagulase-positive β-lactamase-producing *Staphylococci*.
	3.	A useful first choice.
	4.	"Ceporex" is recommended at 15 mg kg^{-1}, but can be used at up to 30 mg kg^{-1} in severe or stubborn cases.
Aminoglycosides	1.	Bactericidal, with a very broad spectrum including coagulase-positive β-lactamase-producing *Staphylococci*.
	2.	Injectable only, nephrotoxic, and most are expensive.
	3.	Gentamycin is relatively inexpensive. It is useful in severe, mixed, or life-threatening infections where the patient is hospitalised.
	4.	For *Pseudomonas*, gentamycin can be used in combination with penicillins.
Chloramphenicol	1.	Bacteriostatic and broad spectrum.
	2.	Inexpensive and may be effective in mixed infections.
	3.	In vivo effectivity does not necessarily follow in vitro sensitivity on culture. This has led to less frequent use.
	4.	Must be used at maximum dose (50 mg kg^{-1} repeated three times daily), if at all.
Potentiated sulphonamides	1.	These combinations of trimethoprim with sulphadiazine or sulphamethoxazole are bactericidal.
	2.	Inexpensive and may be effective in mixed infections.
	3.	In vivo effectivity does not necessarily follow in vitro sensitivity.
	4.	Minimum dose is 15 mg kg^{-1} repeated twice daily; higher (20 mg kg^{-1} repeated twice daily) in severe pyoderma.
	5.	Prolonged use may cause keratoconjunctivitis sicca.

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A variety of bacteria including *Klebsiella, E. coli,* and almost all pathogenic *Staphylococci* on canine skin produce B-lactamases (previously known as penicillins)[8]. Cleavage of the β-lactam ring in certain penicillins and cephalosporins which render these antibiotics ineffective is catalysed by β-lactamases. For successful therapy of pyoderma, a drug with minimal side effects, reasonable cost, likelihood of high efficacy and ease of administration should be selected from Table 4. Clavulanic acid is a substance capable of inhibiting β-lactamases produced by bacteria[8]. The combination of amoxicillin trihydrate and clavulanate potassium (*Synulox*, Beecham) produces enhanced antibacterial activity[8].

In most cases of surface and superficial pyoderma, it is not necessary to select an antibiotic which is bactericidal. Patients that may require a bactericidal drug include the very young, the very old, and the immunoincompetent (many have unfortunately been on long-term immunosuppressant glucocorticoid therapy). Anaerobic infections require the selection of a bactericidal drug since most bacteria within an abscess are in a stationary growth phase[4]. However, most veterinary obligate anaerobic bacteria are inhibited by therapeutic levels of penicillin G[4]. *P. multocida,* a facultative anaerobe present in many animal bite wounds is also susceptible to penicillin[4].

TOPICAL THERAPY

Management of pyodermas may benefit from topical therapy. However, except in surface pyodermas, it is an adjunctive therapy to systemic antibiotics[5]. Medicated soaps such as Gill and povidone-iodine soaks are useful in the removal of crusts, exudates and purulent material.

Benzoyl peroxide is a follicular flushing agent with antibacterial properties[5]. It has been advocated in skin fold pyodermas[5]. The benzoyl peroxide molecule is unstable and the purchase of generic products as well as bulk buying and repackaging has been discouraged[7]. The only shampoos that are recommended are Oxydex (DVM) and Pyoben (Allerderm)[7]. Benzoyl peroxide-containing shampoos further aggravate pre-existing dry skin and should be used with caution.

Topical antibacterial creams, gels and ointments have limited application in pyodermas, since greasiness, matting of hair and stimulation of the lick-itch-lick cycle are frequent sequelae[7].

IMMUNOMODULATION THERAPY

Certain dogs appear to be susceptible to recurrent intractable pyoderma. The white Bull Terrier is well represented and this may indicate a familial immunodeficiency[7]. Careful confirmation of the diagnosis should include examination for precipitating factors including internal disease. Furthermore, individual patients that have been on long-term glucocorticoid "therapy" may suffer *iatrogenic* immunosuppression. Pyoderma in these patients either responds poorly to antimicrobial agents, or responds only to relapse on cessation of therapy. In view of the guarded prognosis, alternatives to lifelong antibiotic therapy may be offered. An immunomodulator such as 2,5% levamisole hydrochloride (Tramisol, Coopers Animal Health) can be administered to dogs at 2,2 mg kg^{-1} orally 3 times weekly[7] in an attempt to stimulate the immunity.

Autogenous bacterin administration is an alternative method of immunostimulation. Only certain laboratories can produce the bacterins. For a bacterin to be effective, the culture should yield a pure growth of coagulase-positive *Staphylococci.* Bacterins are administered subcutaneously in increasing increments according to a pre-arranged schedule. Subcutaneous swellings at the injection site, with possibly fever and malaise, indicate reactions to the antigen. The programme must then be interrupted until these side-effects have disappeared. The owner must be counselled as to the cost, possible side-effects and the duration of the schedule. Simultaneous antibiotic coverage may be necessary during the immunomodulation therapy and hence the efficacy has been difficult to evaluate[7]. However, immunomodulation can be offered as a possible alternative to the less desirable life-long prophylactic administration of antibiotics.

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INTERNATIONAL EMBRYO MOVEMENT

W C D HARE and SARAH M SEIDEL

Published by the International Embryo Transfer Society Printed by Lowe-Martin Company Inc. Ottawa, Canada, January 1988 pp xii and 198, numerous illustrations and tables, price not supplied.

This volume contains the proceedings of a symposium sponsored by the International Embryo Transfer Society in conjunction with XXXIII World Veterinary Congress in Montreal, Canada in 1987. Most chapters are in English with the few in French having English summaries.

The first paper, by R J Mapletoft provides an update on the technology of embryo transfer. This article is probably the most comprehensive and up-to-date review of embryo transfer applications in domestic animals available in current literature. The following articles set out to depict the current status advantages and potential applications of embryo transfer in relation to animal production and species preservation in Africa, Central and South America, North America, Asia, Australia, East Europe and the USSR and North, South and West Europe respectively. The paper on Africa by P. Chicoteau (in French) is marred by the scant reference to embryo transfer activity in South and southern Africa.

The next section deals with the interaction between embryo and pathogens and the potential for disease transmission or control by embryo transfer. Pathogens of cattle, sheep and goats, pigs and horses are discussed in separate papers. The information in this section is comprehensive and up to date. The rest of the volume deals with determining scientifically based regulations for the international movement of embryos, and techniques for optimising the health status of embryos for movement. Experiences of various countries in this regard are also discussed.

This book provides concise, current and valuable information for all concerned in the embryo transfer industry, and will be especially valuable to those concerned with legislation for international embryo movement. For this readership it is highly recommended.

R O GILBERT

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