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TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

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CONTENTS/INHOUD

SOME USES OF ISAVERIN® IN EQUINE COLIC IN SOUTH AFRICA

SUMMARY

Isaverin is often the drug of choice as a spasmolytic and analgesic in equine colics as it causes minimum fall in blood pressure and tolerance is excellent. Because of the spasmolytic and analgesic effects together it has a prolonged action of up to 4 to 5 hours. Some diagnostic points to be taken into account in selection of drug therapy are given and suggested treatment regimes are elaborated.

DIAGNOSTIC POINTS

In addition to the violence of the spasms and the presence or absence of flatulence on abdominal asculation, the selection of drug therapy must be influenced by an accurate evaluation of the severity of the case and state of shock resulting from the pain.

This is indicated by pulse rate and pressure; conjuctival congestion; capillary refilling time of the buccal mucosa as well as any evidence of cyanosis; respiratory rate, distress and exertion; coldness of the ears and limb extremities. More elaborate examinations such as a paracentesis are necessary in unresponsive, violent, or cyanotic cases. The value of the venous haematocrit cannot be overemphasised as the shock indicator. To avoid rectal trauma, great care and lubrication must be emphasised with rectal examination when violent pain is evident.

TREATMENT

1. Kill pain – this not only reduces shock but also violence, which can result in gastric rupture or other trauma. Administer Isaverin.
2. If no flatulence is present then relax the bowel. Hypermotility can result in an intussusception or a volvulus. Isaverin will accommodate these demands at a rate of 15 ml i/v and may be repeated after 4 hours by intra-muscular administration. This will prolong the spasmolytic effect.
3. Drench with lubricants and antiferments to prevent further gas formation and reduce stasis resulting from the spasmolytic drugs. Don't rush to force defaecation with parasympathomimetics.
4. Keep warm (rug up and bandage limbs) to reduce shock. Allow the relaxed horse to rest if he is not attemtping to roll; it is unnecessary to exhaust him by continuous walking.
5. Administer fluid therapy as indicated by the haematocrit. Remember a haemaconcentration is usually associated with an acidosis and therefore the use of sodium bicarbonate i/v is indicated. It may be necessary to administer up to 40ℓ of hypotonic saline or some potassium. The aid of flame photometry for Na and K is emphasised.

CLINICAL FIELD TIPS

1. The earliest clinical observation of cynosis is seen in the buccal mucosa at the points of the gums adjacent to the incisor tooth junction. This can be seen up to two hours before general cynosis is observed as indicated by the buccal capillary refilling time and buccal mucosa.
2. The field haematocrit can be simply conducted. Aspirate the EDTA or Heparinised blood specimen into a disposable syringe barrel. The needle is then pegged vertically into the rubber stopper of the specimen tube in such a way as to seal the end of the needle and keep the barrel upright. The plunger is withdrawn and the sedimented haematocrit observed at thirty minutes. (Bear in mind the state of health and feeding of the patient as well as if warm blooded – or cold blooded to assist your interpretation of the test).
3. Liquid paraffin (Odina 33) is a safe lubricant and half to one gallon is a reasonable dose for spasmodic or impaction colic. The latter case can receive one gallon a day for as much as 5 consecutive days. Impactions have survived 13 days without defaecation and up to 9 gallons of liquid paraffin have been administered to one patient over 21 days without incident. Odina 16 is too thin to lubricate well although it flows readily in a regular stomach tube and funnel. A stomach pump is indicated to administer Odina 33 as this is less viscous.

ACKNOWLEDGEMENTS

The above information is based on Dr. Maurice Azzie's paper on Equine Colic presented at the Annual Congress of the Oranjevaal group of the S.A.V.A. at Potchefstroom on the 15th of May 1976.

CORRIGENDA

1. "Opening Address – Openingsrede:
71ste Algemene Jaarvergadering ens., Bloemfontein, Sept-Okt 1977; 71ste Annual General Meeting etc., Bloemfontein, Sept-Oct 1977" Vol 47 No 4 p 243: Should read **"1976"**
2. "Van die Redaksie: Sonde met die Honde": Jaargang 47 Nr 4 p 239: Para. 5, reël 5: "122,7%" moet lees **"22,7%"**
3. "Fluid therapy in canine babesiosis – C. Button: Vol 47 No 4 p 286: (c) Calories, line 11: "170g of glucose" should read **"175g".** Add as foot note to p 287: Enteren-Vetlab.

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EDITORIAL

STATE VETERINARY GROUP

With the establishment of the Association in 1905 which later became the South African Veterinary Medical Association (presently S A Veterinary Association) the majority of the members were State employees. This situation persisted approximately until the Second World War when a gradual but definite change took place. More opportunities for private practice and employment in the private sector arose which prompted veterinarians to enter the field of farm and small animal practice. The tendency continued to such a degree that the proportion of veterinarians in the profession who are State employed has dwindled to approximately 18%.

Concomitantly the nature and activities of the Association have also changed and expanded. More branches, based on geographical distribution, have been established while groups of members with similar or common interests and specialities formed specific Groups. State Veterinarians have played an important and prominent role in both instances particularly in the organization of branch conferences and continued education courses. Also, at a more central level, State Veterinarians have continued to play a prominent role by serving as presidents of the Association as well as members of Council and its committees.

Nevertheless, and regrettably, some State Veterinarians felt strongly that the nature of the SAVA had changed to such an extent that it no longer adequately served their interests. For this reason some members even went as far as withdrawing their membership, whilst others simply became apathetic towards the SAVA.

In 1975 Professor J H R Bisschop took the initiative and his endeavours led to the establishment of a State Veterinarians Group within the framework of the SAVA at the Association's Annual General Meeting in Durban. Dr C M Cameron was elected as the first chairman of the committee which included Dr T W Naudé (Vice-chairman) and Dr P P Bosman (Secretary).

The objectives of the group are to promote the professional and academic interests of all State Veterinarians. In order to achieve these goals and to place their activities on a sound scientific basis, it arranged for the presentation of a series of papers at Bloemfontein at the Association's A G M in Sept 1976.

The topics are current and also of general interest. The colleagues who compiled and read their papers did so with dedication and enthusiasm and the material is of importance to all veterinarians dealing with farm animals. The contents of the papers are a credit to our profession.

We trust that this effort will be the start of a series of presentations which will lead to a regular exchange of information and ideas. Such endeavour will undoubtedly improve the services which State Veterinarians in the field and at the Onderstepoort Research Institute can offer to the animal industry in South Africa.

The State Veterinarians Group is to be congratulated with this effort and the papers are proudly presented in this issue of the Journal.

VAN DIE REDAKSIE

DIE STAATSVEEARTSGROEP

Met die totstandkoming in 1905 van die Vereniging wat later die Suid-Afrikaanse Veterinêr-Mediese Vereniging (tans S A Veterinêre Vereniging) geword het, was die oorgrootte meerderheid van die lede in diens van die Staat. Hierdie toestand het voortgeduur tot ongeveer die tydperk van die Tweede Wêreldoorlog, toe daar stadig maar seker 'n verandering ingetree het. Geleidelik het daar meer en meer geleenthede vir veeartse in die privaatsektor van die huisdier- en veebedryf ontstaan. Dit het in so 'n mate toegeneem dat die persentasie van lede van die beroep wat in Staatsdiens was, gaandeweg gekrimp het en tans op ± 18% staan.

Intussen het die aard en aktiwiteite van die Vereniging ook verander en toegeneem. Meer en meer takke is op geografiese grondslag tot stand gebring. Daarby het Groepe van lede met gemeenskaplike spesialiteitsbelange tot stand gekom. In albei liggame het Staatsveeartse hulle belangrike rol gespeel o a met die reel en hou van takkongresse en voortgesette opleidingskursusse. Op sentrale vlak dra die staatsverbonde veearts steeds meer as sy deel by deur te dien as presidente van die Vereniging en as lid van die Raad en sy komitees.

Desnieteenstaande was daar by sommige Staatsveeartse ongelukkiglik die sterk gevoel dat die SAVV tot so 'n mate verander het dat lidmaatskap vir hulle nie juis enige besondere voordeel ingehou het nie. Sommige van ons lede het ook a g v hierdie siening hulle lidmaatskap laat verval.

Oud-professor J H R Bisschop het in 1975 die voortou geneem en sy aanvoorwerk het gelei tot die stigting van 'n Staatsveeartsgroep, binne die raamwerk van die S A V V tydens die A J V in Durban. As eerste voorsitter is Dr C M Cameron verkies, en sy komitee (bestuur) bestaan uit Dr T W Naudé (Ondervoorsitter) en Dr P P Bosman (Sekretaris).

Die doelstellings van die Groep is om die professionele en akademiese belange van alle Staatsveeartse te dien. Om hierdie doelstellings te bereik en om die bedrywighede van die Groep op 'n gesonde wetenskaplike basis te plaas, is 'n reeks voordragte in 'n spesiale sessie tydens die A J V te Bloemfontein in Sept 1976 aangebied. Die onderwerpe is tydig en van onmiddelike belang, en het wye byval gevind. Die kollegas het hulle voordragte met ywer voorberei en met geesdrif aangebied. Die materiaal is van belang vir alle veeartse wat met plaasdiere doenig is en strek die professie tot eer.

Daar word vertrou dat hierdie maar die eerste van 'n reeks aanbiedings is wat sal lei tot gereelde uitruiling van inligting en gedagtes. Dit sal ongetwyfeld lei tot verdere verbetering van die dienste gelewer deur Staatsveeartse in die veld en by die Onderstepoort Navorsingsinstituut ter bevordering van die veebedryf in Suid-Afrika.

Die Staatsveeartsgroep word met die poging gelukgewens. Die referate word met trots in hierdie uitgawe van die tydskrif aangebied.

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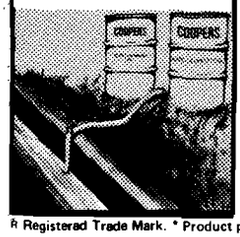

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ADDRESS **VOORDRAG**

HAEMORRHAGIC FEVERS OF AFRICA
AN ACCOUNT OF TWO RECENT OUTBREAKS

J.H.S. GEAR*

My address is concerned with some zoonoses which mainly involve animals but are transmissible to Man. We share a common interest in these infections, their natural history, their reservoir hosts, vectors and transmitters and in their clinical manifestations.

The term "haemorrhagic fever" refers to a syndrome characterized by fever, bleeding from the mucous membranes manifesting as epistaxis, haematemesis and melaena and often purpura. There are many causes of this condition (Table 1).

Our interest in these infections was stimulated by the occurrence in the autumn of 1975 of several cases of haemorrhagic fever each of which posed diagnostic problems.

Marburg Virus Disease

The story of the first outbreak began with a journey undertaken by two Australian students, M.H. and D.O., on a holiday visit to South Africa. On 1st February 1975, they left Johannesburg and hitchhiked to Beit Bridge, Salisbury, Kariba Dam, Victoria Falls, Gwaai, Bulawayo, Zimbabwe and back to Beit Bridge, arriving on 8 February. Possibly of significance was that while waiting for a lift at the Wankie turnoff from the main Bulawayo road, M.H. was bitten or stung on his right flank. He thought a sting had been left in and complained about the irritation for the rest of the day, but no sting could be detected. At Gwaai they slept out in the open on the grass, over which zebra grazed, close to the hotel. At Zimbabwe they were taken to a private game farm for a braaivleis and came into contact with two tame vervet monkeys and the fox terrier which had foster nursed them and at the braai M.H. handled raw eland meat.

From Beit Bridge they returned to Johannesburg and the following day, 10 February, set off for the Natal South Coast arriving at Marburg, Natal§ the same day. On Wednesday M.H. felt ill with chills and muscle and joint pains and headache. On Friday while M.H. was still feeling very ill, they hitchhiked back to Johannesburg. He was admitted to hospital on Saturday 15 February; his condition was provisionally diagnosed as malaria for which he was treated with chloroquin but showed no response.

When this patient was seen on the evening of Monday 18 February, it was clear that he had developed a haemorrhagic state which seemed to be related to a virulent infection. The causes of haemorrhagic fever were listed (Table 1) and amongst them the following were specially considered: Of the arbovirus infections, chikungunya fever was suggested by his flushed face, injected conjunctivae and mottled erythematous rash. The first recognized outbreak in South Africa occurred in 1957 at Easter time affecting visitors to the Kruger National Park and the population living on its borders. In the autumn of 1975 at Easter time, chikungunya fever was again prevalent in the lowveld of the north eastern Transvaal, including the Limpopo valley and in Rhodesia, areas traversed by the patient shortly before his illness.

Of the Group B arboviruses, the possibility of yellow fever was suggested by his high fever with bradycardia and signs of hepatitis with bleeding and vomiting of altered blood, suggestive of coffee grounds and reminiscent of the black vomit typical of this infection. Although yellow fever has not yet been identified south of the Zambesi valley, an epidemic had occurred in 1973 in the vicinity of Luanda in the neighbouring territory of Angola and its spread in the meantime to the south east into Rhodesia could not be excluded. Dengue fever was considered because it has been incriminated as the main cause of haemorrhagic fever in the Far East. However, epidemics of this infection have not occurred in Southern Africa since 1928-1929. Rift Valley fever has caused several extensive epizootics in which hundreds of thousands of lambs have died. These epizootics were associated with many cases involving human beings, affecting in particular veterinary surgeons, farmers and farm labourers who had handled sheep carcasses. At that time, however, it was not known to have caused any deaths in Man.

The next group of virus infections to be considered in the differential diagnosis were those associated with rodents. These include Korean haemorrhagic fever which had affected American soldiers in particular serving in the front line in the Korean war. The aetiology of this infection still has to be fully elucidated. Other infections of this group were Argentinian haemorrhagic fever caused by Junin virus and Bolivian haemorrhagic fever caused by Machupo virus and Lassa fever of West Africa.

Lassa fever was first recognized in 1969, when a nurse Laura Wine became ill in a mission hospital in Lassa in north eastern Nigeria. She was flown for treatment to the hospital in Jos where she died. The sister who nursed her contracted the infection and died and then the third nursing sister Lily Pinneo who attended her in her turn became ill but was flown to the United States. She was admitted to the Intensive Care Unit in the Presbyterian Hospital in New York and after a severe illness recovered. Later two laboratory workers, Dr Jordi Casals and Mr Roman of the Arbovirus Research Unit, in the Department of Epidemiology and Public Health at Yale University School of Medicine, also contracted the infection and

*National Institute for Virology, State Health Department, Johannesburg.

Address to the O.F.S. and North Cape Branch, S.A.V.A., 1975.

§The fact that the patient visited Marburg, Natal whilst affected with Marburg virus is a curious coincidence.—Ed.

Mr Roman died. Dr Casals recovered after receiving transfusions of plasma taken from Sister Lily Pinneo in her convalescence. The virus was isolated and characterized by Dr Casals and his associates and found to be an RNA virus related morphologically and serologically to the virus causing lymphocytic choriomeningitis and to Junin and Machupo viruses causing Argentinian and Bolivian haemorrhagic fevers respectively. These viruses have been grouped together and named the Arenaviruses because of the distinct granules reminiscent of sand seen in the elementary bodies on electron microscopy. Since this original episode there have been several further outbreaks in West Africa. The clinical picture has been characterized in severe cases by fever, severe sore throat and signs of hepatitis, often associated with purpura and bleeding from the mucous membranes and about 50% of the patients admitted to hospital have died.

Lassa fever was seriously considered in this patient, because of his complaint of sore throat, muscle pain, especially in the back and calf muscles, and the finding of lymphadenitis, hepatitis and haemorrhagic diathesis. He also mentioned that he had camped near the Gwaai River. At the time there had been a population explosion in that area, of *Mastomys natalensis,* the multimammate mouse which had proved to be the reservoir of Lassa virus in West Africa.

Finally the possibility that the infection might be caused by Marburg virus was considered. Marburg virus disease was first recognized in 1967 in Marburg, Germany and then in Yugoslavia, where 26 laboratory workers after handling vervet monkeys' tissues and blood, developed an acute febrile illness and seven of them died manifesting signs of severe liver disorder and bleeding from the mucous membranes and an erythematous maculopapular rash. Five of their medical and nursing attendants in turn developed the infection, but all recovered. The virus was isolated in England, Germany and in South Africa by Dr Malherbe and Miss Strickland-Cholmley in the Laboratories of the Poliomyelitis Research Foundation. It was found to be different from all previously known viruses. The source of the infection was traced to vervet monkeys imported from Uganda. When questioned directly M.H. and his companion D.O. said that on three occasions, at Victoria Falls, Zimbabwe and Margate, they had been in the vicinity of these animals, but had not come into close contact with them or with baboons on their journey.

A number of bacterial infections are associated with haemorrhagic rashes and a haemorrhagic state. The best known is meningococcal septicaemia, but cases of staphylococcal and streptococcal septicaemia are often also associated with a purpuric rash. Of particular concern was the possibility of septicaemic plague, because as already noted, the patient had camped and slept on the grass in an area where an outbreak of plague was occurring at the time.

Cases of malaria and trypanosomiasis with heavy parasitaemia and especially patients with *Trypanosoma rhodesiense* infections are liable to develop signs of disseminated intravascular coagulopathy with marked thrombocytopenia and bleeding from the mucous membranes.

Neither malaria parasites nor trypanosomes were detected in the examination of repeated blood films. Blood cultures taken soon after admission to hospital remained sterile, thus excluding meningococcal, staphylococcal and streptococcal septicaemia and also septicaemic plague. It seemed more likely that the patient had a virulent virus infection involving the liver, kidneys and lymphoid tissue, and a tentative diagnosis of hepatitis associated with lymphadenitis and the development of a haemorrhagic state was made. The identity of the virus was obviously an important question.

The patient died on Tuesday evening, 18 February. At post-mortem examination it was ascertained that the immediate cause of death was profuse gastrointestinal haemorrhage and bleeding into the lungs associated with diffuse hepatitis. The spleen and lymph nodes were slightly enlarged. Bacteriological studies, particularly for evidence of plague gave negative results.

On microscopic examination of sections of the liver it was noted that there was a patchy but extensive degeneration of the parenchymal cells, most marked in the midzones of the liver lobules but also involving the centrilobular and subcapsular areas. The affected cells showed marked eosinophilia of the cytoplasm with amorphous eosinophilic masses resembling Councilman bodies. The kidneys showed marked tubular degeneration and some fibrin deposition in the glomeruli. A feature of the spleen and lymph glands was a depletion of lymphocytes, many of which showed lympholysis, pyknosis and fragmentation of the nuclei and many round or oval bodies were seen.

These findings suggested that the patient had died of one of four virus infections known to occur in Tropical Africa namely yellow fever, Rift Valley fever, Lassa fever and Marburg virus fever.

The pathological changes noted in the liver, spleen and lymph nodes were more like those of Lassa fever or Marburg virus disease than those of yellow fever or Rift Valley fever, an opinion confirmed by Professor J. Davis of Albany, New York, at the time visiting the Medical School of the University of the Witwatersrand. As both of these are known to be contagious and dangerous, it was recommended that until and unless the patient's illness was proved to be due to some other cause, the medical and nursing staff should be isolated if a second case occurred.

D.O. was admitted to the Fever Hospital on Saturday with symptoms suggestive of the same disease. One member of the nursing staff, M.C., who had nursed M.H. and then comforted D.O. and then nursed her, took ill the following week. Both D.O. and M.C., after an acute illness characterized by severe headache, muscle pains and an erythematous maculopapular rash and high fever lasting about one week, recovered.

The first clue to the identity of the virus was obtained on Monday 3 and Tuesday 4 March, when Dr Isobel Spence, electron microscopist in the Laboratories of the Poliomyelitis Research Foundation, found arrays of bodies resembling virus particles in electron microscope pictures of sections of the liver and spleen. These structures resembled Marburg virus, but she was hesitant to identify them as such at first. The same evening Drs Dowdle, Murphy and Hufakker of the Center for Disease Control, (CDC), Atlanta, U.S.A., telephoned to say that structures identical to those of Marburg virus had been seen in "Vero" cells inoculated with suspension of the liver specimen from M.H.

In the Laboratories of the Poliomyelitis Research Foundation, the coverslip preparation inoculated on 20 February with suspensions from liver, spleen, kidney and brain when fixed and stained on 6 March with haematoxylin and eosin, showed inclusions resembling

those of Marburg virus infection. A fluorescent antibody test revealed the development of antibodies to M.H. virus in the blood of the two patients who subsequently became ill, the early acute phase serum giving a negative result and the convalescent serum a positive result.

The virus was finally identified by Dr Herta Wulff in the Maximum Security Laboratory of the CDC when she tested the M.H. virus in immunofluorescent antibody tests against the sera collected at intervals from the onset of illness into convalescence from the two patients D.O. and M.C. and against the virus isolated from a patient in the original Marburg outbreak. The titres of reaction given by the sera against both viruses were similar, thus the M.H. virus was shown to be immunologically identical to the virus isolated from the original Marburg outbreak.

Intensive studies have been carried out to find the source of the infection. In journeys retracing the route taken by the two Australian students, numerous bloods were collected from their human contacts and from various animals including the two vervet monkeys and their foster mother the fox terrier at Zimbabwe game farm and from baboons at the Victoria Falls. The sera have been tested in immunofluorescent tests for antibody against Marburg virus, so far with negative results. The source of the infection has not yet been traced, but it seems almost certain that it will be found to be a zoonosis. The problem remains a challenge to both veterinary and medical scientists.

Rift Valley Fever

At the same time as this episode a much more extensive and more serious outbreak of disease was occurring in South Africa involving most of the Transvaal, Orange Free State, and most of the Cape Province. This was an epizootic of Rift Valley fever in which thousands of lambs died, most pregnant ewes and cows aborted and many died and there were many cases of infection in veterinary surgeons, farmers and farm labourers. A team from the State Health Department and the S.A. Institute for Medical Research, went on a journey from Johannesburg to Harrismith, to Bethlehem, Tweespruit, Bloemfontein, Colesberg, Carnarvon and Calvinia returning via Kimberley to Johannesburg and then to Klerksdorp to investigate the epidemic of illness in human beings.

The previous history of Rift Valley fever in South Africa has been published*.

After an apparent absence of 13 years Rift Valley fever reappeared in domestic animals in Southern Africa in 1969. There was a widespread epizootic in Rhodesia and more limited outbreaks in South Africa and Mozambique. In the course of studies of these outbreaks, Rift Valley fever virus was isolated from the following species of mosquito: *Aedes lineato pennis, Anopheles coustani, Culex theileri, Aedes dentatus* and *Eretmopodites quinquevittatus.*

Another extensive epizootic, the most extensive so far experienced in South Africa, occurred in the summer and autumn of 1974/75. The first outbreak was identified as affecting sheep and their human contacts on farms near the Kalahari Gemsbok National Park. The infection was then recognized successively in the north west Cape Province, the western Orange Free State, the south western Transvaal, spreading to involve the whole of the Transvaal, Orange Free State and almost the whole of the Cape Province to the vicinity of Cape Town and Port Elizabeth. Many thousands of newly born lambs died, most pregnant ewes aborted and many died and many cows aborted, but the mortality amongst cattle was not as high as amongst sheep. At the same time there were many cases of human infection involving as in previous epizootics, farmers, farm labourers and veterinary surgeons, nearly all of whom acquired their infection whilst cutting open the carcasses and handling the organs of animals which had died of Rift Valley fever. A few patients may have acquired their infections whilst handling live sheep or from mosquito bites or from drinking infected milk, for they gave no history of direct contact with the tissues of dead animals. Most of them experienced a typical attack. After an incubation period of about 3-7 days, usually four, the onset of the illness was sudden with pains in the muscles and joints, painful movements of the eyes, headache, often severe, and fever. Many patients had a diphasic illness with a recrudescence of their symptoms during the second wave of fever, often associated with nausea and vomiting. The acute febrile stage lasted about one week and was followed in most patients by an uninterrupted but somewhat prolonged convalescence. Several developed complications, however, the most frequent being defective vision which was first noticed towards the end of the acute phase of their illness or early in convalescence. Reports of patients developing this complication were received from Pretoria, Johannesburg, Klerksdorp, Bethlehem, Bloemfontein, Colesberg, Calvinia, Beaufort West and Cape Town. Twenty patients were seen in Klerksdorp in consultation with the ophthalmologists of that town. Each patient gave a history of having had an acute febrile illness followed by loss of vision of one eye in most patients and of both eyes in three. On ophthalmological examination a characteristic "cotton wool" exudate often associated with thrombosis and occlusion of the central branches of the arteries, was seen in the macular region of the retina. Most patients gradually recovered their full vision, but in a few the defect may be permanent.

Several patients developed encephalitis associated clinically with intense headache and a pleocytosis in the cerebrospinal fluid. All recovered except one who in addition developed a haemorrhagic state associated with profuse gastro-intestinal haemorrhage.

Seven fatal cases were notified. The infection was contracted by six of the patients whilst handling sheep carcasses and the source in the seventh case was not known.

During the course of the illness each of these patients developed a haemorrhagic state with profuse gastro-intestinal haemorrhage from which they died. Post mortem histological examination revealed that this was associated with a massive necrosis of the parenchymal cells of the liver showing hyaline eosinophilic degeneration of the cytoplasm with eosinophil bodies resembling Councilman bodies and intranuclear eosinophil granules and inclusion bodies. In the Arbovirus Research Unit, virus was recovered in mice inoculated with suspensions of the liver from three cases and identified as the virus of Rift Valley fever by the histological picture of the liver and specifically in complement fixation tests, testing antigen prepared from liver suspensions against known specific Rift Valley fever antiserum. In another four patients virus isolation was not attempted, but the history of contact with sheep carcas-

(*See *Jl S. Afr. vet. Ass.* (1975) **46**(3):221-225. Editor).

ses, the clinical haemorrhagic state and the pathological picture were so similar that there is little doubt that they also died of Rift Valley fever. These findings emphasize that Rift Valley fever should be added to the list of virus infections to be considered in the diagnosis of patients with haemorrhagic fever.

The contraction of Rift Valley fever is a serious hazard faced by veterinary surgeons and laboratory workers in the course of their work. Fortunately a vaccine is available to protect against this infection. It was developed by Colonel Randall and his team in the Graduate School of Medicine in the Walter Reed Research Institute. The vaccine is a formalin-inactivated suspension of Rift Valley fever virus grown in tissue cultures of vervet monkey kidney. Its administration stimulates a good neutralizing antibody response and it has been free from adverse reactions. There have been no overt laboratory infections in vaccinated personnel and it has been reported that the immunity lasts for at least 18 months. This year the State Health Department offered this vaccine to all veterinary surgeons and laboratory workers. Those veterinary surgeons who have not yet had Rift Valley fever, and perhaps this is a minority of those at this meeting, should avail themselves of its protection. In future, of course, it would be wise to give this vaccine to all veterinary students. The production of enough vaccine to immunize the farmers and their labourers at risk, should also be considered.

May I end by emphasizing once again that more valuable than the protective vaccines which are available and which, of course, do not protect against all the infections to which veterinary surgeons are exposed, is the simple precaution of wearing gloves and goggles and protective gowns when doing post-mortem examinations on animals which have died mysterious deaths.

Table 1: HAEMORRHAGIC FEVERS

Virus infections:

Infectious fevers:
black measles, haemorrhagic smallpox

Arbovirus infections:
Group A: chikungunya fever, Sindbus fever
Group B: yellow fever, dengue fever, West Nile fever
***Ungrouped*: Rift Valley fever**

Virus infections associated with rodents:
Far East: Korean haemorrhagic fever
South America: Argentinian haemorrhagic fever, Bolivian haemorrhagic fever
Africa: Lassa fever

Virus infections associated with monkeys:
Marburg virus disease

Rickettsial infections

epidemic typhus fever
Rocky Mountain spotted fever: tick bite fever
Q fever

Bacteria:

meningococcal septicaemia
septicaemic plague

Protozoa:

Malaria: *P. falciparum*
Trypanosomiasis: *T. rhodesiense*

BOOK REVIEW

BOEKRESENSIE

ZOOTIER KRANKHEITEN

H.-G. KLÖS and E.M. LANG
Paul Parey, Berlin – Hamburg 1976 pp XIV 351 Figs 108 Tabs 32 Price not stated

The subtitle specifies: "Diseases of wild animals in zoo, game park, circus and in private ownership." In separate chapters different groups of wild mammals are dealt with as well as birds, reptiles and amphibians, and fishes. Each chapter is subdivided under the following headings: Special information, Immobilization, Parasites, Infectious Diseases and Vaccinations, Organic Diseases and Intoxications, Deficiencies and Nutritional Problems, Artificial Rearing and Diseases of the Young, Surgery and Obstetrics. In a general part the book also deals with the professional image of the zoo veterinarian, veterinary equipment and aids, general veterinary prophylaxis in zoos and state veterinary interests in the zoo, circus and game park. Tables in the text detail such physiological parameters as temperature, gestation period, age etc. The extensive annexures comprise 1725 literature references as well as indices of drugs, animal species, disease agents and a glossary of general terms.

The book is well written and presented and contains a wealth of information. It is, however, impossible to exhaust the subject in the allocated limited space. Thus the reader is often referred for detailed information to the cited literature. This, unfortunately, necessitates having access to a scientific library and could cause considerable delay if and when this book should be consulted in an emergency. Also, in places, the style of writing becomes telegrammatic which makes certain passages difficult to understand even for somebody fluent in German. Even more to be deplored is the tendency of some of the 24 contributors to summarize in high sounding banalities where detailed information would have been more to the point.

These few shortcomings, however, do not detract from the general high standard of the book and of the ease of accessibility of its contents due to its clear composition and its detailed indices. With the continuing expansion of the horizon of our profession a book like this was badly needed, and authors and publishers are to be applauded for their efforts. This book can be recommended wholeheartedly to the veterinarian, able to read German, who is involved in this particular field of work as well as to the general practitioner.

F.W.H.

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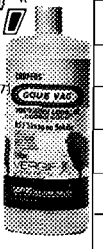

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KEDS	GOLDEN FLEECE D.F.F. SUPADIP D.F.F. DIAZINON D.F.F.	C.I.D.**
TICKS	GOLDEN FLEECE D.F.F. SUPADIP D.F.F. DIAZINON D.F.F.	C.I.D.** or Belly Bath or footbath
BLOWFLY	DIAZINON D.F.F. SUPADIP D.F.F. GOLDEN FLEECE D.F.F.	C.I.D.** (2 month's wool) HAND-SPRAYING (Crutch) JETTING
ITCH MITE	GOLDEN FLEECE D.F.F.	C.I.D.** (off shears) 30 seconds' immersion
*SHEEP SCAB	DIAZINON D.F.F.	2 C.I.D.** (7- 10 days' interval) under Government supervision (off shears)
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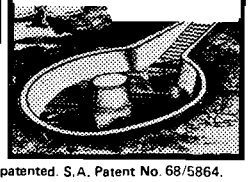

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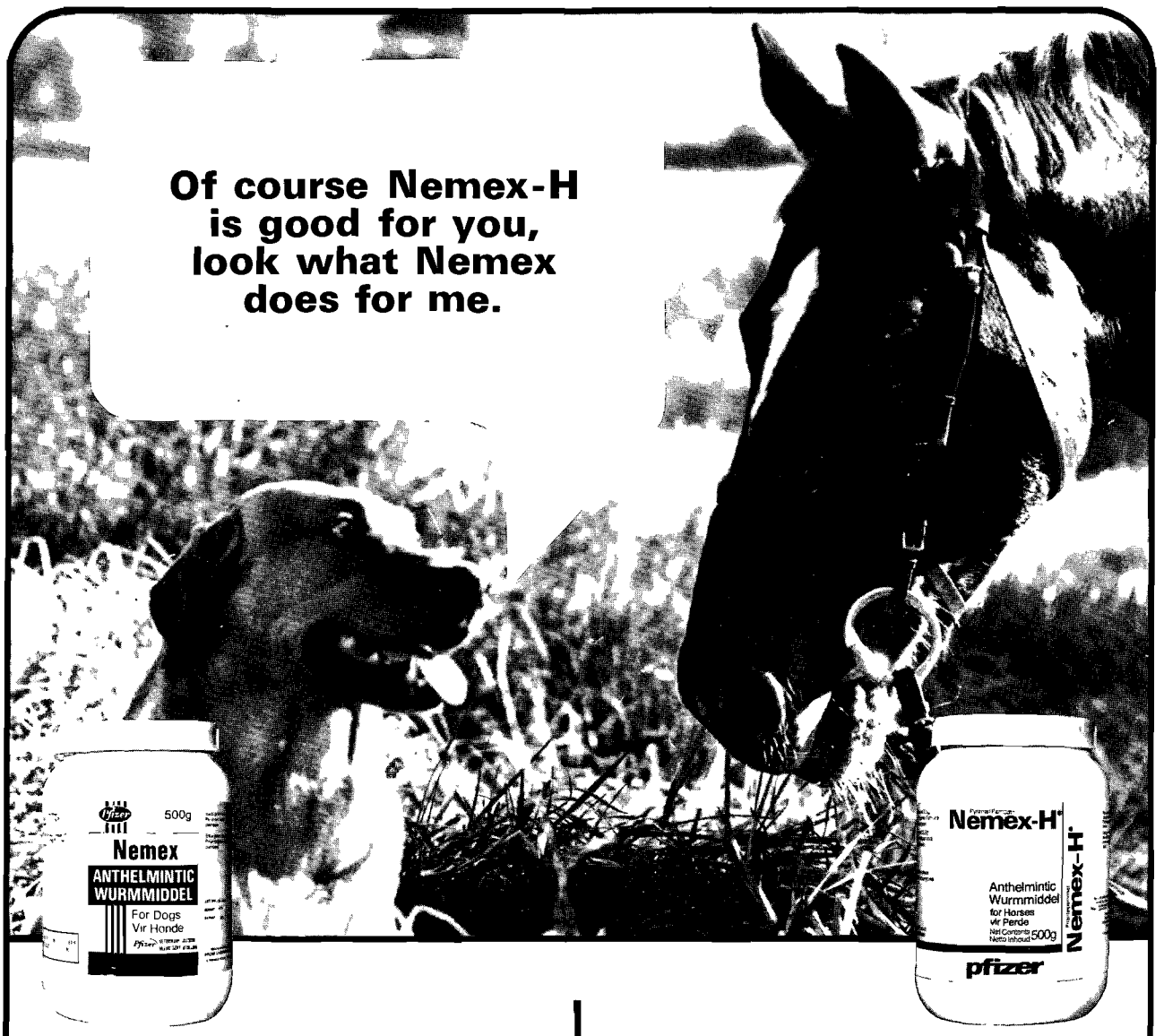

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Jl S. Afr. vet. Ass. 48(1) 11 – 12 (1977)

BOSLUISBESTANDHEID TEEN PLAAGMIDDELS IN DIE R.S.A. – ENKELE WAARNEMINGS

C.J. HOWELL*

ABSTRACT: Howell C.J. **Tick resistance to pesticides in the R.S.A. – some observations.** *Journal South African Veterinary Association* (1977) **48** No. 1, 11 – 12 (Afr) Section Entomology, Vet. Res. Inst., 0110 Onderstepoort, Republic of South Africa.

Observations on the screening for resistance against acaricides in both larvae and adults of tick species commonly found on cattle in South Africa, have shown that widespread resistance of varying degrees occurs in strains of all these species against compounds of the arsenical, organochlorine and organophosphorous groups. With few exceptions the degree of resistance is of a low order and probably indicative of selection at the low acaricide concentrations generally used by stockowners. The distribution of less susceptible and resistant strains is patchy, and fully susceptible strains are found in the same areas.

In Suid-Afrika is die ontwikkeling van bestandheid teen plaagmiddels van besondere belang by bosluise weens die gebruiksfrekwensie van die gifstowwe, die aantal spesies wat beheer moet word en ons totale afhanklikheid van chemikalieë vir bosluisbestryding. Met die doeltreffende aanwending van 'n plaagmiddel moet die lewenssiklus van die bosluisspesies in aggeneem word, veral ten opsigte van die lengte van tyd wat hulle in een of meer parasitiese lewensstadia op die gasheer voorkom. Aangesien ons egter met een-, twee- en driegasheer-bosluissoorte te doen het wat almal in verskillende stadia op die gasheer vir wisselende periodes mag vasheg, is individuele spesiebeheer baie moeilik en is 'n stelsel wat naastenby alle soorte op die dier kan beheer, van groter praktiese waarde. Die tradisionele weeklikse toediening van plaagmiddels gedurende die somermaande is hierop gemik aangesien die meeste parasitiese stadia van meeste bosluis spesies vir ongeveer een week op beeste vasheg voordat hulle volsuig en afval. Doeltreffende beheer vereis dat elke bosluis op die dier met die plaagmiddel kontak maak voordat dit die dier verlaat, en, alhoewel hierdie metode die meerderheid van spesies onder normale omstandighede sal beheer, is daar uitsonderings wat veral in buitengewone nat jare nie beheer sal word nie. Die onvolwasse stadia van bruinoorbosluise is daarvoor bekend dat hulle soms in 3 tot 5 dae kan volsuig en dus blootstelling kan vryspring, terwyl laboratoriumbevindings daarop dui dat bontbosluis larwes ook hierdie neiging mag toon. Waar weeklikse aanwendings van die akarasied dus marginaal is vir die beheer van sommige meergasherige bosluissoorte, sal dit, in teenstelling, bloubosluise, wat hulle totale parasitiese lewensloop van ruim 3 weke op dieselfde dier voltooi, aan oormatige blootstelling van die middel onderhewig maak. In die reël sal lg. dus die eerste spesie wees wat bestandheid ontwikkel as gevolg van hoë seleksiedruk. In die geval van bontpootbosluise daarenteen, heg slegs die volwasse stadia op beeste vas en wel op onbehaarde liggaamsdele waar swak kontak met die plaagmiddel gemaak word. Die seleksiedruk is hier dus laag en die gevolglike ontwikkeling van bestandheid baie stadiger.

Bestandheid teen 'n chemiese plaagmiddel is in Suid-Afrika vir die eerste maal in 1938 vasgestel nadat arseen, wat toe reeds vir meer as 40 jaar in gebruik was, nie meer in staat was om *Boophilus* spesies in dele van die Oos-Kaap te beheer nie. Dit is ook reeds geskiedenis dat hierdie spesies daarna teen elke nuwe groep plaagmiddels wat verskyn het, wisselende grade van bestandheid met verloop van tyd ontwikkel het. In die veebedryf is die een krisis na die ander egter afgeweer deur die toevallige en tydige verskyning van nuwe, anderswerkende chemiese groepe wat die bestande stamme kon beheer. Die vereiste vir die uitskakeling van bestande stamme berus feitlik geheel-en-al op die ontwikkeling van gifstowwe wat op 'n ander wyse doodmaak as dié waarteen seleksie plaasgevind het, en dus ook indirek op die vernuf van die chemiese nywerheid om gedurig nuwe produkte te skep.

Bestandheid het mettertyd ook in meergasherige bosluissoorte ontwikkel. Die graad daarvan hang af van die mate van kontak wat hulle met plaagmiddels gehad het, maar die patroon sal bosluisbeheer praktyke in die toekoms toenemend beïnvloed.

EVALUERING VAN BESTANDHEID

Die enigste betekenisvolle wyse waarop die ontwikkeling, verspreiding en graad van bestandheid in bosluisstamme opgespoor kan word, is deur gereelde en beplande toetsing van bosluise uit alle dele van die land waar bosluisbestryding stelselmatig toegepas word. Die resultate verkry, is om verskillende redes van belang:

1 Dit is noodsaaklik dat die oorsaak van swak bosluisbeheer ondersoek word om gevalle van werklike bestandheid van verkeerde bestuurspraktyke te onderskei. Tydens opnames van dipbakvloeistofkonsentrasies was gevind dat tot soveel as 86% van die totaal teen laer as die aanbevole konsentrasies gebruik was en dat hiervan tot die helfte dipmengsels bevat het wat laer as 50% van die voorgeskrewe konsentrasies was.
2 Die onwilligheid van talle vee-eienaars om bosluisbestryding gedurende die wintermaande vol te hou lei dikwels tot sogenaamde "bevolkingsontploffings" in die vroeë somer en, aangesien hulle met die gewone beheerpraktyke sulke bevolkings nie kan onderdruk nie, word bestandheid voorgehou as die rede vir swak beheer. Aangesien die boere die klein larwes op hulle diere nie kan sien nie en ook nie weet dat die onvolwasse stadia van somer-aktiewe bosluise juis hulle grootste getalle en aktiwiteit gedurende die winter bereik nie, was daar nooit werklik enige bevolkingsontploffing nie, en is die skynbare toename in getalle volwassenes in die somer bloot die gevolg van verkeerde bestuurspraktyke.
3 Plaagmiddels verskil van mekaar in die periode van doeltreffende beskerming en benutting voordat betekenisvolle seleksie vir bestandheid 'n probleem kan word. Sommige bly vir baie jare effektief terwyl ander binne 'n jaar of twee na aanvanklike registrasie minder doeltreffend werk. Dit is dus belangrik dat die bruikbare lewensduurte van elke plaagmiddel opgevolg word in die veld. Bestandheid ontwikkel nie noodwendig in elke stam bosluise wat blootgestel word aan dieselfde chemiese middel na dieselfde periode van gebruik nie, en ook nie teen dieselfde snelheid in die verskillende spesies nie. Slegs gereelde toetsing van die bosluisbevolking kan dus 'n beteke-

Bydrae tot Staatsveeartsgroep-program, Bloemfontein, 1976

*Seksie Entomologie, Navorsingsinstituut vir Veeartsenykunde, 0110 Onderstepoort

nisvolle vertolking van die potensiaal van elke bosluisdoder verseker; iets waarsonder advies aan die vee-eienaar nie veel waarde inhou nie.

Toetsing van veldversamelde bosluisstamme word in die laboratorium met beide larwes en volwassenes van die verskillende spesies uitgevoer aangesien verskillende plaagmiddels verskil in hulle effektiwiteit op die twee lewensstadia. Die toetse berus op 'n vergelyking van die konsentrasies wat benodig word om 'n veldstam en 'n bekende vatbare laboratoriumstam van dieselfde spesie te beheer. Indien die veldstam 'n hoër konsentrasie vereis, word die veelvoud uitgedruk as 'n faktor van bestandheid. Die term "bestandheid" word in hierdie verband ietwat los gebruik omrede dit veral by lae faktore nie onderskei word van lewenstaaiheid (of toleransie) nie. In die geval van bosluise bestaan daar nog onvoldoende inligting om hierdie onderskeid behoorlik af te baken. Dit is veral belangrik by die vertolking van larftoetsing aangesien larwes teen sulke lae konsentrasies gedood kan word met sommige plaagmiddels dat selfs 'n tienvoud faktor van bestandheid nog weinig verband hou met wat in die veld plaasvind. Dit is dus slegs aanduidend van stamme wat nie meer ten volle vatbaar is nie. Die gebruik van volgesuigde volwasse bosluiswyfies vir toetsing is goed vergelykbaar met die aktuele veldtoestand aangesien volwassenes in die reël hoër konsentrasies vereis vir beheer en dus gouer gebruikskonsentrasies in die veld oorlewe. 'n Probleem wat toetsing van volwasse bosluise in die laboratorium baie strem, is die versameling van voldoende getalle bruikbare, onbeskadigde volgesuigde wyfies wat die laboratorium betyds bereik, d.w.s. voordat hulle begin eiers lê. In hierdie opsig het larftoetsing die voordeel van getalle aangesien slegs 'n paar wyfies van 'n spesie duisende larwes produseer wat gebruik kan word om die graad van afwyking aan te dui indien bestandheid nie alreeds daardeur vasgestel kan word nie.

Bestandheidsevaluering word op 'n roetine basis uitgevoer teen sekere middels wat as indikators vir die arseen, chlorinaat en organofosfor groepe dien waarin al die beskikbare plaagmiddels ingesluit word met uitsondering van die nuutste groep, die formamidiene en derivate. Die verdere indeling van die organofosfor groep in Groep 1 en Groep 2 is tentatief en afgelei van die neiging tot onderlinge kruisbestandheid in die Groep 1 middels teenoor die in Groep 2 waar hierdie verskynsel nog nie gevind is nie.

Indien spesifieke klagtes uit die veld ontvang word, en bosluise vir toetsing beskikbaar is, word die betrokke dipbak konsentrasie ondersoek en, indien moontlik, 'n geskiedenis van middels wat gebruik is, verkry. Toetsing van die middel in gebruik word dan uitgevoer en, indien voldoende bosluisgetalle beskikbaar is, word hulle ook aan verdere toetsing met ander middels onderwerp. Tot dusver het bestandheid teen chemiese plaagmiddels slegs in bosluissoorte wat op beeste voed probleme geskep omrede die hoë seleksiedruk veroorsaak deur die blootstellingsfrekwensie. Toetsing word dus meestal uitgevoer op blou-, rooi-, bruin-, bont- en bontpootbosluise.

Opnames vir bestandheid in die verskillende bosluis spesies van beeste is gedurende die afgelope jaar dwarsdeur die streke onderneem waar bosluise gereeld met chemiese plaagmiddels bestry word. Deur die goeie samewerking van die Afdeling Veeartsenydiens wat inspeksiepersoneel vir versameling van bosluise beskikbaar gestel het en die verskaffing van larftoets resultate deur een van die handelsfirmas wat al vir geruime tyd hierdie werk onderneem, kan sekere besliste afleidings gemaak word t.o.v. die invloed van plaagmiddeldruk op die verskillende bosluissoorte uit die gemonsterde gebiede:

1 Bosluisstamme van al die verskillende betrokke spesies wat alreeds wisselende grade van bestandheid ontwikkel het teen die 4 getoetste groepe plaagmiddels, is dwarsdeur die land aangetref.
2 Met enkele uitsonderings waar werklik hoë faktore van bestandheid vasgestel was, is die gemiddelde afwyking relatief laag en moontlik aanduidend van seleksie op 'n lae vlak as gevolg van die gebruik van onder-sterkte dipmengsels.
3 Alhoewel stamme van die verskillende bosluisspesies wat nie meer as ten volle vatbaar beskou kan word nie, dwarsdeur die monstergebiede voorkom, is feitlik net soveel vatbare stamme aangetref uit dieselfde gebiede. Die ontwikkeling van bestandheid met so 'n kollerige verspreiding, waar dit selfs mag wissel van plaas na plaas, word sekerlik grootliks deur die bestuurspraktyke van die verskillende vee-eienaar beïnvloed.
4 Bloubosluise toon 'n hoër persentasie verskuiwing by die ou plaagmiddels wat reeds lank in gebruik is as by nuwere produkte. Hierdie neiging kan ook to 'n mindere mate by die ander bosluissoorte waarge neem word.
5 Die graad van afwyking, selfs massiewe bestandheid in stamme afkomstig van veral die Oos-Kaap-Ciske gebied, was opvallend in larfevaluering met bv. kamfechloor. Faktore van bestandheid van 4 000 to 5 000X is aangeteken, en dit is interessant om te bespiegel oor die rol van chlorinate soos BHC wa vroeër in die gebied gebruik was en dus reeds moontlik geselekteer het vir bestandheid voordat di chemies-verwante kamfechloor in die gebiede i gebruik geneem was. Dit is bekend dat bestandhei teen BHC binne 18 maande aangeteken is na ge bruik.
6 Alhoewel bestandheid ook wydverspreid voorkon teen arseen, die oudste middel wat reeds vir meer a 70 jaar in gebruik is, is dit hoofsaaklik van 'n la graad met enkele hoë faktore in sommige gebiede Dit wil voorkom asof arseen stadig selekteer, en di feit dat dit baie min invloed op bloubosluislarwes he terwyl die volwasse bosluise makliker gedood wor teen die gebruiks dipkonsentrasie, mag hier 'n be langrike rol speel. Ontwikkeling en volsuiging va volwasse bloubosluiswyfies duur ongeveer een wee op die gasheer, maar daarvan is hulle maar vir slegs tot 4 dae vir plaagmiddels vatbaar aangesien hull tydens die nimf-volwasse vervellingsproses en oo weer tydens volsuiging minder vatbaar is. Nalatig heid met dipmengselkonsentrasies maak hierdi middel dus baie minder doeltreffend as in die geva van larf-dodende plaagmiddels.
7 Die wye voorkoms van bosluisstamme wat minde vatbaar is vir plaagmiddels, is 'n duidelike waarsku wing dat seleksie teen *alle middels* gedurig plaasvin en in sommige gebiede reeds probleemstatus berei het. Aangesien daar tans geen alternatief bekend i vir bosluisbeheer nie, is meer noukeurige gebrui van die beskikbare plaagmiddels van besondere be lang, en moet metodes ondersoek word waarmee se leksiedruk verlig kan word. Die grootste knelpunt i onvoldoende kennis van die biologie van die verskil lende bosluissoorte en veral van die meer kwesbar stadia in die lewensloop. Die kennis kan dien as bas vir die beplanning van beheerstelsels waarin weidin en kamp rotasie 'n belangrike rol kan speel.

nisvolle vertolking van die potensiaal van elke bosluisdoder verseker; iets waarsonder advies aan die vee-eienaar nie veel waarde inhou nie.

Toetsing van veldversamelde bosluisstamme word in die laboratorium met beide larwes en volwassenes van die verskillende spesies uitgevoer aangesien verskillende plaagmiddels verskil in hulle effektiwiteit op die twee lewensstadia. Die toetse berus op 'n vergelyking van die konsentrasies wat benodig word om 'n veldstam en 'n bekende vatbare laboratoriumstam van dieselfde spesie te beheer. Indien die veldstam 'n hoër konsentrasie vereis, word die veelvoud uitgedruk as 'n faktor van bestandheid. Die term "bestandheid" word in hierdie verband ietwat los gebruik omrede dit veral by lae faktore nie onderskei word van lewenstaaiheid (of toleransie) nie. In die geval van bosluise bestaan daar nog onvoldoende inligting om hierdie onderskeid behoorlik af te baken. Dit is veral belangrik by die vertolking van larftoetsing aangesien larwes teen sulke lae konsentrasies gedood kan word met sommige plaagmiddels dat selfs 'n tienvoud faktor van bestandheid nog weinig verband hou met wat in die veld plaasvind. Dit is dus slegs aanduidend van stamme wat nie meer ten volle vatbaar is nie. Die gebruik van volgesuigde volwasse bosluiswyfies vir toetsing is goed vergelykbaar met die aktuele veldtoestand aangesien volwassenes in die reël hoër konsentrasies vereis vir beheer en dus gouer gebruikskonsentrasies in die veld oorlewe. 'n Probleem wat toetsing van volwasse bosluise in die laboratorium baie strem, is die versameling van voldoende getalle bruikbare, onbeskadigde volgesuigde wyfies wat die laboratorium betyds bereik, d.w.s. voordat hulle begin eiers lê. In hierdie opsig het larftoetsing die voordeel van getalle aangesien slegs 'n paar wyfies van 'n spesie duisende larwes produseer wat gebruik kan word om die graad van afwyking aan te dui indien bestandheid nie alreeds daardeur vasgestel kan word nie.

Bestandheidsevaluering word op 'n roetine basis uitgevoer teen sekere middels wat as indikators vir die arseen, chlorinaat en organofosfor groepe dien waarin al die beskikbare plaagmiddels ingesluit word met uitsondering van die nuutste groep, die formamidiene en derivate. Die verdere indeling van die organofosfor groep in Groep 1 en Groep 2 is tentatief en afgelei van die neiging tot onderlinge kruisbestandheid in die Groep 1 middels teenoor die in Groep 2 waar hierdie verskynsel nog nie gevind is nie.

Indien spesifieke klagtes uit die veld ontvang word, en bosluise vir toetsing beskikbaar is, word die betrokke dipbak konsentrasie ondersoek en, indien moontlik, 'n geskiedenis van middels wat gebruik is, verkry. Toetsing van die middel in gebruik word dan uitgevoer en, indien voldoende bosluisgetalle beskikbaar is, word hulle ook aan verdere toetsing met ander middels onderwerp. Tot dusver het bestandheid teen chemiese plaagmiddels slegs in bosluissoorte wat op beeste voed probleme geskep omrede die hoë seleksiedruk veroorsaak deur die blootstellingsfrekwensie. Toetsing word dus meestal uitgevoer op blou-, rooi-, bruin-, bont- en bontpootbosluise.

Opnames vir bestandheid in die verskillende bosluis spesies van beeste is gedurende die afgelope jaar dwarsdeur die streke onderneem waar bosluise gereeld met chemiese plaagmiddels bestry word. Deur die goeie samewerking van die Afdeling Veeartsenydiens wat inspeksiepersoneel vir versameling van bosluise beskikbaar gestel het en die verskaffing van larftoets resultate deur een van die handelsfirmas wat al vir geruime tyd hierdie werk onderneem, kan sekere besliste afleidings gemaak word t.o.v. die invloed van plaagmiddeldruk op die verskillende bosluissoorte uit die gemonsterde gebiede:

1 Bosluisstamme van al die verskillende betrokke spesies wat alreeds wisselende grade van bestandheid ontwikkel het teen die 4 getoetste groepe plaagmiddels, is dwarsdeur die land aangetref.
2 Met enkele uitsonderings waar werklik hoë faktore van bestandheid vasgestel was, is die gemiddelde afwyking relatief laag en moontlik aanduidend van seleksie op 'n lae vlak as gevolg van die gebruik van onder-sterkte dipmengsels.
3 Alhoewel stamme van die verskillende bosluisspesies wat nie meer as ten volle vatbaar beskou kan word nie, dwarsdeur die monstergebiede voorkom, is feitlik net soveel vatbare stamme aangetref uit dieselfde gebiede. Die ontwikkeling van bestandheid met so 'n kollerige verspreiding, waar dit selfs mag wissel van plaas na plaas, word sekerlik grootliks deur die bestuurspraktyke van die verskillende vee-eienaars beïnvloed.
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7 Die wye voorkoms van bosluisstamme wat minder vatbaar is vir plaagmiddels, is 'n duidelike waarskuwing dat seleksie teen *alle middels* gedurig plaasvind en in sommige gebiede reeds probleemstatus bereik het. Aangesien daar tans geen alternatief bekend is vir bosluisbeheer nie, is meer noukeurige gebruik van die beskikbare plaagmiddels van besondere belang, en moet metodes ondersoek word waarmee seleksiedruk verlig kan word. Die grootste knelpunt is onvoldoende kennis van die biologie van die verskillende bosluissoorte en veral van die meer kwesbare stadia in die lewensloop. Die kennis kan dien as basis vir die beplanning van beheerstelsels waarin weiding en kamp rotasie 'n belangrike rol kan speel.

NUWERE VETERINÊRE NEUROPATOLOGIESE TOESTANDE IN SUID-AFRIKA

J.G. PIENAAR*

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A brief review is given of some of the more recent findings on lesions of diseases affecting the central nervous system of animals. These include local diseases caused by poisonous plants; *Helichrysum argyrosphaerum, Solanum kwebense, Matricaria nigellaefolia;* infectious diseases e.g. heartwater, cerebral babesiosis and cranial abscesses in goats caused by *Corynebacterium pyogenes.* Reference is also made to some conditions, originally described in overseas countries and which subsequently have been diagnosed in South Africa. Original findings on lesions seen in mycotoxicoses caused by *Fusarium moniliforme* and *Aspergillus clavatus* are included.

Betreklik onlangs nog, was veterinêre neurologie en neuropatologie 'n stiefkind in die veeartsenykundige wetenskap. Baie beperkte en soms baie oppervlakkige aandag is gewoonlik, gedurende opleiding van veeartse aan aspekte soos die anatomie, fisiologie, embriologie, histologie, patologie, ens. van die senustelsel van die verskillende huisdiere gegee.

In teenstelling hiermee neem die bestudering van die senustelsel in al sy verskillende fasette, 'n baie belangrike plek in, in die mediese wetenskap. Daar is reeds 'n fenomenale hoeveelheid kennis oor die menslike en primate senustelsel oor die jare versamel. Die rede hiervoor is voor die handliggend. Neurologiese afwykings is van baie groot belang in die mediese wetenskap omdat die gemeenskap so 'n hoë waarde heg aan die menslike lewe. Eienskappe soos spraak, denke, redenasie ens., wat intiem verweef is in die persoonlikheid van elke mens, kom by diere glad nie in gedrang nie. Episoötiese en ensoötiese infeksiesiektes, waar daar dikwels nie letsels in die senustelsel is nie, is egter by huisdiere, vir ekonomiese redes, natuurlik van die grootste belang.

Die veearts beskik dikwels ook nie oor verfynde hulpmiddels soos analitiese waarnemings op die disfunksie van spraak, gehoor, gesig en sensasie, elektroenkefalografie, arteriografie en ventrikulografie, ens. nie. Hy het wel egter aan die ander hand dié voordeel dat hy geredelik maklik monsters van die senustelsel kan bekom vir ondersoek. Die studie van patologiese veranderinge in die sentrale senustelsel, m.a.w. neuropatologie, is ongetwyfeld dus een van die grootste hulpmiddele in die versameling van inligting oor die senustelsels van die verskillende huisdiere.

Gedurende die afgelope 10 jaar is heelwat nuwe inligting bygevoeg tot die kennis van neuropatologie van huisdiere in hierdie land. Hierdie inligting is bekom deur 'n sistematiese ondersoek van die sentrale senustelsel. Hiervoor is die hele brein en rugmurg, of die grootste deel daarvan nodig, behoorlik gefikseer in 'n groot volume fikseermiddel. Sommige van hierdie toestande is siektes wat reeds bekend was in die buiteland terwyl die meeste egter lokale toestande is, wat vir die eerste keer beskrywe is. Van die belangrikste is die volgende:

PLANTVERGIFTIGINGS EN MIKOTOKSIKOSES

Sewejaartjie – vergiftiging[4] *(Helichrysum agyrosphaerum)*

Die belangrikste letsel hier is 'n *status spongiosum* van die witstof met 'n predileksie vir die areas direk om die ventrikels, (Fig. 1) die optiese bane, optiese chiasma, piramidale bane en *brachium pontis.* Vergroting van die optiese fassikuli veroorsaak malasie, papiledeem en letsels in die retina. In natuurlike uitbreke is amourose en parese die hoof kliniese tekens. Kom voor in Suidwes-Afrika en moontlik ander droëre dele van Suid-Afrika.

Fig. 1. *Helichrysum* vergiftiging. Swelling van periventrikulêre areas en gevolglike knopperige uitbulting van die *corpus callosum.*

Maldronksiekte[20] *(Solanum kwebense)*

Veroorsaak 'n vakuolisasie van groter neurone, veral Purkinje selle van serebellum maar ook tot 'n mindere

Aangebied in Staatsveeartsgroep-program, Bloemfontein 1976.

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mate van ander neurone in middebrein, breinstam en rugmurg. Purkinje selle ondergaan uiteindelik nekrose en verdwyn en gevolglik ontstaan atrofie van die skors van die serebellum wat dan ook die enigste nadoodse letsel is (Fig. 2). Vernaamste kliniese tekens is 'n tydelike verlies van ewewig, en epilepsie-agtige aanvalle, so erg dat aangetaste diere neerslaan. Hierdie aanvalle kan deur verskillende stumuli aangebring word. Hierdie vergiftiging is tot die bosveldstreke van die Noordwes-Transvaal beperk.

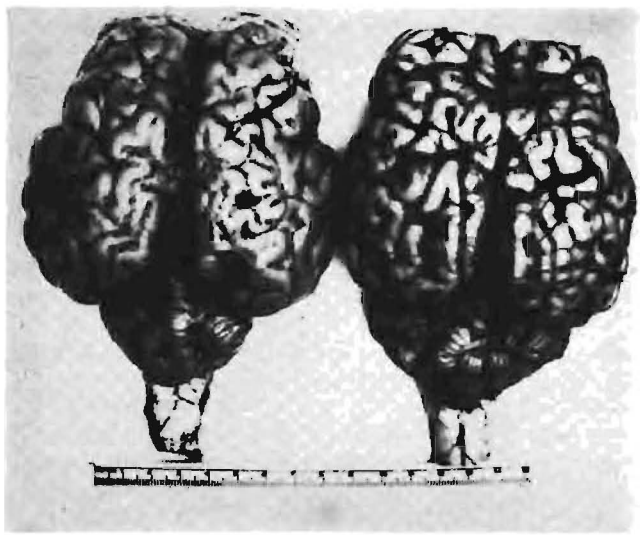

Fig. 2. Maldronksiekte. Atrofie van cerebellum (links). Normale brein aan die regterkant.

Stootsiekte[21] *(Matricaria nigellaefolia)*

Die etiologie, simptome en ander aspekte van dié vergiftiging is reeds in 'n pragtige stuk werk deur Andrews[1] so vroeg as 1923 gepubliseer. Die simptome kon egter nooit verklaar word nie omdat geen patologiese ondersoeke van die brein ooit gedoen was nie. Met onlangse eksperimentele werk te Onderstepoort is gevind dat daar mikrokavitasie van sekere kerne in die middebrein, breinstam en serebellum voorkom, asook 'n uitgesproke perivaskulêre gliose dwarsdeur die sentrale senustelsel. Hierdie letsels kan dus nou gebruik word om hierdie siekte met sekerheid te diagnoseer.

Fusarium moniliforme[15] (Mouldy Corn Disease)

Dit is reeds sedert 1850 bekend dat perde, wat op swambesmette mielielande wei, kan vrek aan 'n siekte wat gekenmerk word deur erge senusimptome. Aangetaste diere raak "mal" en hardloop in heinings, mure, ens. vas en vrek binne 'n paar uur. Kenmerkend is groot areas van enkefalomalasie in die subkortikale witstof van die serebrum vergesel van bloeding (Fig. 3). Eers in 1971 kon Wilson en Maranpot[26] bewys dat *F. moniliforme* die oorsaak van die toestand was. Hulle kon slegs een geval in 'n donkie verwek voordat hul kultuur sy toksisiteit verloor het. Gedurende die afgelope paar jaar is die swam in Suid-Afrika geïsoleer en kon nie alleen die breinsindroom na willekeur veroorsaak word nie, maar is daar ook vir die eerste keer bewys dat die swam ook lewerbeskadiging in perde kan veroorsaak[13].

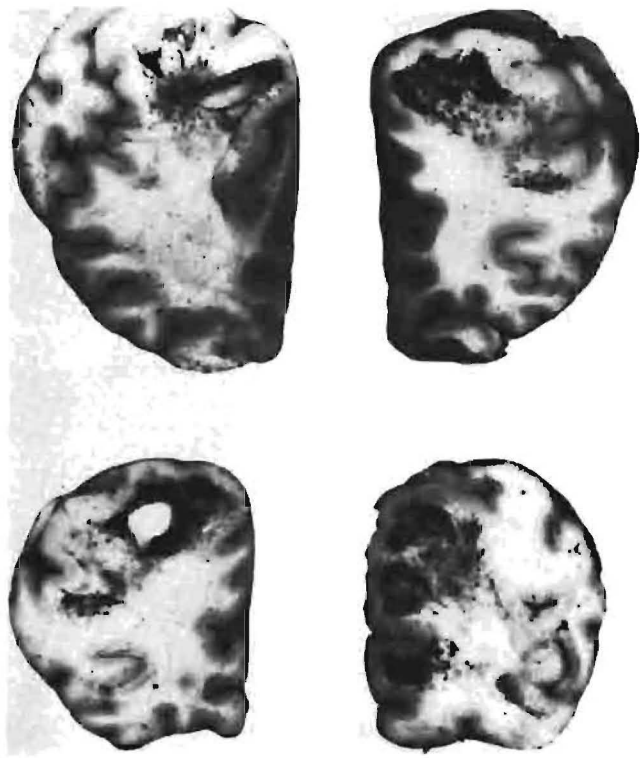

Fig. 3. *Fusarium moniliforme* vergiftiging. Vervloeiingsnekrose van subkortikale witstof in serebrum. Veelvoudige klein bloedings is teenwoordig naby die letsels.

Aspergillus clavatus[14] (Maroekvergiftiging)

In die V.S.A. is gedurende die jongste jare gevind dat etlike spesies van *Penicillium* en *Aspergillus* sekere metaboliete produseer, s.g. "tremorgenic toxins" wat spiertrilling, konvulsies en die dood kan veroorsaak. Hierdie werk was tot dusver beperk tot laboratorium diere.

In Suid-Afrika is verlede jaar vasgestel dat *A. clavatus,* wanneer dit onder sekere omstandighede op maroek groei, 'n onbekende gifstof vorm wat hoë mortaliteit in beeste kan veroorsaak. Kenmerkende simptome is hipersensitiwiteit, stywe stokkerige gang, bewerasie van spiere en verlamming gevolg deur die dood. In die senustelsel is dit hoofsaaklik die laer motoriese neurone wat nekroties word. Dit veroorsaak uitgebreide neurogene spierletsels. Diere mag herstel maar toon dikwels permanente simptome van breinbeskadiging.

INFEKSIESIEKTES

Serebrale babesiose[6] in honde

Alhoewel dié vorm van galkoors reeds baie jare bekend is, is dit eers redelik onlangs aangetoon dat nekrose en bloeding dikwels in die brein voorkom. Hierdie letsels, wat in werklikheid infarkte is, word baie dikwels gesien in die basale ganglia, die dak kerne van die serebellum en in verskillende dele van die serebrale skors. Makroskopies kan die toestand baie maklik verwar word met diamidien-vergiftiging in die hond.

Fokale simmetriese enkefalomalasie in skape[22]

Hartley[10] in Nu Zeeland was die eerste om die toestand te beskrywe. Soos die naam aandui is die letsels van enkefalomalasie, bilateraal en simmetries in verspreiding. Die verspreiding is karakteristiek, nl. interne

kapsel (Fig. 4), dak kerne van die serebellum, middebrein en thalamus. Hierdie toestand kom sporadies voor gedurende uitbreke van bloednier en kan ook eksperimenteel verwek word met trypsin geaktiveerde toksiene van *Cl. perfringens,* tipe D.

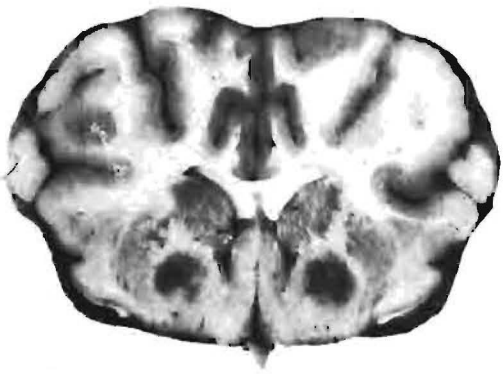

Fig. 4. Fokale simmetriese enkefalomalasie in 'n skaapbrein. Die letsels kan in die interne kapsel gesien word.

Hartwater[19]

Breinswelling gevolg deur breinedeem is die basis van die bekende senusimptome van dié siekte. Bloedinge en klein areas van nekrose volg op die edeem. Baie kenmerkend is verspreide foki van Walleriese degenerasie veral in die interne kapsel in die *corpus striatum.* Daar is ook gevind dat baie van die s.g. 'atipiese' gevalle van hartwater wat ten spyte van korrekte behandeling nog vrek, gevalle is waar brein edeem baie vinnig ontstaan of waar daar te laat of met 'n te lae dosis behandel word. Alhoewel die organismes vernietig word, vrek die diere aan die gevolge van die brein edeem.

Kraniale absesse[18] (Bosluiskop)

Uitbreke kom van tyd tot tyd voor in bokke en soms ook in beeste. Die siekte word dikwels met hartwater en senuvergiftiging verwar. Die uitstaande simptome is aanvanklike ataksie, hipersensitiwiteit en 'n baie opvallende opistotonus met uiteindelike verlamming. Die bok kan soms aanhoudend blêr en een oog kan uitpeul. Besmette bosluis bytwonde aan die kop en veral aan die basis van die horings en in die ore, is waarskynlik die primêre oorsaak. Metastase vind dan plaas na die plexus van bloedvate om die hipofise en/of na die brein waar o.a. inflammasie en absesse kan ontstaan. Dit is nog nie duidelik waarom die bok so besonder vatbaar vir hierdie toestand is nie. *Corynebacterium pyogenes* word gewoonlik uit die letsel geïsoleer.

Hidranenkefalie

Gedurende die 1974/75 lamseisoen het *hydrops amnii* in ooie, gepaard met hidranenkefalie en artrogripose (Fig. 5) in die fetusse taamlik wydverspreid in die noordwestelike skaapstreke van die land voorgekom. Eksperimentele werk het getoon dat beide die attenueerde Wesselsbronsiekte-entstofvirus en die natuurlike Wesselsbronvirus die toestand kan veroorsaak[8]. Slenkdalkoors entstofvirus veroorsaak ook hidranenkefalie en atrogripose, sonder *hydrops amnii*[8]. Ooie is besmet tussen 42 en 74 dae dragtigheid.

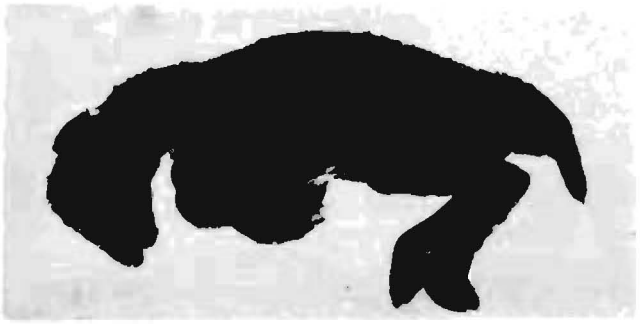

Fig. 5. Artrogripose in 'n lam. Die toestand gaan gepaard met hidranenkefalie in die lam en *hydrops amnii* in die ooi. Distokie vind gewoonlik plaas a.g.v. die verwronge ledemate. Tortikollis en bragignatie is ook teenwoordig.

Heelwat gevalle van hidranenkefalie is ook in kalwers gedurende die afgelope 3 seisoene waargeneem (Fig. 5 & 6). Eksperimenteel is bewys dat bloutongvirus die toestand in beeste kan verwek[2]. Dit is egter nie die enigste, en waarskynlik ook nie die belangrikste oorsaak van hidranenkefalie in kalwers nie. In Japan en Israel is daar sterk aanduidings dat Akabanevirus ook hierdie toestand kan veroorsaak[11].

Fig. 6. Natuurlike geval van hidranenkefalie in 'n kalf. Let op die opistotonus.

Fig. 7. Hidranenkefalie in 'n kalf. Geen serebrum is teenwoordig nie. Dit is vervang met vloeistof wat hier uitgeloop het nadat die breinvliese stukkend gesny is.

Nosematose *(Nosema (Encephalitozoon) cuniculi)*

Die eerste geval van hierdie siekte in 'n hond in die R.S.A. is in 1966 beskrywe[5]. Siektetekens behels blindheid, ataksie en konvulsies. Uitstaande letsels is 'n meningo-enkefalitis met mikrogranulome, gliose asook uitgebreide trombose en bloeding. 'n Interstisiële nefritis is baie dikwels ook teenwoordig. 'n Geval in 'n kat is ook sedertdien beskrywe[24].

VERGIFTIGING DEUR TERAPEUTIESE MIDDELS

Diamidien-vergiftiging[16]

Oordosering en/of herhaling van terapeutiese dosisse veroorsaak in die hond uitgesproke senusimptome soos balanssteurnisse, rolbewegings, ekstensor styfheid, opistotonus, nistagmie en terminale verlamming. Bloeding en enkefalomalasie kom voor in die serebellum, midbrein, thalamus en basaal kerne (Fig. 8). Hierdie toestand word gewoonlik klinies verwar met serebrale babesiose, tetanus of hondesiekte.

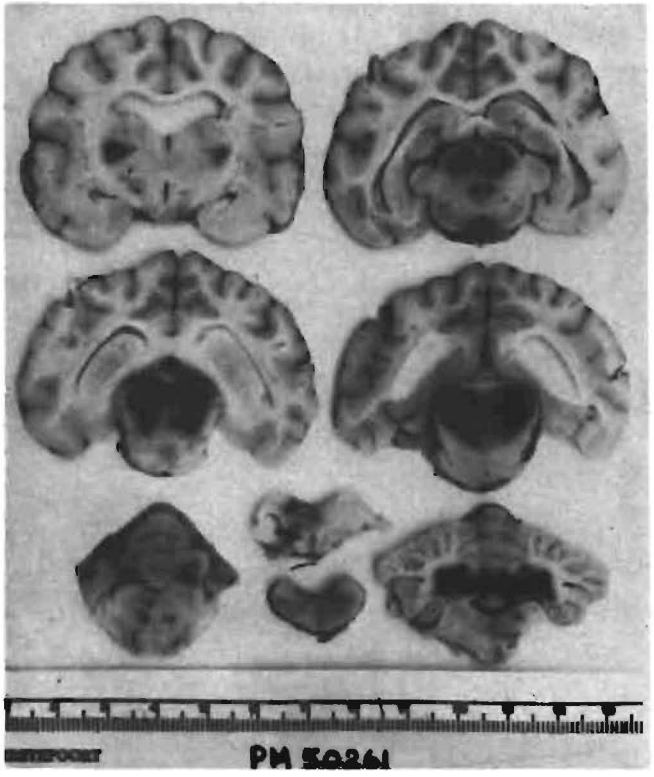

Fig. 8. Diamidien-vergiftiging in die hond. Nekrose en bloeding in die thalamus, *corpora quadrigemina*, middebrein, dakkerne gebied van serebellum en in die *medulla oblongata*.

CS_2-Haloxon-vergiftiging in perde[17]

'n Kombinasie van dié twee stowwe veroorsaak afsterwing van neurone, veral in die breinstam en sakrale gedeelte van die rugmurg (parasimpaties). Die gevolg hiervan is neurogene atrofie van die spiere van die larinks *(crico-pharyngeus, crico-thyroideus, thyro-hyoideus, chondro-pharyngeus* en *crico-arytenoideus dorsalis* en *lateralis)*. In erge gevalle vind daar ook atrofie van die spiere van die slukderm plaas (Fig. 9). Met oefening toon perde "roaring", eintlik 'n geweldige stridor en mag vrek a.g.v. asfiksie. Ander simptome is parese van die stert, anus en blaas, asook ataksie in die agterbene met swelling van die laere gedeeltes van die bene. Interessant is dat die twee middels, wanneer hulle afsonderlik gedoseer word, geen letsels veroorsaak nie.

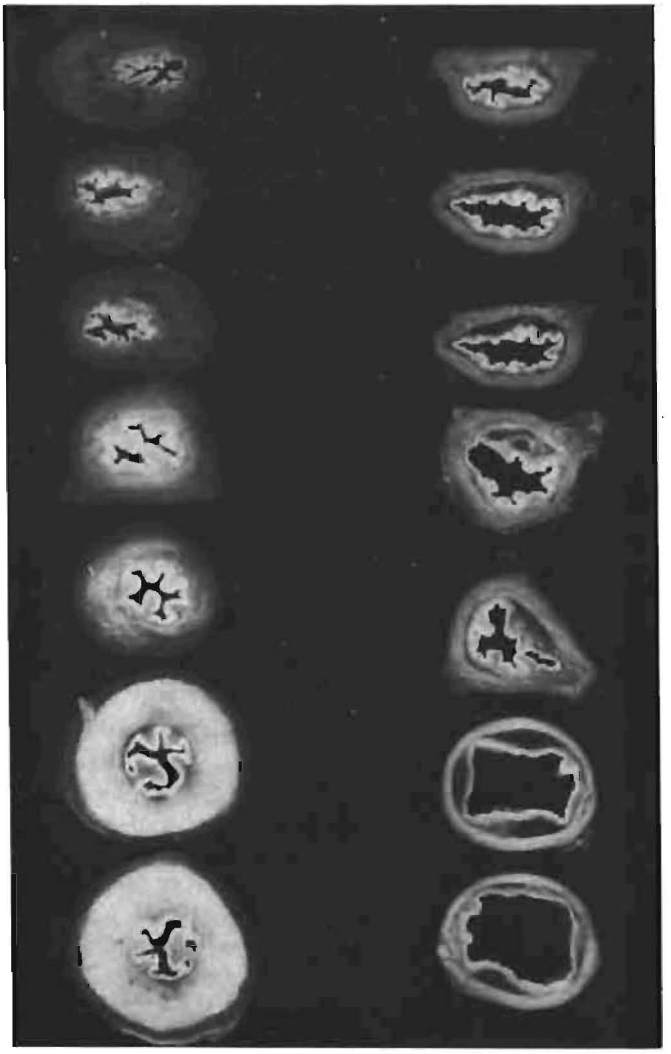

Fig. 9. CS^2-Haloxon-vergiftiging in die perd. Opvallende atrofie van die spierskede van die slukderm in 'n kroniese geval. Normale slukderm op linkerkant.

Rafoxanied-vergiftiging[23]

Gedurende die afgelope paar jaar het verskeie gevalle van vergiftiging met hierdie middel in skape voorgekom. In elke geval was foutiewe dosering die oorsaak. Die uitstaande simptoom is blindheid. Klinies is daar 'n uitgesproke midriase en soms ataksie. Die letsel bestaan uit 'n *status spongiosum* van die witstof, veral om die ventrikels, in die optiese bane, die laterale genikulaat, optiese fassikuli, basaal ganglia, *corpora quadrigemina* en tot 'n mindere mate in die rugmurg. Daar is ook nekrose van neurone in die retina wat later deur 'n gliose gevolg word. Hierdie toestand kan maklik verwar word met *Helichrysum* vergiftiging.

Ander middels

Enkele gevalle van Furazolidone[21] vergiftiging in kalwers is gedurende die afgelope paar jaar waargeneem. Met 'n oordosering van die middel toon kalwers uitgesproke senusimptome a.g.v. 'n brein edeem. Hexachlorophene vergiftiging[7] is ook in beeste teëgekom. Hierdie diere toon stupor en verlamming en histologies is daar 'n erge *status spongiosum* in die brein.

OORERFLIKE SIEKTES

Neuraxiale edeem van Hereford kalwers[9]

Hierdie is 'n autosomale resessiewe oorerflikheidstoestand in poenskop Hereford beeste. Kalwers word

gebore met die siekte, kan gewoonlik nie staan nie en toon uitgesproke ekstensor spasme met stimulasie. Die letsel is 'n uitgebreide edeem van die terminale gemiëlineerde senubondels, wat mikroskopies as vakuoles gesien word. Hierdie toestand is onlangs vir die eerste keer in die R.S.A. waargeneem[7].

Mannosidose (Pseudolipidose) van beeste

Mannosidose in Aberdeen-Angus beeste is in 1957 reeds beskrywe[27]. Hierdie toestand toon 'n groot ooreenkoms met mannosidose in die mens, en is dusver nog net in suiwer Aberdeen-Angus beeste gesien. 'n Toestand wat nie te onderskei is van hierdie nie, is in Aberdeen-Angus kruiskalwers in die R.S.A. gesien[7]. Kruisings met verskillende rasse was betrokke in die kudde. Die kenmerkende letsel is die aansameling van 'n lipiedagtige stof in neurone en RE selle. Kalwers is gewoonlik "dom" en toon verskillende grade van ataksie

Spinale ataksie van beeste[25]

Die teenwoordigheid van hierdie unieke siekte is vir die eerste keer in 'n kudde beeste in die Gibeon distrik van Suidwes-Afrika gedurende 1969 waargeneem. Klinies word die toestand gekenmerk deur wisselende grade van ataksie van die agterhand. Meeste van die multipolêre neurone in die ventrale horings asook die groot neurone in die dorsale en laterale horings van die hele rugmurg word aangetas. Enkele abnormale neurone is ook gevind in die medulla oblongata. Opvallende vergroting, eksentriese kerne, abnormale Nisslgranules, beide in konfigurasie en hoeveelheid asook 'n eienaardige eosinofilêre paranukluêre massa, gewoonlik groter as die kern, is van die belangrikste afwykinge. Laasgenoemde massa is waarskynlik 'n abnormale hoeveelheid neurofibrille wat in die neurone vorm.

ANDER SIEKTES

Serebro-kortikale nekrose (Polio-enkefalomalasie van beeste en skape)

Hierdie siekte is die eerste keer in 1956 in die V.S.A.[12] beskrywe. Sedertdien is dit in baie ander lande gevind en het 'n lywige literatuur daaroor ontstaan. Klinies begin die siekte met spiertrekkings van die ore, ooglede en gesigspiere, gevolg deur blindheid, konvulsies en die dood. Minder erge gevalle mag blindheid toon, die kop word vir lang periodes teen voorwerpe gedruk, doelose koubewegings word gemaak, erge speekselvloei kom voor en eindelik raak aangetasde diere verlam.

Die letsel is 'n vervloeiingsnekrose van die grysstof van veral die serebrale kortex. 'n Tiamien (Vit. B_1) tekort is waarskynlik die onderliggende oorsaak. Behandeling van vroeë gevalle met tiamien gee goeie resultate.

Die toestand is die afgelope paar jaar onder skape en beeste in verskillende dele van die R.S.A. gediagnoseer[18].

Gedoelstiase (Uitpeuloog)[3]

Die eerste stadium larwes van die vlieg *Gedoelstia hässleri* en *G. cristata* veroorsaak a.g.v. migrasie deur die bloedvate ernstige letsels in verskillende organe van vreemde gashere, veral in die skaap, wat dikwels tot die dood mag lei. 'n Trombovaskulitis is die fundamentele letsel wat gloukoom en ander ernstige letsels in die oog, infarksie van die miokard, longe niere en enkefalomalasie van verskillende dele van die brein veroorsaak. In die brein bestaan die letsel uit verskillende ontwikkelingstadia van infarkte gepaard met 'n fokale of verspreide meningo-enkefalitis.

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DIE DIAGNOSE VAN BRUCELLOSE BY BEESTE

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ABSTRACT: Schutte A.P.; Ogonowski K.; Roux D. **The diagnosis of brucellosis in cattle.** *Journal South African Veterinary Association* (1977) **48** No. 1, 19 – 23 (Afr) Sect. Reprod., Vet. Res. Inst., 0110 Onderstepoort, Rep. of South Africa.

Tests currently in use for the diagnosis of brucellosis and the immune response in cattle following exposure to *Brucella* antigens are reviewed. The interpretation and evaluation of results obtained by different tests under conditions where S19 vaccine is used, are discussed. Attention is focussed on the possible role of the bull in disseminating *Brucella* organisms.

Brucellose is 'n dieresiekte wat toevallig die mens besmet. Die besmette koei, bok- en skaapooi is draers van die smetstof en alle diere en die mens word vanuit hierdie bronne blootgestel aan *Brucella* organismes.

Brucellose het 'n wêreldwye verspreiding en in Suid-Afrika word al 70 jaar lank met hierdie siekte geworstel. Ter plaatse word brucellose by beeste slegs met *B. abortus* gekoppel, terwyl in lande soos die VSA ook *B. suis* met die siekte in verband gebring word. Slegs met uitsondering word *B. melitensis* van die bees afgesonder.

Die diagnose van brucellose is geensins 'n maklike taak nie. 'n Indrukwekkende diagnostiese armamentarium is reeds teen hierdie siekte opgebou maar desnieteenstaande, blyk die reeks nog steeds onvoldoende te wees. Daar bestaan geen toets wat op sigself alle *Brucella*-besmette diere kan uitwys nie. Trouens, die laborant word dikwels genoodsaak om 'n battery van toetse en metodes in werking te stel om in sy doel te slaag.

Maar alvorens die huidige diagnostiese metodes evalueer word, is dit wenslik om ter wille van helderheid eers by enkele fasette van die immuunmeganisme van die bees en die *Brucella* organisme as sodanig, stil te staan.

IMMUNOGLOBULIENKLASSE[2 8 9 10 23 24 25]

Plasma proteïene kan op grond van elektroforetiese eienskappe in 4 groepe verdeel word, naamlik albumin en alfa-, beta- en gammaglobulien.

Behalwe vir enkele seldsame uitsonderings word teëliggaam aktiwiteit met laasgenoemde groep in verband gebring en derhalwe staan gammaglobulien dikwels sinoniem vir teëliggame.

Immunoglobulien is saamgestel uit heterogene klasse en subklasse. Vir die bees word IgG_1, IgG_2, IgM, IgA en IgE tans erken.

Immunoglobulien G_1 en IgG_2 word in die serum van immuniseerde beeste aangetref en beweeg vrylik tussen bloedvate en interstisiële vloeistowwe. Beide IgG_1 en IgG_2 is vir virus neutralisasie, toksien neutralisasie en bakteriese agglutinienreaksies verantwoordelik. Slegs IgG_1 kan egter komplement bind. Hierdie immunoglobulienklas is ongevoelig vir die inwerking van tiol reagense (mercapto-etanol) en is ook nie so gevoelig vir hitte, soos byvoorbeeld die IgM klas nie.

Immunoglobulien M is in teenstelling 'n heelwat groter molekule en is veral van belang by agglutinasie en komplementbinding reaksies. IgM is besonder gevoelig vir hitte, lae pH waardes, en die teëliggaamaktiwiteit word vernietig met die toevoeging van mercaptoetanol.

Immunoglobulien A is die belangrikste globulien in die meeste maar nie al die liggaamsekrete. IgA kan nie komplement bind nie en het ook nie 'n opsoniserende funksie nie. Die waarde van IgA lê blykbaar daarin opgesluit dat bakterieë verhoed word om aan die seloppervlakte vas te heg.

DIE *BRUCELLA* ORGANISME[1 13 14 16 17]

Die *Brucella* sel is saamgestel uit 'n aantal antigene, sommige met sterk agglutinogeniese aktiwiteit, terwyl ander weer die produksie van teëliggame stimuleer wat met komplement bind. Hierbenewens, besit van die ander antigene weer die eienskap om die produksie van monovalente of onvolledige teëliggame te bewerkstellig.

Die S-variante (bv. S19 *Brucella* stamme) stimuleer die produksie van al 3 bogenoemde soorte teëliggame. Hierteenoor, blyk dit dat die R-variante (bv. 45/20 *Brucella* stamme) slegs in die teenwoordigheid van adjuvante, serologiese reaksies sal uitlok.

Indien Stam 19 entstof aan verse toegedien word, word komplementbindingsteëliggame, agglutiniene, sowel as onvolledige teëliggame geproduseer. Hierdie primêre stimulasie vir immunoglobulienproduksie duur enkele weke waarna die produksie geleidelik weer afneem. Sou hierdie diere aan 'n sekondêre of tweede dosis blootgestel word, is die teëliggaamreaksie beperk tot die produksie van komplementbinding en onvolledige teëliggame. Die agglutiniene bly relatief konstant.

Die 45/20 adjuvant entstowwe reageer in teenstelling hiermee soos 'n "ware" antigeen in die sin dat met die eerste (primêre) toediening weinig teëliggame geproduseer word. Met die tweede (sekondêre) dosis, word tot 'n groot mate onvolledige en tot minder mate, komplementbindingsteëliggame geproduseer. Prakties geen agglutiniene word geproduseer.

SEROLOGIESE DIAGNOSTIEK[1 4 5 6 15-18 20 27]

Daar is 'n verskeidenheid van serologiese-toetse beskikbaar vir die diagnose van brucellose. Met die gebruik van entstowwe het dit ook nodig geword om metodes te formuleer wat aangewend kan word om die entstofreaksies van aktiewe besmettings te onderskei. Die meer belangrike van hierdie kan kortliks aangehaal word.

DIE SERUM AGGLUTINASIE TOETS (SAT)

Hierdie toets word al jare lank vir diagnostiese doeleindes wêreldwyd in uitroeiingskemas gebruik.

Die FAO/WHO Komitee insake brucellose beveel aan dat 100 internasionale eenhede (IE) as grenslyn vir nie-geënte diere asook diere waarvan geen entingsgeskiedenis beskikbaar is, aanvaar moet word. Hierteenoor word aanbeveel dat 200 IE die grenslyn moet wees vir diere ouer as 30 maande en waarvan die entingsgeskiedenis betroubaar is.

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Sommige lande stel strenger vereistes, en slegs gevalle met 30 of minder eenhede word as negatief geklassifiseer. Diere met titers tussen 30 en 100 eenhede sal negatief geklassifiseer word indien die komplementbindingstoets ook negatief blyk te wees.

Die agglutinasie toets se beperking lê daarin opgesluit dat beide IgG en IgM opgewys word. Derhalwe kan daar nie met hierdie toets tussen infeksie- en entstoftiters onderskei word nie. Eweso kan gevalle in die vroeë stadia van besmetting asook lankstaande chroniese gevalle ook nie met hierdie toets identifiseer word nie.

KOMPLEMENTBINDINGTOETS (KB)

Die KB toets is besonder sensitief en akkuraat en word wêreldwyd vir *Brucella*-diagnostiek gebruik.

Die toets bestaan kortliks uit 2 fasette. Die eerste gedeelte behels 'n reaksie tussen antigeen en spesifieke teëliggame. As komplement (in die vorm van marmotserum) bygevoeg word, "bind" die komplement met die antigeen-teëliggaam kompleks. Die tweede faset of reaksie ontstaan waar die byvoeging van skaaprooibloedselle en anti-skaaprooibloedselle hemolisien as 'n indikator gebruik word om te bepaal of komplement gebind het aldan nie. In die afwesigheid van "vry komplement" word die skaaprooibloedselle nie deur die spesifieke teëliggame gehemoliseer nie, wat dan 'n positiewe reaksie aandui.

Indien geen teëliggame vir die eerste reaksie teenwoordig is nie, is die komplement vry en bind aan die tweede antigeen-teëliggaamreaksie. Hemolise vind plaas wat 'n negatiewe reaksie omskrywe.

In diere wat meer as 6 maande gelede geënt is, verdwyn gewoonlik beide die IgM en IgG teëliggame en die KB wat op hierdie stadium gedoen word, sal gevolglik negatief wees. In teenstelling hiermee bly die IgG teëliggaam vlak in aktief besmette gevalle baie lank positief en gevolglik sal die KB steeds positief wees terwyl die agglutinasietoets negatief blyk te wees.

Hierdie onderskeiding is slegs op 'n kuddebasis geldig en hou nie noodwendig vir individue stand nie. Hierbenewens moet dit ook beklemtoon word dat die KB geen onderskeid maak tussen 'n geval wat slegs *onlangs geënt* was en 'n aktiewe besmette geval nie.

DIE COOMBS TOETS (ANTIBEES-GLOBULIEN, ABT)

Waar die SAT die vlak van agglutiniene (volledige) teëliggame identifiseer en waar die KB toets die vlak van komplementbindingsteëliggame uitwys, illustreer die ABT die totale teëliggame-vlak wat tot stand gekom het met die brucella-antigeen stimulasie. Anders gestel, beteken dit dat die ABT die onvolledige teëliggame wat nie met behulp van die SAT of KB toets opgewys kan word nie identifiseer.

ROSE BENGAL TOETS (RBT)

Hierdie toets is 'n modifikasie van die Bremer kaarttoets en die suur-plaattoets wat Rose en Roepke ontwikkel het vir die uitkenning van nie-spesifieke teëliggame. Die Brucella organismes word met Rose Bengal kleurstof gekleur en aangesuur tot pH 3,6.

Basies behels hierdie toets 'n prosedure waar gelyke volumes antigeen en serum saamgevoeg word en waar die graad van agglutinasie aangeteken word. Die resultate van hierdie toets stem nou ooreen met die KB.

Die RBT identifiseer besmette gevalle op 'n veel vroeër stadium as wat met die SAT moontlik blyk te wees.

In kuddes waar die voorkoms van brucellose redelik laag is en waar kalwers volgens voorskrif met S19 geënt word, blyk hierdie toets ietwat te sensitief te wees. Derhalwe is dit raadsaam om onder sulke omstandighede positiewe RBT gevalle ook aan buisagglutinasie- en komplementbindingstoetse te onderwerp.

MERCAPTO-ETANOL TOETS (ME)

Hierdie toets is gebaseer op die verskynsel waar die aktiwiteit van IgM teëliggame vernietig word met die toevoeging van mercapto-etanol (ME sensitiewe teëliggame) terwyl die IgG teëliggame indien teenwoordig, ongeskonde bly.

Met die gebruik van Stam 19 entstof in kalwers vind daar ten eerste 'n styging van IgM plaas wat enkele dae later deur 'n styging van IgG teëliggame opgevolg word. Laasgenoemde teëliggame verdwyn egter weer gouer as die IgM teëliggame. In chronies besmette gevalle bly die IgG behoue en is soms ook die enigste immunoglobulien wat identifiseer kan word. Beide IgG en IgM kan komplement bind alhoewel IgG meer doeltreffend blyk te wees.

Die toets kan derhalwe gebruik word om IgM (die meer algemene teëliggaam in geënte diere) te identifiseer bloot deur titers voor én na behandeling met mercapto-etanol te vergelyk. So byvoorbeeld kan 'n titer van 1/100 voor behandeling teenoor 'n lesing van 1/25 daarna, dui op immunoglobuliene van die IgM klas en sulke gevalle word as nie-besmet geklassifiseer.

As die titer dieselfde bly of hoër blyk te wees na behandeling, dui dit op die teenwoordigheid van IgG en dié gevalle moet as positief geklassifiseer word.

RIVANOL TOETS

Indien Rivanol (2-etoksie-6, 9 diamienoakridienlaktaat) by serum gevoeg word, presipiteer al die serumproteïene uit behalwe die gammaglobuliene. Sodoende word die IgG van die IgM (betaglobuliene) geskei en dieselfde ondersoeke soos van toepassing op die ME metode kan hier ook aangewend word.

Na behandeling kan 'n plaat- of buisagglutinasie toets onderneem word. In geënte diere (hoofsaaklik IgM) sal daar 'n dramatiese val in die titer aangeteken word terwyl in besmette gevalle (hoofsaaklik IgG) prakties geen verskil opgewys word nie.

IMMUNOFLUORESENSIE (FA)

Hierdie toets behels net soos in die geval van KB en die Coombs twee fasette of reaksies. Serum wat ondersoek word, moet toegelaat word om met *Brucella* antigeen wat op 'n plaatjie vasgeheg is te reageer, waarna die preparaat weer deeglik gespoel word. Die preparaat word hierna met anti-bees gammaglobulien, wat aan isotiosianaat gekoppel is, oorvloei. Indien spesifieke teëliggame teenwoordig is, kan die *Brucella* organismes met gemak identifiseer word.

Net soos in die geval van die Coombs toets reageer die bees anti-Brucella globulien in die eerste deel van die toets as 'n teëliggaam en as 'n antigeen in die tweede deel.

DIE MELKRINGTOETS (MRT)

Hierdie toets is ontwikkel vir die identifikasie van *Brucella*-teëliggame in melk en as sulks is dit 'n handige

en praktiese toets om besmette kuddes te identifiseer. Die toets is afhanklik van:

(a) Vetglobule-agglutinien wat in melk teenwoordig is en wat verantwoordelik is vir die klomping van vetglobules wat na die oppervlakte van die melk monster styg. Hierdie tipe agglutinien word vernietig indien die melk verhit of oormatig agiteer word.

(b) *Brucella*-teëliggame: Word identifiseer deur die toevoeging van *Brucella* antigeen wat met hematoksilien gekleur is. Die antigeen bind met die teëliggame om 'n kompleks te vorm wat aan die vetglobules koppel, na die oppervlakte styg, en 'n blou ring in die roomvlak veroorsaak. Indien geen teëliggaampies teenwoordig is nie bly die gekleurde *Brucella* antigeen in suspensie en kleur die melk onder die roomvlak blou.

Dit is egter belangrik om daarop te let dat hierdie toets nie absoluut betroubaar kan wees nie, aangesien *Brucella*-besmette diere nie noodwendig 'n infeksie van die uier ook het nie.

Hierdie diagnostiese metode word beïnvloed deur die feit dat met mastitis, uitsypeling van serumproteïene in die melk plaasvind, wat dan aanleiding tot vals-positiewe resultate gee. Van ewe veel belang is die voorkoms van bakteriese en sellulêre debris. Die pH en kleurveranderings wat plaasvind, bemoeilik die interpretasie van die resultate. Vars melk (voordat dit afgekoel is) en kolostrum is ook geneig om aanleiding tot vals positiewe reaksies te gee. Hierbenewens is dit ook bekend dat indien S19 entstof aan volwasse diere toegedien word, die MRT vir periodes so lank as 4 maande positief uitval. Hierteenoor het 45/20 entstowwe geen invloed op die MRT resultate nie.

BRUCELLA SERO-DIAGNOSTIEK ONDER PLAASLIKE TOESTANDE[6 7 17 18 21 26 27]

Data soos in Tabel 1 vervat, illustreer resultate wat gedurende die afgelope 2 jaar verkry is onder omstandighede waar die inentingsgeskiedenis onvolledig en deurgaans onbetroubaar was; waar *Brucella*-besmetting in kuddes teenwoordig was: Beide onder ekstensiewe sowel as intensiewe boerderystelsels: Beide onder melk-sowel as vleisbeeste; waar daar aktiewe foki van *Brucella*-besmetting in kuddes was en waar aanteeldiere nie voldoende beskerm was nie; waar S19 entstof nie volgens voorskrif gebruik was nie.

Tabel 1: *BRUCELLA* SERO-DIAGNOSTIEK: RESULTATE SOOS VERKRY MET VERSKILLENDE TOETSE ONDER PLAASLIKE TOESTANDE

		RBT: Positief			RBT: Negatief			
SAT		KB			KB			
Sera	Eenhede	Pos.	V	Neg.	Pos.	V	Neg.	
3 104	100	2 691	40	293	36	2	42	RBT 97% KB 89%
1 108	30 100	276	63	371	74	40	284	RBT 64% KB 40%
235	30	61	14	123	23	14	—	RBT 84% KB 48%
4 447								

SAT = **Agglutinasie**
RBT = **Rose Bengal**
KB = **Komplementbinding**

Bykans 70% van die 4 447 serummonsters wat as positief deur een van die battery van toetse (SAT, KB, RBT, ME, Rivanol) geklassifiseer was, het geblyk om meer as 100 IE te bevat. Maar indien die KB en die RBT resultate hiermee vergelyk word, blyk dit duidelik dat onder hierdie groep nie-besmette gevalle moes gewees het en wat verkeerdelik as positief geklassifiseer was.

Aan die ander ent van die skaal is 235 gevalle streng volgens die SAT as negatief geklassifiseer. Volgens die KB resultate was bykans die helfte van die gevalle *Brucella*-draers.

AFSONDERING EN IDENTIFIKASIE VAN *B. ABORTUS* ORGANISMES UIT FETALE WEEFSEL[1 7 16 17 18 26 27]

Daar is ongelukkig weinig kenmerkende letsels in die verwerpte konseptus te bespeur en ondersoeke na brucellose op sulke materiaal is beperk tot die identifikasie en/of afsondering van die veroorsakende organisme.

Nieteenstaande die tekortkominge, is fetale materiaal gewoonlik voldoende om 'n diagnose van brucellose te stel. Gedurende die afgelope 5 jaar is fetale en plasenta materiaal van 1941 bedrywe aan 'n breë spektrum ondersoek onderwerp. Nege persent (9%) van die bedrywe soos gedurende 1971/72 ondersoek was as *Brucella*-besmette kuddes geklassifiseer. Die syfer vir 1975/76 was bykans dubbel soveel (15%) (Tabel 2).

Tabel 2: ONDERSOEKE NA DIE VOORKOMS VAN *B. ABORTUS* INFEKSIES BY BEESTE

Jaar	*Aantal Bedrywe*	*Aantal bedrywe besmet*
71/72	136	12 (9%)
72/73	364	40 (11%)
73/74	342	34 (10%)
74/75	534	47 (9%)
75/76	565	85 (15%)

Tabel 3: IDENTIFIKASIE VAN *BRUCELLA* ORGANISMES IN DRUKSMERE VAN FETALE WEEFSEL EN VRUGVLIESE GEKLEUR MET DIE GEMODIFISEERDE ZIEHL-NEELSEN KLEURMETODE

	Aantal Aborsies	Positief vir *B. abortus*
Fetusse met vrugvliese	134	34%
Fetusse sonder vrugvliese	427	11%

Dit is egter noodsaaklik dat die laborant aandring dat plasentale weefsel tesame met die fetus vir ondersoek aangebied moet word. Hierdie aspek word baie duidelik deur die data soos in Tabel 3 vervat, geïllustreer. Van die 134 fetusse wat gedurende 1975/76 ondersoek is en wat plasentale weefsel ingesluit het, was 34% op smeerondersoeke as positief geklassifiseer. In teenstelling hiermee kon slegs in 11% van 427 fetusse wat gedurende dieselfde periode verwerk is en wat geen plasentamateriaal ingesluit het nie, as positief op smeerondersoeke geklassifiseer word.

Hierdie aspek word verder uitgelig deur gevalle waar *Brucella* organismes in druksmere van die plasenta identifiseer kon word. Abomasumsmere van 10% van hierdie gevalle en 28% van die longsmere, het negatief geblyk te wees (Tabel 4).

Uit die resultate van ondersoeke wat gedurende die

Tabel 4: IDENTIFIKASIE VAN *BRUCELLA* ORGANISMES IN FETALE MATERIAAL

	Vals negatief		
	Long	Abomasum	Plasenta
A ZN-gekleurde Druksmere	28%	10%	0%
B Isolasies	16%	0%	0%

afgelope 5 jaar versamel is, blyk dit dat die fetale weefsel waaruit *B. abortus* organismes met sukses afgesonder kan word, deurgaans vrugvliese, fetale maaginhoud, melk en *lochia* insluit. Laasgenoemde is besonder belangrik na fetale verwerping of gedurende die eerste 2 weke post partum. Dit is belangrik om daarop te let dat in *Brucella*-besmette kuddes daar 'n aantal besmette koeie teenwoordig sal wees wat geboorte gee aan lewendige kalwers terwyl die vrugvliese en baarmoederafskeidings erg met *Brucella* organismes gekontamineerd is.

Omrede fetale weefsel en meer dikwels vrugvliese wat vir ondersoeke aangebied word erg gekontamineer is, moet selektiewe media waar kiemweermiddels bygevoeg word, gebruik word. Die metodes vir die afsondering van *Brucella* organismes uit fetale weefsel en ander materiaal kan as volg saamgevat word:

VRUGVLIESE

Brucella organismes kan redelik gemaklik van aangetaste cotyledons afgesonder word. Gedeeltes van die plasenta wat inflammatoriese letsels wys, moet vir hierdie doel geselekteer word.

MELK

'n 20 ml hoeveelheid word vir 10 minute teen 1 000 r.p.m. afgeswaai. Die melk onder die roomlaag word afgegooi en die room-sedimentmengsel word op selektiewe media uitgeplant. Inkubeer onder 10% CO_2 by 37°C vir 3-8 dae.

ANDER WEEFSEL

Let wel dat *Brucella* organismes nog uit limfkliere en uierweefsel afgesonder kan word in gevalle waar al die ander weefsel negatief blyk te wees. Pharyngeale- en mesenteriese limfkliere, uterus en testis is ook geskik vir isolasie werk.

Weefsel materiaal word in metanol gedompel en deur 'n vlam getrek. Hierna word die weefsel óf met steriele sand óf met emulsifiseerders fyn gemaal. 0,2 ml word op selektiewe kweekbodems uitgestryk. Die vloeibare gedeelte kan ook in marmotte gespuit word.

Fetale maaginhoud word (na die maagoppervlakte met 'n warm mes geskroei is) met behulp van Pasteurpipette of 'n spuit opgesuig en direk op selektiewe media oorgeplaas.

BIOLOGIESE METODES

Deur van marmotte gebruik te maak, kan *Brucella* organismes uit materiaal afgesonder word wat met direkte isolasie-tegnieke negatief geblyk te wees het. Twee marmotte per monster moet gebruik word en 1-2 ml van die suspensie moet binnespiers toegedien word. Die marmotte word 3 weke later geslag en die milt word op *Brucella* agar plate uitgestryk. Om ondersoeke af te rond, is dit raadsaam om ook serologiese ondersoeke op hierdie marmotte te onderneem.

IDENTIFIKASIE VAN *BRUCELLA* ISOLATE[1 7 16 18]

Op selektiewe media kan *Brucella* kolonies uitgeken word as tipiese ronde gladde opgehewe homogene kolonies, gewoonlik 2 mm in deursnee, blou van kleur en wat duidelik teen die agtergrond afsteek. Die kolonies is gewoonlik heelwat kleiner. Plate moet deeglik ondersoek word omrede groei gerem word en slegs enkele kolonies teenwoordig mag wees wat moeilik waarneembaar is. Verdagte *Brucella* kolonies kan verder identifiseer word deur: (a) Agglutinasie toetse met positiewe *Brucella* serum; (b) Stamp of gemodifiseerde Ziehl-Neelsen kleuring; (c) Immunofluoresensie.

BRUCELLOSE BY BULLE[3 11 12 18 19 22]

Daar heers heelwat onsekerheid oor die juiste rol van die manlike dier in die epidiomologie van brucellose.

Dit word betwyfel of bulle met natuurlike dekking besmet kan raak en of *Brucella*-besmette bulle die siekte veneries kan versprei. Dit is egter waar, dat semen van besmette bulle met intrabaarmoeder plasing, brucellose in koeie kan verwek. Sou die saad egter in die vagina of in die serviks gedeponeer word, sal slegs enkele van die vroulike diere besmet raak.

Gevalle waar jong bulle wat kongenitaal of gedurende die neonatale periode besmet geraak het, en waar die infeksie eers maande later as 'n vesiculitis en/of orchitis waargeneem word, is reeds aangeteken. Eweso is dit ook gedokumenteer dat soortgelyke letsels in die geslagstelsel van bulletjies na die toediening van stam 19 entstof, tot stand kan kom.

Die diagnose van brucellose by die manlike dier berus op agglutinasie toetse soos uitgevoer op serum en seminale plasma. Indien die bul vir KI doeleindes gebruik gaan word, moet semen ook vir die afsondering van *Brucella* organismes (op selektiewe media en biologies) verwerk word.

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BOOK REVIEW

BOEKRESENSIE

ANIMAL DISEASE MONITORING

EDITORS: D.G. INGRAM, W.R. MITCHELL and S.W. MARTIN

Charles C. Thomas, Springfield, Illinois, 1975. Pp x + 215. Figs 35 (14 Graphs) Tabs 36 Publ. Price unstated.

This book is a report on the proceedings of an international symposium held at the University of Guelph on July 4 and 5, 1974 and consists of 24 Chapters by 28 distinguished contributors on monitoring (the watch on events and as a result, advice is given) and surveillance (observation but within a control activity) of animal diseases.

An essential function of animal disease monitoring is the collection of data by as wide a variety of persons and institutions as possible e.g. farmers, practising veterinarians, livestock inspectors, laboratory directors, abattoir superintendents and veterinary faculties. The computer can play an important role in the recording, storage, retrieval, analysis and interpretation of these data. The importance of rapid and regular communication of the information to those who have the responsibility and authority to act on it is stressed. A microscale project concerns a farm, while mesoscale projects deal with abattoirs, laboratories, zoos, clinics etc. and macroscale projects with national surveys.

The subjects dealt with in this book cover a wide range for veterinarians with epidemiological interests. To illustrate this some of the subjects covered are as follows: The veterinary practitioner in disease monitoring; An integrated university system for animal disease data; The use of computer simulations in the design, evaluation and monitoring of animal disease control programs; Brucellosis; Dairy calf mortality; Clinical data; Parasitism in domestic animals; Disease accounting and reporting systems for diagnostic laboratories; Zoo animals; Storage, retrieval and statistical analysis of wildlife disease data; A population-based animal tumor registry; Disease in a primate centre; Bovine leucosis in Canada; International co-operation in animal disease monitoring; The future of disease monitoring in agricultural animals.

The chapter on disease monitoring for disease eradication will interest most veterinarians and deals with surveillance at the farm, the milk ring test for brucellosis, identification of farm animals and disease monitoring at slaughter for the diagnosis of tuberculosis and brucellosis. Concerning the milk ring test it is indicated that a sample of milk from the entire production of the dairy herd was found to be capable of indicating the presence of a single affected cow using one drop of antigen to a composite milk sample of 1 ml milk in herds of 150 animals or less; 2 ml if the herd contains 151 to 450 cows and 3 ml for herds 451 to 700 cattle.

Parts of this book will be useful to students studying the subject of State Veterinary Medicine. Looking at the subjects mentioned above, private practitioners will also find some of interest to them.

It is, however, the epidemiologist and those responsible for policy making in research, faculties, animal disease control and eradication schemes and animal production who will find this book of interest, especially where computers are available or are soon to become available.

A.v.H.

FIG. 1. SKAAPBRANDSIEKTE 1940 TOT 1966 - BESMETTE DISTRIKTE MET JAARTAL VAN UITBRAAK

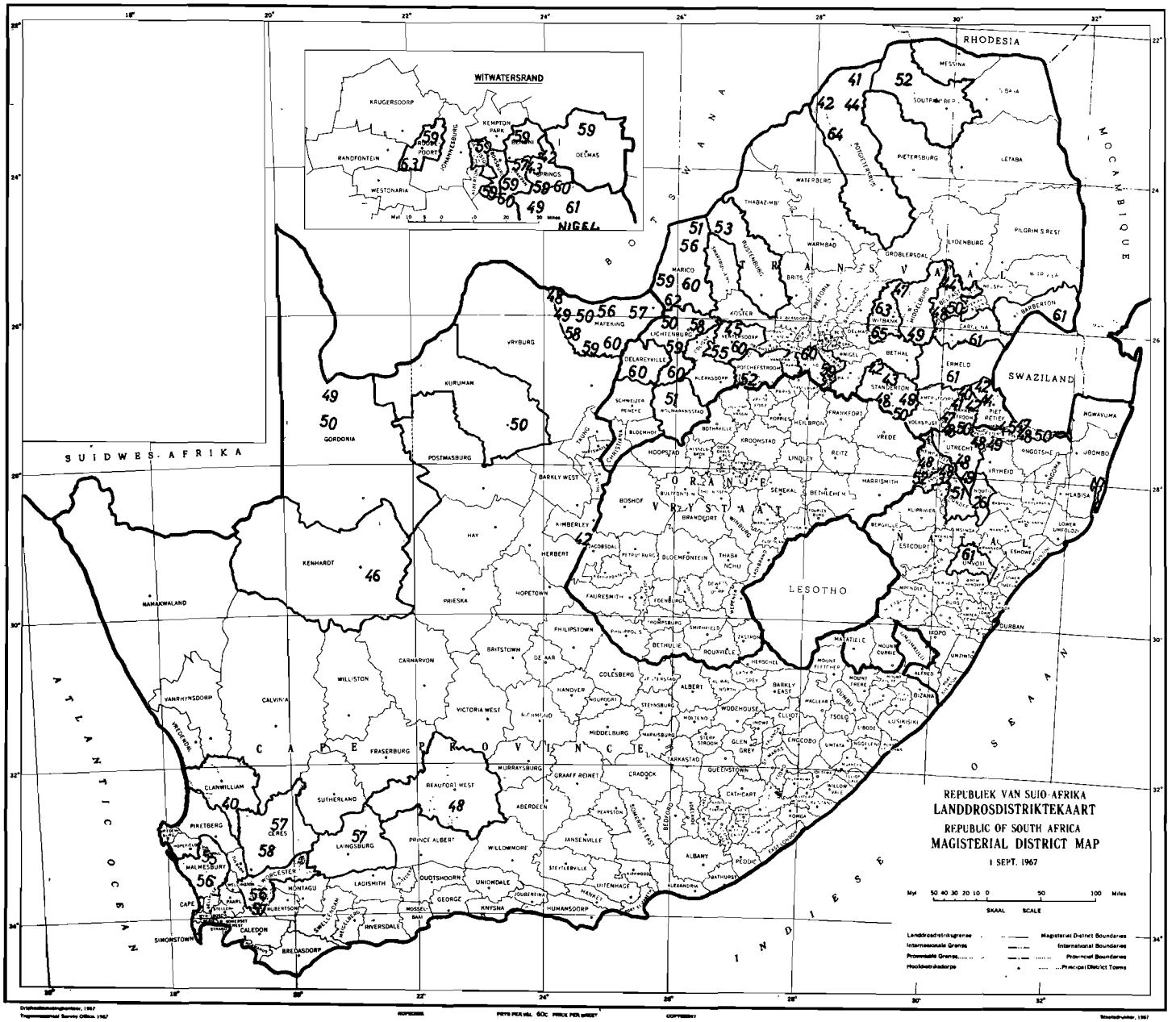
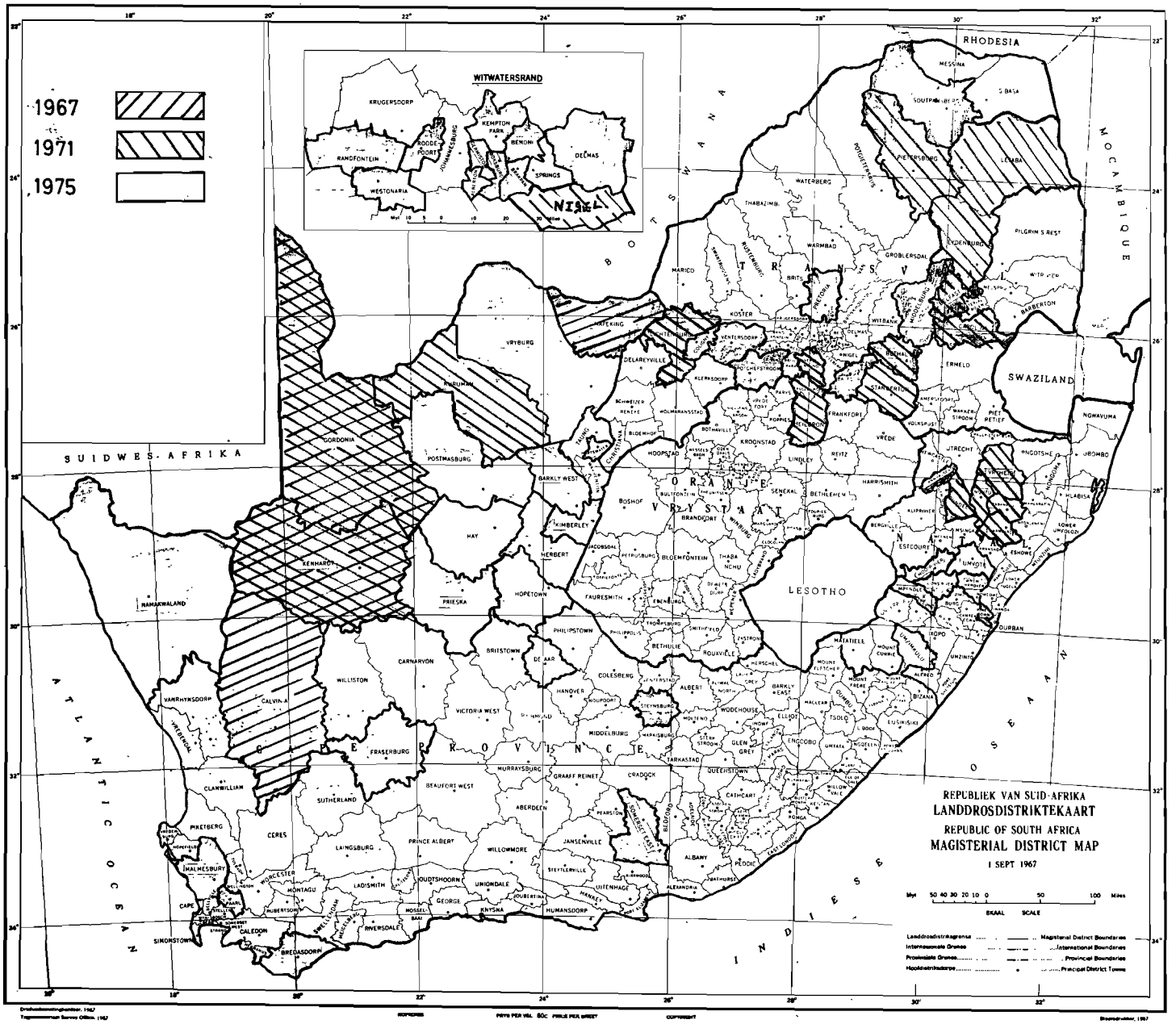


FIG. 2. SKAAPBRANDSIEKTE: DISTRIKTE WAAR BESMETTING VASGESTEL IS



dieselfde volksgroepe en families, wat gevolglik die oorsaak is van onwettige veebewegings.

In Noord-Wes Kaapland grens die *distrikte van Gordonia en Kuruman* ook oor 'n groot afstand aan Botswana. Hierdie gebied van Botswana is ver verwyder van 'n mark en afsetgebied vir vee in die land self en dit skep gunstige omstandighede vir onwettige vervoer na genoemde grensdistrikte in die Republiek.

UITBRAKE TUSSEN 1967 EN 1976

Die byna jaarlikse besmetting wat in sekere distrikte en gebiede voorkom soos in Tabel I aangegee, dui op vermoedelike endemiese besmettings in die gebiede met sporadiese verspreiding daarvandaan na die gewone afsetgebiede in die binneland waar die siekte in sommige gevalle vir bykans 50 jaar nie voorgekom het nie. Hierdie gebiede waar uitbrake gereeld voorkom (Vgl. Fig. 1 & 2) is:

1 *Pietersburg en omgewing* in Noord-Transvaal, waar dit vermoed word dat die jaarlikse groot saamtrek oor die Paasnaweek van 'n godsdiensgroep vanoor 'n wye deel van die land bygedra het tot die oorsprong en verspeiding van die siekte daar.
2 *Belfast en omgewing* in Oos-Transvaal waar die trekgewoonte vir winterweiding na die laeveld nog steeds bydra tot verspreiding van die siekte. Skape uit die dele word hoofsaaklik in Sentraal-Transvaal bemark en kan dus verantwoordelik wees vir sommige uitbrake in laasgenoemde gebied.
3 Alhoewel die aantal gevalle in *Mafeking en omgewing* in Wes-Transvaal verminder het oor die laaste dekade, bly hierdie grensgebied 'n moontlike bron van gevaar vir veral die Sentrale deel van Transvaal.
4 In *Nqutu, Utrecht en Newcastle en omgewing* van Noord-Natal was daar vir bykans 20 jaar sedert 1952 geen gevalle aangemeld nie tot dit weer in 1971 vasgestel is, sonder dat die oorsprong bepaal kon word. As gevolg van omstandighede en 'n moeilike bergagtige terrein is daar probleme om die siekte hier uit te wis en hou die gebied gevaar in vir skaapbewegings op die trekroetes na Suid-Oos Transvaal en vir bemarking na die res van Natal, waar verskeie uitbrake, soos in Tabel I aangedui, die laaste twee jaar voorgekom het.
5 *Kenhardt* en die Botswana-grensdistrikte van *Gordonia* en *Kuruman* in Noord-Kaapland word ook volgens Tabel 1 uitgewys as 'n probleemgebied. Skape uit die gebied word byna na alle windrigtings bemark en verhandel. Daar is aanduidings dat hierdie gebied die bron van besmetting kan wees vir uitbrake wat in Suidwes-Afrika, Vryburg, Sentraal-Transvaal, Oranje-Vrystaat, Oos-Kaapland en Suidwes-Kaapland voorgekom het.
6 Die aantal uitbrake in *Calvinia* toon ooreenstemming met die posisie in die distrikte verder noord in Noordwes-Kaapland en is die voorkoms van die siekte in die res van Suidwes-Kaapland meer sporadies van aard.
7 Die prentjie in *Sentraal-Transvaal* skep die indruk dat die gebied endemies besmet mag wees. Tussen 1960 en 1970 is egter geen gevalle aangemeld nie en die sporadiese uitbrake wat daarna voorgekom het hou verband met handels- en bemarkingspraktyke vanaf die vermoedelik endemies-besmette grensgebiede. Die toestand kan egter verander as daar steeds soveel uitbrake voorkom soos in die afgelope ses jaar.

GEBIEDE WAAR UITBRAKE SLEGS DIE LAASTE PAAR JAAR VOORKOM

1 Die *Oranje-Vrystaat* was skynbaar vir meer as 30 jaar vry van skaapbrandsiekte voor die eerste geval in 1970 in Frankfort aangemeld is en nou al in 22 landdrosdistrikte voorgekom het. Indien Noordwes-Kaapland nie as hoofsaaklike bron van besmetting vir hierdie gebied uitgeskakel word nie, kan die probleem in die toekoms al groter word.
2 Wat *Kaap-Oos en Karoo* betref, geld dieselfde as vir die Oranje-Vrystaat.

OPTREDE WAAR SKAAPBRANDSIEKTE VOORKOM OF VERMOED WORD

Die geweldige toename van brandsiekte die afgelope dekade (Tabel I) noodsaak 'n deeglike inspeksie en ondersoek in al die gevalle waar simptome soortgelyk aan die wat by skaapbrandsiekte gevind word, voorkom. Selfs in die gevalle waar daar met die eerste oogopslag toestande soos skurfte of besmetting met *Melophagus ovinus, Damalinia ovis* en *Psorergates ovis* voorkom, of grassade, klontwol en alopesie as gevolg van anemie of koorssiekte, gevind word, moet 'n ondersoek vir *Psoroptes communis ovis* nie sondermeer agterweë gelaat word nie.

SIMPTOME EN DIAGNOSE

Nadat slegs sporadiese uitbrake van skaapbrandsiekte vir bykans 30 jaar tot 1967 in Suid-Afrika voorgekom het, was daar min boere, veeinspekteurs en veeartse oor met ondervinding van die siekte. Gevalle het dus dikwels eers in 'n gevorderde stadium, met gevolglike wye verspreiding, onder die aandag gekom.

Na besmetting, gewoonlik deur kontakoordraging, veroorsaak *Psoroptes communis ovis* deur middel van sy monddele 'n wondjie in die epidermis. 'n Gelerige pustula vorm wat 'n strooikleurige eksudaat vrystel en na 4 tot 5 dae 'n kors vorm. Die lewenskringloop word gewoonlik voltooi tussen 8 en 10 dae en nuwe letsels ontwikkel op die rand van die kors. Dit word donkerder van kleur en lig saam met die gematte wolvesels van die vel af op of word afgeskuur. By wolskape kan letsels verwag word op die skouers en sye terwyl die stertvou en bors van haarskape noukeurige ondersoek verg.

Die myte veroorsaak intense irritasie, maar vir ondersoekdoeleindes word skape vooraf rondgejaag en dan saamgebondel sodat die hoër temperatuur as gevolg van verhoogde bloedstoevoer na die vel die parasiet stimuleer en sodoende pruritis intensifiseer. Skape wat hulleself dan byt, krap en skuur, sowel as die met 'n versteurde wolvag, kaal kolle op die vel of waar wol tussen die tande vassit as gevolg van knibbel aan die geïrriteerde ligaamsdele word vir noukeurige ondersoek uitgevang. In die somermaande wanneer myte onaktief is moet besondere aandag bykomstig gegee word aan ondersoek van die perineum, liesholte, infraorbitale holte en basis van die horings.

'n Vermoede van skaapbrandsiekte word eers bevestig wanneer *Psoroptes communis ovis* gevind is. Die beste plek om te soek is op die rand van 'n letsel en die parasiet mag moontlik met die blote oog of met behulp van 'n vergrootglas waargeneem word. Myte kan met 'n vuurhoutjie of 'n mespunt opgelig of d.m.v. 'n pipet opgesuig word. Aangesien myte lig van kleur is kan hulle op 'n donker agtergrond soos die swawelkant van 'n vuurhoutjiedosie geplaas en in die son neergesit

word, om indien hulle begin beweeg, die myte te onderskei van velskubbetjies. Vir die maak van skraapsels word die meer aktiewe vogtige deel van die letsel verkies. Dit word, indien myte nie met 'n direkte ondersoek gevind kan word nie, met 10 persent natriumhidroksied behandel, afgeswaai en druppels van die sediment en meniskus geneem vir mikroskopiese ondersoek. Die tipiese pedikel (stingeltjie) met drie segmente en tregtervormige suier aan die pote van myte of dele van myte met pote bevestig die diagnose en help om dit te onderskei van ander myte soos byvoorbeeld *Chorioptes* of ander oorsake van skurfte.

WETLIKE BEHEERMAATREËLS

Waar brandsiekte vasgestel is of vermoed word, kan die nodige maatreëls ingevolge die Wet op Dieresiektes en -parasiete, Wet 13 van 1956 en die Regulasies wat daarkragtens uitgevaardig is, ingestel word om die siekte te bestry. In die verband word verwys na sekere algemene regulasies wat voorkom in Deel VI van die Vaste Regulasie G K No R 1531 van 4 Oktober 1963 en Deel XI paragrawe 18 tot 27. Laasgenoemde handel spesifiek oor brandsiekte.

Deel VI Algemene Beperkings

Skaapbrandsiekte is een van die 32 siektes wat vermeld word in Aanhangsel D van die Vaste Regulasies. Dit kom ook voor in Aanhangsels E en F en kragtens Deel VI bestaan die volgende magte om die siekte te bestry:

1 Wanneer 'n dier besmet of vermoedelik besmet is moet die eienaar dit dadelik by 'n staatsveearts, veeinspekteur of polisiebeampte aanmeld.
2 Die eienaar moet ook ander eienare op dieselfde eiendom en aangrensende plase daarvan verwittig.
3 Elke veearts wat in die loop van sy werk of praktyk die bestaan van die siekte ontdek moet dit dadelik by die naaste staatsveearts aanmeld.
4 Wanneer 'n eienaar vasstel of vermoed dat sy diere besmet geraak het moet hy hulle dadelik afsonder en so hou totdat 'n staatsveearts die vrylating magtig.
5 'n Karkas van 'n dier wat aan die siekte dood is moet verbrand of minstens 1,22 m diep begrawe word.
6 Indien daar die vorige 90 dae besmette diere op die plaas was, kan die staatsveearts vatbare diere op 'n bepaalde gedeelte van die plaas laat afsonder om moontlike verspreiding van die siekte te voorkom.
7 Die eienaar kan ook verplig word om 'n register te hou waarin 'n beskrywing van die diere gegee en alle verminderings en vermeerderings opgeteken moet word.
8 Niemand mag enige vatbare diere na, deur of van die plaas waarop daar besmette diere is, beweeg sonder om 'n vervoerpermit van 'n staatsveearts vir die beweging te verkry nie.
9 Vatbare diere mag selfs nie eers geslag word sonder die staatsveearts se toestemming nie. Die doel hiervan is klaarblyklik om getallekontrole tydens 'n uitbraak moontlik te maak.
10 'n Staatsveearts beskik oor wye magte ter bestryding van siektes en kan instruksies gee vir byvoorbeeld afskeer; ontsmetting; merk van diere; verhindering van toegang tot plekke wat besmet of vermoedelik besmet is met siekte; vernietiging van dierekarkasse, uitwerpsels, afskeidings, kooigoed; ontsmetting van plekke, strukture en vervoermiddels en die herstel, verbetering en skoonmaak van dipbakke. Indien 'n eienaar versuim, uitstel of weier om aan hierdie instruksies gehoor te gee, kan 'n staatsveearts in dringende gevalle die funksies self uitvoer of laat uitvoer en is die eienaar aanspreeklik vir die koste daaraan verbonde.
11 'n Staatsveearts kan 'n eienaar ook skriftelik gelas om diere op 'n bepaalde datum, tyd en plek te toon vir inspeksie deur 'n beampte.

Deel XI 18-27

Hierdie Deel wat spesifiek met die siekte handel omskryf ook die woord "brandsiekte" soos in die inleiding aangedui.

1 Dit meld verder dat 'n skriftelike permit van 'n beampte vir die beweging van skape benodig word indien die skape gedurende die voorafgaande 6 weke in aanraking was met besmette skape, of as van weiding, krale of slaapplek gebruik gemaak is waarop daar in die tyd besmette skape was.
2 Indien brandsiekte onder trekkende skape uitbreek moet die eienaar die plaaslike veeinspekteur om instruksies vra betreffende afsondering, behandeling en verdere beweging van die skape.
3 'n Beampte kan die dip van skape uitstel indien dit volgens sy mening nodig is vanweë hulle swak kondisie, gevorderde dragtigheid, lang wol of weens gure weer. Hy kan egter gelas dat die sigbaar besmette skape op bepaalde tye met 'n doeltreffende middel behandel moet word.
4 Wat die diptussenpose betref word bepaal dat besmette of vermoedelik besmette skape dubbel gedip moet word met 'n tussenpose van nie minder as agt en nie meer as tien dae nie. Verdere enkel- of dubbeldip van die skape kan ook gelas word.

Deel VIII Metode om Skape te Dip

In paragraaf 16(1) word bepaal dat die dipbak self aan amptelike goedkeuring onderworpe is en elke dier minstens een minuut in die dipvloeistof moet wees en tydens die tydperk soveel keer ondergedompel word dat die kop en ore ook deeglik benat word.

Goedgekeurde skaapbrandsiektedipstof: Paragraaf 16(2) bepaal dat kalkswawel en BHC van die voorgeskrewe sterkte goedgekeur is, terwyl 16(2)(d) die Direkteur magtig om enige ander stof en konsentrasie daarvan goed te keur. Hiervolgens is diazinon teen 'n konsentrasie van 500 d p m vir 'n eerste vulling en 1000 d p m vir aanvullings op 23.8.1972 goedgekeur.

'n Beampte kan die eienaar ook gelas om 'n dipbak skoon te maak en met vars dipvloeistof te vul. Hy kan monsters van die dipvloeistof neem en dit is 'n oortreding as dipvloeistof onder die voorgeskrewe sterkte vir die verpligte dip van die diere gebruik word.

Beheermaatreëls: Waar brandsiekte vasgestel is, is dit van belang om na beste vermoë te bepaal hoe oud die besmetting is op die diere wat die oudste letsels toon. Die ouderdom van 'n letsel met 'n deursnit van 2,5 cm word geskat op ongeveer 1 maand met dieselfde toename in deursnit per maand vir die maande wat daarop volg. Toestande soos goeie kondisie van die skape, baie wolvet, droë weer, hitte van die somermaande en waar haarskape besmet raak, is ongunstig vir die parasiet en mag vergroting van die letsels dus nie teen dieselfde

tempo plaasvind nie, terwyl die omgekeerde gunstige toestande die letsels weer vinniger kan laat vergroot. Die bepaling van die ouderdom van die oudste letsels help om die oorsprong of bron van die besmetting op te spoor sodat die nodige ondersoek aldaar ingestel kan word. Alle bewegings vanaf die besmette plaas sedert die vermeende datum van besmetting moet opgevolg word om sodoende die omvang van verspreiding te bepaal. Die skape van al die kontakplase word deeglik ondersoek. Waar daar bewyse gevind word of 'n vermoede is van kontak met die skape van buurplase, kom laasgenoemde vir 6 weke onder kwarantyn en kan die skape ook gedip word. Dit is dikwels gewens om liewer te dip en sodoende 'n sluimerende besmetting uit te wis, as om later te moet terugkeer vir die bestryding van 'n nuwe uitbraak.

Wat die dip self betref, word elke skaap net voor dit die dipbak ingaan gewoonlik met verf voor die kop gemerk. Hierdie kontrole is nodig om te verseker dat alle skape op die plaas gedip word.

Tydens die dip word die skape met groot letsels en korste met die hand behandel en die kors opgebreek of afgeskeer sodat die dipmiddel alle parasiete kan benat.

Voor die gedipte skape na die weiveld teruggaan is dit wenslik om 'n uitgebreide inspeksie op die plaas uit te voer en te bepaal of alle skape vir dip aangebied was. Dit is dikwels die enkele skaap met 'n gebreekte been, die siekes, kruppeles of jong lammers wat, omdat hulle onwettig teruggehou word, die oorsaak is van 'n heruitbraak.

Indien daar bokke op 'n besmette plaas is, word hulle ook gedip om moontlike meganiese oordraging te voorkom.

Na die eerste dip gaan die skape oor na weiding waarop hulle die afgelope maand nie gekom het nie en moet die besmette kraal of slaapplek vir minstens 3 weke nie gebruik word nie. Indien dit nie moontlik is nie, word minstens 'n derde dip 8 tot 10 dae na die tweede toegepas.

Die skape bly onder kwarantyn vir minstens ses weke na die laaste dip. Gedurende die tydperk is alle bewegings van, na en deur die plaas aan 'n vervoerpermit, uitgereik deur 'n staatsveearts, onderworpe. Bewegings na byvoorbeeld 'n abattoir of ander plaas sal slegs oorweeg word mits beheer so toegepas kan word dat verspreiding van die besmetting nie sal plaasvind nie. As daar ses weke na die laaste dip tydens 'n tafelinspeksie van die skape geen tekens van besmetting is nie, word die kwarantyn opgehef. Dit is egter raadsaam om gereeld elke maand of twee opvolginspeksies uit te voer.

Uit die bostaande is dit duidelik dat daar nie 'n gebrek aan wetsmagte is om brandsiekte doeltreffend te bestry nie. Die redes vir die toename in die aantal uitbrake oor die laaste dekade moet moontlik gesoek word by:

1 'n Gebrek aan ondervinding en kennis van die siekte by diegene wat daarmee gemoeid is;
2 Onvoldoende beamptes om inspeksies uit te voer en beheermaatreels toe te pas;
3 Onvoldoende plaaslike kennis en ondervinding oor die biologie van *Psoroptes* en
4 Nie-effektiewe aanwending van dipstowwe.

Die vermeende oorsake van heruitbrake op die plase in Tabel 2 gee 'n aanduiding van sommige van die oorsake.

Tabel 2: HERUITBRAKE VAN SKAAPBRANDSIEKTE

Distrik/Plaas	Gebied	Datums	Opmerkings
Belfast I	O. Tvl	13. 2.74 en 2.12.75	Trekskape na Hoëveld
Middelburg I	O. Tvl	9. 7.75 en 4. 3.76	Belfast veeveiling
Nigel I	Sentr. Tvl	10. 2.73 en 29. 4.76	Spekulant
Babanango I	Natal	27. 4.72 en 11.12.72	Kontak Nqutu
Babanango II	Natal	28. 4.72 en 23.12.74	Kontak Nqutu
Gordonia I	N.W. Kaap	6. 9.74 en 18. 3.75	Opvlam van ou besmetting
Gordonia II	N.W. Kaap	7. 5.74 en 4. 8.75	Opvlam van ou besmetting
Gordonia III	N.W. Kaap	17. 6.74 en 26. 5.75	Kontakbesmetting
Gordonia IV	N.W. Kaap	25. 7.73 en 5. 8.75	Slagter/spekulant
Gordonia V	N.W. Kaap	1.10.67 en 1.10.69	?
Kenhardt I	N.W. Kaap	14. 7.75 en 13. 5.76	Alle skape moontlik nie met eerste uitbraak gedip nie.
Herbert I	N.W. Kaap	15. 7.75 en 4. 3.76	Opvlam – ou besmetting
Brandfort I	O.V.S.	26. 3.75 en 21.10.75	Spekulant
Koffiefontein I	O.V.S.	14. 5.75 en 24.12.75	Kontak albei uitbrake
Bloemfontein I	O.V.S.	28. 1.74 en 10. 6.75	Aankope veeveilings
Bloemfontein II	O.V.S.	28. 5.75 en 29.10.75	Opvlam ou besmetting
Boshof I	O.V.S.	12. 8.75 en 6. 5.76	Dipstofprobleem
Brandfort II	O.V.S.	13. 8.75 en 27. 1.76	Kontak?
Brandfort III	O.V.S.	13. 8.75 en 2. 2.76	Kontak
Calvinia I	S.W. Kaap	21. 3.74 en 23. 5.75	Heruitbraak – baie reën tydens dip – eerste uitbraak
Calvinia II	S.W. Kaap	29. 3.74 en 4. 6.75	Heruitbraak – baie reën tydens dip – eerste uitbraak
Calvinia III	S.W. Kaap	27. 5.74 en 17. 4.75	Opvlam ou besmetting
Calvinia IV	S.W. Kaap	9. 4.75 en 25. 2.76	Alle skape moontlik nie 2 maal gedip met eerste uitbraak
Calvinia V	S.W. Kaap	29. 7.75 en 4. 3.76	Alle skape moontlik nie 2 maal gedip met eerste uitbraak
Paarl I	S.W. Kaap	17. 7.72 en 22. 3.73 en 18. 2.75	Spekulant
Paarl II	S.W. Kaap	21. 7.72 en 24. 2.75	Grens aan I
Tulbagh I	S.W. Kaap	12. 2.74 en 29. 8.74	Nie na skoon weiding oorgeplaas infraorbitale besmetting
Calvinia VI	S.W. Kaap	11. 2.74 en 28. 4.75	Opvlam ou besmetting
Fraserburg I	K.O. & Karoo	9. 4.74 en 16. 4.75	Stroping dipstof
		13. 8.75 en 23. 4.76	Na dip nog lewende myte in infraorbitale holte

Verdere redes waarom brandsiekte toeneem: Die toename in die aantal uitbrake die afgelope tyd en die aantal heruitbrake waar daar geen ander getuienis gevind kon word as 'n opvlamming van die vorige besmetting nie is 'n aanduiding dat sekere gevestigde begrippe oor brandsiekte, die parasiet en die beheer daarvan bevraagteken kan word.

Een daarvan is die idee dat waar brandsiekte gevind en behoorlike dip van skape met 'n goedgekeurde dipstof toegepas word, die siekte in alle gevalle uitgewis kan word.

Verder wys Roberts en Meleney[5] op die gedragsverskille tussen verskillende stamme van *Psoroptes ovis* en bevraagteken die gasheerspesifisiteit van *Psoroptes.* Wat die verskillende stamme betref wys hulle op die volgende:

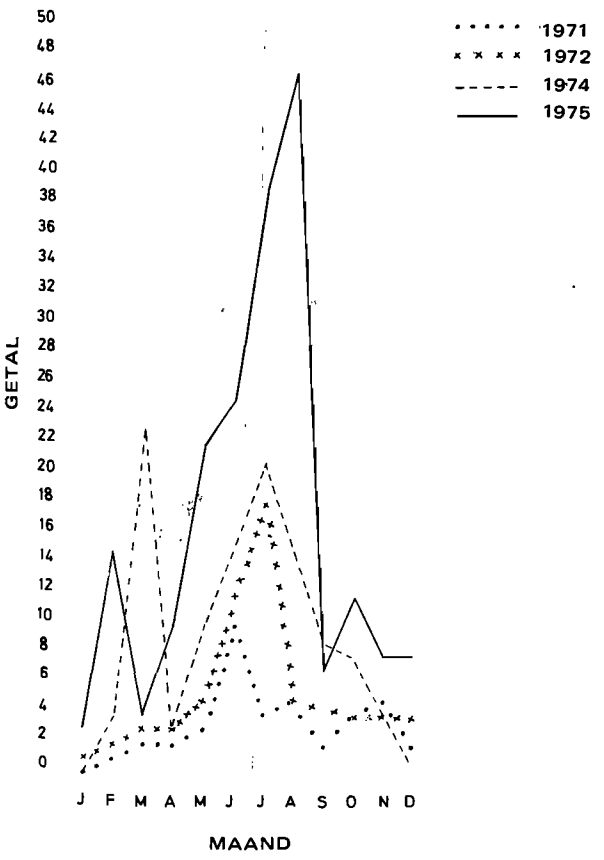
1 *Seisoen:* Sommige stamme weerstaan die populasievermindering gedurende somermaande suksesvoller as andere. Wat besmetting met 'n nie-virulente stam betref het 'n 100% besmetting by 31 skape in die winter afgeneem na nul in die somer. By die virulente stam het besmetting by 22 skape van 100 persent na 40 persent verminder.

   In Suid-Afrika toon 'n grafiek (Fig. 3) van die aantal uitbrake per maand tussen 1971 en 1975 dat die meeste gevalle waargeneem word tussen Mei en Augustus.

2 *Virulensie:* Myte van 'n "aggressiewe" stam weerstaan 'n bepaalde dipstof *beter as* die van 'n nie-"aggressiewe"-stam.

   Geen lewende myte van laasgenoemde stam kon sewe dae na toediening van coumaphos (asuntol) in konsentrasies tussen 0,05% en 0,25% gevind word nie. By die "aggressiewe" stam is konsentrasies tussen 0,1% en 0,4% gebruik en kon daar nie in geslaag word om met enige van die konsentrasies myte totaal uit te wis nie.

FIG. 3. SKAAPBRANDSIEKTE-UITBREKE PER MAAND



3 *Lewensduurte van myte op enkel-geïsoleerde skape:* Van 18 skape, elk apart afgesonder, wat met avirulente myte besmet was het 7 skape na 4 maande geen lewende myte meer getoon nie en na 21 maande was almal vry van myte.

   By die 24 skape besmet met die virulente stam was almal nog besmet na 4 maande, 10 het hulle myte voor 8 maande verloor en op 26 maande was daar nog 1 besmet.

4 *Invloed op beeste:* Van 11 beeste het 8 in 1956 besmet geraak met 'n nie-virulente stam en was nog net 1 bees besmet in 1962. Gedurende 1964 is 10 skape, besmet met 'n virulente stam by 'n ander groep van 20 beeste geplaas. In 1966 was 21 beeste uit 25 waaruit die groep toe bestaan het met *Psoroptes* besmet. Een kronies besmette bul met 'n nie-virulente stam is kunsmatig met hierdie stam besmet en het algemene letsels in so 'n graad ontwikkel dat die dier na 2 maande gevrek het.

   In die artikel word ook daarop gewys dat Sweatman (1958) die begrip van gasheer spesifisiteit verwerp en gevind het dat myte van perde en beeste met gemak op die skaap kon leef.

PSOROPTES EN ANDER DIERSOORTE IN SUID-AFRIKA

In Suid-Afrika is daar nog nie bewyse gevind dat *Psoroptes* wat op ander diersoorte voorkom, die oorsaak is van uitbrake van skaapbrandsiekte nie.

Van der Merwe[7] kon nie in 1948 daarin slaag om myte uit die ore en infra-orbitale fossa van skape oor te dra op die liggaam van skape nie. Hy het wel daarin geslaag om oormyte van skape na die ore van bokke oor te dra, maar nie die omgekeerde nie. Von Maltitz en Bezuidenhout[8] kon ook nie daarin slaag om in 1969/70 brandsiekte te veroorsaak met oormyte van skape wat nie met brandsiekte besmet was nie. Die ondersoek gaan egter voort om d m v die skanderingsmikroskoop en biologiese proewe vas te stel of daar wel enige verskille bestaan tussen die oormyte en die wat brandsiekte veroorsaak[2].

Psoroptes is in 1972 van letsels op 'n steenbok versamel en deur dr Fain van België as *Psoroptes com. ovis* geïdentifiseer[2]. Verder is myte van 'n skaap die afgelope jaar suksesvol na 'n bees oorgedra met die vorming en verspreiding van letsels en 'n geweldige pruritis. Sestig dae later is 9 myte weer na 'n skaap oorgedra, sonder om besmetting by die skaap te veroorsaak. Die klein skaal waarop die werk gedoen is regverdig egter nie bepaalde gevolgtrekkings mbt die herbesmetting van skape nie[2].

In die geheel gesien is daar dus aanduidings van:

1 Aanpasbaarheid van myte afkomstig van een gasheerdiersoort op 'n ander diersoort.
2 Verskille tussen stamme van *Psoroptes ovis.*
3 Die moontlikheid van basterkrag as verskillende stamme kruis.

Wat die oordraging, onder natuurlike omstandighede van *Psoroptes* vanaf ander spesies na die skaap betref, wys Tarry[6] daarop dat, alhoewel myte eksperimenteel van een gasheerspesie na 'n ander oorgedra en 'n toestand wat as brandsiekte by skape gedefinieer kan word, veroorsaak het, ondervinding geensins aandui dat dit onder veldtoestande 'n rol speel nie.

Een rede hiervoor is dat indirekte oordraging, alhoewel teoreties moontlik, in die natuur 'n twyfelagtige rol speel, terwyl direkte kontak tussen verskillende spe-

sies, behalwe miskien die bok en die skaap, selde plaasvind.

DIPSTOF

Kalkswawel

Met kalkswawel as dipstof en die toepassing van verpligte gelyktydige dip van skape is daar in die dertigerjare daarin geslaag om die siekte in Suid-Afrika uit te roei. Uitbrake in die veertigerjare het in verband gestaan met internasionalegrensprobleme en trekskape na buurstate.

B.H.C.

Die einde van 1949 is B.H.C. goedgekeur as 'n dipstof vir brandsiekte. Die algemene gebruik daarvan waar brandsiekte voorgekom het, het met goeie resultate voortgegaan tot 1972 toe dit van die mark onttrek is. Baie skaapboere het die middel gebruik om hulle skape in te dip nadat die diere geskeer was ter bestryding van uitwendige parasiete. Dit het waarskynlik ook 'n invloed op die verspreiding van skaapbrandsiekte uitgeoefen soos duidelik blyk uit die relatiewe klein aantal uitbrake in die 20 jaar waar tydens die middel gebruik is en die groot toename slegs een jaar nadat dit van die mark onttrek is.

In Brittanje word daar nou ook op 'n redelike groot skaal gebruik gemaak van die verpligte enkele dip van skape in gamma-B.H.C. tussen 1 Oktober en 31 Januarie jaarliks wanneer myte die aktiefste is. Aangesien gamma-B.H.C. met die wolvesels afmigreer na oppervlakte aanwending word daar aanbeveel[1] dat skape nie direk na hulle geskeer is behandel word nie terwille van 'n langer retensie van die middel deur langer wol. Die nawerking van B.H.C. op myte word aangegee as ongeveer 56 dae.

In Suid-Afrika het enkele gevalle voorgekom waar lewende myte weer later gevind is nadat skape met B.H.C. gedip was, byvoorbeeld 1 plaas in Gordonia distrik in 1950, en een in Keetmanshoop in 1957 en 'n ander een in 1967. Die redes hiervoor is nie vasgestel nie, maar mag moontlik blyk uit wat later bespreek sal word.

Diazinon

Die onttrekking van B.H.C. het dit noodsaaklik gemaak dat 'n ander dipstof of dipstowwe vir skaapbrandsiekte gevind moes word. Na laboratoriumproewe en veldtoetse in Noord-Natal[3] is diazinon in Augustus 1972 goedgekeur as 'n skaapbrandsiekte dipstof. Heruitbrake het hierna voorgekom soos in Tabel II vermeld. Op Koppiesfontein, Fraserburg tydens die uitbrake van 9.4.1974 en 16.4.1975 is die skape elke keer driemaal gedip in diazinon met 'n varsvulling van 1:600 en 'n aanvulling van 1:300 na $\frac{1}{3}$ van die vloeistof uitgedra is. Voor die tweede dip op 1.5.1975 het Nagy[4] lewende myte in die infraorbitale fossa van 'n skaap gevind. Na lewende myte weer op 13.8.1975 gevind is het Bezuidenhout[2] ondersoek gaan instel na die oorsaak van die heruitbrake en lewende myte op nog 2 skape gevind. Die dip is gemeng en aangevul volgens voorskrif soos hierbo aangedui. Dipvloeistof monsters het 'n konsentrasie van 433 d.p.m. na varsvulling getoon en nie meer as 355 d.p.m. na enige van die aanvullings nie, terwyl dit tot 36 d.p.m. gedaal het net voor die tweede aanvulling. Laboratoriumtoetse het getoon dat 160 d.p.m. effektief is teen *Psoroptes*. In hierdie geval het dit by tye beslis laer gedaal as die effektiewe konsentrasie. Latere waterontledings van die plaas toon 'n tydelike hardheid as $CaCO_3$ van 200 en permanente hardheid van 345.

Veldtoetse met dipstowwe

1 'n Nuwe formulasie *diazinon* wat in April 1976 op dieselfde plaas uitgetoets is met aanvullings na $\frac{1}{4}$ van die dipbakinhoud uitgedra is, het nie laer as 249 d.p.m. in konsentrasie gedaal nie en is dus heelwat bokant die verlangde 160 d.p.m.
2 Op 'n plaas in dieselfde omgewing het 'n bepaalde formulasie van *B.H.C.* teen 'n varsvulling van 1:300 en aanvulling van 1:150 na $\frac{1}{3}$ dipvloeistof uitgedra is, stroping tot 'n konsentrasie van 89 d.p.m. getoon. Dit is baie laer as die effektiewe konsentrasie van 175 d.p.m.
3 *Lindaan* gemeng en aangevul volgens voorskrif het nie in hierdie omgewing laer gedaal as 242 d.p.m. nie, terwyl myte deur 100 d.p.m. B.H.C. (Lindaan) gedood word.

Dipstofvereistes

Vir doeltreffende bestryding en uitwissing van skaapbrandsiekte in Suid-Afrika is 'n dipstof nodig waar die volgende vereistes in ag geneem word:
1 'n Prys wat dit moontlik sal maak om alle skape minstens eenkeer per jaar daarin te dip, ook ter bestryding van meeste ander uitwendige parasiete van skape.
2 'n Nawerking t.o.v. brandsiektemyte wat lank genoeg is om besmetting in 'n kudde na een dip uit te wis.
3 Genoegsame indringing van alle uitwendige holtes om myte wat daar skuil te dood.
4 'n Formulasie wat so is dat dit in enige (sagte en harde) water 'n konsentrasie sal behou wat sal verseker dat 'n effektiewe dosis om myte te dood, op die liggaam deponeer word.
5 Om die laagste effektiewe konsentrasie waarteen die middel myte dood te bepaal, behoort daar van die mees virulente stam van *Psoroptes* beskikbaar gebruik gemaak te word in laboratoriumproewe.

Moontlike Beheermaatreëls

1 Die gedagte om 'n skaapdipmiddel slegs vir registrasie in terme van Wet 36 van 1947 te aanvaar indien dit 'n middel bevat wat skaapbrandsiektemyte uitwis, verdien oorweging.
2 'n Verpligting dat alle skape jaarliks in so 'n dipstof met 'n lang nawerking gedip moet word lyk onafwendbaar.
3 Sekere gebiede soos hierin aangedui, wat skynbaar endemies besmet is, behoort oorweeg te word vir die toepassing van 'n verpligte gelyktydige dubbeldip onder amptelike toesig. Dit en normale inspeksiefunksies sal meebring dat die veeinspeksiepersoneel se getal aangevul moet word.
4 Die gebruik van spesiaal ingerigte vragmotors uitsluitlik vir die vervoer van skape vanaf verskillende plase dra moontlik ook by tot verspreiding van die siekte. Die skoonmaak en ontsmetting van vervoermiddels vir skape behoort dus ook aandag te kry.
5 Skaapboere behoort voorts aangemoedig te word om skaapdippe te herstel, te bou of van die verskuifbare tipes aan te koop.

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PORCINE RESPIRATORY DISEASE IN THE WESTERN CAPE PROVINCE

I.F. ZUMPT*, G.L. MULLER**, S.K. BAKKER*** and W.J.J. VAN RENSBURG*

ABSTRACT: Zumpt I.F.; Muller G.L.; Bakker S.K.; Van Rensburg W.J.J. **Porcine respiratory disease in the Western Cape Province.** *Journal South African Veterinary Association* (1977) **48** No. 1, 35 – 37 (En) Regional Vet. Lab., 7600 Stellenbosch, Rep. of South Africa.

The aetiology and pathogenesis of respiratory syndromes in pigs are reviewed with emphasis on the role of environmental factors and diagnosis.

Therapeutic and control measures are suggested and the cost of various regimens is dealt with in detail.

INTRODUCTION

Respiratory disease syndromes in pigs are probably the result of complex interactions of many factors rather than a specific infection. While a number of specific interactions may be involved, they may be present alone or in combination in herds without obvious respiratory disease problems. Respiratory disease is present to a varying degree in most pig herds in some form or another and is probably the most common disease complex of pigs in the world[3].

It was found that in the United Kingdom at least 10% of lungs had to be condemned at abattoirs[3] whereas this figure used to be less than 2% in the Western Cape[1]. Recently this figure has increased dramatically. Over a period of four months 1 484 of 19 500 slaughtered pigs or 7,6% showed lung lesions leading to condemnations. In some groups of porkers up to 60% of plucks were condemned. In the Western Cape acute respiratory disease leading to death from pneumonia before weaning, is becoming of increasing importance, whereas losses after weaning, contrary to the position in the U.K., are relatively unimportant[2].

A respiratory complex as encountered in the Western Cape will be discussed. On first visiting an infected farm, the first impression is that something is drastically wrong. One or more piglets per litter are visibly affected, in some outbreaks up to 70% of all piglets show a severe setback. After weaning, pigs lose mass with serious retardation of the growth rate.

Correction of housing and management with special emphasis on excessive humidity and inefficient ventilation reduces the general morbidity to some degree, but does not totally eliminate the respiratory complex.

In a few affected units where this complex seemed to have appeared very suddenly, it was found that in each of these outbreaks new stock had been introduced from three to ten months previously. In one of these cases only gilts, which had been quarantined for four weeks, were introduced.

CLINICAL SYMPTOMS

The number of pigs affected and degree of severity of the disease in affected piglets within a herd depends largely on the standard of housing, especially in respect of humidity and ventilation. Litters are normal and healthy at birth. First signs may be noticed as early as the fourth day after farrowing when one or more piglets begin to sneeze, especially following excercise. The odd sneeze then develops into sporadic bouts of sneezing, and with increasing age a dry cough is characteristic. At this stage the temperature may be moderately elevated. From the third week of age the stockman usually notices that something is obviously wrong. Affected piglets seem to lose mass, coughing becomes harsh, often in bouts lasting for up to a minute, breathing is very laboured and often only abdominal..

A dirty, long haircoat, characterised by blackish greasy flakes matted in the hair, is especially noticeable on the dorsum of the back and around the snout. The latter is in the shape of a broad ring half-way between snout and eyes.

Cannibalism, especially tail biting, becomes a serious problem. Some pigs may scour or have a loose stool. A nasal discharge is seldom seen except in the late stage due to secondary complications. The mortality is usually low, whereas the morbidity is high and often the whole litter is affected.

After weaning, severe loss of body mass or retardation of growth and runting are noticed with the occasional death of a severely affected pig. Secondary complications are commonly encountered; skin changes become marked, and affected pigs are often so weak that they cannot compete for food at the self-feeders. From the tenth to twelfth week of age onwards most of the pigs seem to recover, even without treatment, and gains in mass improve. The skin changes gradually to a light-pink colour, the haircoat loses its greasiness and gleams again. From 10 to 30% of affected pigs remain runts and are culled to reduce economic losses.

AETIOLOGY

In the U.K. it was found that besides helminth parasites the following played a role in the respiratory disease complex[2]:

Viruses: Inclusion Body Rhinitis Virus, Parainfluenza 3 Virus, Adeno- and Reoviruses and others.

Bacteria: Bordetella bronchiseptica, Pasteurella multocida, Haemophilus spp., *Pseudomonas* and other secondary bacteria.

Mycoplasmas: especially *M. hyopneumoniae* and *M. hyorhinis.*

Until recently very little diagnostic work on pigs had been done in the Cape.

In modern piggeries the lungworm *Metastrongylus apri* is very rarely encountered, and *Ascaris suum* is seldom a problem.

Virus isolations and serological diagnostic methods are in the planning stage and no work has been done in this respect.

Presented as part of State Veterinarians Group programme, Bloemfontein, Sept 1976.

*Regional Veterinary Laboratory, Stellenbosch
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***Meat Board Veterinarian, Stellenbosch

Various bacteria have been isolated from cases of respiratory disease. *B. bronchiseptica, Haemophilus* spp. and *P. multocida* are of unquestionable importance. Over the past two to three years one or more of these species have been isolated from specimens received from 18 farms which had a respiratory disease complex history. It is doubtful, however, if each of these organisms could be blamed individually for a disease as such.

From specific units under investigation *B. bronchiseptica* and *Haemophilus* spp. were more readily isolated from visually healthy piglets between 4 and 10 days of age, whereas in piglets over the age of 3 weeks *P. multocida* was the predominant organism encountered.

Mycoplasmas have been isolated from various outbreaks of respiratory disease and have been sent to Onderstepoort for identification and typing.

PATHOLOGICAL CHANGES

Macroscopic Lesions

The most important lesions are found in the lungs, which in acute cases show pale, pink-grey raised areas of pneumonia, mainly in the cardiac and apical lobes, where the lesions may be lobar or lobular, often extending into the diaphragmatic lobes, where they tend to be lobular in distribution (checkerboard effect). The areas of pneumonia may contain dull, white foci of necrosis *(Pasteurella)* or small abscesses *(Corynebacterium)* due to secondary infection.

If death was due to pneumonia, the lungs are also oedematous, with froth in the bronchi. A fibrinous pleuritis is occasionally seen.

In chronic cases the pneumonic areas, of similar distribution to acute cases, are dull red and collapsed, sometimes containing small abscesses. Fibrous adhesions are also sometimes seen.

Microscopic Lesions

The lesions seen vary according to the stage of development, severity of the pneumonia and degree of secondary infection.

Basically, the lesions start as an interstitial pneumonia with a peribronchial and perivascular infiltration of round cells, an accumulation of alveolar macrophages and a swelling and thickening of the alveolar walls. These lesions may become progressively more severe until the alveoli are quite consolidated, the bronchial epithelium becomes hyperplastic, and the bronchioli become stenosed by the round cell infiltration as well as developing lymphoid follicles in the bronchiolar walls (similar to those in the spleen and lymph nodes).

If the pig survives, the alveolar reaction subsides, but the lobules remain collapsed for some time, partly because of the abovementioned stenosis of the bronchioles. Alternatively the affected lung tissue is often invaded with bacteria which give rise to a purulent bronchopneumonia with large numbers of neutrophils in the bronchi and the alveoli. The lesions still retain their lobular distribution.

TREATMENT AND CONTROL

Various water and feed premedications were tested taking as principle criteria the effect on daily live mass-gain and marketing time. None of the tested preparations were completely satisfactory, and although the various organisms could not always be isolated during treatment, this was usually possible after medication was stopped. To date the best results have been obtained with a tetracycline medicated sow ration (110 or 220 g active ingredient per tonne) and a tylosin-sulphamethazine (100 g + 100 g/tonne) medicated creep and growth ration. As the latter preparation is not registered in this country yet, the following regime is now advocated:

(a) The sow ration is medicated with 110 or 220 g oxytetracycline*/tonne for the period four weeks before farrowing to the time of weaning five weeks after farrowing, or a total of nine weeks.
(b) The creep and grower rations are medicated with 100 g tylosin**/tonne until the pigs reach a minimum of 12 weeks of age, or if warranted until marketing.

Control measures include correction of housing, especially ventilation and elimination of excess humidity. In many outbreaks of respiratory disease, humid, ill-ventilated and unhygienic conditions were prevalent, but this does not apply to all units. Correction of these factors plus premedication of feeds is essential in the control of the respiratory disease complex.

Further investigation into the epidemiology of this complex will *hopefully* throw some light on to more efficient methods of control or of elimination of respiratory disease conditions.

FINANCIAL IMPLICATIONS

In most erosion diseases economic losses are difficult to measure. In swine financial losses due to respiratory disease complex may be direct or indirect. The latter aspect is very difficult to calculate and includes the costs of additional labour and space and thus loss of interest on capital, costs of extra electricity and water and the effect of downgradings and condemnations at slaughter. Due to the retarded throughput in such a unit, overcrowding results, and escalation of respiratory diseases within the piggery is unavoidable.

Direct losses are more obvious and can be measured. In an acute outbreak of a respiratory disease complex, morbidity in weaners can be as high as 100% with a 30% mortality and permanent stunting of another 30%.

In one specific outbreak where all rations were medicated, it was found that baconers were marketed some 40 days later than normal (period lengthened from 165-180 to 200-220 days). For a 100 sow unit it was calculated that losses amounted to: R15 520.00 not including cost of medication, eg:- 100 sows x 40 days x 2 kg feed at 10,8 cents/kg x 18 piglets/sow/year.

The cost of medication varies greatly depending on the length of treatment, choice of drugs, type of ration medicated and whether rations are home-mixed, delivered in bags, or in bulk. In the outbreak referred to, it was found that the following costs were incurred when one kilogram of feed, delivered in bags, was medicated:

(a) Tylosin at 100g/tonne = 2,32c
(b) Tylosin at 40g/tonne = 0,92c
(c) Oxytetracycline at 220g/tonne = 1,44c
(d) Oxytetracycline at 110g/tonne = 0,72c

*TM50: Pfizer

**Tylan: Elanco

If a baconer consumes 80 kg of a creep ration and 125 kg of a grower ration it would cost the following to medicate all the feed per animal:

(a) Tylosin at 100g/tonne = 205kg x 2,32c = R4.76
(b) Tylosin at 40g/tonne = 205kg x 0,92c = R1.89
(c) Oxytetracycline at 220g/tonne = 205kg x 1,44c = R2.95
(d) Oxytetracycline at 110g/tonne = 205kg x 0,72c = R1.48

It is further advocated that the sow ration be medicated for a period of one month before farrowing up to the time when her litter is weaned. If, therefore, a sow consumes 2 kg per day for the first 4 weeks and an average of 8 kg per day for the next 5 week period the costs will be 2 kg x 28 days x 0,72c plus 8 kg x 35 days x 0,72c = R2.42 if the ration is medicated with 110 g/tonne of oxytetracycline or R4.84 for 220 g/tonne of oxytetracycline.

If medication of the sow ration is prescribed with 110 g oxytetracycline/tonne and the creep and grower ration with 100 g/tonne of tylosin, costs for a 100 sow unit producing 1 800 piglets per year should be 100 sows x R2.42 plus 1 800 x R4.76 or R8 810.00 in total.

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Jl S. Afr. vet. Ass. 48(1) 39 – 40 (1977)

AN OUTBREAK OF CAPRINE LISTERIOSIS IN THE WESTERN CAPE

I.F. DU TOIT*

ABSTRACT: du Toit I.F. **An outbreak of caprine listeriosis in the Western Cape.** *Journal of the South African Veterinary Association* (1977) **48** No. 1, 39 – 40 (En) Regional vet. lab. Bag X5020, 7600 Stellenbosch, Republic of South Africa.

Listerial meningo-encephalitis (circling disease) is reported for the first time in ruminants in South Africa. An account is given of the clinical signs, pathology and bacteriological confirmation of the disease.

INTRODUCTION

In South Africa, listeriosis has only been reported in gerbilles[4] and chinchillas[3] although man, sheep, cattle, goats, horses, pigs, dogs, cats, rabbits and some wild animals are known to be susceptible[1].

In ruminants *Listeria monocytogenes* causes three distinct syndromes which do not occur simultaneously during the same outbreak[1]. The three syndromes include listerial meningo-encephalitis (circling disease), listerial abortion and systemic listeriosis. The organism has a high infectivity but a low pathogenicity. Morbidity is usually only about 10% but mortality in the meningo-encephalitic form may be as high as 100%[1]. The reason for this is not clear but a predisposing cause is suspected[1].

CASE HISTORY

The outbreak occurred in goats on a coastal farm in the Darling district of the Western Cape Province. The affected flock consisted of 70 goats, 15 of which had been bred on the farm and 55 introduced from the Oudtshoorn district during March 1975. The flock was kept in a camp of natural veld through which ran a wide, shallow stream with lush growth on its banks.

During the first week of September 1975, five of the goats became ill and subsequently died.

CLINICAL SIGNS

Affected animals isolated themselves, became depressed and usually carried their heads held high and slightly to one side. They were loath to move but on being disturbed they walked in circles. In the earlier stages, however, they were able to run in a reasonably straight direction when chased.

Partial paralysis of the muscles of mastication and deglutition occurred early in the course of the disease so that a large amount of unchewed material was found in their mouths. The lethargy progressed to a state of advanced somnolence followed by generalized paralysis with occasional slight clonic convulsions. Elevated rectal temperatures of up to 41°C were recorded in the early stages but were normal or subnormal later on. The animals usually died 24-48 hours after the first clinical signs appeared.

A week after the first cases were observed, the flock was moved to another camp. In all, eight of the 70 animals died, the last about 3 weeks after the first. Five cases were examined and necropsied.

NECROPSY

Macroscopic pathology

Congestion of the meninges and oedema of the brain was a consistent finding in all the cases. No further pathology was observed except in one case where there was a raised pale area near the apex of the heart. This lesion extended through the myocardium into the septum.

Microscopic pathology

Pathological changes were present in the brain tissues. These included mild meningitis over the cerebrum and micro-abscessation of the brain parenchyma. The latter, which was most severe in the pons and fairly mild in the thalamus, consisted of gliosis and neutrophil infiltration associated with heavy perivascular cuffing with lymphocytes and histiocytes. In addition there was oedema and encephalomalacia of the white matter in these areas. The myocardial lesion consisted of interstitial myocarditis with purulent foci.

Small Gram positive rods were seen in both the brain and myocardial lesions.

Bacteriology

Cultures were made on blood tryptose agar from the lung, liver, heart, kidney, brain and spleen and incubated aerobically as well as under 10% CO_2.

After two days, small oval haemolytic colonies were observed. These yielded Gram positive rods with rounded ends singly or in pairs. The organisms were motile. Duplicate specimens refrigerated for 3 days and cultured on blood tryptose agar yielded the same organism. Standard bacteriological techniques were employed to establish that the organism was *L. monocytogenes*[2]. This identification was confirmed by the State Pathology Laboratory, Cape Town, and the Bacteriology Department, Onderstepoort, where it was identified as Type 4.

DISCUSSION

The origin of the infection could not be determined but may have come either from rodents inhabiting the vlei area of the camp or may have been introduced by a carrier in the group of goats which had been purchased. It is known that healthy animals may harbour the infection in the nasal mucosa and thus act as carriers[1].

In one case, *Bronhamella (Neiseria) cattarrhalis* was also isolated from the brain, and, as this organism can occasionally be pathogenic, it may have played a contributory role.

There is no known method of control of the disease as vaccination appears to be of little value[1]. No subsequent mortality occurred in the flock after the eight clinically affected animals had died.

ACKNOWLEDGEMENTS

The author wishes to express sincere thanks to Dr J. Dale Kuys for the histopathological examinations, and to Mr W. van Rensburg for the bacteriology.

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BOOK REVIEW

BOEKRESENSIE

COMPARATIVE ANIMAL CYTOLOGY AND HISTOLOGY

ULRICH WELSCH and VOLKER STORCH

Sidgwick and Jackson, London, 1976
pp XIV 343 Figs 174 (black & white), Tabs 0, Publ. Price £3,50 (U.K. only)

This book can be highly recommended on the basis of its comparative approach. The authors, by presenting histological data on tissue and organ structure of invertebrate and vertebrate forms, have succeeded in giving the reader an unsurpassed insight into the evolutionary trends in histology. For those acquainted with mammalian histology this is an entirely new and most challenging approach.

The subject matter is of a highly detailed nature, well illustrated with sufficient emphasis on up-to-date electron microscopical findings. After a brief introduction on technical methods general cytology and ultracytology are dealt with, followed by a chapter on the four elementary tissues. Thereafter the integument with various types of receptor cells; the nervous system; the digestive tract; endocrine, respiratory and circulatory organs and organs of reproduction and excretion are dealt with. The stress is basically on mammalian morphology but the corresponding organs or their homologues in lower vertebrates and invertebrates are always dealt with. A literature reference list has been added at the end of each chapter.

An occasional minor error is encountered, as for instance Fig. 79 reveals myoepithelial cells in merocrine sweat glands but not in apocrine sweat glands. These will hopefully be corrected in later editions.

The book contains an enormous amount of important and diverse information and really fills a longfelt need in all biological sciences. Its use as a textbook or as a reference book can be most sincerely advocated.

W.H.G.

BOOK REVIEW

BOEKRESENSIE

PESTICIDE RESIDUES IN FOOD
TECHNICAL REPORT SERIES 592
REPORT OF THE 1975 JOINT FAO/WHO MEETING

World Health Organisation, Geneva, 1976 pp45, Annexures 3, price not stated.

This booklet contains the report of the 1975 Joint Meeting of the FAO Working Party of Experts on Pesticide Residues and the WHO Expert Committee on Pesticide Residues held in Geneva from 24 November to 3 December 1975.

To meet the increasing need for food there has been a worldwide increase in the use of pesticides in agriculture. Even when applied in accordance with good agricultural practice pesticides sometimes leave residues in food, and man is exposed to them.

This report provides toxicological evaluations aimed at establishing acceptable daily intakes for man and recommends limits of certain pesticide residues in specific foods. The recommendations provide expert guidance for those attempting to control the agricultural use of pesticides. Evaluation of the hazards of pesticides contribute to the health protection of man from the pollution of the general environment by chemicals.

Annexure 1 lists in convenient form recommended acceptable daily intakes and residual limits for a series of pesticides in various foods including poultry and red meat, eggs, fat, milk, milk products and meat products. This makes the booklet invaluable for those interested in the safety of food of animal origin.

L.W. v.d. H.

Jl S. Afr. vet. Ass. 48(1) 41 – 43 (1977)

THE ISOLATION OF *BRUCELLA ABORTUS* BIOTYPE I FROM AFRICAN BUFFALO IN THE KRUGER NATIONAL PARK

D.V. GRADWELL*, A.P. SCHUTTE**, C.A.W.J. VAN NIEKERK* and D.J. ROUX**

ABSTRACT: Gradwell D.V.; Schutte A.P.; Van Niekerk C.A.W.J.; Roux D.J. **The isolation of *Brucella abortus* biotype I from African buffalo in the Kruger National Park.** *Journal of the South African Veterinary Association* (1977) **48** No. 1, 41 – 43 (En) Vet. Invest. Centre, Box 12, 1350 Skukuza, Rep. South Africa.

The isolation of *Brucella abortus* from free living wild African buffalo *(Syncerus caffer)* in the Kruger National Park, South Africa, is described. The four isolates tested proved to be biotype I and it is speculated that the origin of brucellosis in wild animals is from domestic stock.

INTRODUCTION

Indications of infection by *Brucella* organisms in a number of wild animal species has been found by means of serological tests particularly the agglutination test. Guilbride, Coyle, McAnulty, Barber & Lomax[6] found eight of 144 serum samples from hippopotami *(Hippopotamus amphibius)* to give positive agglutination tests for brucellosis and Rollinson[12] found evidence of this disease in game in Uganda. Heisch, Cook, Harvey & De Souza[7] isolated *Brucella* from rodents in East Africa while De Vos & Van Niekerk[5] found 13,7% of hippopotami (51 tested), 14,2% of buffalo (253 tested), one impala *(Aepyceros melampus)* out of 120 tested, and a single waterbuck *(Kobus ellipsiprymnus)* that was tested in the Kruger National Park to show positive reactions to *Brucella* agglutinins in their serum. The latter workers, however, were unable to isolate any organisms from the African buffalo *(Syncerus caffer),* six of which showed hygromata, one a bilateral orchitis and epididymitis and one a purulent endometritis. All these animals were positive reactors to the agglutination test.

Condy & Vickers[3 4], have isolated *Brucella* from a waterbuck and also from two eland *(Taurotragus oryx)* in Rhodesia. These workers also found serological evidence of brucellosis in buffalo, duiker *(Sylvicapra grimmia),* impala, kudu *(Tragelaphus strepsiceros),* sable *(Hippotragus niger)* and zebra *(Equus burchelli).*

MATERIALS AND METHODS

Cotyledons were collected from all pregnant buffalo culled during routine cropping procedures in the Kruger National Park. Two pieces of cotyledon from two separate areas on the membranes were removed immediately after death, placed in sterile bottles and sent to the laboratory at Skukuza unrefrigerated. Cropping took place in the late afternoon and on arrival at the laboratory the specimens were refrigerated and processed the following morning. From each cotyledon sample an impression smear was made, fixed briefly in a flame and stained by a modified Ziehl-Neelsen technique[14]. These were examined for typical intracellular colonies of *B. abortus* under a light microscope. A fresh surface of each specimen was also flamed in alcohol, exposed by means of a sterile blade and smeared on to the surface of selective media described by Morgan, McKinnon, Gill, Gower & Norris[11]. The plates were incubated at 37°C for three days in an atmosphere containing 10 to 15% carbon-dioxide. During the latter part of the experiment Tryptose Soy Agar was used to culture the organisms. Smears were made from *Brucella*-like colonies which appeared and stained using the same modified Ziehl-Neelsen (ZN) staining technique and were considered positive if morphology and colour were typical for *B. abortus.* In each case where a positive culture was obtained the impression smears were also found to be positive and therefore during the later phase of the experiment only cotyledons showing positive ZN smears were cultured for *Brucella.*

For confirmatory identification the four isolates obtained were subjected to tests advocated by Alton & Jones[1] and the WHO/FAO Expert Committee on Brucellosis[2], namely (1) sensitivity to basic fuchsin and thionin dye; (2) sensitivity to penicillin and erythritol; (3) phage typing using $10^3 \times$ RTD of the Tbilisi phage; (4) agglutination with monospecific serum to *B. abortus* and *B. melitensis;* (5) production of hydrogen sulphide; (6) aerobic growth. These tests were also performed on the following reference strains: *B. abortus* 544, *B. melitensis* M 6012, *B. suis* 1330, *B. canis* 666/RM, *B. abortus* S 19, *B. melitensis* Rev I, *B. abortus* 45/20 and *B. ovis* AH/1655/11.

All isolates and reference strains were in addition subjected to immunofluorescence studies using specific conjugates for *B. abortus* and *B. ovis,* prepared according to the method suggested by Schutte[13] for *Campylobacter fetus.*

RESULTS

A total of 136 cotyledons from 68 pregnant buffalo were tested and positive smears and cultures were obtained from four animals; one animal showed positive smears but negative cultures.

Two of the positive animals were from the same herd and in these cases as well as one other, no macroscopic abnormalities could be detected in the foetus, fluids, membranes or uterus. The fourth case, however, indicated that the foetus had been dead before the cow was shot and externally, the uterus appeared normal. On sectioning the uterine wall, the escaping allantoic fluid was seen to be cloudy and tainted brownish-red. The allanto-chorionic membrane was greyish and less transparent than normal and the foetal cotyledons separated easily from the maternal caruncles, which both looked dull yellow. The foetus was ± 50 cm long from crown to rump and was not opened for post-mortem examination.

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Table 1: GENERAL DIFFERENTIAL CHARACTERISTICS OF *BRUCELLA* ORGANISMS

Brucella isolate	Growth on media containing							Direct immunofluorescence		Phage typing $10^3 \times$ RTD	Monospecific sera		H_2S production	Aerobic growth
	Basic Fuchsin		Thionin			Penicillin	Erythritol	*B. abortus* conjugate	*B. ovis* conjugate		*B. abortus*	*B. melitensis*		
	11	111	1	11	111									
AH/660/1	+	+	–	–	–	+	+	3+	0	–	3+	0	(+)	–
AH/660/2 Buffalo	+	+	–	–	–	+	+	3+	0	–	3+	0	(+)	–
AH/660/3 isolates	+	+	–	–	–	+	+	3+	0	–	3+	0	(+)	–
AI/124	+	+	–	–	–	+	+	3+	0	–	3+	0	(+)	–
AH/1472/2 Rhodesian waterbuck	+	+	–	–	–	+	+	3+	0	–	3+	0	(+)	–
B. abortus – 544	+	+	–	–	–	+	+	3+	0	–	3+	0	(+)	–
B. melitensis – M 6012	+	+	+	+	+	+	+	3+	0	–	0	3+	(–)	+
B. suis – 1330	–	–	+	+	+	–	+	3+	0	–	3+	0	(+)	+
B. canis – 66/RM	–	–	+	+	+	–	+	0	3+	+	0	0	(–)	+
B. abortus – S 19	+	+	–	–	–	–	–	3+	0	+	3+	0	(+)	+
B. melitensis – Rev 1	–	–	–	–	–	–	+	3+	0	–	3+	0	(+)	+
B. abortus – 45/20	+	+	–	–	–	+	+	0	3+	+	0	0	(+)	+
B. ovis – AH/1655/11	+	+	+	+	+	+	+	0	3+	–	0	0	(–)	–

Concentrations: 1 = 1:100 000; 11 = 1: 50 000; 111 = 1: 25 000

+ = growth; – = no growth; (+) = H_2S positive; (–) = H_2S negative

3+ = intensity of fluorescence; 0 = no fluorescence

The results of the tests performed on the four isolates are shown in Table 1. All four isolates were found to be identical and classified as *B. abortus* biotype I. The Rhodesian isolate from a waterbuck proved to belong to the same biotype.

DISCUSSION

De Vos & Van Niekerk[5] stated that over a period of 6 months 36,4% of the buffalo cows, in an area where serological evidence of brucellosis was present, had calves running with them. Owing to the inevitably high post-natal mortalities associated with predators in a large game reserve brucellosis was considered to have a negligible effect on the reproduction of buffalo in the Kruger National Park. This is also borne out to a large extent by the fact that despite heavy cropping of buffalo in the Park, aerial censuses show an annual increase in buffalo numbers.

The isolation of *B. abortus* from the membranes of a dead foetus, however, shows that brucellosis must have some effect on the reproductive potential. The foetus under consideration would probably have been aborted at a later date. The effect of brucellosis on a free living buffalo population would probably be to give an apparent later maturing age for females, as a result of the loss of the first or even the second calf. Abortions are seldom if ever noticed where predators and scavengers are prevalent and so the first calf at foot could easily be the result of a second or third pregnancy.

The great importance of brucellosis in buffalo, however, lies in the possibility of dissemination to other game and domestic species. It is well known that buffalo herds wander over extremely large areas and the contamination of these areas will readily occur. It has also been shown that the organism may survive for a considerable time under controlled conditions in nature.[8] This aspect of the disease will have to take prime consideration in any brucellosis eradication scheme in a country where buffalo are prevalent.

Meyer[9 10] states that the original *Brucella* from which all other *Brucella* strains evolved was *B. abortus*, biotype II. It is interesting to speculate about the origin of brucellosis in free living animals and the fact that all four strains tested from bufallo in the Kruger National Park were biotype I and not biotype II seems to indicate that buffalo are not the original reservoir of brucellosis. From this it would seem that buffalo originally obtained the disease from domestic stock and may now reinfest susceptible cattle.

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BOOK REVIEW

BOEKRESENSIE

INTERNATIONAL HISTOLOGICAL CLASSIFICATION OF TUMOURS OF DOMESTIC ANIMALS PART 2

Bulletin of the World Health Organization, Volume 53, No. 2-3, Pages 137-304, 1976

World Health Organization, Geneva. Pp 167. Numerous figures. Price: about Sw.fr. 36

The need for a standard classification and nomenclature of neoplasms of domestic animals has long been needed for obvious reasons. This hiatus in veterinary science has now been filled by two publications of the World Health Organization. The first to appear in this series was Part 1 which appeared in Vol. 50, No. 1-2, Pp. 1-142, 1974 of the Bulletin of the World Health Organization and covered the classification of tumours of 10 body sites. It will require no introduction to those concerned with this particular aspect of veterinary science. Part 2, the volume under review, completes the series and contains the contributions of 27 collaborators on the classification of tumours of 11 body sites, *viz.:* upper alimentary tract; lower alimentary tract; liver and biliary system; pancreas; ovary; female genital tract; adrenal gland and paraganglia; kidney; prostate and penis; nasal cavity; and, bones and joints. The species included are the horse, ox, sheep, pig, dog and cat.

The style and format of Part 2 are very similar to those of the first part. Each section includes an introduction, tabulated classification and histological description of individual tumours which is exemplified by numerous photographs. The authors and editors are to be congratulated on the excellent quality of the latter. References have been kept to a minimum since the publication is not intended to serve as a text book.

This book is a "must" for all those concerned with veterinary pathology, comparative oncology and the teaching of veterinary oncology.

R.C.T.

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AN INACTIVATED RIFT VALLEY FEVER VACCINE

B.J.H. BARNARD* and M.J. BOTHA*

ABSTRACT: Barnard B.J.H.; Botha M.J. **An inactivated Rift Valley fever vaccine.** *Journal of the South African Veterinary Association* (1977) **48** No. 1, 45 – 48 (En) Vet. Res. Institute, 0110 Onderstepoort, Rep. of South Africa.

The immunising potency of an inactivated Rift Valley fever (RVF) vaccine prepared from RVF virus infected mouse brain and RVF infected cell culture was studied in cattle and sheep. Different doses and adjuvants were compared. In laboratory trials both cattle and sheep developed neutralising antibodies against virulent RVF virus and in cattle antibodies were still detectable 9 months after immunisation. Although the immunity produced was inadequate to prevent viraemia after challenge, evidence of protection against clinical RVF was obtained. Field trials in sheep showed that vaccination induced a good immunity in pregnant ewes.

INTRODUCTION

During the summer of 1950-1951 Rift Valley fever (RVF) made its appearance for the first time in South Africa in the northern Cape, the western Orange Free State and in the southern Transvaal[5]. Subsequently there were less serious but confirmed outbreaks during 1952, 1953, 1955, 1957, 1958 and 1959, while unconfirmed outbreaks were reported in 1956 and 1967[4]. During 1970 and 1971 no outbreaks of RVF were reported but the virus was isolated from mosquitoes[7]. Outbreaks of the disease usually occur when climatic conditions favour the breeding of large numbers of mosquitoes. The widespread incidence of the disease during 1974-1975 after an exceptionally wet and rainy season, is clearly reflected in the large number of cases of the disease which were confirmed.

Fig. 1 shows the extent of the outbreaks of RVF during this period. The increased demand for RVF vaccine also indicated the severity of the outbreaks of RVF during the 1975 season. From 1952 to 1973 only 10 million doses were issued, compared with 18 million doses during the period November 1974 to May 1975[12].

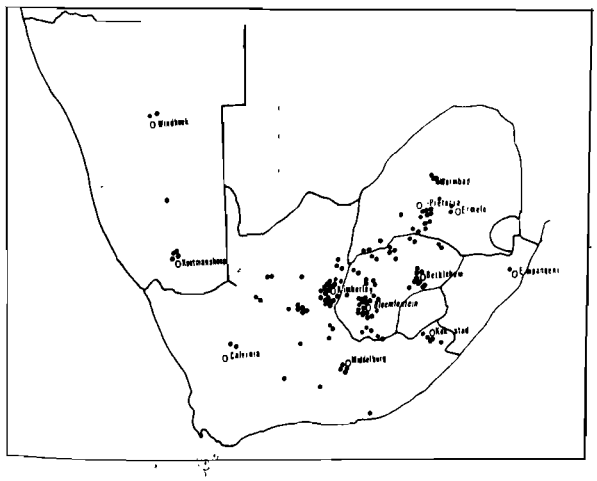

Fig. 1 Confirmed outbreaks of RVF from January 1974–July 1975.

When RVF made its appearance in 1950-1951, a vaccine was not available and stock losses were estimated at 100 000[10]. Although Smithburn's[11] attenuated strain of virus was available at the time, the difficulties encountered in the preparation of sufficient stocks of vaccine in mice at that time prompted Alexander to say: "This summer we may have to sit back and watch the disease take its toll of animals and man"[1]. Nevertheless, during 1952 more than 100 000 doses of RVF vaccine were prepared in mice, using Smithburn's neurotropic strain of virus at the 102nd intracerebral mouse passage. Since this vaccine produced abortions when used in pregnant ewes, it was further attenuated by an additional 50 passages in embryonated hens' eggs and 16 passages in mice after which it was used for the preparation of vaccine from 1953 onwards. In 1958, however, it was conclusively shown that the immunogenicity of vaccine produced with this attenuated strain of virus was inferior[14]. Consequently Smithburn's neurotropic strain was again used for vaccine production. From 1971 onwards, vaccine was prepared from the same strain cultured in cell culture.

Although Smithburn's strain produced a good immunity in sheep, it was not very effective in cattle[6]. Coackley, Pini & Gosden reported that the neutralizing antibody of the sera of cattle to Smithburn's neurotropic strain of RVF virus was low[2 3]. In sheep either a high or low response may occur[3]. As a result of the abortifacient property of Smithburn's strain and its low immunogenicity for cattle, an inactivated vaccine was developed. This paper deals with certain laboratory and field tests carried out in the development of this vaccine.

MATERIALS AND METHODS

Virus

A field strain of RVF virus, isolated from a cow, was passaged six times in mice by the intracerebral (i.c.) route and then freeze-dried in volumes of 0,5 ml as a 10% suspension of infective mouse brain in a phosphate buffer containing 10% peptone and 5% lactose (BLP). The freeze-dried virus was stored at 10°C and used as seed virus for the inactivated mouse brain vaccine and as antigen for serum-virus neutralisation tests. This seed virus was also adapted to cell culture by 2 serial passages in roller tubes of BHK 21 cells maintained in Hank's medium without serum. At the second passage a clear cytopathic effect was obtained in 48 hours. This virus suspension was mixed with an equal volume of BLP and freeze-dried in volumes of 0,5 ml stored at 10°C and used as a seed virus vor the preparation of inactivated cell culture vaccine. Live mouse brain and cell culture vaccines were prepared from Smithburn's neurotropic attenuated strain at the 102nd intracerebral mouse passage.

For the challenge of immunity, sheep were injected subcutaneously with 1,0 ml infective sheep blood. The

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blood was obtained from an experimentally infected sheep, bled at the height of the febrile reaction and it had an infectivity titre of 10^7 mouse LD_{50}. The blood was collected in 1% citrate and stored at 10°C.

Experimental animals

Locally-bred albino mice were used for serum-virus neutralisation tests, virus titrations and the preparation of vaccine.

Young Afrikaner type cattle held under field conditions in a RVF free area, were used for immunity studies, while adult Merino sheep were used for field experiments and laboratory tests.

Serum-virus neutralisation tests

Blood was collected, where applicable, before and at various intervals after inoculation. Using the sera so obtained, neutralisation tests were carried out according to the constant serum, virus dilution procedure. Serial tenfold dilutions of virus were prepared in BLP and a unit volume of each dilution was mixed with an equal volume of undiluted serum and incubated at 37°C for 1 hour. Each serum-virus mixture was injected into two families each consisting of seven, two-day-old mice, each mouse receiving 0,03 ml intraperitoneally. Deaths were recorded from the 2nd to the 7th day. The endpoints were calculated according to the method of Read & Muench[9] and the neutralisation indices were expressed as $\log_{10}$ Mouse LD_{50} of the virus neutralized.

Virus assay

In the laboratory experiments conducted in sheep, blood was collected daily after challenge and assayed for virus.

The virus suspensions used for the preparation of the different vaccines were also assayed for infectivity before inactivation. Serial tenfold dilutions of these samples were prepared in BLP and 0,03 ml of each dilution was injected i.c. into each of two families of two-day-old mice. Deaths were recorded from the 2nd to the 7th day. The endpoints were calculated and expressed as $\log_{10}$ Mouse LD_{50}/ml.

Preparation of vaccines

(a) The inactivated vaccines were prepared from infected mouse brain and in cell culture.

Three-week-old mice were injected i.c. with 0,03 ml of a 1:1 000 dilution of seed virus in BLP. When 15% to 20% of the mice showed nervous symptoms, all mice were killed with ether and their brains collected aseptically. A 10% brain suspension in BLP was prepared and titrated i.c. in mice.

Seed virus was inoculated into roller bottles of BHK 21 cells maintained in Hank's medium without serum. When approximately 75% of the monolayer showed cytopathic changes, the culture was harvested and diluted with 9 parts BLP. The diluted mouse brain and cell culture suspensions each with an infectivity titre of at least 10^7 Mouse LD_{50}/ml was inactivated separately by the addition of formalin to a final concentration of 0,2%. After incubation at 37°C for 12 hours without agitation the suspensions were tested for the absence of infectious virus by injecting 0,03 ml i.c. into each of two families of baby mice and 0,5 ml intraperitoneally into 10 three-week-old mice. The mice were observed for 7 days and the vaccine considered safe if no deaths as a result of RVF occurred. Brain tissue of any mice that died was passaged in baby mice to determine the presence of RVF virus. The unfiltered inactivated virus suspensions were then mixed with the following adjuvants as required.

Aluminium hydroxide ($Al(OH)_3$) adsorbed vaccines: Either mouse brain or cell culture suspensions were mixed with an equal volume of Alhydrogel*.

The oil adjuvant vaccine consisted of an emulsion of 30% mouse brain suspension, 2% Lissapol**, 8% Lubrol** and 60% Bayol F* as mineral oil.

(b) The live mouse brain vaccine was prepared as described previously[13]. The live tissue culture vaccine was prepared by seeding roller bottles of BHK 21 cells with Smithburn's neurotropic strain. Both live vaccines were freeze-dried and reconstituted to ensure that each animal received at least 10^4 Mouse LD_{50} per ml. This is the standard accepted for the Onderstepoort live RVF vaccine.

RESULTS

The immunogenicity of inactivated vaccines in cattle

Three groups of six cattle each were injected subcutaneously with 2 ml and 4 ml Al $(OH)_3$ vaccine and 2 ml oil emulsion vaccine respectively. The mean antibody response of the three groups of animals is indicated in Fig. 2. There appears to be little difference in the degree and duration of the antibody response. The antibody concentration reached a peak between 2 and 3 months depending on the vaccine and then gradually declined. After a period of 9 months, antibodies were still detectable in all the vaccinated cattle.

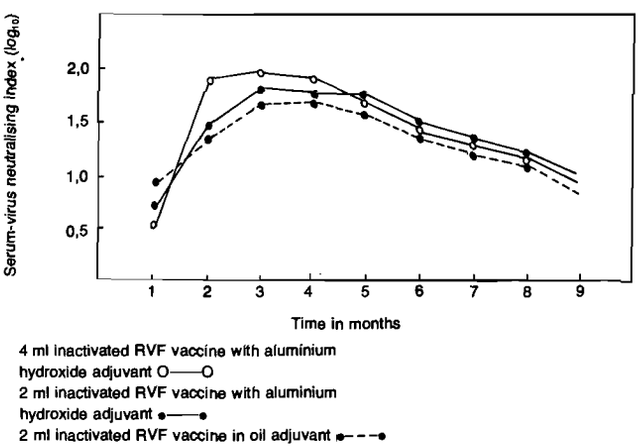

Fig. 2. The immunogenicity of three inactivated R.V.F. vaccines in cattle.

The effect of a booster dose

Eight cattle were each injected subcutaneously with 2,0 ml of the inactivated mouse brain vaccine with aluminium hydroxide adjuvant. After 3 months four cattle received a second injection of the same vaccine. The mean antibody response of the 2 groups of animals is presented in Fig. 3. The booster dose given at 3 months, did not significantly affect the antibody response, although slightly higher levels of antibody persisted over a period of 4 months in those animals which received a booster injection.

A comparison of live and inactivated vaccines

Four groups of sheep were used to compare the immunogenicity of four different vaccines, namely a live

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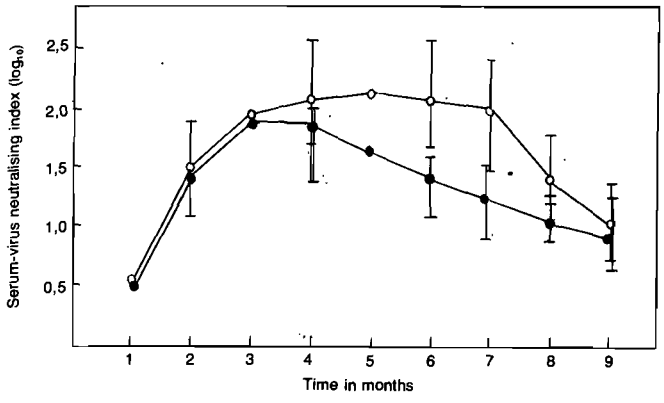

Primary dose of inactivated RVF vaccine •——•
Primary dose of inactivated RVF vaccine followed by a booster dose after 3 months O——O

Fig. 3 The effect of a booster dose 3 months after administration of a primary dose of inactivated R.V.F. vaccine with aluminium hydroxide adjuvant in cattle.

and inactivated mouse brain vaccine as well as a live and inactivated cell culture vaccine. $Al(OH)_3$ was used as adjuvant in the case of the inactivated vaccines.

Blood was collected from the sheep 17, 25 and 32 days after administration of the vaccines, the serum neutralizing antibody indices determined and their immunity was challenged on the 32nd day. Four sheep were included as controls.

It is clear from Table 1 that sheep which had been injected with the live mouse brain vaccine developed higher neutralizing antibody indices than the other groups. The group injected with the inactivated mouse brain vaccine, developed only low neutralizing indices. Within the groups themselves there was a marked variation in antibody response. Some sheep, viz. 2089, 2084 and 2097, had higher neutralizing antibody indices as early as the 17th day after inoculation, while others, 2104, 2105 and 2101 first showed a significant antibody response on the 32nd day. In spite of the fact that 2104 and 2065 had neutralizing antibody indices of 1,5 and 3,5 respectively, an infectivity titre of 0,6 MLD_{50} of challenge virus could be demonstrated on the 3rd day after challenge. On the other hand, sheep 2067, 2098 and 2094 with neutralizing antibody indices of less than 1,0 developed no viraemia after challenge.

The immunised sheep showed little or no clinically significant reactions after challenge and only two sheep, viz. 2091 and 1072, which had received the live cell culture vaccine, developed a transient febrile reaction. In contrast, all the uninoculated control sheep showed febrile reactions, anorexia and high concentration of circulating virus after challenge.

Vaccination during an outbreak of RVF

During an outbreak of RVF on the farm Palmietfontein in the Boshof district, 437 Merino ewes in advanced pregnancy were each injected with 2,0 ml of the inactivated mouse brain vaccine with aluminium hydroxide adjuvant. A group of 110 ewes was not inoculated and kept as control animals in the same flock. All abortions, deaths and births were recorded from the 7th to 30th day after inoculation. The mounting number of abortions and deaths among the ewes was

Table 1: THE IMMUNE RESPONSE OF SHEEP TO THE ADMINISTRATION OF 4 EXPERIMENTAL RVF VACCINES

Vaccine	Sheep No.	Neutralizing antibody indices			Temperature and Viraemia after challenge				
		Days after inoculation			Days after challenge				
		17	25	32	1	2	3	4	5
Live mouse brain vaccine	2 089	3,0	3,0	3,5	0*	0	0	0	0
	2 104	<0,5	1,0	1,5	0	0	0,6	0	0
	2 065	<0,5	3,0	3,5	0	0	0	0	0
	2 084	3,0	3,0	3,5	0	0	0	0	0
Live cell culture vaccine	2 091	<0,5	<0,5	<0,5	3,5	0	0T	0	0
	2 072	<0,5	<0,5	<0,5	3,0	0	0T	0T	0
	2 097	2,5	1,5	2,0	0	0	0	0	0
	2 073	1,0	1,5	3,5	0	0	0	0	0
Inactivated mouse brain vaccine with aluminium hydroxide	2 071	<0,5	<0,5	<0,5	3,4	3,6	0	0	0
	2 067	<0,5	<0,5	<0,5	0	0	0	0	0
	2 101	<0,5	<0,5	1,5	0	0	0	0	0
	2 098	<0,5	<0,5	1,0	0	0	0	0	0
Inactivated cell culture vaccine with aluminium hydroxide	2 105	<0,5	<0,5	2,1	0	0	0	0	0
	2 090	<0,5	<0,5	1,0	0	0	0,6	0	0
	2 094	<0,5	<0,5	1,0	0	0	0	0	0
	2 096	2,0	1,0	1,0	0	0	0,5	0,6	0
Challenge controls	647	Not immunised			4,4	0	0,5T	0T	0
	8 109				3,8	6,0	5,1T	0T	0T
	6 202				5,5	7,5	6,5T	3,0T	1,5T
	1 108				5,0	4,5	3,5	0T	<0,5T

*Infectivity titre in baby mice log base10; 0=<0,5
T = temperature elevated above 39,9°C.

expressed as a percentage of the total number of lambs which were born alive. The 7th day was taken as the starting point for observations since it had seemed unlikely that the vaccine could have any effect before that time. From the summary of the results presented in Fig. 4, it is evident that the immunized ewes and their lambs were protected against the disease since fewer than 3% died, compared with 50% losses amongst the control ewes and 31% amongst their lambs. Only 11% of the immunized ewes aborted, compared with 37% of the control ewes.

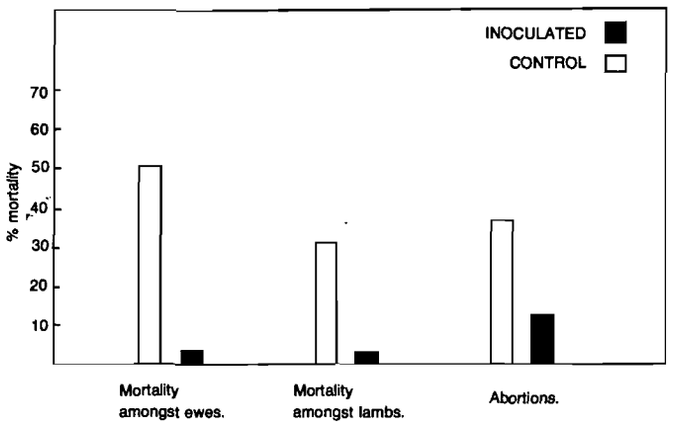

Fig. 4 Abortions and mortalities amongst ewes and lambs in inoculated and controlled ewes during a natural outbreak of Rift Valley fever.

DISCUSSION

The presence of antibodies as determined by the serum neutralization test in mice has been extensively used as a measure of immunity in RVF[13]. Work carried out by Coackley, Pini & Gosden[2 3], however, showed that sheep with viraemia developed high neutralizing antibody indices, whereas only low neutralizing antibody indices were found in sheep without preceding detectable viraemia. Nevertheless, they remained immune for at least 2 years.

From the results reported in this paper it is evident that although there is a considerable individual variation in the antibody response to different vaccines, live RVF vaccines in general produce a higher neutralizing antibody index than the inactivated vaccines. Despite the low serum neutralizing index produced in sheep after administration of inactivated vaccines, most of the sheep were able to resist natural as well as artificial infection with RVF virus. On the other hand, two sheep 2104 and 2065 with a high neutralizing index developed a viraemia after challenge. This observation confirms the finding of Pini, Lund & Davies[8] in that the serum virus neutralization test is insufficiently sensitive and/or that factors other than neutralizing antibodies play a role in resistance against RVF infection.

The value of an inactivated RVF vaccine is reflected in the results obtained in the field experiment. Pregnant ewes could be injected without the risk of complications attending the administration of a live vaccine. Although the involvement of other viruses could not be excluded, the abortion rate in the immunized sheep was considerably lower than in the non-immunized sheep. The reason for the relatively high abortion rate of 11% in the immunized sheep can possibly be attributed to the fact that viraemia can still occur in immunized sheep, despite the presence of circulating neutralizing antibodies as was the case in sheep 2104 and 2065. If this virus passes through the placenta, as often occurs, death of the foetus and abortion may result.

Neutralizing antibodies could be shown to persist in cattle for at least 9 months after administration of the inactivated vaccine and there is every reason to believe that the immunity is sufficient to protect cattle for at least one season. Annual immunisation before the beginning of the rainy season should thus suffice to protect sheep and cattle against clinically detectable RVF during the period of risk during the ensuing summer and autumn, but some abortions may still be expected.

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THE EFFECT OF SAMPLE SIZE AND CULTURE METHOD ON THE RECOVERY OF *SALMONELLA* spp. FROM NATURALLY CONTAMINATED CARCASE MEAL

L.W. VAN DEN HEEVER* and H.N. VAN DER MADE**

ABSTRACT: Van den Heever L.W.; Van der Made H.N. **The effect of sample size and culture method on the recovery of Salmonella spp. from naturally contaminated carcase meal.** *Journal of the South African Veterinary Association* (1977) **48** No. 1, 51 – 52 (En) Fac. Veterinary Science, Univ. of Pretoria, Box 12580, 0110 Onderstepoort, Rep. of South Africa.

The results of parallel examination by two methods of 223 consignment samples of carcase meal were compared. Selective pre-enrichment of 5 g of sample prior to plating on to a solid media disclosed that 2,7% of consignments were contaminated with *Salmonella*. Non-selective pre-enrichment of 100 g followed by selective enrichment prior to plating found 21,5% of the consignment to contain *Salmonella*.

INTRODUCTION

Carcase and other meals derived from animal by-product rendering plants are generally required to be free from pathogenic micro-organisms so that they may safely be used for compounding livestock feed and licks. For the purpose of safeguarding animal health the primary emphasis is usually placed on the absence of pathogenic sporeformers such as *Bacillus anthracis* and *Clostridium* spp.

Because the time-temperature relationships obtained in the rendering process is designed *inter alia* to destroy the spores of these organisms, the presence of non-spore formers in the final product is generally regarded to be the result of post-sterilising recontamination. Such contamination may be of considerable magnitude. Keil & Keller[1] examined 480 bacterial organisms from 78 samples of animal meals: 29,5% were Gram-positive cocci; 28,2% were Gram-positive rods and 42% were Gram-negative rods; total counts varied from 9 000 to 2X 10^{10}/g meal. The presence of a large proportion of the Enterobacteriaceae in such meals indicates considerable faecal contamination.

Numerous publications have appeared concerning the incidence and significance of *Salmonella* in carcase and other meals[6]. Van Keulen[8] has reported on the significance of salmonellosis as a zoonosis. Surveys in various parts of the world indicate the incidence of *Salmonella* contamination of animal meals to vary from 0,65% of samples examined to as high as 92%. On examining 35 to 50 gram samples in a nation wide survey in the United States of America, Moyle[4] found that 12,57% of meals contained *Salmonella*. Tittiger[7] examined 3 to 5 samples from 180 batches of animal meal from five Canadian plants: *Salmonella* contaminated meals emanated from four of the plants.

Lee[2] has reported on the incidence of *Salmonella* in feeds containing animal meals in England and demonstrated the positive epidemiological correlation of the presence of these organisms in animal feeds in relation to the cause and incidence of human salmonellosis following ingestion of food of animal origin.

Morehouse and Wedman[3] emphasize the need for standardisation of examination procedures designed to recover *Salmonella* from carcase and other animal meals. In view of the tonnage of meals produced and handled at most rendering plants, it may be expected that the magnitude of recontamination would be of a rather low order. As the moisture content of the meals is usually in the vicinity of perhaps 10%, significant growth of the recontaminating bacteria is unlikely. The availability of a series of representative random samples of carcase meal from various rendering plants presented on opportunity for comparing two methods of examination of the samples. The results are the subject of this report.

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MATERIALS AND METHODS

The samples were drawn by means of a sterile graduated tubular sampling device inserted into the core of the meal from the top of the opened bag. Aseptically compounded samples consisting of a minimum of 500 g were submitted in sterile containers to the laboratory.

After thorough mixing of the contents of the sample container the meal sample from each consignment was examined by means of both the following two methods:

Method I:

100 g was suspended in 1 ℓ of sterile isotonic peptone water buffer (IPWB)[5] of pH 7,0 brought to room temperature and magnetically stirred during incubation at 37°C for 6 to 8 h. The suspension was then allowed to stand at room temperature for 20 minutes and the supernatant transferred to 80 ml tubes for centrifugation at 3 000 rpm for 30 minutes. From each tube 10 ml of the supernate was mixed with the sediment, and one drop of the mixture was transferred to each of the following enrichment broths:

(a) 5 ml Stokes and Osbornes modified selenite brilliant green mannitol enrichment broth (SO)[5]; and
(b) 5 ml Müller – Kaufmann's modified tetrathionate brilliant green bile enrichment broth (MK)[5]

The inoculated enrichment broths were incubated at 37°C for 18 to 24 h, after which 0,01 ml quantities of each broth were plated on to each of the following solid media:

(i) Salmonella-Shigella agar (SS); and
(ii) Taylor's xylose lysine decarboxy cholate agar (T)[5]

The plates were incubated at 37°C for 18 to 24 h. Non-lactose fermenting colonies were transferred to Triple Sugar Iron (TSI)[5] agar slants. Cultures from slants showing the characteristics of growth of *Salmonella* after 18 to 24 h at 37°C were tested for lactose and sucrose fermentation and production of urease. Suspect cultures were tested against polyvalent "O" antiserum and finally submitted to Dr C.M. Cameron of the Salmonella Reference Centre, Veterinary Research

Institute, Onderstepoort for serotyping.

Method II:

Five g of meal and 1,9 g of dehydrated brilliant green broth (Difco) were suspended in 20 ml of sterile phosphate buffer (pH 7,0); after incubation at 37°C for 48 h a loopful of broth was plated on to SS agar plates which were again incubated at 37°C for 18 h to 24 h. Colourless lactose negative colonies were transferred to TSI[5] slants and suspect cultures were submitted for final identification and serotyping as above.

RESULTS

By use of Method I, 48 (21,5%) of 223 consignment samples of carcase meal yielded *Salmonella* spp. Method II revealed only 6 (2,7%) samples to be so contaminated. Three of the 6 samples which were positive to *Salmonella* according to Method II failed to yield the organisms after examination by Method I. Combined use of both methods disclosed that 54 (24,2%) of the 223 samples contained *Salmonella.*

Comparison of the two enrichment broths used in Method I revealed that sole use of MK-broth failed to identify five contaminated meal samples. Conversely, sole use of SO-broth failed to identify 24 contaminated meals.

All but three of the 48 *Salmonella* contaminated meal samples yielded a single serotype. Two samples, obtained on separate occasions from one particular plant, each yielded *S. thompson* and *S. marylebone.* A sample from another plant proved to be contaminated with both *S. dublin* and *S. friedenau.*

Whereas Method I involved the parallel use of two enrichment broths and plating onto two types of solid media, the results were not identical. Comparison of the recovery of *Salmonella* from the respective solid media reveals the following:

1. After enrichment in MK-broth, 43 samples yielded *Salmonella* on one or both solid media. There was no growth, however, on SS agar in 10 instances and no growth on Taylor agar in seven.
2. After prior enrichment in SO-broth, 22 samples were found to yield *Salmonella* on one or both types of solid media. In this case, four of the positive samples failed to yield the organism when plated on SS agar and eight samples were negative on Taylor agar.

The frequency of isolation of the various *Salmonella* serotypes isolated from contaminated meal samples may be summarised as follows:

METHOD I:			
S. oranienburg	**9**	***S. rotterdam***	**3**
S. dublin	**5**	***S. marylebone***	**2**
S. typhimurium	**5**	***S. gwaai***	**1**
S. thompson	**8**	***S. senftenberg***	**1**
S. kinshasa	**5**	***S. anatum***	**1**
S. virchow	**3**	***S. ? (rough)***	**1**
S. lexington	**1**	***S. berta***	**1**
S. friedenau	**1**	***S. pretoria***	**1**
METHOD II:			
S. oranienburg	**2**	***S. zega***	**1**
S. thompson	**2**	***S. ? (rough)***	**1**

S. oranienburg and *S. thompson* were recovered by both Methods I and II in all samples from which these organisms were isolated.

DISCUSSION AND CONCLUSIONS

The advantages of non-selective preliminary enrichment in buffered peptone water of 100 g of sample over direct selective enrichment of 5 g of meal are clearly demonstrated by the fact that the former identified nearly 22% of the series of samples as *Salmonella* contaminated whereas only about 3% were disclosed by the latter method and 24% by combined use of both methods. In view of the fact that the order of magnitude of *Salmonella* recontamination of sterilised carcase meal is probably rather low in most instances, this result is not unexpected. It does however, emphasize that regardless of the eventual method of enrichment and cultural examination employed, a relatively large sample is necessary if the results of any bacteriological monitoring system are to be meaningful.

Standard method 759 of the S.A. Bureau of Standards[5] recommends examination of 20 g for detection of the presence of viable *Salmonella* organisms in food for human use. Such foods usually have a rather high water content and multiplication of contaminating organisms in the food is during storage accordingly possible. The water content of carcase meal varies from 6 to 25%, average 13,5% (unpublished data). Such low A_w-values do not favour bacterial growth, and examination of a larger amount of carcase meal appears to be justified.

Although not statistically evaluated it is clear that secondary selective enrichment in Müller-Kaufmann broth yielded measurably better results than did the use of Stokes-Osborne broth. Although not greatly so, SS-agar more frequently yielded *Salmonella* from contaminated meals in this investigation than did Taylor-agar.

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AN ABATTOIR SURVEY OF HELMINTHS IN CATTLE IN SWAZILAND*

J.R. MITCHELL

ABSTRACT: Mitchell J.R. **An abattoir survey of helminths in cattle in Swaziland.** *Journal of the South African Veterinary Association* (1977) **48** No. 1, 53 – 54 (En) Box 241, 1740 Krugersdorp, Rep. of South Africa.

In an abattoir survey involving 5 886 bovine carcasses the incidence of helminths which are responsible for condemnation of meat was determined. The prevalence of helminths according to the origin of carcasses is summarised and discussed.

INTRODUCTION

Helminth infestation in cattle results in losses to the meat industry not only through reduced productivity but also through condemnation of carcasses or parts thereof at the abattoir.

A survey was conducted at the Swaziland Meat Corporation Abattoir to determine the incidence of those infestations which result in meat condemnation. Besides *Cysticercus bovis,* hydatid cysts and *Fasciola* spp. infestation, *Oesophagostomum radiatum* was included, because of the lesions it causes in the intestines. *Cordophilus saggitus,* which has recently been recorded in Swaziland[3], was also included, because of the heart lesions it may cause.

MATERIALS AND METHODS

At the abattoir the routine meat inspection procedures adopted in the Republic of South Africa were followed. The hearts, however, were examined more thoroughly for the presence of *C. saggitus.*

The 5 886 carcasses included in the survey were grouped according to origin. These groups were: (1) Swazi owned cattle from various kraals in different geographical locations; (2) Cattle from Government Holding Grounds (fattening ranches); (3) European owned cattle; (4) Cattle from ranches of the Swaziland Meat Corporation; (5) Cattle imported from the Republic of South Africa.

The latter group was included for comparative purposes and also because all were derived from a feedlot in the highveld of the Transvaal. This group was fed for approximately 3 months on maize, molasses and poultry droppings.

The prevalence of *C. bovis* was recorded from the following sites: masseter muscles, heart and carcass incisions which included shoulder muscles and diaphragm. The findings were grouped as: (a) one to five cysts; and (b) more than five cysts. The presence of *C. bovis* in liver, lungs and other sites was ignored as having no bearing on records of prevalence, and because *C. bovis* does not normally appear in these sites[1].

Hydatid cysts were recorded from lungs and livers. No attempt was made to differentiate between *F. hepatica* and *F. gigantica,* evidence of infestation being simply recorded as fascioliasis. The lesions of "pimply gut" in the intestines were accepted as evidence of infestation with *O. radiatum.*

A special attempt was made to find *C. saggitus* in the heart, but because of difficulties in locating this helminth no claim is made for the absolute accuracy of these findings.

The survey proceeded throughout the year whenever material from any particular group of cattle was available.

Table 1: THE PREVALENCE OF HELMINTHS IN 5 886 BOVINE CARCASSES

C. bovis			Hydatid cysts		*O. radiatum*	*Fasciola* spp.	*C. saggitus*
Total	1–5 Cysts	>5 Cysts	Lungs	Liver			
13,75%	11,20%	2,55%	10,82%	0,27%	15,58%	10,26%	0,32%

RESULTS

The prevalence of helminth infestation in all the carcasses examined is summarised in Table 1, while Table 2 summarises the data according to the origin of the carcasses.

Table 2: PREVALENCE OF HELMINTH INFESTATIONS IN CARCASSES FROM DIFFERENT SOURCES

Origin of cattle	*C. bovis*			Hydatid cysts		*O. radiatum*	*Fasciola* spp.	*C. saggitus*
	Total	1–5 cysts	>5 cysts	lungs	liver			
Swazi cattle (1704)	16,25%	13,20%	3,05%	11,30%	0,12%	19,07%	16,26%	0,59%
Swaziland Meat Corporation (1625)	15,14%	12,12%	3,02%	13,42%	0,68%	17,23%	11,51%	0,49%
European owned cattle (533)	10,13%	8,07%	2,06%	8,63%	0,0 %	8,26%	7,05%	0,0 %
Government Holding Grounds (447)	15,66%	13,42%	2,24%	11,86%	0,45%	17,23%	7,16%	0,0 %
Cattle imported from R.S.A. (1577)	10,15%	8,50%	1,65%	7,99%	0,06%	12,11%	8,18%	0,06%

( ) = No. examined

*This survey was conducted while the author was employed as a Veterinary Officer in charge of Meat Hygiene at Manzini, Swaziland.

DISCUSSION

Cysticercosis

Taking into account the origin of cattle the overall prevalence figure differs little from previously recorded findings in Swaziland and in nine other countries in Africa[2].

Hydatid cysts

The highest prevalence was recorded in animals which originated from the ranches of the Swaziland Meat Corporation. A possible explanation is that the infested animals came mainly from one particular ranch, situated in the north eastern region along the border of Mocambique, where there is an abundance of game animals and many hyenas, which may serve as hosts for *Echinococcus* spp. The same reason may apply to the high incidence of hydatid cysts in the livers of cattle from ranches also supporting game animals.

Oesophagostomosis

Oesophagostomum radiatum can be controlled by anthelminthic treatment and the results of the survey show that under systems of husbandry where some form of treatment is practised, the prevalence of infestation appears to be lower. It was ascertained that the cattle from European owned ranches and from the feedlot in R.S.A. had been dosed twice with a broad spectrum anthelminthic.

Fascioliasis

The variation in prevalence is probably due to the geographical location and the type of farming practised in the various areas from which the cattle originated. The dry land pasturage and low rainfall at one of the two Government Holding Grounds reduced the prevalence of *Fasciola* spp. in cattle from this station.

Cordophilosis

Little is known of the effect of *C. saggitus* on carcass quality. The fact that it is seldom recorded in other parts of Africa does not exclude the possibility that it is simply not recognised during routine meat inspection procedures.

The number of cattle included in the survey represents approximately 2 months throughput of the Swaziland Meat Corporation and the loss through condemnation for helminth infestations amounted to: Hydatid cysts – 2 089 kg of lungs and 78 kg of liver; Fascioliasis – 2 470 kg of liver; *O. radiatum* – 12 478 kg intestines.

In addition the 807 carcasses infested with *C. bovis* required refrigeration for 14 days and occupied space which could otherwise have been utilised for the freezer storage of sound meat. Finally such losses can only be tolerated in those establishments with facilities to utilise condemned meat in their by-product plants e.g. pets food, carcass meal etc.

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SCHISTOSOMA MATTHEEI INFESTATION IN THE OX: THE INTESTINAL SYNDROME

J.A. LAWRENCE*

ABSTRACT: Lawrence J.A. *Schistosoma mattheei* **in the ox: the intestinal syndrome.** *Journal of the South African Veterinary Association* (1977) **48** No. 1, 55 – 58 (En) Veterinary Research Laboratory, P.O. Box 8101, Causeway, Rhodesia.

Twenty-eight Friesland calves were infested at 7 to 11 months of age with 5 000-45 000 cercariae of *Schistosoma mattheei.* At 7 to 8 weeks post-infestation the majority developed an acute intestinal syndrome characterised by diarrhoea or dysentery, anorexia and loss of condition, from which they recovered spontaneously. The severity and duration of illness was proportional to the level of infestation. Two heavily infested animals on a low plane of nutrition developed a subacute syndrome in which the initial acute disease was succeeded by prolonged unthriftiness, without diarrhoea, from which recovery occurred when the nutritional status was improved. Both acute and subacute forms of the syndrome were encountered in natural outbreaks.

INTRODUCTION

Schistosoma mattheei Veglia & le Roux (1929) is a common parasite of the ox in Southern Africa, incidence rates as high as 92% having been recorded in cattle in Rhodesia[5]. Clinical schistosomiasis is rare, but a few outbreaks have been reported in recent years in Rhodesia and South Africa[13 16 20]. A comprehensive investigation has been undertaken by Lawrence[12] and this paper presents clinical observations on the intestinal syndrome which follows primary infestation. The syndrome was reproduced experimentally and was also encountered in naturally occurring outbreaks. Clinical pathological, serological and pathological features of the syndrome will be published elsewhere.

EXPERIMENTAL OBSERVATIONS

MATERIALS AND METHODS

Investigations were carried out in 38 Friesland steers reared under conditions of minimal exposure to trematode parasites, of which 28 were infested at 7 to 11 months of age. The animals were fed on coarse hay and a commercial cattle meal and for several weeks prior to infestation the quantity of meal fed was adjusted to provide one of two planes of nutrition; either a low plane which supplied little more than maintenance or a high plane which provided a mass gain of 0,7-0,9 kg/day. These planes were maintained for 18 to 41 weeks after infestation, after which the animals were brought back to a common intermediate plane. The calves were divided into five groups of six to nine animals each, depending on age, time of infestation and plane of nutrition, and each group included two uninfested controls.

Infective cercariae of *S. mattheei* were obtained from aquarium-reared *Bulinus (Physopsis) globosus* snails infested with a strain of parasite originating from cattle at the Salisbury abattoir and maintained thereafter in cattle and snails. Animals were infested with between 5 000 and 45 000 cercariae (24-341 cercariae/kg body mass) through the skin. The number of parasites which became established was proportional to the number of cercariae applied[10].

Animals were monitored daily for rectal temperature, appetite, consistency of faeces and clinical appearance, and body mass was recorded every one or two weeks. Data from the calves in the five groups were pooled to obtain a comprehensive picture derived from a large number of animals. Because of the variation in growth rate between the different groups, and within the same group at different times, it was necessary to relate the mass of the infested calves to that of the controls. The mean change in mass from one observation to the next was calculated for the controls in each group and was regarded as the expected change in the infested calves in the same group. From the initial mass and the expected change the "expected mass" of each infested calf was calculated for each observation, commencing approximately one week prior to infestation. The "actual mass" at each observation was expressed as a percentage of the "expected mass", and this figure was used in all further calculations.

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RESULTS

Clinical Signs During the Prepatent Period

The only clinical sign observed during the prepatent period was a reaction at the site of infestation. In animals in which the site was not pigmented slight reddening of the skin became apparent within 30 minutes of infestation. After 24 hours the reddening was marked but there was no evidence of swelling, heat or pain. Reddening was less marked at 48 hours and had usually disappeared by 72 hours. There was no evidence of irritation during the period of infestation and only occasional evidence of slight irritation during the following 24 hours.

Clinical Disease

Eggs were first detected in the faeces 6 to 7 weeks after infestation and the first sign of clinical illness, looseness of the faeces, appeared at 7 to 8 weeks, being earlier in the more heavily infested animals. In the most lightly infested animals the faeces remained loose for only one or two weeks and fresh blood was occasionally seen. There were no other signs of illness. In the most heavily infested animals severe diarrhoea developed within a few days, and was occasionally accompanied by straining and evidence of abdominal pain. The animals were weak, listless and tucked-up, with a staring coat, and showed a reduction in appetite and a marked loss of condition. Fresh blood was seen in the faeces and occasionally blood and mucus alone were passed from the anus. There was no frank febrile reaction but a rise in rectal temperature of up to 0,5°C was noted consistently. Signs were at their most severe at 8 to 9 weeks after infestation and thereafter there was a gradual recovery. The severity of the diarrhoea di-

minished and appetite returned slowly, but blood continued to be passed in small amounts in the faeces, even for several weeks after the consistency had returned to normal. General appearance and bodily condition gradually improved to normal but marked stunting was evident when the animals were compared with uninfested controls. Mild diarrhoea, occasional blood in the faeces and a degree of anorexia persisted for up to 23 weeks post-infestation in a few of the most heavily infested animals.

The visible blood in the faeces apparently originated from the distal part of the large intestine as it was usually passed at the end of a faecal motion and was seen as a blob or streak on top of the pat of dung. Most calves showed occult blood in the faeces when they were first tested with ortho-tolidine at nine weeks post-infestation. Thereafter the incidence of occult blood diminished sharply, although visible blood could still be detected occasionally in some animals. It is probable that the occult blood, admixed with the faeces, originated from the more proximal intestine, and that haemorrhage from this site ceased at an earlier stage than that from the distal large intestine.

The severity and duration of diarrhoea was proportional to the level of infestation in all but two calves. In these animals, infested with 45 000 cercariae on a low plane of nutrition, diarrhoea persisted for only 4 or 5 weeks but was followed by a prolonged period of unthriftiness. The animals were thin, stunted, pot-bellied, stary coated and apparently dehydrated and they became very weak. Appetite, albeit on a restricted ration, was normal but the rumen was distended and the contents hard. The faeces were firmer than normal. This condition persisted until 33 weeks post-infestation when one of them was slaughtered. The other showed a rapid improvement when the plane of nutrition was raised.

In one heavily infested calf, anal polyps appeared at 22 weeks post-infestation as irregular red outgrowths at the rim of the anus and persisted until the animal was slaughtered 11 weeks later. They did not appear to be a source of discomfort.

Body Mass

Mean body mass (%) (see Materials and Methods) for 10 infested calves observed over a period of 78 weeks is plotted against weeks post-infestation in Fig. 1. There was a sharp fall between 6 and 10 weeks, reflecting either a reduction in growth rate relative to the controls or a loss of mass, and this accompanied the period of most severe clinical illness. Thereafter body mass (%) remained steady, indicating a return to a growth rate comparable to that of the controls. Resting body mass (%) was calculated as the mean for the period 0 to 5 weeks post-infestation, and the difference between this and mean body mass (%) for the period 10 to 15 weeks was regarded as a measure of the effect of infestation. There was a significant reduction of body mass (%) in 15 of 27 calves, as estimated by Students "t" test ($P<0,05$). Body mass (%) was significantly reduced in all but one of the calves infested with more than 90 cercariae/kg, while only one calf infested with less than 90 cercariae/kg was significantly affected.

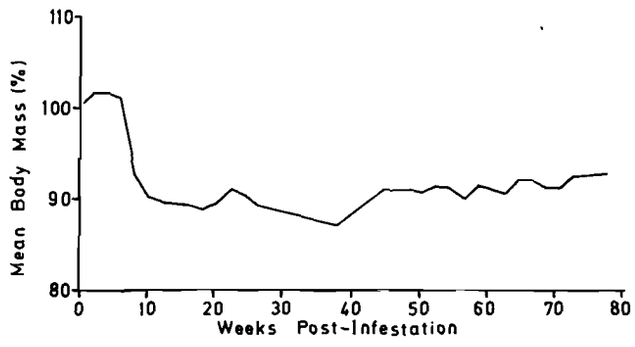

Fig. 1 Changes in mean body mass (%) in 10 calves over 78 weeks after infestation.

The greatest effect in calves on the high plane of nutrition was seen in an animal infested with 45 000 cercariae, where the actual mass fell from 200 kg to 191 kg between 6 and 12 weeks after infestation, while the two controls in the group gained a mean of 37,6 kg over the same period. On the low plane an animal infested with 30 000 cercariae dropped from 155 kg to 134 kg between 6 and 11 weeks while the controls gained a mean of 3,4 kg.

The difference in body mass (%) between 0 to 5 and 10 to 15 weeks (Dm) is plotted against infesting cercariae/10^3 (C) in Fig. 2. The two planes of nutrition have been considered separately and in each case the effect on body mass increased with the level of infestation, the points conforming to the following regressions:

high plane $Dm = 1,278 - 0,561\ C$ ($r = -0,776$, $P<0,001$)

low plane $Dm = 4,062 - 0,476\ C$ ($r = -0,831$, $P<0,01$)

The reduction in body mass (%) was greater in the calves on the high plane than those on the low plane, the difference between the regressions being highly significant ($P<0,001$).

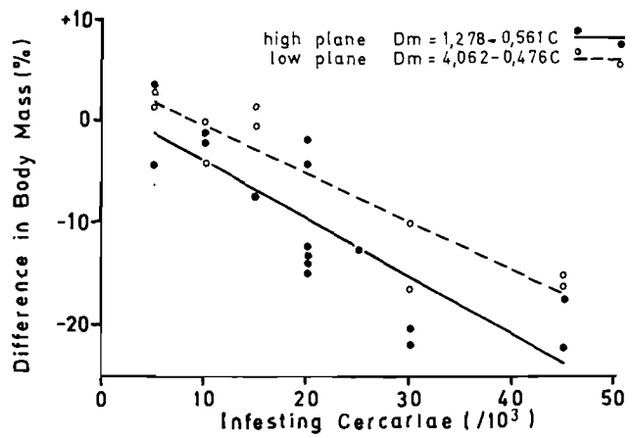

Fig. 2 Difference in body mass (%) between 0-5 weeks and 10-15 weeks post-infestation in calves on two planes of nutrition.

FIELD OUTBREAKS

A severe outbreak of acute intestinal schistosomiasis was encountered on a farm in northern Mashonaland, 175 km north-west of Salisbury. The farm ran 700 head of Hereford-type cattle watering from a variety of sources which were all infested with snails. In June, 1971, it was noted that approximately 50 of the 275 head in the "weaner" herd (9 to 18 months old) were showing diarrhoea, blood in the faeces, emaciation, anaemia and debility, the older animals being more severely affected than the younger ones. A certain amount of coughing was reported but this was trans-

ient. The adult cattle were not affected. Routine anthelminthic therapy had no beneficial effect and the condition of the cattle deteriorated over the next 2 months. Three animals died and one was slaughtered *in extremis,* and a very heavy infestation of schistosomes was detected in two of them at post-mortem examination. A third animal was slaughtered at the Veterinary Research Laboratory and 40 679 parasites were recovered by perfusion.

The clinically affected animals were penned and given supplementary feed but were not treated with a schistosomicide. The majority made a slow recovery but a small number remained emaciated and stunted for a long period and showed occasional episodes of diarrhoea and blood in the faeces. Routine molluscicide treatment of all permanent water sources on the farm was introduced to control snails and the condition has not recurred.

A second outbreak occurred on a farm within a few kilometres of Fort Victoria on which ran 500 head of mixed cattle watering from dams, some of which were heavily infested with snails. In October and November, 1970, a total of 17 animals died, most of them being cows. Post-mortem examination of some of them revealed large numbers of schistosomes and lesions of schistosomiasis. Other cows were in very poor condition in spite of apparently adequate supplementary feeding, but showed no specific clinical signs. The cattle were not treated and their condition improved with the advent of the summer rains in December and the improvement in the grass. Routine molluscicide treatment of the dams was introduced and the condition has not recurred.

DISCUSSION

Clinical illness in the ox following primary infestation with *S. mattheei* is a relatively acute condition characterised by diarrhoea and has been called the "intestinal syndrome" to differentiate it from the "chronic hepatic syndrome" which sometimes follows reinfestation (Lawrence, in preparation). It occurs during the period of high faecal egg output in the first weeks after patency and can be attributed mainly to the passage of large numbers of eggs through the intestinal wall. The severity of the diarrhoea, however, begins to diminish while faecal egg counts are still high, suggesting that factors in addition to the number of eggs passed are responsible for the diarrhoea. Other factors which have been implicated are a hyperreactive response by the host to the eggs and a shift of egg laying to the distal large intestine (Lawrence, in preparation).

Spontaneous recovery occurred in all the experimental animals, confirming the observation of McCully & Kruger[14] in one experimental ox. The acute fatal cases produced by van Wyk & Bartsch[19] died at 2 months post-infestation, the time when clinical signs were most severe in the non-fatal cases in this study. It would appear that after primary infestation death will occur at 8 to 9 weeks post-infestation, if at all, and survival beyond this time will be followed by recovery. The lethal level of infestation is clearly greater than the highest rate used in this experiment, 341 cercariae/kg body mass, and this agrees with the observation that an infestation of 436 cercariae/kg body mass is fatal[19].

Most of the animals in the experiment exhibited an acute intestinal syndrome and diarrhoea was the predominant clinical sign. A similar clinical picture was seen in one of the natural outbreaks. In the two most heavily infested animals on the low plane of nutrition in the experiment, however, diarrhoea was of relatively short duration and was followed by a prolonged period of unthriftiness. This suggests that a variation of the syndrome may occur, a subacute intestinal syndrome, in which debility is a more prominent feature than diarrhoea. Insufficient cases of this type were seen amongst the experimental cattle for the syndrome to be characterised, but evidence from the second field incident and from a previous incident[13] suggests that it does occur in cattle under nutritional stress and that recovery follows when the nutritional status is improved.

The acute intestinal syndrome follows primary infestation only. It did not occur in cattle exposed to heavy reinfestation by van Wyk & Bartsch[19], nor in animals that had recovered from the acute syndrome and were infested for a second time with 15 000 to 21 000 cercariae 20 and 95 weeks later by Lawrence[12]. This is because egg output of reinfesting parasites is suppressed and faecal egg counts remain low[10]. In the first natural outbreak described above the older cattle, which were exposed to the same challenge, but had presumably been infested previously, were unaffected, lending support to the experimental observations. In the outbreaks of the subacute syndrome which were encountered the affected cattle were adult, but since their previous infestation history is not known no conclusions can be drawn as to whether this syndrome is likely to follow reinfestation as well as primary infestation.

In the experiment it was apparent that *S. mattheei* caused a loss of mass or reduction in growth rate in heavily infested cattle, the threshold of significant infestation being 90 cercariae/kg body mass. Animals with lighter infestations were not affected to a significant extent, but it is possible that other aspects of productivity, such as milk yield, might be more sensitive. In the field the effects of the parasite might be increased by the stress of exposure to inclement weather and the necessity of walking long distances for food and water, and by pregnancy and lactation, as in sheep (personal observation). Indirect losses might be caused by an increased susceptibility to other infections, e.g. piroplasmosis[18], while it has been found that infestation with *Schistosoma indicum* in sheep predisposes to increased gastro-intestinal parasitism[17]. For these reasons it is not possible to predict the level at which *S. mattheei* becomes significant in the field, but it appears to be high in relation to the general level of infestation and is probably attained infrequently in nature.

The effect of the parasite on body mass was in part caused by inappetence, which is a common sign of parasitic disease and may be caused by pain and by the effect of intestinal lesions on peristalsis reducing the rate of emptying of the rumen[3]. Other factors may also have been involved as mass loss in sheep infested with *S. mattheei* has been found to be greater than that in controls consuming the same amount of food[15]. Berry *et al.*[4] attributed this to muscle wasting, from mobilisation of protein for synthesis of plasma proteins and haemoglobin in response to losses into the gastro-intestinal tract, and to impaired digestion and/or malabsorption. Another factor which might cause reduced food utilisation is an increase in energy metabolism from local nerve irritation, intestinal inflammation and increased intestinal activity[1,2]. The raised rectal temperature recorded in the calves in this experiment does suggest that there was an increase in the metabolic rate.

It is generally considered that a high plane of nutrition exerts a protective effect in helminthiasis, and a

reduction in the pathogenic effects in well nourished cattle has been demonstrated with *Fasciola gigantica*[7] and gastro-intestinal nematodes[6 8]. There was no evidence of such an effect in the present experiment. In fact, Fig. 2 indicates that animals on the high plane of nutrition suffered a proportionately greater mass loss than those on the low plane. This can probably be explained by a higher egg output by the parasites[11] and a more intense tissue reaction in the calves on the high plane of nutrition (Lawrence, in preparation) exerting a greater pathogenic effect which was not entirely compensated for by the better nutritional status. A similar balance between the pathogenicity of the parasite and the capacity of the host to tolerate it under differing nutritional conditions has been demonstrated with *Schistosoma mansoni* in mice by Knauft & Warren[9].

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RESISTANCE TO TOXAPHENE BY THE BONT TICK, *AMBLYOMMA HEBRAEUM* (KOCH)

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ABSTRACT: Baker J.A.F.; Thompson G.E.; Miles Janet O. **Resistance to toxaphene by the Bont tick, *Amblyomma hebraeum* (Koch).** *Journal of the South African Veterinary Association* (1977) **48** No. 1, 59 – 65 (En) Coopers (SA) (Pty) Ltd, Greenfields, 5208 East London, Rep. of South Africa.

A series of *in vitro* and *in vivo* trials confirmed resistance by the three-host Bont tick, *Amblyomma hebraeum* (Koch), to Toxaphene in the Cape, Natal and Transvaal provinces of the Republic of South Africa, and in Swaziland and Transkei. Resistance was present in 80 of 97 field strains examined. Lindane and DDT resistance was present in the one field strain tested.

The results of comparative *in vivo* hand spraying and plunge dipping trials confirmed those of the larval *in vitro* tests, thus validating the usefulness of this latter technique for identifying changes of susceptibility in field tick strains to ixodicides.

Selection of Toxaphene resistance in *A. hebraeum* occurred within a period of four years when a 14-day interval of treatment was used continuously. The presence of all three tick instars in all stages of engorgement at the time of each treatment under this regimen is thought to have enhanced this selection.

INTRODUCTION

The three-host Bont tick, *Amblyomma hebraeum* (Koch) poses a serious threat to successful stock farming in those areas of South Africa in which it occurs. In cattle, sheep and goats it transmits trans-stadially, *Cowdria ruminantium,* the causal organism of Heartwater, a fatal disease characterised in the host by a marked febrile reaction.

Secondary infection at the site of attachment of adult ticks is usually encountered, leading to abscessation and the formation of suppurating lesions most attractive to myiasis-producing flies. These conditions are exacerbated by the tendency of these ticks to form large, tightly-packed clusters on predilection sites. Attachment of adult ticks to the teats of female animals is a frequent cause of intramammary infection and subsequent loss of one or more udder quarters. Hide damage can be severe with affected areas being quite unsuitable for use.

In man, *A. hebraeum* is an important vector of Tick Bite Fever, (synonyms: Tick Typhus, Boutonneuse Fever), a trans-ovarially transmitted disease caused by *Rickettsia conori.*

Immature stages of *A. hebraeum* will successfully feed on a wide range of hosts including birds, reptiles, rodents, antelopes, carnivores, equines, primates and most domestic stock[10]. Adult ticks occur most frequently on game animals and domestic stock[10]. Bovine animals are the preferred hosts on which large numbers of all three tick stages may be encountered.

The predilection sites of attachment on cattle are the belly, groin, perineum and axilla for adults[5 11], the feet, axilla, sternum, belly, groin and legs for nymphae and, almost equally favoured, the feet, muzzle, legs and head for larvae[5].

The distribution of *A. hebraeum* within the Republic of South Africa is tied to the areas of woodland savannah of Acacia or Mopane covering much of the Eastern and Northern Transvaal bushveld and to the coastal regions stretching from Mossel Bay in the South to the lowveld areas of Zululand. It also occurs in the middleveld and lowveld areas of Swaziland. It is absent from open savannah or steppes[10]. In Natal there are, however, indications that its distribution limits are spreading[5]. More recent data confirms this trend in this and other provinces (J.B. Walker – Personal Communication, 1976).

The seasonal activity of *A. hebraeum* in Natal is larvae, February to May; nymphs, May to September and adults, September to January[5]. However, despite the year-round treatment programme necessitated by such activity, the failure of most stockowners to recognise any but the adult stage, results in Bont tick control measures being confined principally to the summer months. This factor, together with a widespread desire to confer premunity in free-ranging calves to *C. ruminantium* by the maintenance of 'light' *A. hebraeum* infestations in the field, has been instrumental in establishing significant infestations of these ticks, together with an endemic Heartwater disease situation, throughout their distribution limits in Southern Africa.

A. hebraeum was long considered the most difficult to control of all tick species infesting domestic stock. Serial treatments in arsenic failed to remove feeding females, although initially a good kill of males occurred, and tightly packed clusters of engorging ticks resulted[6 14]. Although DDT was quite inadequate for Bont tick control[6], gamma-BHC with or without the addition of DDT proved considerably superior to arsenic[6 14] and was widely used for this purpose. In the early 1950's, Toxaphene replaced gamma-BHC as the ixodicide of choice for the control of *A. hebraeum* due to the efficacy of this compound against this tick species[6 12].

Chemical resistance in *A. hebraeum* has previously only been shown to arsenic[7]. Although this finding is of recent origin, it is suspected that this resistance has long been present. Over the past decade, however, verbal reports of failures to achieve satisfactory control of *A. hebraeum* in the field with Toxaphene have been regularly received.

In the course of a survey of Toxaphene susceptibility in *A. hebraeum* undertaken over the past four years, significant variations in the response of some field strains to Toxaphene were observed.

Both these factors have been further investigated.

METHODS

Comparative laboratory tests on larvae, nymphae and adults were undertaken. In addition, an *in vivo*

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hand spraying and plunge dipping trial was carried out. Details of the methods used were:

1. Unfed larvae

Larvae, 14 to 21 days old, bred from engorged female ticks received from field collections, were immersed between two filter papers in a series of concentrations of ixodicidal wash for a period of 10 minutes. The larvae were then removed from the wash, dried carefully and a number placed in a folded filter paper packet sealed to prevent escapes.

The packets were maintained at 24°C and 80% R.H. for 72 hours when percentage mortality was assessed. The technique used is described by Shaw[9]. Water controls were run concurrently with each test and corrections for control mortality made using Abbot's formula[1].

A comparison of the susceptibility of two strains to a number of different compounds was also undertaken: (i) the known Toxaphene sensitive Kwanyanga laboratory reference strain which has been cultured at this laboratory for more than two years, (ii) the Lowhills strain from the Barberton District, Transvaal, which had a history of Toxaphene tolerance in the field.

2. Engorged larvae and nymphs

Kwanyanga and Lowhills strains of engorged larval and nymphal ticks, fed on the ears of calves, were harvested after dropping overnight and tested within two hours of collection. After immersion for 60 seconds in the concentration of Toxaphene wash under test, the larvae and nymphae were dried on blotting paper whilst exposed to an air-stream at room temperature. The ticks comprising each treatment group were placed in a 100 ml Erlenmeyer flask, the base of which was lined with a single 7,0 cm Whatman's No. 1 filter paper and the top sealed with a single layer of organdie. They were then incubated at 24°C and 80% R.H. Observations were made of nymphal emergence from the larvae, and adult emergence from the nymphae, the final assessments being undertaken two weeks after final emergences in the respective water control groups had occurred..

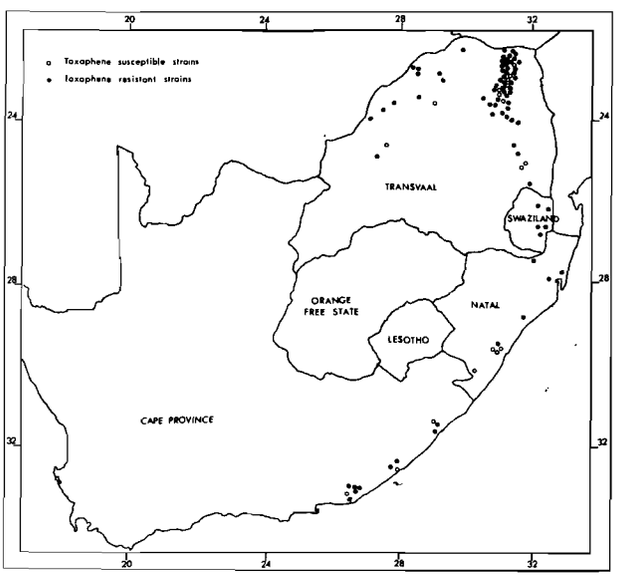

Fig. 1 Geographical distribution of the Toxaphene susceptible and resistant strains appearing in Table 1.

3. Unfed adults

Kwanyanga and Lowhills strains of engorged nymphal ticks, fed on the ears of calves, were harvested after dropping, and groups of 25-50 placed in cotton wool-stoppered 100 ml Erlenmeyer flasks and incubated at 24°C and 80% R.H. 14-21 days after adult emergence a series of wash concentrations of Toxaphene of 25 ml volume were introduced into the flasks which were swirled by hand. After 60 seconds the contents of the flask were emptied onto a sieve having a nominal aperture of 1,25 mm and the treated adults removed and dried on blotting paper whilst exposed to an air-stream at room temperature. The ticks were sexed, placed in clean 100 ml Erlenmeyer flasks with organdie covers and returned to the incubator. Mortality was assessed daily for five days and again on the seventh day post-treatment.

4. *In vivo* trial

Counts and assessments of *A. hebraeum* adults and nymphae were made pre- and 48 hours post-treatment on groups of three cattle hand-sprayed or plunge-dipped in 0,25% Toxaphene washes. Untreated control groups were run concurrently with each test. The techniques used for tick counts and assessments, and for the hand-spraying are those described by Baker *et al*[4].

Investigations were undertaken on two properties. At Gulu, East London District, Cape Province, from which property strains of *A. hebraeum* had been judged to be Toxaphene sensitive in the larval test, a hand-spray group only was employed. At Tarbet, Hhohho District, Swaziland, from which property strains of *A. hebraeum* had been judged to be Toxaphene resistant in the larval test, a hand-spray and a plunge-dipped group were used.

RESULTS

Unfed larvae

The results, expressed as LC50 and LC99 values, are the product of at least two replicates for each observation. The 95% confidence limits for each of these values are also summarized in Table 1. The values for each field strain are compared at both levels with those obtained for the Kwanyanga reference strain and factors of resistance thus obtained are shown. The data were analysed by computer, using a probit analysis programme.

The susceptibility of 97 field strains of *A. hebraeum* to Toxaphene are given in Table 1. Of these, only nine were as susceptible as the Kwanyanga strain. The remainder were less susceptible to varying degrees. The geographical distribution of all strains is illustrated in Fig. 1.

The susceptibility of larvae of the Kwanyanga strain to a range of ixodicides is compared to that of a field strain from Lowhills, Barberton District, in Table 2.

A high factor of resistance against Toxaphene as well as resistance against lindane and DDT are shown by the Lowhills strain. Neither strain shows resistance to the three organophosphorous ixodicides used, with, in fact, slightly increased LC99 values being obtained for the Kwanyanga strain as compared to the Lowhills strain.

Table 1: THE SUSCEPTIBILITY OF 97 FIELD STRAINS OF LARVAE OF *AMBLYOMMA HEBRAEUM* TO TOXAPHENE

Field Strain Sample Number	Origin of Strain		LC50(%)	95% Fiducial Limits		FOR	LC99(%)	95% Fiducial Limits		FOR
	Kwanyanga*		0,007	0,007	0,009		0,14	0,1	0,22	
1	East London**	Cape	0,008	0,0071	0,0081	1,0	0,043	0,037	0,051	1,0
2	East London	Cape	0,03	0,014	0,062	3,9	1,6	0,42	39	11
3	East London	Cape	0,14	0,058	0,42	17	39	5,0	>100	280
4	Bathurst	Cape	0,032	0,006	0,11	4,2	2,0	0,3	>100	14
5	Warmbaths	Transvaal	0,005	—	—	—	0,051	—	—	—
6	Pietersburg	Transvaal	0,014	0,012	0,017	1,8	0,33	0,23	0,54	2,4
7	Soutpansberg	Transvaal	0,023	0,018	0,03	3,0	2,7	1,5	5,9	20
8	Messina	Transvaal	0,025	0,021	0,03	3,2	0,44	0,31	0,74	3,2
9	Brits	Transvaal	0,034	0,012	0,085	4,4	4,6	0,8	>100	32
10	Soutpansberg	Transvaal	0,039	0,031	0,049	5,1	1,1	0,64	2,0	7,6
11	Thabazimbi	Transvaal	0,041	0,027	0,064	5,3	5,06	1,7	27	36
12	Potgietersrus	Transvaal	0,046	0,024	0,093	6,0	6,6	1,5	>100	47
13	Waterberg	Transvaal	0,072	0,06	0,086	9,4	2,6	1,6	4,6	18
14	Barberton	Transvaal	0,44	0,17	2,3	58	>100	45	>100	—
15	Ngotshe	Natal	19	5,4	>100	2500	>100	—	—	—
16	Kentani 352	Transkei	0,014	0,008	0,023	1,8	0,42	0,16	2,7	3,0
17	Kentani 350	Transkei	0,023	0,012	0,045	3,0	3,6	0,88	62	25
18	Willowvale 588	Transkei	0,008	0,004	0,013	1,0	0,35	0,13	2,4	2,6
19	Willowvale 589	Transkei	0,033	0,024	0,046	4,3	1,3	0,72	3,2	9,6
20	Victoria East 1	Ciskei	0,013	0,007	0,023	1,7	0,56	0,18	5,1	4,1
21	Victoria East 3	Ciskei	0,02	0,012	0,034	2,6	1,2	0,43	9,9	9,3
22	Victoria East 2	Ciskei	0,023	0,012	0,053	3,0	0,65	0,17	26	4,7
23	Zwelitsha 22	Ciskei	0,025	0,02	0,033	3,2	5,3	2,8	11	38
24	Keiskammahoek 2	Ciskei	0,25	—	—	32	>100	—	—	—
25	Middledrift 12	Ciskei	0,07	0,056	0,087	9,1	4,6	2,7	9,2	33
26	Umbumbulu 126	KwaZulu	0,01	0,002	0,028	1,3	0,24	0,059	>100	1,8
27	Mapumalanga 880	KwaZulu	0,009	—	—	1,2	0,29	—	—	2,1
28	Mapumalanga 224	KwaZulu	0,013	0,011	0,015	1,7	0,22	0,15	0,4	1,6
29	Mapumalanga 221	KwaZulu	0,014	0,012	0,017	1,8	0,53	0,34	0,9	3,8
30	Mapumalanga 148	KwaZulu	0,016	0,013	0,019	2,1	0,82	0,52	1,4	5,9
31	Hlabisa 526	KwaZulu	0,031	0,014	0,063	4,0	1,2	0,33	17	7,9
32	Mtunzini 632	KwaZulu	0,031	0,025	0,037	4,0	3,3	1,9	6,3	23
33	Ubombo 767	KwaZulu	0,032	0,016	0,055	4,2	1,9	0,8	9,5	14
34	Giyani	Gazankulu	0,039	0,017	0,087	5,1	7,0	1,4	>100	50
35	Giyani 23	Gazankulu	0,047	0,039	0,06	6,1	1,7	1,2	2,9	12
36	Giyani 19	Gazankulu	0,061	0,027	0,15	7,9	7,1	1,3	>100	51
37	Giyani	Gazankulu	0,068	0,05	0,092	8,8	22	9,0	70	158
38	Malamulele	Gazankulu	0,018	0,015	0,022	2,3	1,0	0,66	1,7	7,3
39	Mhala 220	Gazankulu	0,032	—	—	4,2	0,85	—	—	6,1
40	Bolebedu 13	Lebowa	0,09	0,025	0,29	11	31	3,0	>100	220
41	Bolebedu 10	Lebowa	0,14	0,11	0,2	19	53	20	>100	380
42	Bolebedu 12	Lebowa	0,21	0,13	0,47	28	>100	30,0	>100	—
43	Bosbokrand 214	Lebowa	0,007	0,001	0,024	1,0	0,19	0,04	>100	1,4
44	Bosbokrand	Lebowa	0,015	0,013	0,018	1,9	0,55	0,36	0,92	4,0
45	Bosbokrand	Lebowa	0,028	0,012	0,069	3,6	1,9	0,42	88	13
46	Bosbokrand 463	Lebowa	0,057	—	—	7,4	1,6	—	—	11
47	Mokerong	Lebowa	0,024	0,007	0,1	3,1	0,69	0,14	>100	5,0
48	Mokerong	Lebowa	0,043	0,002	0,33	5,6	1,7	0,26	>100	12
49	Mokerong	Lebowa	0,092	0,018	0,35	11	2,1	0,46	>100	15
50	Mokerong 760	Lebowa	0,099	0,032	0,28	12	13	2,0	>100	93

Field Strain Sample Number	Origin of Strain		LC50(%)	95% Fiducial Limits		FOR	LC99(%)	95% Fiducial Limits		FOR
51	Sibasa 12	Venda	0,006	0,0003	0,028	1,0	0,31	0,046	>100	2,3
52	Sibasa 11	Venda	0,007	0,003	0,015	1,0	0,46	0,13	8,8	3,3
53	Sibasa 15	Venda	0,008	0,002	0,035	1,0	0,25	0,049	>100	1,8
54	Sibasa 24	Venda	0,009	0,002	0,022	1,2	0,33	0,081	64	2,4
55	Sibasa 18	Venda	0,01	0,004	0,023	1,3	0,91	0,19	59	6,6
56	Sibasa 19	Venda	0,018	0,01	0,03	2,3	0,86	0,24	14	6,3
57	Sibasa 33	Venda	0,019	0,015	0,024	2,5	1,7	0,98	3,4	12
58	Sibasa 28	Venda	0,021	—	—	2,7	2,0	—	—	15
59	Sibasa 23	Venda	0,022	0,018	0,027	2,9	1,0	0,67	1,9	7,8
60	Sibasa 44	Venda	0,023	0,012	0,05	3,0	1,5	0,37	42	11
61	Sibasa 26	Venda	0,026	0,003	0,34	3,4	12	0,62	>100	88
62	Sibasa 37	Venda	0,032	0,003	0,14	4,2	2,7	0,36	>100	19
63	Sibasa 27	Venda	0,039	0,029	0,051	5,1	9,9	4,8	25	71
64	Sibasa 30	Venda	0,042	0,035	0,052	5,5	2,7	1,6	5,1	19
65	Sibasa 14	Venda	0,051	0,037	0,075	6,6	9,7	2,9	70	70
66	Sibasa 34	Venda	0,051	0,022	0,13	6,6	5,7	1,0	>100	41
67	Sibasa 22	Venda	0,055	0,017	0,16	7,1	3,9	0,7	>100	28
68	Sibasa 36	Venda	0,056	0,014	0,2	7,3	8,4	1,1	>100	61
69	Sibasa 3	Venda	0,056	—	—	7,3	0,64	—	—	4,6
70	Sibasa 21	Venda	0,060	0,049	0,074	7,8	2,5	1,5	5,1	18
71	Sibasa 25	Venda	0,062	0,027	0,15	8,1	7,7	1,4	>100	55
72	Sibasa 8	Venda	0,064	—	—	8,3	1,6	—	—	11
73	Sibasa 38	Venda	0,066	0,032	0,14	8,6	53	8,0	>100	380
74	Sibasa 31	Venda	0,067	0,011	1,2	8,7	36	1,6	>100	260
75	Sibasa 13	Venda	0,072	0,063	0,083	9,4	0,85	0,63	1,2	6,1
76	Sibasa 4	Venda	0,09	0,029	0,54	11	25	2,1	>100	180
77	Sibasa 5	Venda	0,097	0,04	0,29	12	3,4	0,74	>100	25
78	Sibasa 16	Venda	0,1	—	—	13	1,0	—	—	7,7
79	Sibasa 35	Venda	0,1	0,08	0,13	13	10	4,5	33	71
80	Sibasa 17	Venda	0,14	0,06	0,36	18	33	5,0	>100	230
81	Sibasa 32	Venda	0,16	0,06	0,52	21	>100	15	>100	—
82	Sibasa 41	Venda	0,24	0,11	0,69	31	>100	81	>100	—
83	Sibasa 20	Venda	0,43	0,14	9,8	56	>100	6,5	>100	—
84	Vuwani 2	Venda	0,006	0,001	0,02	0,1	1,2	0,18	>100	9,2
85	Vuwani 11	Venda	0,01	0,005	0,019	1,3	0,51	0,15	8,5	3,7
86	Vuwani 3	Venda	0,011	—	—	1,4	0,21	—	—	1,6
87	Vuwani 5	Venda	0,028	—	—	3,6	0,48	—	—	3,5
88	Vuwani 18	Venda	0,03	0,025	0,037	3,9	1,9	1,2	3,6	14
89	Vuwani 4	Venda	0,04	0,014	0,15	5,2	7,5	0,86	>100	54
90	Vuwani 22	Venda	0,048	0,005	0,25	6,2	1,2	0,23	>100	8,7
91	Vuwani 6	Venda	0,055	—	—	7,1	1,1	—	—	8,6
92	Dzanani 17	Venda	0,005	0,00002	0,19	1,0	0,073	0,016	>100	1,0
93	Lubombo 243	Swaziland	0,061	0,047	0,08	7,9	15	7,0	41	110
94	Lubombo 265	Swaziland	0,36	0,75	2,9	170	>100	—	—	—
95	Lubombo	Swaziland	4,3	—	—	560	>100	—	—	—
96	Hhohho***	Swaziland	0,47	0,12	—	62	>100	—	—	—
97	Hhohho	Swaziland	>1,0	—	—	>20	>100	—	—	—

FOR = Factor of resistance determined by comparison with the Kwanyanga strain
* = Kwanyanga Research Station Reference Strain
** = Gulu Strain
*** = Tarbet Strain
— = Confidence limits not calculated, data heterogeneous

Table 2: A COMPARISON OF THE SUSCEPTIBILITY OF LARVAE OF THE KWANYANGA AND LOWHILLS STRAINS OF *AMBLYOMMA HEBRAEUM*

Chemical	KWANYANGA STRAIN						LOWHILLS STRAIN						FOR Based on LC50 (%)	FOR Based on LC99 (%)
	LC50(%)	95% Fiducial Limits		LC99(%)	95% Fiducial Limits		LC50(%)	95% Fiducial Limits		LC99(%)	95% Fiducial Limits			
Toxaphene	0,0077	0,007	0,009	0,14	0,1	0,22	5,8	1,9	46	>100	—	—	750	>700
DDT	0,004	0,002	0,005	0,018	0,01	0,08	0,017	0,008	0,029	0,94	0,6	1,9	4,3	52
Lindane	0,00019	0,0002	—	0,0004	—	—	0,008	0,003	0,014	13	3,0	>100	42	29000
Dioxathion	0,0002	—	0,0002	0,001	0,0009	0,001	0,0002	—	—	0,0006	—	—	1,0	1,0
Chlorfen-vinphos	0,0005	0,0002	0,001	0,001	0,0008	0,0015	0,00031	0,0003	0,00033	0,00053	0,00048	0,0006	1,0	1,0
Dioxathion/ Chlorfen-vinphos	0,00009	0,00003	0,00014	0,00047	0,00024	0,041	0,0004	—	—	0,0009	—	—	1,0	1,0

— = Confidence limits not calculated, data heterogeneous. FOR = Factor of resistance determined by comparison with the Kwanyanga strain.

Engorged larvae and nymphs

Engorged larvae and nymphae of the Lowhills strain resisted much higher levels of Toxaphene than did those of the Kwanyanga strain (Table 3).

Unfed adults

Unfed male and female ticks of the Lowhills strain successfully survived immersion in concentrations of Toxaphene which killed those of the Kwanyanga strain (Table 3).

In vivo trial

Neither the 0,25% Toxaphene hand-spray wash nor 0,25% Toxaphene plunge-dip wash treatments at Tarbet had any marked effect on light infestations of *A. hebraeum* nymphal and adult ticks. In comparison, excellent results against these two tick stages were obtained by the 0,25% Toxaphene hand-spray wash against a heavy field challenge at Gulu (Figs. 2 & 3).

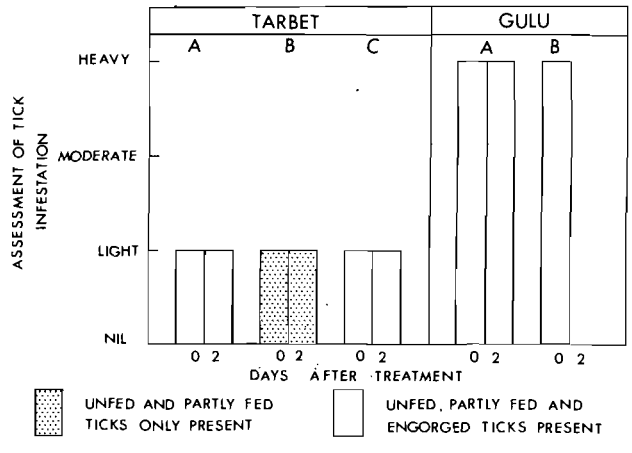

Fig. 2 A comparison of the assessments of *A. hebraeum* nymphs on groups of cattle at 'Tarbet' and 'Gulu' before and 2 days after treatment. A: Untreated Controls; B: 0,25% Toxaphene spraywash; C: 0,25% Toxaphene plunge dipwash.

Table 3: A COMPARISON OF THE EFFECT *IN VITRO* OF A RANGE OF TEST CONCENTRATIONS OF TOXAPHENE TO ENGORGED LARVAE AND NYMPHAE AND UNFED ADULTS OF TWO STRAINS OF *A. HEBRAEUM*

	PERCENT MORTALITY									
Percent Toxaphene	0,001	0,003	0,01	0,03	0,10	0,30	1,0	3,0	10,0	Water Control
Strain										
Engorged Larvae										
Kwanyanga	0	2,9	20	25,7	31,4	82,9	94,3	100	100	0
Lowhills	*	*	0	0	2,0	16,0	20,0	82	100	4,0
Engorged Nymphae										
Kwanyanga	*	*	0	2,0	0	0	56,7	100	100	0
Lowhills	*	*	*	*	0	0	0	0	100	0
Unfed Adults										
Kwanyanga	*	*	*	*	*	*	27,3	100	100	0
Lowhills	*	*	*	*	*	*	0	**14,8	**70,6	0

*Not tested. **Survivors from these groups subsequently used successfully for further strain culturing.

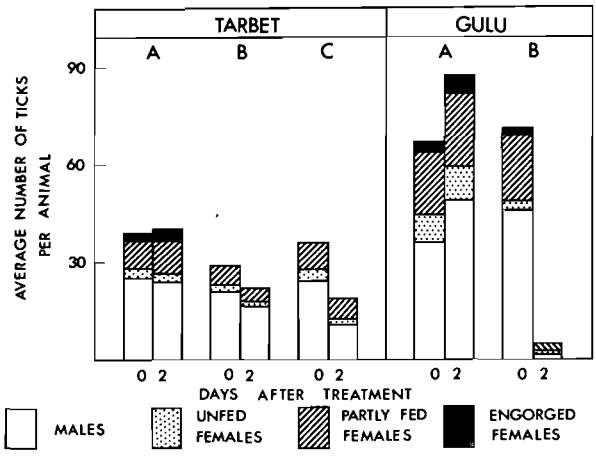

Fig. 3 A comparison of the counts of *A. hebraeum* adult ticks on groups of cattle at 'Tarbet' and 'Gulu' before and 2 days after treatment. A: Untreated Controls; B: 0,25% Toxaphene hand spraywash; C: 0,25% Toxaphene plunge dipwash.

DISCUSSION

A marked resistance by *A. hebraeum* to Toxaphene at all three stages of tick development is shown by the results.

Confirmation of the larval *in vitro* test results by the Tarbet and Gulu *in vivo* trials fully validates the usefulness of this technique for identifying changes of susceptibility in field tick strains to ixodicides. This is further substantiated by the comparison of the Lowhills laboratory results and field experience.

It follows that similar variations in response shown in the larval test by other strains will also indicate a similar level of field resistance. Thus, by using the LC50 value for Tarbet, 0,478 (Table 1), as a guide, those five strains which show larval survival in excess of this value can also be expected to produce a marked resistance to Toxaphene in the field.

Some of the tested strains not having an LC50 value greater than that of Tarbet do, however, have LC99 values often grossly in excess of this value. It follows further that numbers of larvae from these latter strains will also successfully survive field treatment with Toxaphene. These strains must, therefore, also be classified as resistant. On this basis, a further 75 strains, or 80 out of 97, would show levels of resistance to Toxaphene in the field (Fig. 1).

Resistance to Toxaphene in other African multi-host tick species[2 13] has shown that this occurs in larval, nymphal and adult stages. This is shown to be equally true for *A. hebraeum*.

In the absence of any previous dipping history from Lowhills, it is impossible to say whether the increased tolerances shown by this strain to lindane and DDT are as a result of chemical cross-resistance or not. As the usage of both these chemicals was widespread for many years in the district in which the farm is situated, it is possible that this strain was exposed to both these compounds at one time or another.

The excellent clearance of ticks afforded by Toxaphene at Gulu is in accordance with that previously observed against Toxaphene susceptible *A. hebraeum* strains[3 6 12]. In sharp contrast was the poor control obtained at Tarbet especially of the usually easily killed male ticks.

The larval test results from Gazankulu and Venda (Table 1), indicate an unusually rapid development of field resistance to Toxaphene by *A. hebraeum*. After many years of arsenic usage, all cattle dipping tanks in these areas were changed to Toxaphene early in 1970. From four to six years of serial 14-day interval dippings followed until late in 1975, when a change to an organophosphorus ixodicide was made (J.C. Jacobs – Personal Communication, 1976). The purchase of cattle dipping compounds for all dipping tanks in these areas is on a contract basis, thus little or no deviation from the designated usage of ixodicides occurs.

The Gazankulu strains tested all show a similar degree of resistance. Although greater variations exist in the Venda strains, the results conform to much the same pattern of resistance recorded in the Gazankulu ticks, indicating that selection pressures and the subsequent evolvement of resistance had been similar.

The year-round two-week interval of treatments undertaken in Venda and Gazankulu is not common. This regimen might even be expected to reduce selection for resistance as compared to that of a seven-day treatment interval. Weekly applications of 0,25% Toxaphene washes on susceptible *A. hebraeum* populations will provide seven-day protection against larval and nymphal stages and five days against adult stages[12]. Selection for resistance at this treatment interval is thus curtailed.

Engorged *A. hebraeum* larvae have been shown *in vitro* to be more difficult to kill than their unfed counterparts (Tables 1 & 3). In the writers' experience the same is true of all stages *in vivo*. On the host, larvae can be expected to feed to repletion within 6-8 days, nymphs 6-9 days and females 6-12 days[8]. At a 14-day treatment interval, therefore, all tick stages are likely to be present on the host in all stages of engorgement. Under the climatic conditions that exist in parts of these two regions, all stages may be found together on the host at any time of the year. Selection for resistance could thus be considerably enhanced at every 14-day interval treatment.

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TOEKENNING

AWARD

SAVV-GOUE MEDALJE: VYFDE TOEKENNING

Die Raad het in 1976 voorstelle ingewag vir hierdie hoogste toekenning van die Vereniging. Ooreenkomstig die Raad se eenparige aanbeveling is die SAVV Goue Medalje vir "UITMUNTENDE DIENS AAN DIE VEEARTSENY PROFESSIE" deur die President, Dr. B.H. Pappin, oorhandig aan

SAVA GOLD MEDAL: FIFTH AWARD

In 1976 Council invited proposals for the award of the SAVA's premium award "FOR DISTINGUISHED SERVICE TO THE VETERINARY PROFESSION". On Council's unanimous recommendation the President, Dr. B.H. Pappin, presented the SAVA Gold Medal to

DR. JOHN HUXLEY MASON

Die plegtigheid het plaasgevind tydens die Algemene Jaarvergadering in Bloemfontein in Sept 1976.

at the opening of the Association's Annual General Meeting held in Bloemfontein in Sept 1976.

John Huxley Mason is in 1899 te Glasgow gebore en het sy skoolopleiding daar ontvang. In 1920 gradueer hy as veearts (MRCVS) na studie aan die Universiteit Glasgow. Nadat hy as assistent in verskeie praktyke in die Verenigde Koningryk vir drie jaar werksaam was, keer hy terug na die Universiteit vir 'n nagraadse kursus in bakteriologie. Hierdie verdere opleiding stel hom dan in staat om vir 'n tydperk as lektor in bakteriologie aan die Glasgow Veterinary College op te tree.

In 1924 het Jack, soos hy aan sy kollegas en 'n wye vriendekring bekend gestaan het, by die personeel van die Wellcome Physiological Research Laboratories te

John Huxley Mason was born in Glasgow in 1899 where he subsequently received his scholastic education. In 1920 he qualified as a veterinarian (MRCVS) at the University of Glasgow. After serving as an assistant to various practitioners in the United Kingdom for three years, he returned to university for a postgraduate course in bacteriology. His higher qualification in the field of bacteriology enabled him to serve as lecturer in this subject at the Glasgow Veterinary College for some time.

In 1924, Jack, as he was known to his colleagues and wide circle of friends, joined the staff of the Wellcome

Beckenham, Kent, aangesluit. Destyds was die studie van die toksigene anaerobe bakterië nog nie ver gevorderd nie. In die Wellcome Research Laboratories het hy in aanraking gekom met welbekende navorsers op die gebied van anaerobiese bakteriologie. Tesame met persone soos Thomas Dalling, A.T. Glenny en Mollie Barr, om slegs 'n paar te noem, word Jack Mason as een van die pioniers in die verband beskou. Hy het dan ook 'n beduidende bydrae tot sy krediet geplaas deur sy rol in die verduideliking van die oordrag van immuniteit in herkouers d.m.v. kolostrum – iets wat vandag sonder meer aanvaar word.

Intussen is 'n Fellowship van die Royal College of Veterinary Surgeons in 1927 aan hom toegeken, en in 1928 is hy verkies tot 'n Fellow van die Royal Society van Edinburgh.

In 1931 is Jack Mason as Empire Marketing Board Research Fellow aan die Veeartsenykundige Navorsingsinstituut te Onderstepoort aangestel. In 1936, met beëindiging van die Fellowship, word hy 'n lid van die personeel van die Navorsingsinstituut. Op Onderstepoort het hy sy werk i.v.m. die *Clostridium spp* voortgesit en hom veral toegespits op hulle toksienes en die verwekking van immuniteit teen hierdie stowwe. Hierdie studies was toendertyd van besondere belang vir die veenywerheid. Sy navorsingswerk het egter ook hartwater, bloutong in skape en perdesiekte ingesluit. In 1936 verwerf hy die graad D.Sc aan die Universiteit van Suid-Afrika.

Gedurende 1940 het Dr. Mason by die personeel van die Suid-Afrikaanse Mediese Navorsingsinstituut te Johannesburg aangesluit. Daar was hy verantwoordelik vir die produksie van perd-antisera teen die toksienes van *Clostridium* spp wat die mens aantas, die toksien van *Corynebacterium diphtheria* en verskeie slanggifsoorte. Hy het *Salmonella typhi* bestudeer en die produksie van immuniteit teen tifoiedkoors van die mens. Verskeie ander onderwerpe is ook deur hom nagevors, o.a. vlooitifus en bosluisbytkoors in die marmot en die isolering van enkele bakteriese selle.

In 1955 is Dr. Mason bevorder tot adjunk-direkteur van die S.A. Mediese Navorsingsinstituut en in 1975 het hy met pensioen afgetree. Hy het gedurende sy dienstermyn aan die Instituut oortuigende bewys gelewer van die waarde van 'n veearts in 'n span mediese navorsingswerkers.

Gedurende sy aktiewe lewe as navorser het Dr. Mason bekend geraak as 'n laboratoriumswerker, 'n kritiese wetenskaplike en 'n persoon wat duidelik onderskei tussen dinge wat in die wetenskap belangrik is en dinge wat onbelangrik is. Hy is, alleen of met ander, die skrywer van 110 wetenskaplike publikasies. Onder sy kollegas en in sy wye vriendekring is hy bekend vir sy skerpsinnigheid en sy fyn humorsin.

Dr. Mason is nog altyd toegewyd aan die belange van sy professie. Hy was vir 4 jaar President van die S.A. Veterinêre Vereniging en is tans Ere-Lewensvisepresident. Sy lidmaatskap van 6 ander wetenskaplike verenigings getuig van sy wye belange.

Hy is deurgaans deur sy vrou bygestaan en aangemoedig. Dr. Mason is inderdaad die Vereniging se hoogste toekenning waardig.

Physiological Research Laboratories at Beckenham, Kent. In those days the study of the toxigenic anaerobes was in its infancy. At the Wellcome Research Laboratories he became associated with well-known research workers in the field of anaerobic bacteriology. Together with people like Thomas Dalling, A.T. Glenny and Mollie Barr, to mention only some, Jack Mason is regarded as a pioneer in this respect. A significant contribution which stands to his credit is his rôle in elucidating the transfer of colostral immunity in ruminants – something which is taken for granted in modern times.

In the meanwhile he obtained the Fellowship of the Royal College of Veterinary Surgeons in 1927, and was elected a Fellow of the Royal Society of Edinburgh in 1928.

In 1931 Jack Mason was appointed an Empire Marketing Board Research Fellow at the Veterinary Research Institute, Onderstepoort, and in 1936, on the expiration of the Fellowship, became a member of the staff of the Veterinary Research Institute. At Onderstepoort he continued his studies on the clostridial anaerobes, their toxins and the production of an immunity against them. These studies were of considerable importance to the livestock industry at that time. He also conducted research on heartwater and bluetongue in sheep and on horsesickness. During 1936 he obtained a D.Sc degree from the University of South Africa.

During 1940, Dr. Mason joined the staff of the Serum Department of the South African Institute for Medical Research at Rietfontein, near Johannesburg. Here he was responsible for producing antisera in horses against the toxins of the clostridia affecting man, the toxin of *Corynebacteriom diphtheriae,* and different snake venoms. He studied *Salmonella typhi* and the production of immunity to typhoid fever in man. Many other research topics e.g. flea typhus and tick-bite fever in the guinea-pig and the isolation of single bacterial cells, received his attention.

Dr. Mason was made a deputy director of the South African Institute for Medical Research in 1955 and retired in 1975. During his term of office at this Institute he proved convincingly the value of a veterinarian in a medical research team.

During his active research life Dr. Mason became known as a meticulous laboratory worker, a critical scientist and a person able to distinguish between the things that matter in science and those that do not. He is author or part author of 110 scientific publications. He is generally known among his many friends for his keen wit and sense of humour.

Dr. Mason has always been dedicated to the interests of the veterinary profession. He was President of the S.A. Veterinary Association for 4 years and is now an Honorary Life Vice President. His membership of six other professional or technical societies bears testimony to his wide interests.

Through all these times he has been supported and encouraged by his wife. Dr. Mason is undoubtedly a most worthy recipient of the Association's premier award.

FAO/UNEP/WHO FIELD CONTROL OF TAENIASIS & ECHINOCOCCOSIS: AN APPEAL

Dear Sir,

In June 1976, a Joint FAO/UNEP/WHO Consultation of experts was held in Nairobi, Kenya, covering all aspects of problems relating to *Taeniasis/Cysticercosis* and *Echinococcosis/Hydatidosis*. The special purpose of this Consultation was to formulate practical recommendations for an integrated, multidisciplinary, innovative, environmental approach to research leading towards better understanding and the planning of successful control of these two parasitic zoonoses. The resulting *"Report of the Joint FAO/UNEP/WHO Consultation on Field Control of Taeniasis and Echinococcosis"* is available, free of charge, to all interested parties.

Anyone engaged in any capacity on work on these conditions – whether the approach be administrative, laboratory, epidemiological, environmental or socio-economic – is cordially invited to communicate with the under-signed. The objective of this contact would be to establish links and to contribute to an urgently needed break-through in these challenging problems; *Cysticercosis* today is, not only a serious impediment to the development of sound livestock industry in developing countries, but is spreading to developed countries, due to tourist traffic, migrant labour forces and the influx of students as well as the unreliability of the standard sewer treatment, thereby posing a serious threat to well-established livestock industries throughout the world. *Hydatidosis* is still rampant in many parts of the globe. An urgent need exists for refining diagnostic procedures and the development of non-surgical means of prevention and/or treatment in humans. Similarly, the prevention of animal *Hydatidosis*, which leads towards heavy losses of badly needed protein, requires urgent action.

A Reference Library is being established, the principal aim of which is to serve the needs of those confronted with the problems of *Taeniasis/Cysticercosis* and *Echinococcosis/Hydatidosis* in developing countries. As a contribution to the Reference Library, any relevant material, whether statistical data, scientific reports, published or unpublished, would be gratefully received and would be of great value.

Dr. I. Mann
Consultant, FAO/UNEP/WHO
Field Control of Taeniasis and Echinococcosis
United Nations Environment Programme
P.O. Box 30552 NAIROBI Kenya

Cow infertility problems?

Now there's a simple and effective new treatment "Estrumate"—ICI's synthetic prostaglandin analogue

Due to its high potency as a leuteolytic agent, Estrumate has proven an important advance in the treatment of many infertility problems in cattle.

Estrumate has a wide margin of safety and is predictable in its effects.

"ESTRUMATE"—CLINICAL INDICATIONS

Suboestrus ("No Visible Oestrus")

The "Silent Heat" cow usually has normal ovarian cyclicity but the manifestation of oestrus is either very mild or absent, making it difficult to detect for the use of A.I. Production losses can often occur.

Two injections of Estrumate, 11 days apart, followed by fixed time A.I. will overcome this condition.

Chronic Purulent Endometritis (Pyometra)

When this condition exists and a persistent corpus luteum is present, successful treatment is possible with a single injection of Estrumate.

Removal of Mummified Foetus

The mummified foetus will remain in utero as long as the corpus luteum remains functional. An injection of Estrumate can result in the expulsion of the mummified foetus into the vagina—where it is then removed manually.

ADMARK 1592

Termination of normal but unwanted pregnancies

Where accidental mating of very young or immature heifers has taken place, considerable economic loss may be experienced. Termination of pregnancy up to the 150th day can be affected by a single injection of Estrumate.

Luteal Cysts

In treatment of cystic conditions of the ovary, where the condition is due to luteal cysts, a single injection of Estrumate will result in luteolysis followed by the onset of oestrus.

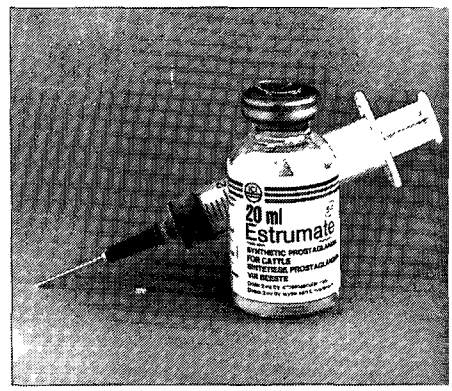
Other Uses

Estrumate has also been successfully used for synchronising of donors and recipients for ovum transplant.

Estrumate may also be used for regulating oestrus in cows to avoid "heat" periods during transportation, exhibiting at shows, etc.

Estrumate has already proven its worth to veterinarians and their clients in a number of countries in combating infertility problems such as these. Now it is available to you.

For further information, write to the address below or contact your local ICI representative or veterinary wholesaler.



ICI **ESTRUMATE**

Veterinary Products Division, ICI House, 1 Leyds Street, Braamfontein, Johannesburg. Tel. 725-4800.

IN MEMORIAM

JOHN ROBERT FREAN
7.5.1900-23.10.1976

John Robert Frean, MRCVS, was born at Ventersdorp, Transvaal on 7 May 1900. Shortly afterwards a detachment of British troops arrived. "Oh, what a tiny baby," said an old Tommy, "just like a pom-pom shell". For the rest of his life he was known as "Pom".

Pom went to a Milner school at Ventersdorp. From there he went to St. Andrew's College, Grahamstown, and later to the Potchefstroom Boys' High School.

In 1917 he proceeded to Britain where he joined the Royal Flying Corps and had scarcely got his wings when the war ended. He then enrolled at the Royal Dick Veterinary College, Edinburgh, in 1919. After qualifying he was engaged in the current foot and mouth disease campaign in the British Isles.

On his return he was quarantined in Cape Town docks because he had contracted FMD, one of the few humans to do so. One of his sisters visited him there and wrote back to the family that he did not look too bad but his Scots accent was terrible!

He joined the Government service and first went to Vryburg where he met his wife, Amalie Marie Meissner.

He was transferred many times, from Vryburg to Sabie, Lydenburg, Stellenbosch, Dundee, Ixopo, Ladysmith, Potchefstroom, Ermelo, Rustenburg, Potchefstroom and Robertson. At Potchefstroom part of his duties was the teaching of veterinary science at the College of Agriculture, where he was extremely popular with both students and staff. His final post was at Onderstepoort where he acted as Public Relations Officer until 1969. He retired to Park Rynie on the South Coast of Natal.

His devotion to duty and love of animals were characteristic of Pom. He had a strong sense of humour and a rich fund of stories, which he usually told with relish in a broad Scots accent.

In spite of failing health in later years, he continued to the last with a modest small animal practice, more out of love for animals than for material gain.

An extract of his obituary in the local church newsletter, by his parson, sums him up very aptly: "A honey of a man, a saint of a man. His passing is to me a personal loss .... His ministrations to God's dumb creatures was a lesson in ministry .... A man whose kindly heart o'er flowed to all distressed – be it of creature, kind or human species."

With his passing, a further link with the past has been severed and to his widow, May, and two sons, John and Robert we extend our sincere sympathy.

Pom served his country well.

CANDUR

CANDUR prevents distemper, hepatitis and leptospirosis

Researchers at the Behring Institute established that by combining an inactivated hepatitis virus with a live distemper virus there is a potentiation of the antigenicity of the distemper fraction, giving a firm, long lasting immunity against distemper, and a solid immunity free from side effects against hepatitis.

without CANDUR, puppies wouldn't stand a dog's chance

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SILASTIC JOINT PROSTHESES

Dr. Thomas David, a veterinary specialist from Vienna, has discovered a new and successful method of implanting artificial joints in dogs and other domestic animals suffering from articular diseases. Now it is possible to implant an artificial joint made from surgical silicone (silastic). Silastic is not rejected by the body; it merges organically with the bone without the need for any kind of adhesive which might later be subject to fatigue and break.

Silastic powder, when mixed with a catalyst, forms a paste which soon assumes a malleable consistency and can be easily moulded. Even when silastic is completely set, it can still be cut with a sharp blade so that its final form can be tailored to meet individual requirements. The two sections of the diseased joint are removed and the exposed sections of bone hollowed out somewhat. The sections of the artificial joint, moulded beforehand on the basis of X-ray photographs, are then inserted. The resumption of movement in the joint means an end to the muscular atrophy which automatically sets in with immobility. Muscles and tendons remain unaffected by the operation since they are not attached to the joint itself but to the adjacent bone at points some way above and below the joint.

The artificial joint is considerably superior to those used until now. Since there is no friction at any point, there is no pain. The joint is more efficient than any other and costs only 200 Austrian Schillings, compared with 20 000 to 40 000 Schillings for the traditional artificial joint.

This development also opens up new perspectives in the field of human surgery. The perfection and the low cost of this new aid are unparalleled. Hospitals need only send X-ray photographs of a diseased joint to a central workshop where a replacement can be made to measure.

(Fed. Press Dept., Rep of Austria Je0051/0004F 28/6/1976)

THE AGRICULTURAL AND VETERINARY CHEMICALS ASSOCIATION OF S.A.

The Agricultural and Veterinary Chemicals Association is an association of manufacturers. Its members are responsible for the manufacture and marketing of the overwhelming majority of products to be found in the field of agricultural and veterinary chemicals. The aims of the Association can be summed up as follows:

1. To promote and maintain the highest production and marketing standards, thereby ensuring that products of the highest quality are consistently available.
2. Initiating and maintaining codes of practice for the industry to ensure that service to the consumer and to the different professions remains the primary consideration of members. The Association encourages a healthy exchange of knowledge and ideas between members to the ultimate improvement of the industry's standards and of the service provided by members. The Association attempts to promote constructive relations between its members and to protect the commercial interests of members in general, provided, however, that the Association will not, under any circumstances, interfere with the internal affairs or the individual commercial policies of its members. The Association will not seek to advance the interests of a particular member to the detriment of the industry as a whole.
3. To negotiate with government departments, institutions and agencies on those matters affecting the industry and in this respect the Association represents its members in discussions with all authorities and bodies in the field of agricultural and veterinary chemicals, including professional and consumer organisations.
4. To ensure that adequate supplies of strategically important products are available at all times.
5. To correct many problems encountered by its members and the industry as a whole in the field of agricultural and veterinary chemicals.

The current Executive Council consists of Mr. A.N. Tooley (Hoechst SA) (President), Mr. J. Mullen (Vice-president) and Messrs. T.R. Conroy, L.S. Nayler, L.M. van Essen and Dr. P. Pullinger.

The following firms are members of the Association:

Agricura Limited
Agritek Triomf (Pty.) Limited
Bayer S.A. (Pty.) Limited
Beecham Animal Health
Carst & Walker Chemicals (Pty.) Ltd.
Celamerck (Pty.) Limited
Chemveld Agricultural Services (Pty.) Ltd.
Ciba-Geigy (Pty.) Limited
Coopers (S.A.)(Pty.) Limited
Datons Insecticides & Fungicides (Pty.) Ltd.
Ethnor Laboratories (Pty.) Limited
Fisons Agrochemicals (Pty.) Limited
Goldfields Veterinary Medical Supplies (Pty.) Limited
Hoechst S.A. (Pty.) Limited
ICI S.A. (Pharmaceuticals) Ltd.
Lilly Laboratories (S.A.)(Pty.) Ltd.
Maybaker (S.A.)(Pty.) Limited
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Monsanto S.A. (Pty.) Limited
MSD (Pty.) Limited
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Pfizer Laboratories (Pty.) Ltd.
Repvet (Pty.) Limited
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Shell Chemical S.A. (Pty.) Ltd.
Smith Kline Animal Health Products
Taeuber & Corssen (Pty.) Limited
Triomf Crop Protection (Pty.) Ltd.
Union Carbide (S.A.)(Pty.) Ltd.