IMPORTANT ECONOMIC DISEASES IN BUFFALOES

LÁU, H.D.

EMBRAPA-CPATU
66.075-900, Belém, Pará, Brazil

INTRODUCTION

The actual world population of buffaloes is probably 150 to 180 thousand of heads. India has nearly 50% of the world population of this animal species. The buffalo has come to occupy an important place as a milch, meat and draught production in certain developing countries. During the past 10 years, much research were contributed for the scientific knowledgement of buffalo. Investigative works on physiology, production, reproduction and nutrition has been conducted in many countries. However, less attention has been given on pathology these animals. This probably occur due to the general opinion that diseases of buffalo are identical with those of cattle and that buffaloes showed high resistance to a number of these diseases. The literature reveals however that the aetiological agents of infections, infestations, deficiencies and injuries really are the same for both animal species, but in respect of a buffalo pathologic process there are differences on the prevalence, pathogenicity and symptomatology. Therefore, the clinical measures for diagnosis, prevention and control of buffalo diseases are not quite similar with those of cattle. This paper reports on principal disease problems with cause economic losses in buffalo production.

PARASITIC DISEASES

Ascariasis

Caused by *Toxocara* (Syn. *Neoascaris*) *vitulorum*, a large round intestinal worm. It is the most common and pathogenic parasite of young buffalo calf. It is the major cause of buffalo calves mortality in countries like Pakistan (1), Ceylon (2), India (3), Malay (4), Egypt (5), Philippine (6), Thailand (7) and Brazil (8). Patent infections are most common in calves less than three months. In not controlled buffalo herds, the morbidity and mortality rates in this age group are often approaches 100% and 40%, respectively. Thereafther it declines rapidly and the infections are rare in animals over the age of six months. Many authors (9), (10), (11), (12), (13), (14), (15) have attributed that the usual route by with buffalo calves become infected is tansmammary via. The infective larvae present in the colostrun are transmited from cow to calf.

Index terms: Buffalo pathology, Clinical findings, Parasitic disease, Infection disease.

Others authors (3), (16) reported the prenatal infection. The foetus is infected by ingestion of larvae present on amminiotic fluid. The infected animals become extremely emaciated and unthrifty, showed smelling coulored watery faeces, rough air, anorexia, convulsions and coma. The principal cause of death is due to obstruction of the intestinal tract caused by adult worm balls. Some authors (17), (18), (19) have associated coliform (*Escherichia coli*) with the mortality of paratised animals. The control measure depend exclusively on the regular deworming scheme, because the larvae are transmitted to calves through to dam. However, hygienic condition is fundamental on prophylaxis measures. (20) have indicate on suitable deworming program in buffalo calves before weaning at 15, 30, 60 and 180 days old, orally.

Coccidiosis

High mortality ocurrence of buffalo calves infected by *Elmeria* species (*E. zuernii, E. bovis, E. auburnensis, E. ellipsoidalis, E. bareilly*) is reported in Ceilon (21), India (22), Italy (23), and Brazil (24). Coccidiosis is commonly a disease of young buffalo from 1 or 2 months to 1 year of age, specially in herds under insanitary conditions. The disease usually is most prevalent durig the winter (25) or during the less raniny period (24). The pathogenic coccidia may cause damage to the mucosa of the lower small intestine, cecum and colon. In light infections the most characteristic sign is watery faeces and little or no blood is apparent in the faeces. Severely affected animals may develop a diarrhea consisting of thin bloody fluid or thin faeces containing streaks or clots of blood, sherds of epitelium and mucus. The buffaloes loses it is appetite becomes depressed and dehydrated, loses weigth and the hindquarters and tail become soiled with fecal discharges. Death may be occur during the acute period. Prophylaxis of clinical coccidiosis is based on good feeding practics, good mamagement and attention to the principles of animal sanitation. Treatment with sulfonamides give satisfactory results agaist the coccidiosis.

Parasitic gastrointestinal

Infection by *Trichostrongyles* nematodes in buffaloes have been recorded in India (26; 27; 28), Yougoslavia (29), Australia (30), Egypt (31) and Brazil (32; 15). Subclinical infections with consequent stunting and unthriftiness has long been described as the classic picture of gastrointestinal parasitism in buffaloes. The most important stomach worm are *Haemonchus contortus* and *Trichostrongylus axei*. Though losses from these parasites have occurred more commonly in young than in mature animals. In the small intestine are common *Bunostomun plebotomun* and *Cooperia punctata*. In the large intestine, *Oesophagostomum radiatum* is more numerous. Anemia, anorexia, weakness, diarrhea, dehydration and progressive weigth loss are the outstanding sings of this disease. For the preventive control it is necessary the treat the entire herd at certain periods of the year to prevent accumulation of debilitating infection. The time for these routine treatments should be determined on the basis of the conditions in the area and the specific herds. Buffaloes with remaining in swampy areas of Amazon region do not suffer from helminthosis, probably because nematode eggs may fail to develop due to submersion in water and lack of oxygen. Adequate nutrition is most important in control of parasitic gastrointestinal disease, because to increase the resistence of animals to parasitisms or to the effects of parasitic infections. .

Fascioliasis

According to Griffiths (33) the buffalo in it is natural habitat, in which the snail vectors of trematode parasites are likely to occur in large numbers, is subject to heavy infection with *Fasciola* species (*F. hepatica, F. gigantica*). In many countries such as Philippines (34), Egypt (35), Singapore (36), Turkey (37), Taiwan (38), Brazil (39), Iraq (40), Pakistan (41) and India (42) high economic losses due to fascioliasis occur in form of reduced milk production, poor body weight, poor carcass quality and condenation of livers. A positive correlacion of dissease incidence to minimum temperature, morning and evening humidity and raifall has been recorded. The occurrence was mor frequently recorded in adult buffaloes below two years of age. In Brazil, fascioliasis was been seen in South region, whereas in Northern region (Amazonian) pratically not exist mybe because nedd a multifactorial system which compuses hosts, parasites agents and transmission process.

Pediculosis

Lice infestation caused by the sucking louse *Haematopinus tuberculatus* is most serious buffalo ectoparasitic disease in many parts of the word, such as India (43), Pakistan (44), Brazil (45), Egypt (46) and Argentina (47). The infestations are determined more by the type of dairy farm (unhygienic farms). That louse infestation lower milk production and tard gains in dairy herds. Calves usually harbour more lice than adults. Large infestations with this parasite contribute to unthrifiness, irritation and worry caused by th constant presence on the body and their bites. The host does not appear to suffer so much from the loss of blood suked. The adult female lays eggs firmily attached in very large numbers to the sparse hairs of the parasited animals. Infestations within a herd usually are not uniform; some animals are heavily infested, others are practically free of louse. The sunlight, the high skin temperature and the dry season limits the louse population in tropics. The usual method of transmission from one host to another is by contact. Excellent results have reported by (48) in control with Trichlorphon wasches. Two treatment was necessary; the first killed all nymphs and adults; the second 18 days later, killed all lice hatching out. A dose of 0.4 mg of Ivermectin per kg of live weight subcutaneousy also presented better result in control of this ectoparasitic disease.

Acariasis

Mange in buffaloes caused by genus *Sarcoptes* has long been recognised as a common dermatological problem in India (49). Usually the impact of mange in buffalo lies on their general health, growth and productivity, but in severe infestations, especially in young stock, the disease may terminate fatally (50). The disease is more common in animals below the age of one year and in confided animals. Fluctuations in environmental temperature affect the incidence. The prevalence is higher in minimum temperature. Relative humidity not affect the incidence level. The lesions caused by mange consist in numerous papules followed by formation of haemorragic crusts. Hairs started falling out. The lesions was present principally at the base of horns and region of auricula nears, may be because the physiological buffalo habits of wallowing in water. It is contended that body regions remaining above the water during wallowing. However the lesions also appear on dorsum, venter and legs. Accordig to

(51) and (52) the treatment was made through spray aplications with inseticides. Since the mite develop from egg to ovigerous female in 10 days (53), three aplications seen to be necessary at the weeky intervals. One or two aplications did not give complete cure.

Trypanosomiasis

The infection caused by *Tripanosoma evansi* has been in buffaloes in India (54, 55, 56) and by *T. vivax* in Brazil (57, 58, 59, 60, 61). The infection in buffaloes is usually latent and symptomless. Natural habitat of buffalo in swampy and marshy land areas is favourable for transmission by tabanid flies. The first evidence of the infection being rapid and progressive emaciation with depression and progressive anaemia. The hind quarters sway from to side with knuckling of the pasterns. Temperature is high at certain stages but is liable to considerable fluctuation every four or five days. Oedema of the eylids accompained by mucopurulent conjutivitis is a another common syptoms. The urine is yellow and turbid and occasionally contains albumin and blood. Usually the disease runs a chronic course, lasting from 1 to 6 months. It is probably transmitted from animal to animal solely by the bites of flies and sucking lice. Treatment consists of isolating individual cases, meatime treating with injections of Diminazene and Oxytetracicline (61) or Berenil at the rate of 0.8/100kg body weigth (62).

Filariasis

Incidence of different filarial worms genus (*Setaria, Stephanofilaria, Onchocerca, Parafilaria, Eleophora, Thelazia*) was reported in buffaloes in some countries such as India (63, 64, 65, 66), Egypt (67,68, 69), Australia (70), Brazil (71) and Philippines (72). The clinical symptoms are diverses, in conformity with the parasitic genus. Dermatitis, bursitis and aortitis are the most common symptoms. Nodular and haemorrhagic cutaneous eruptions is quite familiar in buffaloes paratised by *Parafilaria bovicola*. The nodules usually appear on the axilla, groin and prepuce. The filaricid treatment with A large number of drugs includind ditrazin, sulphoxide , thiabendazole , azinidine, berenil, chlorophos and ivermectin have been tried for the treatment of filariasis. The transmission is by vectors such as tabanids , lice and mosquitoes.

Horn fly

Of the various species of flies that infest and injure buffaloes, the principals are blood suckers with causing annoyance and pain due to their bites. The Horn fly (*Haematobia irritans*), introduted into South America through Colombia and now is found in Brazil. is the serious problem for the buffalo herds. The annoyance and irritation caused by horn flies are responsible for considerable losses during the season when the flies are abundant. It interfers with host feeding, resting and others normal activities. Buffaloes often refuze to graze during the day and seek protection by hinding in brush or tall grass until nightfall when the flies are less active. High populations of the parasites occur in Marajó Island. The black color of buffalo apear attracted the fly, however the animal odor appear to chase away the parasitic. More prevalence at the beginning and end rainy season. The control is basically carried out using spray insecticides.

BACTERIAL DISEASES

Brucellosis

There is a general impression that bacteria *Brucella abortus* was less prevalent in buffaloes than in cattles. However the brucellosis has been considered one of the most important zoonotic disease of buffalo in some countries such as Egypt (73), Italy (74), Brazil (75), Pakistan (76) and India (77). Associated with abortion, retention of placenta and infertility, the brucellosis cause serious economic impact on buffalo production. The diagnosis of brucellosis based on the presence of Brucella antibodies in the blood serum apparently is doubful and needed further investigation. According to (78) the tube agglutination test results in false negatives findings and the card test result in false positives findings. The rivanol test appeared to be superior to others tests in accurancy. According to (79) counter-immunoeletroforesis test is mor suitable for the diagnosis of brucellosis in buffaloes. No practical effective treatment is known and efforts are directed at control and prevention. Vaccination with *B. abortus* Strain 19 is widely used in buffaloes and is effective in increasing resistence to infection. Vaccinated animals are usually resistant to infection (celular immunity) for about 8 at 10 years. Another vaccine *B. abortus* 45/20 bacterin in adjuvant, has gained widspreat acceptance in some countries. According to (80) that vaccine may be induce immunity comparable with that of Strain 19.

Tuberculosis

The buffalo was susceptibile to *Mycobacterium bovis* as the catle. However, the disease in buffaloes is less prevalent in some countries, may be because their extensive mode of life (81, 82). Among the conditions of environment that aid the development of tuberculosis are stabling with lack of ventilation, damp buildings and the keeping of many animals together. The tuberculin test show a greater degree of non-specific allergic reaction, probably because of their habit of wallowing in mude, locals with greater number of bacteria similar to *M. bovis*. Therefore the simultaneous tuberculin testing (mammalian and avian PPD) are recommended in doubtful cases. The interpretation of tuberculin test based upon accepted protocols in cattle is incorrect, because the severity of the reaction was much greater than usually seen in cattle. The oedema is more extensive. In the buffaloes positive reactors the mammalian tuberculin reaction were hot, oedematous and painful. Accordig to (83) tuberculous lesions in buffalo were most commonly found in the retropharyngeal and mediastinal lymph nodes. (84) reported to the antituberculosis drug isonicotic acid hydrazide there was no practical therapeutic agent for yhe treatment of buffaloes tuberculosis.

Haemorrhagic septicaemia

There is general agreement that buffaloes are more susctible to haemorrhagic septicaemia caused by *Pasteurella multocida* than cattle, althougth quantitative evidence is scarce. The disease seriously affects buffaloes especially in India (84), Ceylon (85), Japan (86) Philippines, Indonesia, Malaysia and Thailand (87). Accordig to (88) low sporadic occurrence is reported from some European States, as well as the

URSS. Endemic status is also reported in many South American States, but no confirmation of the disease by serotype identification has been reported. This disease is most prevalent in situation where husbandry practices are poor. Is usually associated with wet, humid weather and an increased incidence is recorded during wet seasons. Most countries recognize higher morbidity rates among animals under 2 years of age (89). In a typical case of pasteurellosis, the first obvious lesions is subcutaneous oedema with serogelatinous fluid particularly in the submandibular throat and brisket region. The subcutaneous connective tissues may be dotted with petechial haemorrages and the lymph nodes may be swollen. Once visible signs appear treatment is of little value. The only practical method is to commence treatment immediately as a temperature elevation is observed. At this stage antibacterial therapy is effective (87). Sulphadimidine intravenously and broad spectrum antibiotic intramuscularly are effective and convinient (90). In the several types of vaccines are used. Alum precipitated and aluminium hydroxide gel vaccines are presentely the most widely used vaccines. Immunity for 4 at 6 months is generally claimed.

Mastitis

Udder infections is the important economic disease confronting the dairy buffalo farms, althought the incidence of mastitis in buffaloes is much less than in cattle. In spite of the teats of buffaloes is relatively pendulous and long, more liable to injury than cows, the teat sphincters contain more smooth muscle, fibres and blood vasels than of cows which might function as a better barrier against infections. Buffaloes was affected most from subclinical mastitis. The most adequated tests for diagnosis of subclinical mastitis in buffaloes are Modified Whitside Test, California Mastitis Test and Cloride Count. These tests are not quite satisfactory for the pre-detection of clinical infection of udder , however, their adoption in the routine examination did help in sorting out the animals likely having mastitis (91). According to (92), the main organisms responsable for latent mastitis in buffaloes are staphylococci (74.71 %) and streptococci (21.13 %). Treatment with appropriate antibiotic intramamary result in complete recovery. The most important single factor for a successful treatment of mastitis is the complete and almost continuous removal of secretion from the affected quarter.

Blackleg

The disease caused by *Clostridium chauvoei,* usually occurs in raining station and areas where flood and swamps. Most cases occurs in animals from 6 to 18 months of age, but some cases in young calves as 6 weeks was been seen. The symptoms was caracterized by emphysematous swelling in the heavy muscles. Blackleg develop spontaneously and generally in best buffaloes of their group. Outbreaks occur in which a few cases found each day for several days. The occurrence of a rapidly fatal febrile disease in wellnourished young animals with crepitant swellings of the heavy muscles suggests a diagnosis of blackleg. The affected muscle is dark red to black, dry and spongy, has a sweetish odor. With few exeptions the disease terminates fatally in 12 to 36 hours after the first appearance of the symptoms. In the control, calves should be vaccinated twice, 2 and 6 months of age. In high-risk areas revaccination may be nacessary at one years thereafter. Treatment of clinical cases may be attempted with penicillin in large doses parenterally.

Leptospirosis

The disease has assumed economic importance with intensification of buffalo dairy herds in many countries. Caused by one or more leptospial serotypes principally *Leptospira pomona*, the disease is usually asymptomatic in older buffaloes. However in acute form the symptoms are prostration, anorexia, haemoglobinuria, anaemia and icterus. The common mode of natural infection is from contact with urine or by intake of urine-contaminated feed or water. No form of treatment will have much effect on the course of the disease.

VIRAL DISEASE

Foot and mouth disease

The disease generally assumes a milder form in buffaloes as compared to catle. The susceptibility vary according to the vaccination status, virulence of the virus, age of animals and certain streess factors such as poor nutritional status. The disease in buffaloes usually have a shorter duration with rapid recovery. The clinical signs of the disease was similar to those commonly observed in cattle, but the mouth lesions may be more severe than on the feet. In acute cases the buffaloes started for long period wallowed in water or mud. The fever and the difficulty of eating cause a rapid loss in flesh. Affected buffaloes may suffer from a considerable drop in a milk yeild. Secondary bacterial invasion of the rupted vesicles, particularly on the feed may occur. The period of incubation is variable, usually from 3 to 6 days. In exceptional cases, it may not appear for 18 to 20 days. Chemically inactived vaccines prepared from virus propagated in cattle and suspended in an adjuvant have been uded with considerable success in some countries. According to (93) although the buffaloes may ppear to recover completly from FMD infection, yet a number of animals usually become carrier for long period the virus, serving as focus for new outbreaks of the disease.

Rabies

The disease occur ocasionally in buffaloes in areas where is enzootic. Pratically, all cases of buffalo rabies are transmitted by Vampire Bat *(Desmodus rotundus),* via saliva itself is infected. The commonest sites of bitten are around the eyes and vulva region. Symptoms do not usually follow the picture of furious rabies. Buffaloes generally shows symptoms of ataxia or incordination with a wide based stance of the forelegs. Tympany, grinding of the teeth, anorexia and constipation also has been seen. Death soon superveness. Buffaloes usually showed th symptoms 20 to 25 days after infected. Prevention was made with annual vaccination on all animals.

Buffalo Pox

The disease appear to be specific to buffaloes and has been reported in epidemic form, in some countris such as India (94), Indonesia (95), Italy (96) and Pakistan (97). Was considerd to be caused by cow-pox virusesbecause of it is close clinical proxinity to cow-pox virus infections (98). The disease is zoonotic in nature since man gets infected through contact with infected animals or material. The typical pox lesions occur on the teats, udder and around the nostril, however in generalized form the

lesions under various stages are present all over the body. Thickening of the teats, stenosis of the milk ducts and mastitis are a common sequel to this infection (99). Although the disease is considered an economic disease for many researchers, programe to control it have yet to be formulated. There is need to develop a suitable vaccine and to carry out epidemiological studies in order to envolve an effective programme for controlling it. According to (94), the treatement with regular antiseptic and antibiotic dressings result in complete recovery.

Cutaneous papillomatosis

The disease, commonly known as warts is a wide spread disease with considerable economic importance at least for the hide industry. The cause of warts in buffalo was established as a virus. A favorite location for the papilloma in buffaloes are the shoulder, chet and neck. These are usually much softer and are well supplied with blood vasel. It is not uncommon for them to be penduculated or stemmed. Their color is cloudy gray or greyish-brown. Their consistence varies. Traction, ligation or surgery are not always practicable measures in dealing with skin papillomatosis in buffaloes. The spontaneous recovery was oftenly delayed for as 18 to 24 months. The treatment by auto-haemotherapy had good response to papillomatosis cure.

REPRODUCTIVE DISORDERS

Various reproductive anomalies of the male and female buffaloes have been recorded (100, 101, 102,103,104,105,106). The majority of abnormalities are anatomical and functional and are commonly encountered on female animals. In the pluriparus buffaloes the most common obtetrical problem are prolapse of vagina and retention of placenta. In the primiparus buffaloes, cervico-vaginovulvar lesions and dystocia are the most big problems. Metritis and infertility are the common sequels of this reproductive disorders. Usually dystocia is due to abnormal position or posture of the foetus (56.62 %), torcion of the uterus (27.72 %), caquexia (9.64 %) and embriopaties (6.02 %). The incidence of retained placenta was highest during the rainy season. Stillbirth was significantly higher among buffalo calving for the first time. The sex of the calf had influence on incidence of different types of calving disorders. The incidence of stillbirth, distocia and prolapse of the uterus was significantly higher among heifers carrying male calves.

MINERAL DISTURBANCE

Buffaloes on severe phosphorus deficient ares are most affected by lack of this element than of energy and protein. According to (107) a phosphorus deficiency is manifested by excessive proliferation of uncalcified bone, low growth, lamness and enlarged joints. It is commonly observed under field condition caquexia (100 %), anorexia (84 %), bristed and dull hair (78 %), claudication (34 %), sustenance in the carpal articulation (23 %), kiphosis (17 %) and allotrophagy which leads to ingestion of wood, dirt and rocks (6 %). Anestrus and low conception rates may be occurs. The phosphorus content of the milk does not decline. Calcium deficiency is not usually oberved in buffaloes. A simple mineral mixture with macronutrients like phosphorus,

calcium, sodium, potassium and cloride and with micronutrients such as copper, zinc, fluorine, iodine and manganese are of concern in buffalo production and health.

POISONOUS PLANTS

Probably more animals are poisoned from this source than from all others combined. Plant poisoing may result from a shortage of more palatable feed, allowing hungry animals to graze in ares where poisonous plant exist or carelessness in leaving poisonous plant material. In Amazonian region (108) described extensive photosensitization in buffaloes intoxicated by *Lantana camara* and *Pithomices chartarum*. Thhe animals also showed jaundice, rogressive ematiatin, uneasiness and anorexia. Many deaths also was been seen in buffaloes intoxicated by *Arrabidaea bilabiata*. Buffalo intoxication from *Mimosa invisa* (Var. *inermis*) was related by (109). The symptoms include salivation, stiffness, lack of mastigation, muscular tremor, dyspnea and killed after the animals had become recumbent. The toxic elements of this plant are cyanide and nitrite.

CONGENITAL ABNORMALITIES

In buffaloes all congenital malformations are not genetically initiated. They have been also attributed to maternal nutritional deficiency, toxic substances and others enviromental factors. May affect a single structure or function, involve several body systems or combine structural and functional alterations. May be lethal, semilethal and non-lethal. The body system mor affected is musculoskeletal (53.7 %). The common defects are agnathia, brachygnathia, prognathia, arthrogryposis, spastic paresis and hernia umbilical. On the intugmentary system the principal deffects are hypotricosis, photosensitvity and albinoidism. On the occular and digestive systems are anophtalmia and atresia ani, respectively.

CALF DISEASES

Diseases of buffalo calves has been posing a serious problem in the buffalo breeding herds. Large morbidity and mortality rates are higher in new born animals than in any other age group. Accurate calf-mortality figures are unavailable for the total buffalo population, but it is well recognized that calfhood disease takes a terrific toll. Mortality is estimed to be 31.45 % in India (110), 37.70 % in Egypt (111)and 13.80 % in Brazil (112). High mortality rate occur in the age group of 1 to 3 months. Mortality was maximum (73.76 %) during dry-hot period. Young calves are suscptible to many diseases and frequenttly suffer from several problems, but ascaridiasis caused by (*N. vitulorum*) is the prime importance. Ascariasis, coccidiosis and *Escherichia coli* infection was responssible for fatal enteritis. Onphalophebitis is not of major aconomic imporance but it can be and occasionally is a serious problem in individual herds. The poor management and unhygienic conditions are principal factors with contribute for the highest mortality rate of buffalo calves.

CONCLUSIONS

Much scientific information about prevalent diseases and parasites in buffaloes is necessary. Until very recent time buffalo reseach has been sparse and limited to only a few situations and sites. A number of obcure diseases await elucidation. Before the magnitude of buffalo disease problems and economic losses suffered as a result from this diseases, it is hoped that more efforts would be directed towards the diagnosis, prognosis, treatment and specific measures of immunization and chemoprophylaxis. Good management and nutrition programs thereford are essential for preventing development and spread the buffalo disease.

REFERENCES

1. SARWAR, M.M.; NAWAZ, M. **Agric. Pakistan, 2**: 74, 1951.
2. SINNIAH, S.A. **Ceylon Vet. Ser. Rep., 2**: 20, 1954.
3. DAS, K.M.; SINGH, G.B. **Brith. Vet. J., 3** (8): 342, 1955.
4. LANCASTER, W.E. **J. Malay.Vet. Med. Assoc., 1**: 151, 1957.
5. SELIM, M.K. ; TAWFIK, M.A.A. **Indian Vet. J., 43**: 965, 1966.
6. DE LEON, D.D.; JUPLO, R.J. **J. Parasitol., 52**: 1214, 1966.
7. SUKHAPESNA, V. **Buffalo Bull., 2** (1): 12, 1983.
8. LÁU, H. D. EMBRAPA-CPATU, 1987. 12P. (EMBRAPA - CPATU. Boletim de Pesquisa, 83).
9. WARREN, E.G. **Intern. J. Parasitol., 1**: 85, 1971.
10. CHAUHAN, P.P.S.; AGRAWAL, R.D.; AHLUWALIA, S.S. **Current. Science, 43** (15): 486, 1974.
11. MIA, S.; DEWAN, M.L.; UDDIN, M.; CHWDHURY, M.U.A. **Trop. Anim. Health Prod., 7**: 153 - 1975.
12. GAUTAN, O.P.; MALIK, P.D.; SINGH, D.R. **Curr. Scince, 45** (9): 350, 1976.
13. BANERJEE, D.P.; BARMAN ROY, A.K.; SANYAL, P.K. **J. Parsitol., 69** (6): 1124, 1983.
14. CONNAN, R.M. In: GAAFAR, S.M.; HOWARD, W.E.; MARSH, R.E. **Paras. Pest and Predators.** Amesterdam. Elsevier Science, 1985. p.265.
15. LÁU, H.D. EMBRAPA-CPATU, 1993. 38p. (EMBRAPA-CPATU. Documentos, 72).
16. SUKHAPESNA, V. **Buffalo Bull., 4** (4): 77, 1985.
17. VERMA, P.C.; KARLA, D.S. **Indian Vet. J., 52**: 605, 1975.
18. CHAUDHRY, N.I. **Pakistan j. Science, 30**: 120, 1978.
19. LÁU, H. D. EMBRAPA-CPATU, 1987. 12P. (EMBRAPA - CPATU. Boletim de Pesquisa, 83).
20. LÁU, H.D. EMBRAPA-CPATU, 1984. 15p. (EMBRAPA-CPATU. Circular Técnica, 49).
21. SHANMUGALINGAM, K.; SENEVIRATNE, P. **Ceylon Vet. Serv. Reporter, 2**: 151, 1954.
22. BHATIA, B.B.; PANDE, B.P.; CHAUHAN, P.P.S.; ARORA, G.S. **Acta Vet. Hung., 18**: 115, 1968.
23. RESTANI, R.; TASSI, P. **Atti Soc. Ital. Sci. Vet., 23**: 881, 1969.
24. LÁU, H.D. EMBRAPA- CPATU, 1982. 11p (EMBRAPA - CPATU. Boletim de Pequisa, 42).
25. KPAHRA, S.S.; SINGH, J. **Buffalo Bull., 5** (1): 9, 1986.
26. PANDE, B.P.; CHAUHAN, P.P.S. **J. Anim. Sci., 39**: 79,1969.
27. BHOPALE, K.K.; JOSHI, S.C.; KALAMAPUR, S.K. **Indian J. Anim. Res., 5** (1):19, 1971.
28. CHAHABRA, R.C.; GILL, B.S. **J. Res. Punjab, 12** (2): 184, 1975.
29. PAPILOVIC, W.H. **Vet. Glasnik, 29** (3):209, 1975.
30. BRYAN, R.P.; BAINBRIDGE, M.J.; KERR, J.D. **Australian J. Zool., 24**: 417, 1976.
31. TAWFIK, M.A. **Assiut Vet. Med. J., 3** (5):161, 1978.
32. STARKE, W.A.; MACHADO, R.Z.; HONER, M.R.; ZOCOLLER, M.C. **Arq. Bras. med. Vet. Zoo., 35** (5): 665, 1983.

33. GRIFFTHS, R.B. In: COCKRILL, W.R. **The husbandry and health of the domestic buffalo.** Rome. FAO. 1974. p 236.
34. REFUERZO, P.G. **Philipp. J. Sci., 77**: 25, 1947.
35. EL-SHERIF, A.F.; ABODOU, A.H.; EL-SAWI, M.F. **J. Egypt. Vet. Med. Ass., 19**: 211, 1959.
36. BALASINGAM, E. **Ceylon Vet. J., 10**: 10, 1962.
37. GÜRALP, N.; OZCAN, C.; SIMMS, B.T. **Am. J. Vet. Res., 25**: 196, 1964.
38. OTTE, E.; SHIEN, Y.S. **Mem. Coll. Agric. Nat. Taiwan Univ., 9** (2): 41, 1968.
39. SIQUEIRA, P.A. de; SERRA, O.P.; ROCHA, U.F.; SERRA, R.G. **Bol. Ind. Anim. s/n** : 27, 1970.
40. AL-BARWARI, S.E. **Bull. End. Diseases, 18**:75, 1977.
41. SHAIKN, H.U.D.; HUQ, M.M.; KARIM, M.J.; KHAN, M.M.H. **Pakistan Vet. J., 3**: 23, 1983.
42. SWARUP, D. ; PACHAURI, S.P. **Buffalo Bull., 6** (1): 4, 1987.
43. CHAUDHURI, R.P.; KUMAR, P. **Indian j. Vet. Sci., 29**:1, 1959.
44. CHAUDHURY, N.I. **Pakistan j. Sci., 30**:120, 1978.
45. LÁU, H.D.; COSTA, N.A. da; BATISTA, H.A.M. EMBRAPA-CPATU, 1980. 12p (EMBRAPA-CPATU. Circular Técnica, 1)
46. EL-METENNAWY, T.M.A.; SELIM, M.K.; TAWFIK, M.A.; HASSAN, A.A. In: WORLD B BUFFALO CONRESS, 1, Cairo, 1985. **Pap...**, Cairo, International Buffalo Information Center, 1985, p 27.
47. MUÑOZ COBENAS, M.E.; BARCI, D.; POPOVICI,A. **Vet. Argentina, 4** (38): 724, 1987.
48. LÁU, H.D. **A Hora Vet., 76**: 53, 1993.
49. TIKARAM, S.M.; RUPRAH, N.S. **Trop. Anim. Hlth. Prod., 18**: 86, 1986.
50. SATIJA, K.C.; DATT, S.C.; TIKARAM, S.M. **Haryana Vet., 2**: 125, 1981.
51. DISSAMARN, R. In: PACIFIC SCIENCE CONGRESS. 9. Thailand, 1960. **Proc...**, Pacific Science association, 1960, p. 106.
52. LÁU, H.D.; COSTA, N.A. da. EMBRAPA-CPATU, 1979. 5p. (EMBRAPA-CPATU. Comunicado Técnico, 25).
53. MASKE, D.K.; RUPRAH, N.S. **Indian Vet J., 61**: 740, 1984.
54. VERMA, B.B.; GAUTAM, O.P. **Indian Vet. J., 45**: 648, 1982.
55. SINGH,D.; GAUR, S.N. **Indian j. Anim. Sci., 53**: 498, 1983
56. SINGH, V.; RAISINHANI, P.M. In: WORLD BUFFALO CONGRESS, 1. Cairo, 1985. **Abst...**, Cairo, p. 862.
57. SHAW, J.J.; LAINSON, R. **Ann. Trop. Med. Parasitol., 66** (1): 25, 1972.
58. MASSARD, C.L.; REZENDE, H.E.B. BRITO, D.B.; LANDY, M. In: CONGRESSO DA SOCIEADE BRASILEIRA DE PARASITOLOGIA, 4, São Paulo, 1979. **Anais...**, São Paulo , 1979. p. 1.
59. SERRA FREIRE, N.M.da; MELLOE SILVA, A. DE ; MUNIZ, J.A.C.P. In: CONGRESSO BRASILEIRO DE PARASITOOGIA, 5, Rio de Janeiro, 1980. **Anais...** p. 26.
60. MELLO E SILVA , A. de. Rio de Janeiro, Universidade Federal Rural do Rio de Janeiro, 1980. 89p. Tese de Mestrado.
61. LÁU, H.D. EMBRAPA-CPATU, 1988. 13p. (EMBRAPA-CPATU. Boletim de Pesquisa, 90).
62. SINGH, C. **Indian Vet. J., 54**: 492, 1977.
63. PATNAIK ,M.M.; PANDE, B.P. **J. helminth., 37**(4): 343, 1963.
64. MALVIYA, H.C. **Indian J. Helminth., 24** (2): 68, 1972.
65. SINGH, D.V.; JOSHI, H.C.; SHIVNANI, G.A. **Philippine j. Vet. Res., 11**: 336, 1973.
66. CHAUHAN, P.P.; ARORA, G.S.; AHLUWALIA, S.S. **J. Helminth., 28**: 289, 1974.
67. HELMY, N.; FOUAD, K.; KHAMIS, Y.; HOSNY, Z. **Vet. Med. J. Giza, 17**: 69, 1970.
68. KHAMIS, Y.; HELMY, N.; FAHMY, L. **Not. Méd. Vet., 4**: 301, 1973.
69. FOUAD, K.; SALEH, M.; KBAMIS,Y.; SHOUMAN, T.; FAHMY, L. **J. Egypt. Vet. Med. Assoc., 34** (3/4): 154, 1974.
70. LETTS, G.A. **Aust. Vet. J., 38**: 74, 1962.
71. LÁU, H.D.; SINGH, N. P. EMBRAPA-CPATU, 1987. 14p. (EMBRAPA-CPATU. Boletim de Pesquisa, 80).
72. TONGSON, M.S.; GEGATO, J.; CANILLAS, J. **Phil. J. Vet. Med. , 20** (1): 38, 1981.

73. AWAD, F.; AMIN, M.M.; SHAWKAT, M.E.; FAYED, A.A.; MATTER, A.A. **Egypt. J. Vet. Sci., 14** (2): 135, 1977.
74. RANIA, U.; APREA, M.; DELLE DONNE, G. **Atti. Soc. Ital. Sci. Vet., 31**: 781, 1977.
75. LÁU, H.D.; SINGH, N.P. EMBAPA-CPATU, 1986. 15p. (EMBRAPA-CPATU, Boletim de Pesquisa, 76).
76. AJMAL, M.; ARSHAD, M.; AHMAD, M.D.; RIZVI, A.R. In: WORLD BUFFALO CONGRESS, 2. New Delli, 1988. **Abst....**, New Delli, p. 326.
77. KULSHRESTHA, R.C.; KALRA, D.S.; ARORA, R.G.; GUPTA, R.K. **Indian Vet. J., 50**: 228, 1973.
78. NICOLETTI, P. In: WORLD BUFFALO CONGRESS, 1. Cairo, 1985. **Abst....**, Cairo, p.830.
79. CHAND, P.; KHANNA, R.N.S.; SADANA, J.R. **J. Appl. Bac., 64**: 445,1988.
80. FENIZIA, D.; GUARINO, A.; ARUSSO, A.; DE FRANCISCIS, G.; MIGLIACCIO,G. In: WORLD BUFFALO CONGRESS, 3. Varna, 1991. **Abstract...**, Varna, p. 1076.
81. MOHAN, R.N. **Vet. Bull. , 38** (10): 647, 1968.
82. LALL, J.M. **Vet. Bull., 39** (6): 385, 1969.
83. SMALL, K.J.; THOMPSON, D. **Buffalo Bull., 5** (3): 62, 1986.
84. SINGH, N.D. **Indian j.Vet. Sci., 18**: 77, 1948.
85. PILLAI, C.P.; THAMBIAYAH, V.S. **Ceylon Vet. J., 5**: 24, 1957.
86. OCHI, Y. **Bull. Off. Int. Epiz., 47**: 103, 1957.
87. DE ALWIS, M.C.L. **Br. Vet. J., 148**: 99, 1992.
88. FAO. Animal Health Yearbook, 1989.
89. DE ALWIS, M. C.L.; VIPULASIRI, A.A. **Ceylon Vet. j., 28**: 24, 1980.
90. KHENG, C.S.; PHAY, C.H. **Vet. Rec., 75**:155, 1963.
91. LÁU, H.D.; SINGH, N.P.; HESS, S.J. EMBRAPA-CPATU, 1986. 13p. (EMBRAPA-CPATU, Boletim de Pesquisa, 77).
92. SINGH, N.; SHARMA, V.K.; RAJANI, H.B.; SINHA, Y.R. **Indian Vet. J., 59**: 693, 1982.
93. REDA, I.M.; HEGAZI, A.G. In: WORLD BUFFALO CONGRESS, 1. Cairo, 1985. **Abst...**, Cairo, p. 722.
94. MALLICK, K.P.; DWIVEDI, S.K, **Indian Vet. J., 59**: 397, 1982.
95. MANSJOER, M. **Hamera Zoa, 58**: 547, 1951.
96. MAMMERICKS, M. **Bull. Agric. Congobelge, 51**: 171, 1960.
97. MAQSOOD, M. **Vet. Rec., 70**: 321, 1958.
98. LAL, S.M.; SINGH, I.P. **Trop. Anim. Hlth. Prod., 9**:107, 1977.
99. KAUSHIK, A.K.; GROVER, Y.P.; PANDEY, R. **Buffalo Bull., 2** (4): 3, 1983.
100. MAURYA, S.N.; BHALLA, R.C.; SONI, B.K. **Indian J. Vet. Sci., 38**: 238, 1968.
101. DWIVEDI, J.N.; SINGH, C.M. **Indian Vet. J., 46** (2): 115, 1970.
102. SHARMA, A.K.; GUPTA, R.C. **Andrologia, 10** (6): 479, 1978.
103. GUPTA, R.C.; SHARMA, A.K.; VERMA, S.K.; KHAR, S.K.; DATT, S.C. **Philip. J. Vet. Med., 20** (2): 133, 1981.
104. PANDIT, R.K.; GUPTA, S.K.; PATTABIRAMAN, S.P. **Indian Vet. J., 60**: 463, 1983.
105. RIBEIRO, H.F.L. UFRRJ, 1986. 90p. **Tese** (Mestrado).
106. VALE, W.G.; SOUSA, J.S.; OHASHI, O.M.; RIBEIRO, H.F.L. **Pesq. Vet. Bras., 1** (3): 101, 1981.
107. LÁU, H.D. EMBRAPA-CPATU, 1988.14p. (EMBRAPA-CPATU, Boletim de Pesquisa, 89).
108. LÁU, H.D. EMBRAPA-CPATU, 1990.18p. (EMBRAPA-CPATU, Documentos, 54).
109. TUNGTRAKANPOUNG, N.; RHIENPANISH, K. **Buffalo Bull., 11** (2): 30, 1992.
110. VERMA, G.S.; SADANA, D.K.; BASU, S.B.; SARMA, P.A. **Indian J. Dairy Sci., 33** (1) :87, 1980
111. ASKER, A.A.; EL-ITRIBY, A.A. **Emp. J. Exp. Agric., 25** (98); 151, 1957.
112. LÁU, H.D.; SINGH, N.P. In: SIMPÓSIO DO TRÓPICO ÚMIDO, 1, Belém, 1986. **Anais**, Belém, p.259.