Chapter XIII

BUFFALO PATHOLOGIES

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The water buffalo is susceptible to most diseases and parasites that afflict cattle, although the effects of disease on the buffalo and its productivity are sometimes less evident. Generally *Bubalus bubalis* is a healthy animal, in spite of a natural habitat consisting of hot and humid regions that are very favourable to micro organism and parasite proliferation.
The diseases affecting buffaloes have been subdivided as follows:

Viral diseases

- Foot-and-mouth disease
- Rinderpest
- Malignant catarrhal fever
- Infectious bovine rhinotracheitis/ infectious pustular vulvovaginitis
- Blue tongue
- Bovine viral diarrhoea
- Rabies
- Ephemeral Fever
- Buffalo pox

Neonatal diarrhoeal diseases

- Rotavirus
- Salmonellosis
- Colibacillosis
- Cryptosporidiosis

Bacterial diseases

- Bovine brucellosis
- Tuberculosis
- Paratuberculosis
- Haemorrhagic septicaemia
- Chlamydiosis
- Leptospirosis
- Contagious bovine pleuropneumonia
- Anthrax

Parasitic diseases

- Trypanosomiasis

- Ascaridiosis
- Fasciolosis
- Babesiosis
- Theileriosis
- Strongilosis
- Coccidiosis
- Echinococcosis/hydatidosis
- Mange

Fungal diseases

- Deg Nala disease

VIRAL DISEASES

Foot-and-mouth disease

Etiology- Foot-and-Mouth Disease (FMD) is an infection caused by the Aphtovirus, an RNA virus pertaining to the Picornaviridae family that affects cloven hoofed animals. The virus occurs in seven main serotypes: O, A, C, SAT 1, SAT 2, SAT 3 and Asia 1. Every serotype comprises various immunological subtypes of different virulence. Hajela and Sharma (1978) reported that Asia 1 is more severe in buffaloes than in cattle.

Epidemiology- The disease is present worldwide except in North and Central America (north of Panama), Australia, New Zealand, Japan, Great Britain and Scandinavia. The European Union (EU) countries are generally free from FMD. Severe forms of the disease have been reported in the indigenous Swamp buffaloes of India, Egypt and Romania. The disease is acute and highly contagious and may spread over huge areas due to movement of infected or contaminated animals, products, objects, and people.

The susceptibility of buffaloes to FMD has shown to vary according to the country and the various strains of virus. Cattle and buffaloes are mainly infected by inhalation due to aerosol transmission, often from pigs, which excrete large amounts of virus by respiratory aerosols and are considered highly significant in the diffusion of the disease. It also spreads through ingestion or direct contact. Large amounts of virus are excreted by infected buffaloes and cattle before clinical signs are evident, and winds may spread the virus over long distances. During the acute stage of infection, the disease has been transmitted from cattle to buffalo and vice versa (Gomes et al., 1997); occasionally buffaloes remain unaffected although in direct contact with cattle. In Nepal the clinical disease was comparatively mild and mainly affected buffaloes as a severe decrease in milk production, even for those in close contact with cattle, that exhibited the classical lesions.

People can be infected through skin wounds or the oral mucosa by handling diseased stock, the virus in laboratories, or by drinking infected milk, but not by eating meat from infected animals. The human infection is temporary and mild, so FMD is not considered a public health problem, but, due to the range of species affected, the high rate of infectivity, and the fact that the virus is shed before clinical signs occur, FMD is one of the most feared reportable disease. An outbreak of FMD involves very high costs due to lost production, which includes milk and draught power, loss of export markets, and loss of animals during the eradication of the disease. The significance of many other reportable diseases is due to their similarity to FMD and the importance of differentiating between them at the first indication of an unusual disease outbreak.

The incubation period is 2 to 21 days (average three to eight). The rate of infection (morbidity) can reach 100 percent, however mortality can range from 5 percent (adults) to 75 percent (suckling pigs and sheep). It may rise up to 20 to 25 percent in buffaloes. Persistent infection

in buffaloes during the first 35 days following infection is similar to that in cattle (Gomes et al., 1997). Recovered cattle and buffaloes may be carriers for 18 to 24 months, sheep for one to two months, while pigs are not carriers. Vaccinated buffaloes may also be carriers whenever exposed to infection.

Clinical findings- Clinical signs in cattle are salivation, depression, anorexia and lameness caused by the presence of painful vesicles in the skin of the lips, tongue, gums, nostrils, coronary bands, interdigital spaces and teats. Fever and decreased milk production usually precede the appearance of vesicles. Vesicle rupture leaves large denuded areas which are then liable to infection. Foot-and-mouth disease has been described in Indian buffalo (*Bubalus bubalis*) with the same features as that in cattle in terms of temperature, viraemia, virus replication in pharyngeal area, excretion, antibodies cynetics and titres, and persistent infection; differences were the presence of minor tongue lesions and initial scaly foot lesions eventually becoming vescicular in buffalo (Gomes et al., 1997). Milk production can be affected by up to 30 percent in buffaloes. All age groups are equally affected but in suckling buffalo calves the disease may result as more severe and a high mortality rate may sometimes ensue (Sharma and Kumar, 2003). Post mortem lesions are characterized by vesicular eruptions and erosions. In calves there can be hyaline degeneration of the myocardium (tiger heart).

Diagnosis- The suspect deriving from clinical findings can be confirmed by foot-and-mouth virus isolation in cell culture and laboratory animals. Up to five days are necessary before a negative tissue culture result can be given. Serological methods consist of complement fixation (CFT), plaque reduction assay, virus neutralization, radial immunodiffusion, virus infection associated antigen test and ELISA. With the latter, results may be available within four hours, differentiating FMD from the Swine vesicular disease (SVD) antigen and, if positive for FMD, it will indicate the serotype of the FMD antigen involved. ELISA has been found to be more sensitive than the microserum neutralization test.

Negative results may be due to poor samples rather than to freedom from infection, so the quality of the submitted material is extremely important. The FMD viral genome can be detected through the PCR technique. Polymerase chain reaction: This technique relies on the amplification of a selected region of the FMD viral genome. A pair of nucleotide primers is selected with sequences related to the nucleotide sequence at the start and end of the selected region. Following reverse transcription of the viral RNA into DNA, the selected region between the pair of primers is amplified by repeated cycles of heating and cooling in the presence of a heat-stable DNA polymerase. The resulting mixture is run on an agarose gel and a positive result is recorded if a band of the appropriate size is observed. Confirmation of the result can be obtained by nucleotide sequencing of the proposed FMD-specific band. The advantage of such a technique is that no live virus presence is required. FMD viral RNA can therefore be identified in material which has been inactivated or poorly preserved. The above technique is extremely sensitive and allows the FMD viral genome to be identified when insufficient virus is present to initiate infection in tissue culture. However, using current techniques, the presence of inhibitory substances in some samples means that not all samples that contain virus or viral genome will give a positive result by PCR.

Therapy- FMD lesions should be disinfected and treated with emollients; the administration of antibiotics could prevent secondary bacterial infections. Sometimes non-specific immunomodulators proved useful (Sharma and Kumar, 2003).

Prophylaxis- Strategies for the control of foot-and-mouth disease can be based on different measures depending on the objectives and on the existing sanitary situation. Eradication implies a policy in which the presence or possible incursion of the virus is not tolerated, while control implies that the presence of the virus might be tolerated but the effects of the disease are minimized by vaccination and other zoosanitary measures. Control by 'Stamping Out' is applied when outbreaks occur in countries which are otherwise free from FMD. It is also applied as the final stage in an eradication campaign to eliminate the virus once the disease

has been controlled. Control of the movement of animals, import controls, removal of the source of infection (slaughter of all infected and in-contact stock), and epidemiological investigations are essential elements in the eradication of FMD.

Methods of immunization have been studied taking into account the antigenic diversity among serotypes. The control of the disease should be effected through inoculation, twice a year, with a cocktail vaccine containing the specific locally circulating serotypes. Molecular studies pointed towards VP1 capsid protein as the major immunogenic site, and it has been tested for vaccines with encouraging results (Sharma et al., 2001).

Rinderpest

Etiology- The causative agent is an RNA virus, belonging to the Paramyxoviridae family, Morbillivirus genus. The disease is characterized by a high morbidity rate. The mortality rate is also high with virulent strains but variable with mild strains, that have almost identical immunological properties.

Epidemiology- Hosts are represented by cattle, zebus, water buffaloes, sheep and goats and many species of wild animals e.g. African buffaloes, eland, kudu, wildebeest, various antelopes, bushpigs, warthog, giraffes, etc. Buffalo susceptibility is variable: Egyptian and Turkish buffaloes seem to be reasonably resistant, while the Far East species appears to be highly susceptible. In India buffaloes are three times more susceptible than cattle; this is perhaps due to host specificity of strains of the virus (Sharma and Kumar, 2003). The disease has been eradicated in most parts of the world. However, among countries involved in buffalo breeding, exceptions are represented by India, Pakistan, the Philippines and Turkey. The virus has never been encountered either in the Americas nor in Australia or New Zealand.

Transmission is due to direct or close indirect contacts through tears, nasal secretions, saliva, urine and faeces, vaginal exudes and milk. Blood and all tissues are infectious before the appearance of clinical signs and excretion is usually limited to two to three weeks after infection. The primary site of invasion is the epithelium of the upper or lower respiratory tract. The incubation period is 3 to 15 days, in buffaloes three to seven days have been reported, but this may vary due to the differences in innate resistance (Adlakha S.C. and Sharma S.N. 1992).

Clinical findings- A febrile period (40-42°C) with depression, anorexia, reduction of rumination, rough hair coat, and an increase in the respiratory and cardiac rate. After two to three days a mucous membrane congestion (oral, nasal, ocular and genital tract mucosae), intense mucopurulent lacrimation and abundant salivation, anorexia, necrosis and erosion of the oral mucosae can be witnessed. Following this gastrointestinal signs appear as the fever drops, with profuse haemorrhagic diarrhoea containing mucus and necrotic debris. Severe tenesmus, dehydration, abdominal pain, abdominal respiration, weakness, recumbency and subnormal temperature occur a few hours prior to death, which transpires within seven to twelve days. The mortality rate is high. In India it is approximately 77.5 percent (Sharma and Kumar, 2003). In rare cases, clinical signs regress by day ten and recovery occurs by day 20 to 25. In the peracute form no prodromal signs or high fever (>40-42°C) are observed; but sometimes the mucous membranes are congested, and death ensues. This form occurs in highly susceptible young and newborn animals. The subacute form is characterized by only one or more of the classic signs and has a low mortality rate. Finally the atypical form is characterized by irregular pyrexia, and mild or absence of diarrhoea, cutaneous eruptions on the perineum, around the udder, scrotum and between legs, abortions and neurologic signs. The lymphotropic nature of the rinderpest virus favours recrudescence of latent infections and/or increased susceptibility to other infectious agents. Lesions are represented by either areas of necrosis and erosions, or congestion and haemorrhage in the mouth, intestines and upper respiratory tracts; enlarged and oedematous lymph nodes; white necrotic foci in Peyer's patches; 'zebra striping' in the large intestine; carcass emaciation and dehydration.

Diagnosis- Laboratory diagnosis involves identification of the agent by antigen detection (Agar gel immunodiffusion test; direct and indirect immunoperoxidase tests; counter immunoelectrophoresis; immunohistopathology); virus isolation and identification in VERO or bovine kidney cell cultures; virus RNA detection (cDNA probes; PCR); serological tests (enzyme-linked immunosorbent assay (ELISA), virus neutralization, agar gel precipitation test, neutralization of inhibition of haemoagglutination, counterimmunoelectrophoresis (CIEP), complement fixation test (CFT), fluorescent antibody test (FAT)). Using the agarose immunodiffusion test (AGID), serum should be taken three to five days following the onset of fever (Sharma and Kumar, 2003). Differential diagnosis is necessary for the mucosal virus disease complex that resembles rinderpest but that has no antigenic relationship to it.

Therapy- No specific treatment. When diarrhoea is present, lost fluids should be replaced by saline or lactated Ringer's solution.

Prophylaxis- Prevention and control is possible thanks to sanitary prophylaxis, isolation or slaughtering of sick and in-contact animals, destruction of cadavers, disinfection, protection of free zones, medical prophylaxis. The virus is in fact very fragile and sensitive to common disinfectants, heat treatment and drying. Cell-culture attenuated virus vaccines are highly effective.

The commonly used vaccine is an attenuated strain of rinderpest virus. In some countries a mixed rinderpest/contagious bovine pleuropneumonia vaccine is used. Immunity lasts at least five years and is probably life-long. Annual revaccination is recommended in order to obtain a high percentage of immunized animals in an area.

Genetically engineered thermostable recombinant vaccines have passed all the safety and efficacy trials recommended by the OIE and are currently undergoing limited field trials. Animals immunized with recombinant vaccinia virus developed immunity and were resistant to infection with virulent RP virus. The immunity lasts for at least one year following one single vaccination (Sharma et al., 2001).

Malignant catarrhal fever

Etiology- Also known as malignant head catarrh, malignant catarrhal fever (MCF) is a generalized viral disease caused by a highly cell-associated lymphotropic herpes virus of the subfamily Gamma herpesvirinae. Two viral strains have recently been designated: alcelaphine herpes virus-1 (AHV-1) and alcelaphine herpes virus-2 (AHV-2), although some continue to designate this agent as bovid herpes virus-3. In addition, a sheep associated form (SA-MCF) identified as ovine herpes virus-2 (OHV-2), represents a worldwide problem in cattle and buffaloes. Seroepidemiological studies implicate goats as possible carriers of SA-MCF (Sunil-Chandra, 2000).

Epidemiology- The virus can be carried as a latent infection by African antelope of the family Bovidae, subfamily Alcelaphinae which includes wildebeest (Connochaetes sp.), hartebeest (Alcelaphus sp.), and topi (Damaliscus sp.), that are considered carriers of the alcelaphine MCF virus. There is serologic evidence that several other African wild ruminants, such as various species of oryx and addax, may also be reservoir hosts, although the MCF virus has not been isolated from these species. Domestic and wild sheep and goats are also considered reservoir hosts for the MCF virus. Sheep-associated MCF occurs worldwide. In cattle the alcelaphine antelope-associated form chiefly occurs in Africa, in the natural habitat of wildebeest, hartebeest, and topi. This form of MCF has, however, been observed in zoos and wild animal parks that keep wildebeest. There is increasing serologic evidence that cattle may develop low levels of neutralizing antibodies following exposure to MCF, especially of sheep or goat origin, without manifesting the clinical disease. There is evidence that stress or some other immunosuppressive factors may be necessary as a precursor of clinical sheep-associated MCF. The MCF virus in wildebeest, hartebeest, and topi is largely cell-associated in adult animals

and hence rarely transmissible. However, neonatal wildebeests have been found to shed cell-free MCF virus through nasal and ocular secretions and faeces. Cell-free MCFV has also been evidenced in nasal secretions of captive adult wildebeests after stress or the administration of corticosteroids. Transmission to cattle or other susceptible species may occur by inhalation of cell-free virus in infectious aerosol droplets, ingestion of food or water contaminated with infectious secretions or faeces, or possibly mechanically by arthropods. The mode of transmission of sheep-associated MCF remains unknown, although relatively close contact between cattle and sheep, especially lambing ewes, is believed necessary. Sheep associated MCF is of major economic concern in Indonesia where susceptible animals, Balinese cattle and water buffalo, are commonly housed together with sheep and goats (Sunil-Chandra, 2000). MCF-affected cattle appear to shed only cell-associated virus, and thus cattle to cattle transmission is thought to be rare or nonexistent, although there are documented instances where this has occurred. There is no evidence that MCF is infectious for humans. Many exotic ruminant species in zoos have been reported affected with MCF, including several wild bovines such as bison, water buffalo, gaur and banteng, and several deer (including white-tailed deer) and antelope species. Morbidity in nonalcelaphine MCF outbreaks in Malaysia ranged from 28 percent to 45 percent. MCF affects all ages, breeds, and sexes; buffaloes are more susceptible than cattle with a morbidity ranging from 20 to 50 percent; the disease is particularly common in the late winter/spring months (Sharma and Kumar, 2003). The prognosis in MCF is poor. Once clinical signs are observed, mortality is usually greater than 95 percent (90-100 percent). The incubation period in natural cases is not known, but epidemiologic evidence indicates it may be as long as 200 days. Experimentally, the incubation period varied from 9 to 77 days. The disease has been reported from most countries breeding buffaloes.

Clinical findings- Clinical signs in domestic cattle and buffaloes and in many species of wild ruminants are characterized by high fever, profuse nasal discharge, corneal opacity, ophthalmia, generalized lymphadenopathy, leukopenia, and severe inflammation of the conjunctival, oral, and nasal mucosas with necrosis in the oral and nasal cavities sometimes extending into the esophagus and trachea. Occasionally central nervous system (CNS) signs, diarrhoea, skin lesions, and non-suppurative arthritis are observed.

Clinical MCF in cattle can be divided into four types: 1. Peracute: Fever, severe inflammation of the oral and nasal mucosas and haemorrhagic gastroenteritis with a course of one to three days. 2. Intestinal: Fever, diarrhoea, hyperemia of oral and nasal mucosas with accompanying discharges, and lymphadenopathy with a course of four to nine days. 3. Head and eye: This is the typical syndrome of MCF with fever, nasal, and ocular discharges progressing from serous to mucopurulent and purulent. Encrustation of the muzzle and nares occurs in later stages, causing obstruction to the nostrils, dyspnea, open-mouthed breathing, and drooling. There is intense hyperemia and multifocal or diffuse necrosis of the oral mucosa (usually on the lips, gums, and hard and soft palate) and buccal mucosa. Erosion of the tips of buccal papillae, leaving them reddened and blunted, is often encountered. Ocular signs referable to ophthalmia include lacrimation progressing to purulent exudation, photophobia, hyperemia, and edema of the palpebral conjunctiva and injection of scleral vessels. Corneal opacity, starting peripherally and progressing centripetally, results in partial to complete blindness. Hypopyon may also be seen. Corneal opacity is usually bilateral but can occasionally be unilateral. Fever is common and usually high (104-107°F [40-41.6°C]) until the animal turns moribund, at which time it shifts to hypothermy. Clinical features at early onset of the disease have included reddening of the udder skin, the coronary bands and interdigital spaces, and marked hyperemia of the oral cavity. Increased thirst accompanies fever, and anorexia is seen in late stages. Constipation is common in this form of MCF, but terminal diarrhoea is sometimes observed. Nervous signs are not frequently seen but may be manifested by trembling or shivering, uncoordinated gait, and terminal nystagmus. Necrotic skin lesions are occasionally seen, and horn and hoof coverings may be loosened or sloughed in some cases. The course of the "head and eye" form, which is invariably fatal, is usually 7 to 18 days. 4. Mild: These are syndromes caused by the experimental infection of cattle with attenuated viruses and are usually nonfatal.

The manifestations of the "head and eye" form of MCF are considered the typical syndrome in cattle, but clinical signs in exotic ruminants are often less dramatic and not usually diagnostic. In buffaloes the disease is seen in head and eye or intestinal forms (Sharma and Kumar, 2003). In Indonesian swamp buffaloes, hyperaemia of the skin, enlargement of the lymph nodes and depression were described (Hoffmann et al.,1984; Adlakha and Sharma, 1992). Gross lesions vary considerably, depending on the form or severity and course of the disease. Animals that die with peracute disease may have few lesions other than a haemorrhagic enterocolitis.

In the more protracted acute to subacute disease (intestinal and head and eye forms), the carcass may be normal, dehydrated, or emaciated. The muzzle is often encrusted and raw. Cutaneous lesions sometimes occur as a generalized exanthema with exudation of lymph causing crusting and matting of the hair. Where skin is unpigmented, hyperemia is apparent. These lesions are frequently seen in the ventral thorax and abdomen, inguinal region, perineum and loins, and sometimes on the head. Enlarged lymph nodes are characteristic findings in MCF. All nodes may be involved, but those in the head and neck and periphery are the most consistently prominent. Affected nodes are grossly enlarged and edematous and sometimes have patchy reddened or beige-brown areas on cut surfaces. Hemolymph nodes are also enlarged and prominent. The spleen is slightly enlarged, and Malpighian corpuscles are prominent. Pale areas may be seen in the heart muscle; serofibrinous epicarditis and myocarditis were found in Indonesian swamp buffalo (Hoffmann et al., 1984). Lesions in the respiratory system range from mild to severe. When the clinical course is short, there is slight serous nasal discharge and hyperemia of the nasal mucosa. Later the discharge becomes more copious and mucopurulent to purulent and is accompanied by intense nasal mucosal hyperemia, edema, and small focal erosions. Occasionally a croupous pseudomembrane formation is seen. Lesions in the nasal passages and turbinates may extend to the frontal sinuses. The pharyngeal and laryngeal mucosas are hyperemic and edematous and later develop multiple erosions, often covered with grey-yellow pseudomembranes. Inflammation and sometimes petechiation and ulceration are seen in the tracheobronchial mucosa. The lungs are often edematous and sometimes emphysematous but in some cases may appear normal. A bronchopneumonia may complicate chronic cases. The alimentary tract mucosa may have no gross lesion in peracute cases. When the course of the disease is protracted, the alimentary lesions are commensurately more severe and include mild to severe mucosal inflammation (hyperemia and edema), erosions, and ulcerations - especially on the dental pad and gingival surfaces, the palate, tongue, and buccal papillae. Mucosal inflammation, haemorrhage, and erosions may also be found in the rest of the digestive tract including the esophagus, rumen, omasum, abomasum, small intestines, colon, and rectum. Petechiation may be seen. Faeces are usually scant, dry, pasty, or blood stained. Urinary tract lesions include hyperemia and sometimes marked distention and prominence of bladder mucosal vessels and mucosal edema, perhaps with petechial to severe hemorrhage and occasionally epithelial erosion and ulceration. Kidneys may appear normal or mottled with patches of beige, discoloured raised areas. Petechiae or ecchymoses may occur in the renal pelvis and ureters. The liver is usually slightly enlarged, and, upon close examination, has a prominent reticular pattern. There may be hemorrhages and erosions in the gall bladder mucosa. In most cases, small arterioles are very prominent and tortuous and have thickened walls. This is usually seen in subcutaneous vessels and those in the thorax, abdomen, and CNS. Fibrinous polyarthritis is seen in many cases of MCF.

Diagnosis- Presumptive diagnosis of MCF can be based on clinical history and gross necropsy lesions.

A history indicating contact with sheep, goats, or alcelaphine antelope, especially around the period of parturition, associated with typical clinical features of MCF, provides grounds for a tentative diagnosis of MCF. The inability to differentiate the alcelaphine clearly from the sheep-associated MCF by clinical observations, lesions, or laboratory means presents an enigma in evaluating the possibility of a foreign animal disease. Based on existing knowledge of the disease, history of association with sheep, goats, or with alcelaphine antelope remains the only practical means of differentiating one form from another. Laboratory diagnosis can

support the field diagnosis if there are microscopic lesions of an extensive fibrinoid necrotizing vasculitis, perivasculitis, and lymphoreticular proliferation in lymphoid organs with mononuclear infiltrations in the kidney, liver, adrenals, CNS, etc., that are pathognomonic for MCF and are a sound, practical basis for a confirmed diagnosis. The disease should be distinguished from BVD mucosal disease, bluetongue, rinderpest, vesicular diseases (FMD, vesicular stomatitis (VS)), infectious bovine rhinotracheitis, haemorrhagic septicemia, ingested caustics and some poisonous plants and mycotoxins.

Virologic and serologic examinations provide additional information that may also ultimately lead to a better understanding of the epizootiology and differences between viral strains and the clinical manifestations. Methods used consist in virus isolation in calf thyroid tissue culture, identification of viral isolates, demonstration of the appearance or rising titers of MCF antibodies and molecular techniques using viral DNA probes, or target DNA amplifying methods such as the polymerase chain reaction (PCR). A single antibody positive serologic sample is of limited value in establishing an etiologic diagnosis. The PCR method for demonstrating MCF DNA segments is proving to be useful for identifying MCF carriers as well as diagnosing overtly diseased animals from formalin-fixed and paraffin-embedded tissue blocks.

Therapy- No specific treatment; supportive treatment can be effected with the use of broad-spectrum antibiotics, fluid therapy and non steroid anti-inflammatory drugs to relieve discomfort.

Prophylaxis- The control of the disease is possible by separating cattle and buffaloes from potential reservoir hosts such as sheep, goats, and wildebeest - especially during lambing, kidding, or calving seasons, respectively. The stocking of cattle ranches with alcelaphine antelope, wild sheep, or goats should be discouraged; in any event a negative MCF serologic test should be required and this preferably by the serum-virus neutralization method. A negative PCR test for any wild ruminant assigned to such a facility should be exacted. Similar testing of such wild ruminants before being placed in, or transferred between, zoos is also recommended as a means to prevent the introduction of potential carriers of the MCF virus.

Containment of an outbreak usually means the immediate separation of cattle or susceptible host from sheep and goats in the case of domestic disease and the susceptible host from alcelaphine or wild ruminants in the case of alcelaphine MCF.

No effective vaccine is available for MCF. Some viral strains have undergone limited attenuation after serial passage in cell cultures and represent a prospect for a future modified live virus vaccine. Experimental killed virus vaccines have been inconsistent in inducing protection against this virulent virus challenge, although some have induced significant titers of serum virus neutralizing antibodies.

Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis

Etiology- The Infectious Bovine Rhinotracheitis/Infectious Pustular Vulvovaginitis (IBR/IPV), sometimes called Red Nose, is an infectious disease of cattle due to Bovine Herpes virus 1, belonging to the Herpesviridae family, sub family Alphaherpesvirinae. The virus can infect the upper respiratory tract or the reproductive tract. Mortality is low but the economic loss can be considerable. In Australia buffaloes' alphaherpesvirus has been differentiated from BHV-1 on a viral DNA restriction profile, indicating that it could be a distinct species (Sunil Chandra N.P., 2000). Like the homologous human herpes viruses, BHV-1 spreads through monocytes and other white blood cells and through peripheral nerves producing a latent infection in neuronal cells of the trigeminal and sacral ganglia. The latent infection allows the virus to persist in the infected hosts for indefinite periods. Reactivation may occur either spontaneously or induced by natural or artificial immunosuppressive stimuli (parturition, transport, dexamethasone). It leads to virus replication and re-excretion with its spread in the environment.

Epidemiology- Susceptible species are domestic and wild bovines; under experimental conditions goats, sheep and pigs have also demonstrated infection.

Transmission occurs through aerosols, direct and indirect (over short periods of time) contact and venereal viae, also artificial insemination, since large quantities of virus are shed in respiratory, ocular and reproductive secretions (amniotic liquid, placenta, foetus and semen) of the infected animals, for ten to fourteen days after infection, even if asymptomatic. During latent infections shedding is not so abundant and long lasting as during the acute phases. BHV 1 transmission is also possible due to milking machine cups (Sunil Chandra, 2000). The incubation period ranges from two to twenty days. It is a worldwide disease. The virus was first isolated in Australia (1972), then in Malaysia, India and Egypt. In Brazil, in the central Amazon region, antibodies against IBR/IPV were detectable in 59 percent of serum samples using the ELISA test (Munchow and Pisarz, 1994), in India 21.1 percent of buffaloes were positives (Costa, 2002). Serological investigations in Northern and South Central Italy evidenced seroprevalence rates of 78.4 percent and 51.1 percent for herds and animals, with a more active viral circulation where buffaloes and cows were reared together (Cavirani et al. 1997). In Southern Italy the prevalence was 82.9 percent among farms and 66.1 percent for the animals (Galiero, 1998). Finally, in Central Italy, the prevalence was 59 percent and about 30 to 80 percent (Fagiolo et al., 2001; Fagiolo and Roncoroni, 2003). A diffuse circulation of the virus is observed in the buffalo species.

Clinical findings- The severity of symptoms very much depends on the strain of the virus and the susceptibility of the cattle. In the respiratory form the symptoms include: fever (up to 42°C), general depression, drop in milk production, anorexia and emaciation, severe hyperaemia of the nasal mucosa (Red nose) with numerous clusters of greyish foci of necrosis on the mucous membranes, abundant serous discharge from nose and eyes, conjunctivitis, hyper salivation, tachypnea and tachycardia, sometimes mastitis, short explosive cough and, rarely, death due to obstructive bronchiolitis or bronchopneumonia from secondary bacterial infection. An abortion form can complicate the respiratory form with late abortion (between the fifth and eighth month of pregnancy) and placenta retention. It can be the only manifestation.

In the genital form of the disease, it lasts for two to three weeks and symptoms include: moderate fever, hyperaemia of genital mucosa with vesicles of one to two mm., white discharge of the vulva, pollakiuria, and reduction in milk yield. Males exhibit a balanoposthitis.

In the young calf, less than six months old, the disease is more severe: meningoencephalitis (lack of coordination, hyperexitation and depression), salivation, blindness, and a high mortality rate.

In newborn calves the disease causes: fever and lack of appetite, salivation, inflammation of the nasal mucosa, conjunctivitis, erosions of the mouth mucous membranes covered with mucopurulent exudate, and respiratory distress is common due to swelling of the larynx and pneumonia. Some calves may develop diarrhoea. Pathological manifestations have been observed mainly in bovines, while the pathogen role in buffaloes is less clear.

Lesions are usually restricted to the upper respiratory tract and include: swelling and congestion of mucosa, sometimes with necrotic foci, petechiae, profuse and fibrinopurulent exudate in severe cases. Differential diagnosis for the respiratory form: enzootic bronchopneumonia, Bovine Virus Diarrhoea/Mucosal Disease, gangrenous coryza, rinderpest, theileriosis; for the abortion form: Bovine Virus Diarrhoea/Mucosal Disease, brucellosis, listeriosis, leptospirosis, coxiellosis.

Diagnosis- The virus can be isolated from blood on EDTA, nasal, pharyngeal, conjunctival swabs, aborted foetus, placenta, vaginal swab, prepuce washing fluid and semen. Techniques include neutralization or antigen detection methods using monospecific antisera or monoclonal antibodies; PCR is also used for detection on semen. Samples should be stored in a transport

medium (cell culture medium containing antibiotics and two to ten percent foetal bovine serum to protect the virus from inactivation), cooled at 4°C, and rapidly submitted to the laboratory. Serum can be submitted for virus neutralization test and ELISA. The ELISA test allows the detection of antibodies in milk. A delayed cutaneous hypersensitivity test has also been proposed for IBR/IPV diagnosis.

Therapy- No specific treatment. Broad spectrum antibiotics can prevent bronchopneumonia from secondary bacterial infection.

Prophylaxis- IBR/IPV is most likely to be introduced by importation of infected animals and semen. Once introduced IBR/IPV is difficult and expensive to eradicate especially due to the fact that when the disease is established, animals tend to become unapparent carriers. Systematic testing and elimination of positives has been successful in some countries such as Denmark and Switzerland (Ackermann et al. 1990).

Different types of inactivated vaccines are available. Officially free countries restrict the use of them.

IBR is present worldwide, very few countries have eradicated it (Austria, Denmark, Finland, Sweden, Switzerland). Many Pacific countries or territories have reported serological evidence without clinical cases (Fiji, Guam, Niue, Samoa, the Solomon Islands, Tonga, and Vanuatu), New Caledonia has identified clinical cases.

Blue tongue

Etiology- Blue tongue is a vector-borne disease of ruminants caused by a virus of the Reoviridae family (24 serotypes have been identified), genus Orbivirus and biologically transmitted by five species of Culicoides mosquitos, small biting midges. It is not transmitted by direct or indirect contact between animals in the absence of these insects. The vector competence of all the different species of Culicoides has not yet been totally explored however new species are regularly found to be potential vectors.

Epidemiology- The virus may rarely be excreted in the semen when males are viraemic. Contaminated semen may infect recipient cows but would be unlikely to settle in an area unless abundant vectors were present. Blood is also an infective material. Hosts are represented by all domestic and wild ruminants: sheep, goats, cattle, buffaloes, dromedaries and wild ruminants. The incubation period ranges from five to twenty days. The mortality rate is normally low in sheep but can be up to 10 to 30 percent in some epizooties. The distribution of Blue tongue disease is substantially related to the distribution of the Culicoides vector. It can be introduced to new regions by the importation of infected animals, but it will not survive unless competent vectors are present and sufficient susceptible hosts are available.

Blue tongue occurs as a clinical disease in Africa, the Middle East, the Indian subcontinent, China, USA, Mexico and southern Europe. Serological evidence has also been found in South East Asia, northern South America, northern Australia, the Solomon Islands and Papua New Guinea and, on buffaloes, in Egypt, India, Botswana and Papua New Guinea, while all buffalo serum samples tested resulted negative in Iraq and India during the outbreaks of catarrhal fever of the ovine (Adlakha and Sharma, 1992; Capezzuto and Galiero, 2001).

Clinical findings- The disease is characterized by the inflammation of mucous membranes, congestion, swelling and haemorrhages. Sheep are generally the worst affected. The disease can be quite variable showing commonly: fever (42 °C), loss of condition and emaciation, inflammation, ulcers and necrosis in and around the mouth (gums, cheeks and tongue) and, in a small percentage of cases, cyanotic tongue that appears purplish-blue, reddening and haemorrhages of the coronary band (above the hoof) causing lameness. Abortions and congenital malformations can also occur and sometimes pneumonia.

Infection is generally sub-clinical in cattle and buffaloes. Cattle can remain a source of infection for sheep for some time. In about 5 percent of cases, fever, salivation, congestion and swelling and ulcers inside the mouth may occur. The varying reactions of buffaloes to the infection could depend on the serotype, on the infectant dose and on the involved species infecting the buffalo, whether it is only a carrier-amplifier of the virus or a serological responder without viraemia (Capezzuto and Galiero, 2001).

In sheep, most deaths occur as the result of secondary pneumonia. Hence severe, bilateral pneumonia is a common finding. Other findings may include: haemorrhages in the heart, swelling and necrosis of the muscles, enlarged lymph nodes and swelling and congestion of the spleen and liver.

Diagnosis- Serum can be used for the competitive ELISA, a serological test which is the OIE reference test, or Agar Gel Immunodiffusion. Virus neutralization and Complement Fixation can also be performed. Serum samples should be paired to demonstrate a rising antibody titre. Isolation of the agent is possible from organs and whole blood, by inoculation of sheep, intravascular inoculation in ten to twelve day old embryonated chicken eggs and in tissue cultures. The same matrices can be used for identification by PCR. For identification plaque reduction serum neutralization can be used but for serotyping there are too many cross-reactions.

Therapy- No efficient treatment is available.

Prophylaxis- In disease-free areas prevention consists in quarantine and serological survey and vector control. In infected areas sanitary prophylaxis can only be represented by vector control, for example putting sensitive animals during the night into closed rooms protected by mosquito nets. A medical prophylaxis is possible by vaccination with modified live virus vaccine or deadened polyvalent vaccines. Serotypes incorporated into the vaccine must be the same as those causing infection in the field. Attenuated vaccines are widely and effectively used in southern Africa, Europe and the USA, but present a number of disadvantages. Vaccination of pregnant ewes should be avoided because of the risk of foetal abnormalities and abortions. Inactivated vaccines are not used in endemically infected countries, since effective ones are yet to be developed. New immunizing drugs, based on the use of proteic viral fractions will probably prove to be a valid alternative to the traditional vaccine (Capezzuto and Galiero, 2001).

Bovine viral diarrhoea

Etiology- The BVD virus belongs to the Pestivirus genus (Flaviviridae family) and is closely related to the classical swine fever virus, the border disease of sheep and hepatitis C. These viruses are related antigenically as well as genetically. It is a group of small enveloped viruses with a single stranded, positive sense RNA genome. They all have a similar genomic structure and protein composition, the virus particle comprising a single capsid protein surrounded by an envelope containing two or three glycoproteins. The bovine viral diarrhoea virus has the ability to develop many different variants. If the virus finds itself in another situation, another virus type may take over the population with shifts in its pathogenic or antigenic properties.
BVDV also presents different biotypes: one producing cytopathologic effects (CPE) in cell culture, versus another one that does not (non-CPE). This is important as persistent infections are always due to non-CPE viruses that also cause foetal malformations, while CPE viruses are responsible for mucosal disease (MD) (Farina and Scatozza, 1998). Type I and Type II BVDV are related, but distinctly separate. Some Type II isolates do not cause severe clinical disease, and some Type I isolates indeed do, and both Type I and Type II BVD viruses present cytopathic and non-cytopathic forms. In both cases, the non-cytopathic form is the natural, more common state in cattle.

Epidemiology- The bovine viral diarrhoea (BVDV) infection is a major worldwide problem affecting different species of ruminants (Pringle 1999). Two biotypes of the virus, cytopathic

and non-cytopathic, are identifiable based on their lythic activity in vitro cultures (Meyers and Thiel, 1996). The disease is associated both with acute and persistent infections and, depending on epidemiological circumstances, may manifest as outbreaks affecting large numbers of animals or a continual low incidence of cases within endemically infected herds. The significant economic impact inflicted is due to productive and reproductive losses: by a reduced milk yield, reduced conception rate, abortion, foetus mummification, congenital malformations, weak calves and increased animal mortality.

Apart from cattle, other species that have been infected with BVDV include sheep, goats, lamas, pigs, giraffe, captive and free-ranging deer, antelope, elk, buffalo, water buffalo, reindeer and wildebeest. Persistently infected calves derive by in utero exposure at a certain period of gestation. These calves are persistently infected as they become immunotolerant, so they recognize the virus as "self" and never clear the viral infection. For that reason, they shed considerable quantities of virus, however this may not be done continuously. BVDV can be transmitted in many ways among the cattle population, such as through foetal infection and the shedding of virus in secretions. BVDV is primarily maintained in the population by persistently infected animals. Persistent infection (PI) is probably the primary mode of transmission among herds, but acute infections can also be involved in the transmission. The high prevalence of cattle herds infected with BVDV in many countries throughout the world is believed to be a consequence of the ability of non-cytopathic BVDV (ncpBVDV) to establish lifelong infections following in utero infection in early pregnancy, thus generating a reservoir of persistently infected animals (Brownlie et al. 1989).

This biotype, which is most frequently isolated during investigations is characterized by a vertical virus transmission and is responsible for a permanent BVDV circulation in the cattle population (Brownlie, 1991 and Booth et al. 1995). The investigations on the spread of BVDV, its manifestation and distribution depend not only on the properties of the agent, ways of transmission or immune status of the animal, but can also be associated with demographic risk factors.

Clinical findings- BVDV infection can be described as an acute, chronic, persistent, mucosal disease and haemorrhagic syndrome. It is now possible to find a much broader spectrum of disease than before. The clinical aspects of the infection are influenced by host factors such as immune status to BVDV, that is immunocompetence or immunotolerance of the animal, the gestation period, and by the BVDV-genotype involved. Estimates of economic losses due to BVDV infection vary, depending on the immune status of the cattle population and the pathogenicity of the virus strains (Houe 1999). In buffaloes, clinical signs are mild compared to those in cattle, but they are similar in chronology. In a study on buffalo calves experimentally infected with BVDV, an initial diphasic rise in temperature of about 40°C was evidenced as well as nasal and lacrimal discharge; only some of them had transient diarrhoea during and after the febrile phase; erosions and/or ulcers were seen in the surface of the lips and dental pad associated with congestion of the nasal and gum mucosa. In the first 11 days of infection a mild leukopenia (neutropoenia and lymphopoenia) was evidenced later turning to leukocytosis (neutrophilia) from day 15 to 32. The phagocytic activity decreased starting from the first day post infection and the T-lymphocyte population was severely affected (Hegazy et al. 1991).

Diagnosis- The primary dilemma for veterinary clinicians and diagnosticians is the detection of the virus in samples obtained from clinically ill, as well as subclinically infected cattle (Bolin, 1995) and the control of the viral disease, especially in reproductive-age cattle. Although acute infections with non-CPE BVDV are often asymptomatic or produce only mild clinical symptoms, there is evidence that they result in immunosuppression. The problems associated with the BVDV infections during pregnancy are complex. Pregnant, immunologically naive cattle are at risk of acquiring BVDV infections early (<100 days gestation), resulting in abortion; or mid-gestation (100-125 days), resulting in calves that are congenitally infected and are born persistently infected (PI); or later in gestation (>125 days), resulting in weak calves. There are at least two factors associated with the virus and two associated with the pregnant

animal that contribute to the vulnerability of the host to infection and disease. The two viral factors are immunosuppression and strain variation while the two host factors are the immune status of the heifer entering pregnancy and the physiologic immunosuppression which occurs during pregnancy. Laboratory tests can confirm clinical diagnosis and detect persistent infections. The samples are represented by oral swabs, faecal samples and blood with EDTA; spleen, brain, lung and kidney from foetuses and spleen, lymph nodes, gut and abomasum from adult animals. The samples are submitted to cell cultures with labelled antibodies. Sereological diagnosis can be performed with serum neutralization and ELISA.

Therapy- The primary treatment for the BVD virus arises from prevention since no good therapy exists.

Prophylaxis- Careful culling with proper diagnosis is therefore most important. Prevention focuses on good management and a vaccination programme. Management involves minimizing exposure to the virus from potential sources:

1. All ruminants; large or small (sheep and goats), wild (deer) or domestic movement of animals into a closed herd.
2. Modified live vaccines have been sources in the past.
3. Careful embryo transplant technique.

Additionally, good management must minimize stress in order to enable an optimum immune response to vaccines. Identifying persistent carriers in the herd is a good procedure but may not be practical for most herds. Whenever a case of BVD is confirmed or suspected, especially in a pregnant animal, identification of suspect carriers and culling is necessary.

A perfect vaccination programme has not been effected to date. However, careful administration of quality vaccine products with precise handling will provide a good deal of protection. Traditionally modified live vaccines have proved best in stimulating antibody response. The protection provided by the vaccines is a function of the strains in the product as well as the strains encountered in the field. Although only partial protection is accomplished with vaccines, they will in any case reduce the incidence. A good vaccine schedule should start at four to six months of age after colostral protection is ending and this should be repeated prior to breeding. A killed product should be used in pregnant animals. A killed product can be used if the facility permits easy handling of livestock and allows vaccination to be repeated since the duration of immunity is shorter than a modified live (ML) product. Since no pathognomonic clinical signs of infection with BVDV are to be evidenced, diagnostic investigations rely on either laboratory-based detection of the virus, or virus-induced antigens or even antibodies in submitted samples. In unvaccinated dairy herds, serological testing of bulk milk is a convenient method for BVDV prevalence screening. Alternatively, serological testing of young stock may indicate if BVDV is present in a herd. In BVDV positive herds, animals persistently infected (PI) with BVDV can be identified by the combined use of serological and virological tests for examination of blood samples. ELISA has been used for rapid detection of both BVDV antibodies and antigens in blood, but should preferably be supported by other methods such as virus neutralization, virus isolation in cell cultures or amplification of viral nucleic acid.

Rabies

Etiology- The Rhabdovirus (genus Lyssavirus) responsible for the disease is a truly neurotropic virus that causes lesions only in nervous tissue. It may be eliminated by the use of standard disinfectants and heat treatment.

Epidemiology- The source of infection is always represented by infected animals and it spreads mostly by saliva through contamination of wounds or bites or ingestion. The excretion in milk is low and does not cause the disease. Reports of this disease in buffaloes are not very numerous since they defend themselves well from rabid animals and no cases of rabies have

been reported due to transmission by bats as is the case in cattle. However mortality in buffaloes is 100 percent. Rabies occurs in most countries of the world except on islands where rigid quarantine measures are guaranteed. The incubation period is almost three weeks.

Clinical findings- The symptoms resemble those in cattle. The disease may present a paralytic (drooling of saliva, eructation, grinding of teeth, tail movement, anorexia, stiffness of hind limbs, paralysis and recumbency, death in two to three days) or furious form (alert state, hypersensitivity, sexual excitement, inability to swallow, ramming of head on fixed objects, loud bellowing, collapse and death) (Adlakha and Sharma, 1992).

Diagnosis- It can be made based on clinical symptoms. For confirmation: negri bodies evidenced by brain microscopic examination, impression smears from brain tested by FAT, CFT and ELISA, direct immunofluorescence test and PCR. The biologic test on mice is also important.

Therapy- Wounds should be irrigated with a soap solution and water. Post exposure vaccination can be performed. Suspected animals should be kept under close observation avoiding euthanasia.

Prophylaxis- Destruction of wild fauna around animal holdings and vaccination of cats and dogs are important. Vaccines from chick embryo origin and tissue culture origin can be used (Sharma and Kumar, 2003).

Ephemeral Fever

Etiology- Bovine Ephemeral Fever (BEF) is a viral disease of cattle and buffalo, caused by an arthropod-borne rhabdovirus that features four serovars (Sharma and Kumar, 2003). It is also referred to as an arbovirus in so much as biting insects spread it.

Epidemiology- The most likely insects to transmit the disease are blood sucking flies or mosquitoes, such as Culex annulirostris. Biting midges may also play a role in disease spread, and it is possible that some vectors have still not been identified. Adult buffaloes are those primarily affected while those below six months of age are not.

The distribution of the above mentioned insects varies according to climatic conditions; this, in turn, will influence the pattern of disease spread and time of occurrence. In fact most cases occur under hot and humid conditions. Mortality is low, although the morbidity rate is considerable, which entails enormous economic losses in terms of a significant reduction in production, disruption of national and international trade resulting in a variety of complications and such inconveniences have drawn appreciable attention to this disease. Subtropical and temperate regions of Africa, Asia and Australia have experienced the main epidemics of bovine ephemeral fever (Nandi and Negi, 1999).

Clinical findings- A sudden onset of fever (41°C) can be observed. The first sign in milking cows is a sudden and severe drop in milk production. Buffaloes in advanced stages of pregnancy may sometimes abort, this is probably due to the fever, rather than to a specific effect of the virus. Animals stop eating and drinking and become depressed, start drooling saliva, and develop a stringy nasal discharge. Lameness may not appear prior to the second day of illness but may cause the typical posture of laminitis. Muscular areas over the shoulder, back and neck regions show swelling. Shivering, stiffness and clonic muscular movements are also manifest (Sharma and Kumar, 2003). By day three the affected animal is usually standing again and will begin to eat. However, lameness and weakness may last for another two or three days. The disease can vary in severity. Some animals may show only slight symptoms for about 24 hours, while a small number may be affected for many weeks. The disease is usually milder in calves below 12 months of age. Milk production should return nearly to normal after about three weeks, but cows which are affected late in lactation, often become dry. Mastitis sometimes

develops, with a marked rise in the somatic cell count.

Diagnosis- When an outbreak occurs in unvaccinated cattle not previously exposed to the virus, a diagnosis of BEF can often be accomplished based on clinical signs and the brevity of the illness. Blood analysis evidences leukocytosis, neutrophilia, lymphopenia and increased fibrinogen.

Laboratory confirmation is possible by agar gel precipitation, complement fixation, ELISA and fluorescent antibody tests (Sharma and Kumar, 2003). This is usually undertaken by taking two blood samples - one during the very early stages of the illness, and another one three weeks later. If BEF is responsible, BEF antibody levels will be much higher in the second test than in the first one.

Therapy- Medical treatment is often unnecessary for non-lactating stock. However, bulls and high-producing cows, in early to peak production, should have supportive treatment. It consists in relieving temperature and muscular stiffness with paracetamol and phenylbutazone. Broad spectrum antibiotics (streptopenicillin, tetracyclines) prevent secondary bacterial complications.

Affected animals should not be drenched or force fed as BEF can impair the swallowing reflex, so this may result in the inhalation of food or water and pneumonia.

Prophylaxis- Both live and inactivated vaccines against BEF are available: In cattle the live vaccine gives at least 12 months' protection following two doses, the killed one only gives about six months' protection. Cattle can be vaccinated beginning from six months of age and should then be revaccinated each year to ensure continued protection.

Buffalo pox

Etiology- Orthopox virus, family Pox Viridae. It is resistant to inactivation by ether but sensitive to a change of pH, chloroform and bile salts. It only affects buffaloes. Rabbits and infant mice can be experimentally infected. The infection has been reported in man, among persons handling affected animals.

Epidemiology- The disease is usually present in an endemic form. Morbidity can be as high as 70 percent but the mortality rate is known to be low. It has been reported in India, Indonesia, Italy, Pakistan, Russia and Egypt (Sharma and Kumar, 2003).

Clinical findings- It can be observed in both a localized and generalized form. The pox skin lesions are mainly on the teats, udder and thighs and manifest the typical stages and heal in three to four weeks. About 50 percent of affected animals show mild to severe mastitis. Signs of the disease are represented by fever, anorexia, dullness, depression and congestion of conjunctivae (Adlakha and Sharma, 1992).

Diagnosis- The presence of lesions on the udder and teat could lead to suspicion. Isolation can be performed on chick embryo or cell culture. Confirmation is performed by precipitation, complement fixation and neutralization tests. ELISA has been found to be more sensitive.

Therapy- No specific treatment is applicable. Antibiotics can control secondary bacterial contamination

Prophylaxis- The control of the disease is based on strict hygienic measures since no suitable vaccine is available (Sharma and Kumar, 2003).

NEONATAL DIARRHOEAL DISEASES

Etiology- The greatest buffalo losses are often among calves. Farm records show that losses in buffalo calves can reach 22.2 percent in the first month of life or in the fourth to sixth month (Sunil Chandra and Mahalingam, 1994). Newborn buffalo calves, like bovine calves, can succumb in large numbers to viruses, bacteria, and poor nutrition. This is largely due to poor management during the calf's first two months of life, especially with regard to males. For example, in some countries, breeders often sell the valuable buffalo milk, thus depriving the calves. Apart from poor management, among other causes of calves' mortality, an important problem is represented by neonatal diarrhoea. It heavily affects both buffalo and cattle herds, causing extensive economic losses due to mortality and cost of treatment. 23.7 percent of neonatal buffalo deaths are due to enteric disease (Sunil Chandra and Mahalingam, 1994). Diarrhoea should be studied as a syndrome since it is related to a complex etiology. During outbreaks of buffalo neonatal diarrhoea, different pathogens have been detected: several bacteria, viruses and fungi. Viral agents such as Rotavirus, *Coronavirus* and *Calicivirus* are spread worldwide and have been serologically evidenced in buffaloes (Giglio et al., 1994). E. coli and Cryptosporidium have also been detected (Cavirani et al. 1997) but in other cases the main causes have turned out to be colibacillosis and salmonellosis. E. coli incidence appeared higher in the first week and salmonellosis in the third, but E. coli seems to be the major cause of diarrhoea with an incidence of 54 to 58 percent compared to 13 to 14 percent for salmonellosis. Haemorrhagic diarrhoea has also been reported in buffalo calves due to Clostridium perfringens types A, C and D. Pasteurella, Klebsiella, Proteus vulgaris and Citrobacter have also been recorded (Sharma and Kumar, 2003). Parasitic agents such as Eimeria spp., Strongyloides papillosus, Toxocara vitulorum and Cryptosporidium parvum are commonly found in both diarrhoeic and non-diarrhoeic calves. This demonstrates the significance of all calves as potential sources of infection. E. coli, E coli ETEC, E. cloacae, K. Pneumoniae and Citrobacter spp. show a higher rate of occurrence in diarrhoeic animals but, except for E. coli ETEC and Citrobacter, they are also present in the enteric microflora of the buffalo calves (Ribeiro et al. 2000). Multiple infections due to enteropathogenic viruses and bacteria are more common than single ones.

Epidemiology- The incidence and severity of the disease depends on several factors: colostral immunity, overcrowding, parity of dam, age, sex, birth weight, quality of diet, meteorological conditions and the general care provided to calves. Studies have shown that mortality among buffalo calves is also related to the season; higher mortality, in India, was registered during the months from July to September. Another major factor is colostrum: inadequate feeding on the first day of life makes the calf more susceptible because of the associated low level of serum immunoglobulins (Yadav, 2003). Calves delivered from heifers were more susceptible being weak at birth because of dystokia and the enteric infections were higher in very young calves (El-Garhi et al. 1994). Overall mortality in buffalo calves registered by different authors ranges from 7 to 33.97 percent and among these deaths the major cause is enteritis. Mortality from salmonellosis ranges from 40 to 72 percent, that from colibacillosis is 47 percent. The newborn buffaloes usually present diarrhoea during the first four weeks of life (Ribeiro et al. 2000).

Clinical findings- Generally calves show acute, profuse diarrhoea, sometimes with dysentery, cyanotic mucous membranes, depression, weakness, lack of coordination, severe dehydration and acidosis leading to death within a few days. Sometimes calves also develop pneumoenteritis.

Diagnosis- It can be confirmed by isolation and identification of the organisms.

Therapy- Usually this is based on diet, administration of fluids, electrolytes, antibiotics and intestinal protectants.

Prophylaxis- This is based on cage comfort and hygiene, adequate colostrum administration and increasing non-specific and specific resistance with dams' or calves' vaccination. Colostrum

should be provided within the first two hours of the calves' life, in a quantity of 50 ml/kg, corresponding to 1/20th of the body weight. It has also been proved that calf mortality decreases when feeding is always regularly performed by the same person and that proper housing and ventilation are important as they contribute to reducing stress (Yadav, 2003).

Rotavirus

Etiology- Rotaviruses belong to the Reoviridae family and are characterized by a genome consisting of 11 segments of double-stranded RNA (dsRNA) enclosed in a triple-layered capsid. Its structure accounts for a high resistance in the ambient, seven to nine months at 18 to 20°C, and to various disinfectants. Rotaviruses include seven different serotypes identified by the letters from A to G. Group A rotaviruses are the main cause of acute viral gastroenteritis in humans and animals throughout the world, particularly in newborn animals and among those, in buffaloes. The inner capsid protein VP6 bears the subgroup (SG) specificities that allow the classification of group A rotaviruses into SGI, SGII, both SGI and SGII, or neither SG based on the reactivity with SG-specific monoclonal antibodies. The serotyping and genotyping of rotaviruses within the group A is determined by cross neutralization and by sequence comparison respectively. There is also a classification system based on glycoprotein VP7 and protease sensitive virus protein that recognizes 14 G types and 19 P types in group A rotaviruses (Sunil Chandra, 2000).

Epidemiology- The epidemiology of buffalo rotaviruses is still widely unknown. Rotaviruses are transmitted by the ingestion of viral particles due to oral-faecal contamination. It affects the buffalo calf when it is about one to three weeks old. Rotavirus environmental infecting power is generally high due to the viral excretion from the infected calves, although healthy looking, viral elimination from mothers just before calving, and high viral resistance in the environment. After ingestion, rotaviruses infect the small intestine, multiply in the mature epithelial cells at the tips of the villi, causing the microvilli to degenerate and the release of a large amount of viral particles. The destroyed cells are replaced by underlying ones that are lacking in digestive and absorption functions. So cell death and desquamation cause reduced digestion, malabsorption and villous atrophy. The consequent reactive crypt cell hyperplasia and the increased secretion contributes to the severity of the diarrhoea. Rotavirus antigens or antibodies have been detected in buffaloes faecal or serum samples in Iraq, India, Italy, Sri Lanka and Bulgaria. Buffalo calves showed a greater number of positives compared to calves from cattle. Infection rates were higher in winter than in summer and the highest rates were found in two-week old calves (Adlakha and Sharma, 1992). BRV is pathogenic for calves for the first three weeks of life, then the infection becomes clinically irrelevant. The morbidity for buffalo calves is 43 to 45 percent; mortality is 25 percent. BRV is not highly virulent but proves to be serious in the case of mixed infections. 91 percent of mixed infections comprehend BRV. The viral antigen was detected in 36.6 percent of diarrhoeic calves aged from 10 to 51 days, and only in 11.9 percent of non diarrhoeic calves (Sunil Chandra and Mahalingam, 1994). The incubation period is assumed to range from one to three days.

Clinical findings- The onset of anorexia, depression, sometimes fever, and most of all watery or pasty diarrhoea that can show traces of blood, and dehydration, is rapid. The peak of the diarrhoea is at seven to eight days, afterwards BRV shedding may occur but the clinical disease is not evident. Many pathogens of low pathogenicity can, together with BRV, produce a severe syndrome of diarrhoea, dehydration and acidosis, often resulting in death in young calves. Lesions are essentially intestinal, including dilatation, yellowish content and thinned sections.

Diagnosis- It is based upon virus identification in the faeces, which should be performed at the beginning of the diarrhoea and prior to any antibiotic therapy in order to verify the presence of bacteria. Necroscopy should be carried out within 24 hours. Polyacrilamide gel electrophoresis (PAGE) allows the detection of group A, B and C. Antigenic types are identified with a monoclonal antibody, screening and blocking ELISA and DNA probes. The BRV positives can be confirmed by dot-ELISA and immunofluorescence. Immunoperoxidase focus neutralization

assay can determine the serotype specificity of the rotavirus specific colostral antibody. Rotavirus isolation can be performed using cell lines of a foetal Rhesus monkey kidney and foetal calf kidney (Adlakha and Sharma, 1992).

Therapy- No specific drug has shown to be efficient. It is essential to provide calves with liquids during diarrhoea in order to replace the lost fluids and restore the right hydrosaline balance by saline or lactated Ringer's solution intravenous, subcutaneous or by oral solutions. The use of antibiotics allows the control of secondary bacterial infection in a damaged intestine.

Prophylaxis- Direct prophylaxis is aimed at the maintenance of a high environment hygiene. Buffalo calves should be placed in single disinfected cages which have not been used for 15 to 20 days. High quality colostrum is essential for the prevention of neonatal disease. Various preparations with more antigen valences are sold for passive immunization of calves through vaccination of pregnant female buffaloes. The first two feeding supervisions should permit the attainment of the maximum number of calves with maternal antibodies as maternal vaccinations are not effective if colostrum is not provided in sufficient quantities during the first six hours of life. The amount should be equal to five to ten percent of the calf's body weight or to two litres on the first day (Galiero, 2000).

Salmonellosis

Etiology- Salmonella is a Gram negative bacteria comprehending more than 2 000 species. In the buffalo species several isolations have been reported, but no salmonella serotype seems to be host-adapted. Among the most disseminated serotypes there are S. typhimurium, S. dublin, S. newport, S. bovis morbificans, S. weltevreden, S. abortusbovis, S. java, S. paratyphi B, S. panama, S. enteritidis, S. reading, S tafo, S. kottbus, S. weybridge, S. muenster, S. abortus ovis, S. cholerae suis, S. pullorum (Galiero G., 1999; Sharma and Kumar, 2003; Bahiraie and Moghaddam, 2004). Infection becomes possible due to the presence of adhesines and the different invasive ability in the various strains. Some strains produce enterotoxins and cytotoxic toxins. Salmonella has a good resistance, it can survive for one year in the ground. It is sensible to one percent formaldehyde solution, one percent glutaraldheyde solution and formalin. Salmonellosis is an important zoonoses deriving from contact with infected animals or contaminated equipment.

Epidemiology- The most affected animals are those ranging from one to twelve weeks of age. Older animals generally show no clinical signs but are very significant for the spread of the infection in the herd since one gram of faeces may contain more than ten thousand million of Salmonella bacteria. Other sources of infection are contaminated forages and water as well as rodents, wild winged animals, insects and man. In a study of 250 diarrhoeic calves, bacteriological examination revealed the Salmonella species in 0.8 percent of them (Hegazy et al. 1991).

Clinical findings- Symptoms are anorexia, fever, diarrhoea and dehydration. Diarrhoea, mucous at first, becomes bloody and fibrinous with shreds of mucosa. It sometimes causes sudden death without symptoms. During septicaemia articulations, the lungs and meninges are also involved. The inflammatory lesions are markedly present in the small intestine, but they could also be found in the abomasum and colon. There is congestion, whitish mucus, pseudo-membranes easily removable or not, and blackish or spotted mucosa. The diagnosis should differentiate salmonellosis from colibacillosis, rotavirosis, kriptosporidiosis, coccidiosis, mucous membrane disease, colisepticaemia and clostridiosis.

Diagnosis- It should be performed on fresh faeces from not treated animals, the liver, the liquid of the joints, intestine or whole carcass. The samples should be refrigerated but preferably not frozen. The bacteriological examination is performed through the use of different media: a pre-enrichment, a selective enrichment, solid selective; solid and non selective and media for confirm tests. Biochemical reactions and other tests are also usually employed.

Serotyping of isolated salmonellae is carried out by a test of Fast agglutination on a slide.

Therapy- It is aimed at restoring the correct hydro-saline balance and fighting the infection. Rehydrating therapy is always necessary, while antibiotics are indicated only in cases of severe diarrhoea with a rise in body temperature. Antibiotic resistance is increasing in anti salmonellosis therapy, and for this reason it is preferable to make the choice of the molecule on the basis of the literature reports, farm experiences and antibiogram results. It is important to provide a mass treatment calculating the adequate posology and administrating for at least four to five days.

Prophylaxis- Antibiotic treatment to all neonates prevents serious symptoms and spread of the infection. Direct prophylaxis is always important. It consists in the control of animal introduction with a quarantine period allowing for the execution of three bacterial examinations at a distance of two weeks one from the other, since excretion can be intermittent. Contact with adult and synantrhopical animals, should be avoided, even indirectly. The water should also be analysed. Indirect prophylaxis could be provided on the farm with vaccines prepared from the insulated strain but this implicates a long preparation (Galiero, 1999).

Colibacillosis

Etiology- Escherichia coli is a Gram negative bacillus, asporigenous, facultative aerobe, motile or not, fermenter, characterized by different antigenic structures of the cell wall (antigen O), capsule (antigen K), flagels (antigen H) and fimbriae (antigen F). It includes about 50 000 serotypes of which only a few strains are pathogens. In fact it is a usual species of the intestinal aerobic flora in humans and animals. The pathogenic action relies on the production of virulent structural or secreted factors like adhesines, endotoxins, enterotoxins, cytotoxic toxins, haemolysin. Cytotoxic toxins are responsible for all enterotoxigenic Escherichia coli (ETEC) strains symptoms, heat labile producer strains are rarely isolated from buffalo calves less than one month old; heat-stable toxins producer strains are not very widespread either and affect few day-old buffalo calves. The production of cytotoxic and necrotic factors (CNF) was predominant (21.5 23.2 percent) in comparison with other toxins; VT producing resulted to be 6.5-7.7 percent, Hly 22 percent. Vero cytotoxins 1 (VT1) are those mainly isolated in buffaloes. The buffalo calf is not considered to be a reservoir of serovar O:157, which is an important zoonosic agent (Galiero et al. 1997; Galiero et al. 1999).

Epidemiology- The disease derives from a combined action of different factors: environment, microclima, management, nutrition and sanitary status. It is in fact considered to be a typical conditioned pathology (Galiero, 1998). Morbidity in buffalo calves may be high and mortality may be higher than 50 percent (Galiero et al. 1997). In a study on diarrhoeic calves E. coli was isolated in 4 percent of the animals (Hegazy, 1991).

Clinical findings- Diarrhoea, dehydration, hypothermia and sometimes hypovolemic shock in the presence of enterotoxins. Endotoxins cause fever, diarrhoea, fall in pressure, haemorrhages and vascular thrombosis. The action of cytotoxic toxins as Vero cytotoxins (VT) and cytotoxic and necrotic factors (CNF), results in bloody diarrhoea, weakness, emaciation and anaemia for the first ones and systemic pathologies associated to haemorragic colitis and pulmonary disease. The presence of haemolisin seems to enhance the pathogenic action of the other factors.

Diagnosis- E. coli strains can be isolated from various organic districts (intestine, mesenteric lymph nodes, liver, gall bladder, lungs). Thermolabile toxins (LT), VT and CNF production can be tested on Vero cell cultures; -hemolysin (Hly) on sheep's blood agar; thermostable toxins (ST) on blocking ELISA.

Therapy- It is aimed at re-establishing the electrolitic balance and counteracting the bacteria with antibiotics. The antibiotic choice should be made on the basis of an antibiogram.

Prophylaxis- It is hygienic for calving structures and calf stalls. As for all neonatal pathologies a correct colostrum administration is essential. Vaccines for buffalo species are available.

Cryptosporidiosis

Etiology- Cryptosporidium parvum is a coccidian protozoan parasite of mammals, birds and reptiles. It affects man and all domestic species. The life cycle lasts four to seven days and takes place in the microvillous borders of the enterocytes. It is considered a single-species genus lacking host specificity. Oocysts are extremely resistant to commonly used disinfectants. Their viability is reduced only by five percent ammonia, ten percent formaldehyde and steam under pressure.

Epidemiology- Parasites were evidenced in buffalo in all the countries where it is bred. Interfarm prevalence resulted to be 23-38.8 percent (Galiero, 1999). The ingested oocysts release four infectious sporozoites, after bile salts and trypsin action; they penetrate and reproduce in epithelial cells, then they sporulate within the intestine lumen re-infecting the host or other animals through elimination in the faeces. The transmission is faecal-oral as the hosts start shedding sporulated oocysts at the beginning of the diarrhoea and it persists for 3 to 13 days generally depending on the diarrhoea duration, but it can also happen in asymptomatic carriers. The most affected are one to four week old calves. The incubation period is about two to seven days. The infection has zoonotic implications.

Clinical findings- Enterocolitis is characterized by increased defecation of yellow and watery stools, tenesmus, weight loss, anorexia, depression, weakness and dehydration. Symptoms last more than ten days. Macroscopic lesions are not pathognomonic being represented by an enteritis in particular of the ileus.

Diagnosis- Giemsa stain and modified Baxby or Ziehl-Neelsen techniques can detect cryptosporidial oocysts in faecal samples and preparations of the intestinal mucosa. The oocysts may be concentrated from the faecal matter by centrifugal flotation in a high specific-gravity salt solution (Abd El-Rahim, 1997). Considering that autolytic phenomena take place six hours after death and that freezing destroys the parasite, it is important to preserve samples, for a maximum of 120 days, mixing a part of the faeces with two parts of a 2.5 percent solution of bichromate potassium.

Therapy- No specific drugs are indicated for cryptosporidiosis therapy in the buffalo calf. As in all diarrhoeic pathologies all interventions that restore the hydrosaline balance are useful.

Prophylaxis- Direct prophylaxis aimed at improving management and sanitary levels is useful. Immediate isolation from mothers, division into homogeneous groups by age, isolation of infected animals, sanitation of rooms and equipment are useful measures in every neonatal pathology. Steam jets should be preferred to common disinfectants (Galiero, 1999).

BACTERIAL DISEASES

Bovine brucellosis

Etiology- This disease, generally known as brucellosis, is also called Bang's disease, malta fever and undulent fever (in man), contagious abortion and infectious abortion. It is caused by Brucella abortus (B. abortus), a small gram negative, non-motile, non-sporulating coccobacillus organism that can be easily killed by heat, direct sunlight or common disinfectants and pasteurization. In dry conditions they survive only if embedded in protein, while in tap water they resist for several months at 4-8°C, 2.5 years at 0°C, and several years in frozen tissues or medium. Brucellae can also survive up to 60 days in damp soil, and up to 144 days at 20°C and 40 percent relative humidity. Brucellae can survive 30 days in urine, 75 days in aborted

foetuses and more than 200 days in uterine exudate. In bedding contaminated with infected faecal material Brucella will be destroyed at 56°C-61°C within 4.5 hours. It has been found that Brucella can survive in faeces, slurry, or liquid manure up to 85 to 103 days in the winter, 120 to 210 days in spring, 30 to 180 days in summer, and 50 to 120 days in autumn, indicating that the survival of Brucella is subject to seasonal influences.

Epidemiology- It affects many animal species on every continent and it has been described in all the countries where this species is bred. It is a major pathology concerning buffalo, and it is a zoonose of great economic importance, as well as a public health hazard. Mediterranean buffaloes, not having a specific-species strain, present higher possibilities of contracting this disease especially when it is present in the other receptive species, such as bovine and ovine, and there is promiscuous breeding or uncontrolled livestock movement (Fraulo and Galiero, 1999). Buffaloes are very susceptible both to Brucella abortus, that is the main cause of this disease in bovines, and to Brucella melitensis, more frequent among the ovine species. Frequently in buffaloes biotypes 1, 3 and 6, and less the 7, have been isolated for B. abortus, only 1 and 2 for the B. melitensis (Fraulo and Galiero, 1999). B. abortus appears to be the main species in buffaloes and biotype 1 is that most frequently isolated (Costa, 2002). Although B. abortus is relatively resistant and may survive for a considerable time, the environment is not considered to be an important source of infection. Age, sex, stage of pregnancy and natural resistance to Brucella may influence the progression of infection. Heifers born of infected dams usually test seronegative for Brucella for a long period as the stage of pregnancy at the time of infection determines the incubation period. Abortions in cattle caused by B. abortus seldom occur before the fourth or fifth month of pregnancy. Pregnant females are more likely to become infected than non-pregnant cattle or males. This is because a gravid uterus sustains growth of the organism. Furthermore, the course and incidence of the disease is also influenced by natural resistance to Brucella infection. Finally, the success of the Brucella infection depends on exposure dose, virulence of the organism and natural resistance of the animal, based on its ability to prevent the establishment of a mucosal infection by the destruction of the invading organism. Transmission of B. abortus is very likely to occur via the oral route as cattle tend to lick aborted foetuses and the genital discharge of an aborting cow. So in the animals the infection usually occurs through ingestion of feed and water contaminated with the uterine discharges of an aborted animal or its foetus. Contamination of a cowshed or pasture takes place when infected cattle abort or have a full-term parturition. Although it is generally accepted that B. abortus is not excreted for any considerable time before abortion occurs, excretion in the vaginal discharge of infected cattle may occur as early as 39 days after exposure. A massive excretion of Brucellae starts after abortion and may continue for 15 days. Once the foetal membranes are expelled the uterine discharge diminishes and the number of Brucella organisms excreted decreases rapidly. Although the infectious material from the genital tract usually clears after two to three months, some infected cattle become carriers of Brucella and excrete it intermittently for many years. Congenital infection can be seen in calves as exposure to Brucella organisms is also likely to occur in utero, or when calves born of healthy dams are fed on colostrum or milk from infected dams. Other sources of spread are represented by semen from infected bulls or contaminated udder during milking. The organism can also penetrate intact skin or mucous membranes. Invading Brucella usually localize in the lymph nodes, draining the invasion site, resulting in hyperplasia of lymphoid and reticuloendothelial tissue, and infiltration of inflammatory cells. Survival of the first-line of defence by the bacteria, results in a local infection and the escape of brucellae from the lymph nodes into the blood. During the bacteraemic phase (which may last two to eight weeks) the bones, joints, eyes and brain can be infected, but the bacteria are most frequently isolated from supramammary lymph nodes, mammary lymph nodes, milk, iliac lymph nodes, spleen and uterus. The tropism of Brucella to the male or female reproductive tract was thought to be by erythritol, which stimulates the growth of the organism, but Brucella has also been found in the reproductive tract of animals with no detectable levels of erythritol. In the acute stage of infection, abortion occurs at four or five months of pregnancy, and cattle usually abort only once. Proliferation of Brucella in the uterus induces necrosis and destruction of the foetal and maternal placental membranes resulting in death and then expulsion of the foetus. Excretion

of Brucella after parturition may re-occur after any consecutive normal parturition. Infected cattle excrete Brucellae in the colostrum or milk although it cannot always be detected. Humans usually acquire brucellosis by consumption of raw milk or milk products, but it is also an occupational hazard for farmers, veterinarians and workers in the meat industry within areas with enzootic B. abortus. Farmers and workers in the meat industry may contract brucellosis by percutaneous, conjunctival or by nasal mucous membrane infection. Veterinarians may become infected when handling aborted foetuses or apparently healthy calves born to infected cows and by performing gynaecological and obstetric manipulations or rectal examination of infected cattle. There are definite host preferences. Secondary hosts play a small part if any in the maintenance or spread of a particular Brucella species. Brucella abortus mainly infects cattle and is the main cause of contagious abortion in cattle, however, sheep, goats, dogs, camels, buffaloes as well as feral animals may also contract B. abortus infections. Although sheep do not easily become infected with B. abortus they may become carriers and excrete Brucellae for up to 40 months once they have acquired the infection. The low prevalence of naturally acquired B. abortus infections reported in goats makes this animal species irrelevant as a host for B. abortus. Swine, horses and camels may acquire infection with B. abortus, however, their significance as a host for B. abortus is doubtful while dogs with naturally acquired B. abortus infections play an important role in the epidemiology of cattle and buffaloes brucellosis. Moreover, while indirect exposure to Brucella organisms could be mediated by wildlife, birds and waterways (contaminated with urine, uterine discharge, or slurry from aborting cattle) it seems that only dogs carry pieces of placenta or aborted foetuses from one place to another causing direct exposure. Also feral animals such as buffalo, swine, deer, fox, hare and rodents are susceptible to Brucellae . The significance of fowl as a reservoir of Brucella is unclear. Flies, arthropods and other parasites may be susceptible to Brucella infection. The disease is prevalent in buffaloes throughout the world: in India the incidence has been reported to be 3-5 percent, in Egypt 20-25 percent, in Iraq 4-5 percent, it has also been reported in buffaloes in Vietnam (Sharma and Kumar, 2003), USSR, Turkey, Philippines, Pakistan, Indonesia (Adlakha and Sharma, 1992), Venezuela, where it is increasing more rapidly among buffaloes than among cattle with as many as 57 percent of Venezuelan herds being infected; in Italy, in 1995, up to 3 percent of the tested animals were infected (D'Apice et al., 1997), in Sri Lanka, 4.2 percent not substantially different from that of cattle (4.7 percent) (Silva et al., 2000) as was also the case in India. The seroprevalence is twice as high in mature buffaloes (over three years) as infection occurs in animals of all ages but persists only in sexually mature animals. B. melitensis has also been isolated from the genital tract of female buffaloes (Adlakha and Sharma, 1992).

Clinical findings- Brucellosis in animals is primarily a reproductive disease characterized by abortion, retained placenta and impaired fertility in the principal animal host. The clinical findings mostly depend on immune status and the physiological status of buffaloes: susceptible pregnant females suffer from abortions after six months, retained placenta and catarrhal metritis. After the first abortion the animal can give birth to full term calves. In bulls epididymitis and orchitis may occur involving one or both scrotal sacs with painful swellings and infection of the accessory sex glands. It has been established that brucellosis in bulls does not always result in infertility, although semen quality may be affected. Bulls that remain fertile and functionally active will shed Brucella organisms with the semen during the acute phase of the disease. Shedding, however, may cease or become intermittent. In contrast to artificial insemination, bulls used in natural service may fail to spread the infection, as the infected semen is not deposited in the uterus. Mild cases are characterized by sinovitis and painful swelling of affected joints. The economic loss from brucellosis in developed countries arises from the slaughter of cattle herds that are infected with Brucella. The economic loss from brucellosis in developing countries arises from the actual abortion of calves and resulting decreased milk yield, birth of weak calves that die soon after birth, retention of the placenta, impaired fertility and sometimes arthritis or bursitis. It is difficult to estimate the financial loss caused by brucellosis, as it depends on the type of cattle farming, herd size, and whether it is an intensive or extensive cattle farm. Furthermore, although it is very difficult to estimate the financial loss incurred by human brucellosis there is no doubt that it is substantial.

Symptoms of acute brucellosis in man, caused by Brucella abortus, are 'flu-like' and highly non-specific, while chronic brucellosis is insidious as the vague symptoms might be confused with other diseases.

Diagnosis- Demonstration of characteristic clumps of Brucella organisms in stained smears of hygroma fluid, chorionic epithelium, or the use of fluorescent antibody techniques to examine foetal stomach content and uterine, or vaginal exudate may provide a tentative diagnosis. Bacteriological examination of lochia of aborting cattle is the method of choice for early infections but it is laborious, time consuming and costly. Moreover, negative culture results do not exclude infection.

Several serological tests are used to detect brucellosis in body fluids such as serum, uterine discharge, vaginal mucus, milk, or semen plasma, as infected cattle may or may not produce all antibody types (M, G 1, G 2, and A types) in detectable quantities.

The commonly used tests are the milk ring test (MRT), serum agglutination test (SAT), Rose Bengal (RB) plate test, complement fixation test (CFT), anti globulin (Coombs) test, 2-mercaptoethanol, rivanol and the enzyme-linked immunosorbent assay (ELISA). The use of several tests to reliably detect brucellosis suggests shortcomings in each of the tests. The ELISA has proven to be specific and as sensitive as the MRT and SAT in detecting Brucella antibodies in milk and serum. ELISA results are usually also in agreement with CFT results.

The test can be used for screening and confirmation of brucellosis in both milk and serum. However, it may test false positive or false negative. It seems less sensitive than the CFT. The main advantage of the ELISA when compared with the CFT lies in its relatively simple test procedure. The assay is very costly when only a few samples are tested, therefore, it is unsuitable for testing individual animals but it is the ideal test for screening suspected herds. The reliability of serological tests to detect brucellosis depends on the antibodies present at the time of examination, inevitably some infected animals may elude detection. The skin delayed-type-hypersensitivity (SDTH) test is independent of circulating antibodies, it should be used to confirm serologic test results. Other tests have been developed and tested, for example flow cytometry (D'Apice et al., 1997); Mathias et al (1998) has compared competitive enzyme immunoassay (CEIA) with the complement fixation test (CFT) and the Rose Bengal test (RBT) obtaining a sensitivity of 100 percent and a specificity of 98.55 percent, suggesting that it could be a useful tool for diagnosis of brucellosis in buffaloes. Other serological test evaluations (competitive ELISA, indirect ELISA, agglutination) have been performed by Molnar et al. (2002).

Therapy- Treatment should be prolonged and with a high dosage of antibiotics, with risks for the human food chain and the possibility of relapses.

Prophylaxis- Efforts are directed at prevention or eradication of brucellosis. Suspect herds must be tested at regular intervals until all the animals test negative. Animals that test positive should be removed from the herd. In areas with endemic brucellosis only vaccination will control brucellosis. Vaccination reduces the number of infected animals and eventually permits disease control. It does not prevent buffaloes from becoming infected with B. abortus, but it prevents abortions protecting 65-75 percent of the herd from infection. Brucella vaccines in use are the live B. abortus Strain-19 vaccine and to a lesser extent the whole cell killed adjuvant B. abortus 45/20 vaccine, and the recently introduced B. abortus vaccine RB51. During vaccination trials, the animals vaccinated with B19 showed a lower percentage of seropositives after 18 months, while the ones vaccinated with 45/20 still had a high amount of seropositive animals (Fenizia, 1991). No scientific tests exist, showing the administration methods and the possible protection given by the RB51, for buffaloes. So its use is not authorized in buffaloes. The use of a reduced dosage strain 19 vaccine has proved to provide comparable protection to the original one (Costa, 2002).

Tuberculosis

Etiology- Bovine and buffalo tuberculosis is caused by Mycobacterium bovis. In Australia Mavium, M. fortui and M. flavescens have also been isolated from buffaloes' tubercles. M. bovis is a long, slender, rod-shaped organism, belonging to the acid-fast group of bacilli; it may be slightly curved and occur in small groups. It is moderately resistant to heat, desiccation and many disinfectants, but not to direct sunlight. In warm and moist places it can survive for long periods: in stagnant drinking water up to 18 days (Adlakha and Sharma, 1992); and in the soil for up to two years. It is considered a disease of socio-economic and public-health importance as M. bovis is a zoonotic bacteria and it is the major cause of human infection in developing countries (Tariq Javed et al. 2004).

Epidemiology- An infected animal is the main source of transmission. The organisms are excreted in the exhaled air and in all secretions and excretions (sputum, faeces, milk, urine, vaginal and uterine discharges and open peripheral lymph nodes). Mycobacteria invade cattle by respiratory (90-95 percent) and oral routes (5-10 percent). Inhalation is the chief mode of entry (it accounts for 97 percent of infections in Australia) and for calves infected colostrum/milk is an important source of infection. Other identified sources of infection are represented by infected flies and birds (Tariq Javed et al. 2004). When infection has occurred, tuberculosis may spread by primary complex (lesion at point of entry and the local lymph node) and by dissemination from primary complex. Tuberculosis lesions can be classified as acute miliary, nodular lesions and chronic organ tuberculosis. Some risk factors influence the development of a clinical or non-clinical form, including calving place, calves' group size, types of concentrates, breed, presence of cattle, herd size, exposure to water supplies, housing system, summer mountain pasture and possibility of contact with wild animals (Tariq Javed et al. 2004). Tuberculosis in small ruminants is rare. In pigs the disease may be caused by the bovine and avian types. Superinfection is specific in cattle. The disease occurs in every country of the world. It has been described for buffaloes in Pakistan, India, Egypt, Australia and Thailand, sometimes as being more severe and with higher incidence than in cattle; in other circumstances as lower compared to cattle and Zebu cattle (Sharma and Kumar, 2003). Despite some claims to the contrary, the water buffalo is susceptible to the bovine strain of tuberculosis (Mycobacterium bovis). Previously it was thought that buffaloes were less susceptible or more resistant to the disease. However, a high incidence of TB has been observed in wild buffaloes. In Pakistan the prevalence in two experiment stations resulted to be 8.48 percent and 2.45 percent. Its incidence is generally higher in buffaloes of around six years of age (Tariq Javed et al. 2004).

Mycobacterium bovis has been isolated in buffalo (*Bubalus bubalis*) in Thailand and Australia (Kanameda and Ekgatat, 1995; Hein and Tomasovic, 1981). Morbidity rates are 20 to 50 percent, but mortality rates are 60 to 80 percent. It is a significant zoonotic disease as man is susceptible to the bovine type and infected buffaloes represent a source of infection for human beings. The incidence of human tuberculosis caused by Mycobacterium bovis has markedly dropped with the pasteurization of milk. It has also dropped in areas where tuberculosis eradication programmes have been developed.

Clinical findings- It is a chronic disease running a course of a few months to years. The first appreciable sign in buffaloes is a general malaise. In the pulmonary form a low grade fever, loss of appetite, emaciation, chronic intermittent hacking cough and associated pneumonia, difficult breathing, weakness, dryness of skin and swelling superficial body lymph nodes are observable, especially the supramammary one when mammary glands are affected and painlessly enlarged. In these cases there are signs of mastitis and the milk becomes watery. The intestinal form is characterized by persistent diarrhoea. More indicative are the lesions: tuberculous granuloma in the lymph nodes of the head, lungs, intestine and carcass. These usually have a well-defined capsule enclosing a caseous mass with a calcified centre. They are usually yellow in colour in cattle, white in buffaloes and greyish white in other animals. Active lesions may have a reddened periphery and caseous mass in the centre of a lymph node; inactive lesions may be

calcified and encapsulated. There can be nodules on the pleura and peritoneum, lesions in the lungs, liver, spleen and kidney, a firmer and enlarged udder, particularly hindquarters, bronchopneumonia, lesions in the meninges, bone marrow and joints. The lesions in buffalo are paler and less calcified than those in cattle, exhibiting a lardaceous consistency. Localizations are mostly thoracic, in mediastinal and bronchial lymph nodes, then abdominal, in the liver, and in the head, retropharyngeal lymph node, and deep inguinal lymph node.
The carcass of an animal affected with tuberculosis requires additional postmortem examination of the lymph nodes, joints, bones and meninges. Carcasses are condemned when the natural prevalence is low (in the final stages of eradication) or in high prevalence areas (early stages of eradication). The carcass of a reactor animal without lesions may be approved for limited distribution, but if there is a good economic situation, this carcass should be condemned. Heat treatment of meat is suggested during the early and final stages of an eradication programme: in low and high prevalence areas where one or more organs are affected, and where miliary lesions, signs of generalization or recent haematogenous spread are not observed. If the economic situation permits, then the carcass is condemned. In some countries, the carcass is approved if inactive lesions (calcified and/or encapsulated) are observed in organs and without generalization in the lymph nodes of the carcass.

Diagnosis- Cases are generally detected in vivo by an intradermal tuberculin skin test and clinical findings and, post-mortem, by lesions. Delayed hypersensitivity reactions can be used as a single intradermal test or comparative test with tuberculins of various origin; also short thermal test and Stormont test are used. The buffalo reaction is more pronounced than that in cattle and can last for more than ten days (Sharma and Kumar, 2003; Tariq Javed et al. 2004). The diagnosis may be confirmed by making a smear of the lesions or excretions; coloured with Ziehl-Neelsen, the TB bacterium appears as a very small red staining bacillus. Inoculation in laboratory animals, like the Guinea pig, can also be used. For a differential diagnosis all these diseases should be considered: lung and lymph node abscess, pleurisy, pericarditis, chronic contagious pleuropneumonia, actinobacillosis, mycotic and parasitic lesions, tumours, caseous lymphadenitis, Johne's disease, adrenal gland tumour and lymphomatosis.

In cattle, lesions of tuberculosis caused by the avian type are commonly found in the mesenteric lymph nodes. For the in vivo diagnosis, apart from the intradermal tuberculin skin test, the indirect hemagglutination test is sensitive for early and advanced cases (Sharma and Kumar, 2003). The use of a dot blot immunoassay has been suggested for the detection and quantification of circulating Ag85. A positive relationship was found between comparative intradermal tuberculin positive tests and serum total proteins and globulins. An inverse relationship was found with the monocyte percentage (Tariq Javed et al. 2004).

Therapy- No treatment is performed since it would be too long and expensive. In animals of high genetic value some therapeutic protocols can be suggested: streptomycin and para-amino salycilic acid; isonicotinic acid and dihydrostreptomycin; isoniazid; streptomycin 2.5 g IM associated with rifampicin 1.5 g and isoniazid 2.0 g PO (Sharma and Kumar, 2003).

Prophylaxis- It is based on eradication: prevention of any introduction or spread of the disease, removing infected animals by slaughter of any positive animal, both clinical and tuberculin. Physical separation for the rearing of offspring.

Paratuberculosis

Etiology- Paratubercolosis is a serious bacterial disease of ruminants caused by Mycobacterium avium subspecies paratuberculosis, developing as chronic granulomatous enteritis and clinically manifested by emaciation and diarrhoea (Chiodini et al. 1984). Cattle become infected at an early age and clinical signs develop after a long incubation period lasting years. In an affected herd most animals are clinically healthy and only occasionally the causative agent is demonstrable in faecal samples (Whitlock et al., 2000).

M. paratuberculosis is an aerobic, non-spore forming, Gram, non motile, acid fast bacillus that is a slow growing intracellular parasite. It is closely related to M. avium and the wood pigeon bacillus M. silvaticum from which it can be separated by DNA techniques such as restriction endonuclease analysis, restriction fragment length polymorphism (RFLP) analysis, pulsed field gel electrophoresis and field inversion gel electrophoresis.

Epidemiology- Johne's disease affects livestock welfare and productivity by way of direct effects on growth and production and indirectly through restrictions on trade. It occurs in a range of animal species, especially ruminants. The most important source of infection is faeces from animals with M. paratuberculosis infection. Early in the disease shedding in faeces may be intermittent. The number of organisms in faeces increases as the disease progresses and may increase when infected animals are subject to stress. Most animals become infected by ingesting the organism in contaminated feed or water. Cattle are usually infected as young calves and develop resistance to infection with age. The survival of M. paratuberculosis in the environment is favoured by low temperatures, moisture and protection from solar radiation. Some animals may become infected in utero and the chance of this occurring increases as the disease progresses in the dam.

Clinical findings- Clinical Johne's disease in cattle typically presents syndromes of chronic and progressive emaciation and persistent diarrhoea. The faeces are usually green and bubbly and do not contain blood or mucus. Faecal consistence may improve over short periods and then diarrhoea may return with increased severity. Affected animals are bright and alert and eat well throughout the course of the disease but in advanced cases, submandibular oedema may be observed. On rectal examination the mucosa may feel thickened or corrugated. The age of onset of clinical signs can be quite variable. In most cases, clinical signs do not appear until animals are more than three or four years of age, but in some herds the onset of disease has been seen in two year old animals.

Although animals with advanced Johne's disease may have bacteraemia, the only specific lesions are found in the intestine and associated lymph nodes. Early in the course of M. paratuberculosis infection, gross lesions may not be evident but, in clinical cases, the mesentheric lymph nodes are enlarged, pale and oedematous. In all host species, specific intestinal lesions are usually more developed in the lower jejunum and ileum. The ileocaecal valve may be enlarged, but the presence of specific lesions in the valve and immediately adjacent tissues is not constant and a wider range of specimens must be examined to ensure a reliable diagnosis. The classical intestinal lesion is diffuse thickening of the intestinal mucosa with development of transverse folds or corrugations. The crests of the rugae may be congested and the mucosal surface is velvety. Necrosis rarely occurs in cattle, and unlike sheep and goats, there is no calcification or caseation.

Diagnosis- Diagnostic test results should be interpreted in the light of epidemiological, clinical and pathological findings. The tests comprehend:

- histopathological techniques (Ziehl.Neelsen staining method).
- bacteriological method (Herrold'egg yolk agar).
- BACTECO (Radiometric culture: growth in liquid medium is identified by the detection of radiolabelled metabolites).
- DNA detection (PCR).
- Intradermal test (no longer widely used because of its poor sensitivity and specificity in individual mammals).
- Interferon test (it is marketed for bovine tuberculosis detection and includes M.bovis
- PPD (purified protein derivative) and M.avium PPD).
- AGID (agarose immunodiffusion test)
- CFT (no longer recommended for diagnosis or certification).
- ELISA (the most sensitive and specific test for serum antibodies).

Therapy- M. paratuberculosis is naturally resistant to many commonly used antimicrobial

drugs. Information about the susceptibility of M. paratuberculosis to antimicrobial drugs is minimal. This is largely due to the fact that treatment of animals with Johne's disease is considered to be too costly. In fact, it has not been considered economically prudent to treat animals with Johne's disease. The chances of curing the animal are low, the cost of the drugs is high and the meat and milk derived from animals treated with the kind of potent drugs required are not suitable for human consumption. Hence, very little research has been done to establish a profile of drug susceptibility based on laboratory tests (i.e. in vitro drug susceptibility testing). In a trial on bovine calves, rifampicin, streptomycin and pyrazinamide were used at doses respectively of 30-25-50 mg/kg/day for a period of seven months and proved successful (Arrigoni et al. 1995).

Prophylaxis- Vaccinating against Mycobacterium paratuberculosis reduces the average economic loss and, therefore, turns out to be profitable. For cows with clinical paratuberculosis the decrease in milk production in the vaccinated group was 13 percent, while it was 21 percent in the non-vaccinated group (Kalis et al. 1995).

Haemorrhagic septicaemia

Etiology- Haemorrhagic septicaemia (HS) is a contagious bacterial disease, also known as shipping fever, caused by two serotypes of Pasteurella multocida, B2, E2. It is a small gram negative, bipolar coccobacillus, not resistant to heat, which in favourable surroundings can survive as long as one week.

Epidemiology- It is regarded as one of the most serious diseases of large ruminants in south east Asia. It affects cattle (*Bos taurus* and *B. indicus*) and water buffaloes (*Bubalus bubalis*) which is the most susceptible species, with a high mortality rate in infected animals. In buffaloes it is mainly caused by type B2 (Sharma and Kumar, 2003). Carriers are the source of microorganism and can include, apart from cattle and buffalo, pigs, sheep, goats and horses. The nasopharynx is the main route of entry by aerosol, vectors are not considered significant but indirect transmission is possible.

HS is principally a disease of animals under stress. In endemic areas about 2 percent of healthy cattle and buffalo carry the organism in the lymphatic tissue of the upper respiratory tract. Intermittently, even in the presence of a circulating antibody, the organisms invade the nasopharynx and are excreted in nasal secretions. These episodes may be triggered by stress. Infection is transmitted by: direct contact between animals or contaminated feedstuffs or water. The bacterium does not survive in the environment for more than a few days. The disease occurs in South and South East Asia, the Middle East and most of Africa. It has also been reported to occur occasionally in Southern Europe.

Clinical findings- Some studies indicate that endotoxin plays a role in the pathogenesis of HS (Horadagoda et al. 1997). Hot, hard, painful swelling of the ventral neck region from throat to dewlap are the most conspicuous signs of HS. The tongue is swollen and mucous membranes are hyperaemic. Breathing is laboured and painful. HS is an acute febrile disease causing heavy mortality in younger animals. In the febrile stage hepatic damage causes bilirubin and other bile salts in blood to increase and cholesterol to decrease. Hot humid conditions favour the spread of the disease. Most cases are acute or peracute, showing: high fever (42°C), depression, reluctance to move, salivation and nasal discharge, painful, oedematous swelling of the throat, extending to the brisket, congested mucous membranes, respiratory distress; calves may have haemorrhagic gastro enteritis. Death occurs in 6 to 48 hours after onset of clinical signs. Recovery is rare. Lesions are mainly represented by oedematous swellings of the throat, brisket containing a clear, straw-coloured serous fluid, blood-tinged fluid in body cavities, pharyngeal and cervical lymph nodes are swollen and congested, subserosal petechial haemorrhages, generalized congestion of the lungs, variable congestion of the abomasum and intestinal tract (calves may have haemorrhagic gastro enteritis). In the case of a quick death the findings could be minimal.

Diagnosis- The organisms disappear from dead animals after some time. Smears should therefore be done immediately after death. In any case they sometimes reveal to be negative in buffaloes. In these cases cultural and biological tests can be performed.
The diagnosis is based on the isolation of Pasteurella multocida from heparinized blood or affected tissues. Samples should be collected aseptically and kept cool. Tissue samples can also be used for histology and immunohistochemistry (Horadagoda et al. 1991).

A differential diagnosis is necessary for blackleg, rinderpest and anthrax.

Therapy- Prompt veterinary care is effective and helpful but treatment is meaningful only in the preliminary stage. Early cases can be treated with sulphonamides coupled with antibiotics like oxytetracycline (Adlakha and Sharma, 1992).

Prophylaxis- In free areas, restriction of imports of live animals from endemically infected countries would keep the risk of introduction at a low level as the micoorganism hardly survives outside the host, but it would not exclude it completely; in fact healthy animals are liable to carry the organism. For the control of HS, three types of vaccines are commonly in use: broth bacterin, alum precipitated and oil adjuvant vaccines. Annual immunization using adjuvant vaccines gives good control in endemically infected areas. Some studies indicate that oil adjuvant vaccines induce a higher response than alum precipitated and lyophilized ones (Farrag et al. 1991), providing immunity for six months to a year, instead of five months (Sharma and Kumar, 2003). Preventative vaccination is usually undertaken annually, one month prior to the monsoon.

Chlamydiosis

Etiology- Chlamydia psittaci is a small bacterium that does not replicate in traditional in vitro media but only in living organisms. It resists up to four months in dung and litter, 17 days in surface water and 10 days in carcasses.

Epidemiology- It mainly affects calves aged three to ten months. The disease has a seasonal trend increasing in autumn. The morbidity is up to 10 to 15 percent and the mortality can reach 100 percent (Adlakha and Sharma, 1992; Galiero and Sica, 1997). Immunosuppression may aggravate the extension and severity of lesions (Gupta et al. 1991). It has been diagnosed in Bulgaria, Brazil, India and Italy. The seroprevalence is high: 13.9 percent in Campania with high detected titres (Baldi et al. 1997).

Clinical findings- Reproductive disorders, abortion, respiratory diseases, kerato conjunctivitis (Baldi et al., 1997). In the buffalo calf, encephalomyelitis at the beginning of the disease causes depression, prestomaches atony followed by the suspension of rumination and constipation without fever. The signs progress from anorexia, sialorrhoea, hard swallowing, pupil dilatation and blindness to evident nervous symptoms: shaky gait, head stretched on the neck (opisthotonos), turning around, paralysis of back limbs, lying down on their back and death after 10 to 15 days, sometimes death occurs in two to three days. At necropsy only hyperemia of the meninx and necrotic foci on the cerebral parenchyma are seen. Cerebral lesions consist of meningoencephalitis lymph-plasmacellular associated with necrosis foci with areas of malacia on the hippocampus, bridge and cerebral cortex (Galiero and Sica, 1997).

Diagnosis- It is based on germ isolation, microscopic lesions, serum conversion. Only laboratory tests can differentiate it from a lack of thiamine (vitamin B1) or an excess of sulphur in the ration.

Therapy- Chlamydia psittaci is sensitive to tetracycline and oxytetracycline (Fenizia et al., 1991). 15 to 20 mg/kg every 12 hours for five days should be administered early, within the first 24 hours from the appearance of the nervous signs. A supportive therapy including cortisone, re-hydration and detoxicants allows a quicker resolution.

Prophylaxis- It is actually based on environmental hygiene using 2 percent formaldehyde, hypochlorite calcium or caustic soda, and destruction of infected animals carcasses, dung and litter (Galiero and Sica, 1997).

Leptospirosis

Etiology- Leptospira spp. are the causative agents that induce infection with various serovars of bacteria. All leptospire are now classified into one species: Leptospira interrogans. It includes over 180 serovars which are divided into 16 different serogroups.

In Brazil, Langoni et al. (1995) found mainly wolffi serovar in 16 farms in Sao Paulo State. Recently the most frequent serovar to which feral buffaloes reacted is pomona (Girio et al. 2004), while from urine samples collected from female adult buffaloes located in a farm, a leptospira strain was isolated belonging to serogroup sejroe which is closely related to serovar guaricura. In a previous study carried out in different buffalo premises in central Italy, serovar tarassovi and hardjo were documented (Autorino et al. 1991). Clinical disease in buffalo seems to result most frequently from Pomona and hardjo infections (Costa, 2002). Leptospiras can survive for months in moist and humid environments, particularly in swamps, ponds and streams or poorly drained pastures.

Epidemiology- Leptospirosis is an important and relatively common disease of domestic and wild animals and humans. It is also considered one of the most important pathologies concerning buffaloes for public health reasons. In fact it is a zoonosis and also represents an occupational hazard for farmers, veterinarians and butchers. Human infection may occur by contamination with infected urine and urine contents. The bacteria may also be found in milk in acute cases, however, it does not survive for an extended period of time in milk. Pasteurization will also kill leptospiras.

Animals contract the disease by eating and drinking leptospira-contaminated urine, water, or by direct contact of broken skin or mucous membranes with mud, vegetation or aborted foetuses of infected or carrier animals. Recovered animals and animals with unapparent (subclinical) leptospirosis frequently excrete billions of leptospiras in their urine for several months or years.

The wallowing habit of buffaloes makes them prone to leptospiral infections since water sources are often contaminated by rodents and wildlife, that are natural carriers of the organism. Buffaloes are important carriers and shedders especially in rice-growing countries. The disease has been reported in India, the USSR, Bulgaria, Romania, Brazil and Egypt.

Clinical findings- In cattle it is manifested by interstitial nephritis, anaemia and mastitis and abortion in most species. The symptoms in the acute and subacute forms are: transient fever, loss of appetite, mastitis, lactating cows may stop milking and milk may be yellow, clotted and frequently blood stained. If animals are severely affected there could be jaundice and anaemia, pneumonia, abortion with frequent retention of the placenta (afterbirth). In young calves the severe illness may be associated with yellowish discoloration of mucous membranes and reddish-brown urine before death. The most indicative symptoms are represented by haemorrhages of mucosa, haemoglobinuria and icterus. In the chronic form there are mild clinical signs and only abortion may be observed. If meningitis occurs, the animal may show lack of coordination, salivation and muscular rigidity.

Lesions are commonly: anaemia and jaundice, subserosal and submucosal haemorrhage, ulcers and haemorrhages in the abomasal mucosa, rarely pulmonary edema or emphysema, interstitial nephritis and septicaemia The carcass of an animal affected with acute leptospirosis is condemned. A chronic and localized condition may warrant an approval of the carcass.

Diagnosis- Direct microscopic examination can be performed on body fluids. Other diagnostic methods are bacteriological culture, biologic test (animal inoculation) and serological tests (dark-field microagglutination test: MAT).

Acute and subacute forms are to be differentiated from babesiosis, anaplasmosis, rape and kale poisoning, bacillary haemoglobinuria, post parturient haemoglobinuria and acute haemolytic anaemia in calves. The presence of blood in the milk is a characteristic clinical sign which will differentiate leptospirosis from other infectious diseases.

Therapy- Antibiotic therapy: Streptomycin: 12 to 15 mg/kg BW, twice daily for three days, or 25 mg/kg BW in a single dose to eliminate infection in carriers; also Dihydrostreptomycin at a dose level of 10 g/1 000 pound cow, has been reported to be effective for termination of the carrier or shedder state; other antibiotics used are chlortetracycline or oxytetracycline. The therapy should be given early, before kidney or liver damage occurs. A supportive therapy for an early recovery should consist of liver tonics and haematinics (Sharma and Kumar, 2003).

Prophylaxis- Livestock herds can be protected against leptospirosis by a combination of proper management and vaccination procedures. Prevention and control is substantially based on periodic testing in endemic areas, elimination or treatment of carrier and clinically infected animals, hygienic measures, and vaccination of susceptible animals.Vaccination should be performed in animals over four months of age and with a booster dose to be given every six months thereafter, as it is not unusual to diagnose abortions caused by pomona in dairy cows vaccinated 8 to 12 months previously. Vaccination programmes can help to control this disease. Usually this is undertaken with formalin inactivated bacterin with either aluminum hydroxide or Freund's complete adjuvant. The latter gives a better serological response. The protocol starts at four to six months of age, followed by annual revaccinations (Sharma and Kumar, 2003). The vaccine should be given to all susceptible livestock on the premises where infection has been identified and the vaccine used in infected herds should be identical with the serotype causing the diseases, as there is little or no cross-protection between vaccine serotypes. In endemic areas a bivalent vaccine should be used. Hardjo is poorly antigenic and does not prevent infection, leptospiruria (shedding), abortions and neonatal weakness for six months. In the case of hardjo infected herds booster vaccination should be performed at three month intervals (Costa, 2002). The future breeding efficiency of herds that have experienced leptospirosis is usually unaltered. Animals should not be culled because they have had the disease. In fact, their value may be enhanced because they are solidly immune against re-infection with the same serotype.

Contagious bovine pleuropneumonia

Etiology- The causative agent is Mycoplasma mycoides subsp. mycoides SC (small colony type) (bovine biotype); there is only one antigenic type.

Mycoplasmas are microorganisms deprived of cell walls that are, therefore, pleomorphic and resistant to antibiotics of the beta-lactamine group, such as penicillin. They cannot survive for more than three to four hours outside the host and are easily killed by heat treatment or by common disinfectants.

Epidemiology- Not being resistant in the environment, the transmission requires close contact and it is aerial, due to droplets emitted by coughing animals, saliva, and urine. Transmission up to several kilometres has been suspected under favourable climatic conditions. Also transplacental infection can occur. Water buffalo (*Bubalus bubalis*) is present among hosts of the disease, while wild bovids and camels are resistant. Buffaloes of all age groups are equally susceptible but once infected, they become immune for subsequent infections. It is of little significance in buffaloes as they are more resistant than cattle, show milder clinical findings and have a higher rate of recovery than cattle (Sharma and Kumar, 2003). However, since international buffalo exports are becoming more common, and since buffaloes may

transmit the infection to cattle, the disease should be taken into account.

CBPP is widespread in Africa and it is also present in other regions of the world, including southern Europe, the Middle East and parts of Asia. Periodically, CBPP occurs in Europe, and outbreaks have occurred in Spain, Portugal, and Italy. Contagious bovine pleuropneumonia was eradicated from the United States in the Nineteenth century. Currently, CBPP is not present in the Western hemisphere.

During an outbreak of natural disease, only 33 percent of animals present symptoms (hyperacute or acute forms), 46 percent are infected but have no symptoms (subclinical forms) and 21 percent seem to be resistent. The incubation period is one to three months (sometimes longer).

Clinical findings- Symptoms are represented by moderate fever with polypnoea, cough (at first dry, slight, and not fitful, becoming moist), characteristic attitude: elbows turned out, arched back, head extended. After exercise breathing becomes laboured and grunting can be heard; at percussion, dull sounds can be noticed in the low areas of the thorax. Infected calves generally present arthritis with swelling of the joints. The disease is difficult to produce experimentally in the buffalo species.

Characteristic lesions are: important amount of yellow or turbid exudate in the pleural cavity (up to 30 litres) that coagulates to form large fibrinous clots; fibrinous pleurisy; interlobular oedema, marbled appearance due to hepatisation and consolidation at different stages of evolution usually confined to one lung; sequestrae with fibrous capsule surrounding grey necrotic tissue in recovered animals.

Diagnosis- Laboratory diagnosis consists of: Identification of the agent: Isolation of pathogen and identification by metabolic and growth inhibition tests; MF-dot; Polymerase chain reaction (PCR).

Serological tests: Complement fixation that should be used only at herd level and never for individual diagnosis; Competitive ELISA (under validation by the International Atomic Energy Agency and several reference laboratories), and haemagglutination; and agglutination test can be used as penside test in active outbreaks at the herd level.

Samples: Lung lesions, pleural fluids, lymph nodes, lung tissue exudate - frozen for isolation of the organism; acute and convalescent sera.

Therapy- There is no efficient treatment. Mycoplasma mycoides mycoides (SC type) is susceptible to a variety of antimicrobials, including streptomycin, oxytetracycline, and chloramphenicol, but antimicrobial therapy may only serve to slow the progression of the disease or may even in some cases favour the formation of sequestra. In the case of chronically affected animals or subclinically affected carriers, the organisms may be in an inaccessible location within an area of coagulative necrosis, which by definition is not served by a blood supply. That is why antibiotic treatment should be prohibited.

Prophylaxis- Sanitary prophylaxis in disease-free areas should consist in quarantine, serological tests (complement fixation) and slaughtering of all animals of the herd in which positive animals have been found. Control of cattle movements is the most efficient way of limiting the spread of CBPP.

In infected areas a CBPP vaccine containing T1 strain is widely used, while a CBPP-rinderpest combined vaccine is sometimes used. Immunity subsequent to vaccination is generally good and lasts at least 12 months. It is advisable to vaccinate animals for export to CBPP free areas.

Anthrax

Etiology- The causative agent is Bacillus anthracis, a gram positive spore forming rod. Anthrax bacilli spores contaminate soil for many years, in fact it can survive from 15 to 20 years in soil. The organisms possess a capsule producing a toxin.

Epidemiology- The disease occurs sporadically. Cattle are generally more susceptible than buffaloes. This is an acute bacterial infection of humans and animals which may be rapidly fatal. The disease occurs worldwide and is an occupational hazard for persons such as wool-sorters, farm workers and veterinarians in contact with infected animals or their by-products. All domestic, zoo and wild animals are potentially at risk of infection. It can be transmitted to humans through blood, meat, hides, etc. The infection in man usually occurs by inoculation from direct contact with infected animals, carcasses or animal products and contaminated soil. Inhalation or ingestion of spores may occur. Animals are infected from contaminated feed, forage, water or carcasses. Insects like biting flies have been shown to be capable of transmission. The disease has a worldwide distribution. Outbreaks are more common in warm and humid conditions like rains after droughts (Sharma and Kumar, 2003).

Clinical findings- It is a febrile disease with high temperatures and swelling of the neck, thorax and lumbar region. Cutaneous anthrax causes localized ulceration (sores) and scabs with fever and headache which may be followed within a few days by severe illness such as septicemia and meningitis. Inhaled anthrax causes fulminating pneumonia. Intestinal anthrax is associated with acute gastroenteritis (nausea, vomiting, and bloody diarrhoea). It is characterized by an abnormal enlargement of the spleen. Blood discharge from natural orifices is common and mortality is very high. In buffaloes there are acute and peracute forms. The first one has a course of about 48 hours with body temperature of 42°C, depression, deep and rapid respiration, congestion of mucous membranes with haemorrhagic spots. Milk can be blood tinged, there can be diarrhoea and edema of tongue, throat, sternum and perineum. In the peracute form death is sudden after convulsions and collapse, without signs except for loss of blood from nostrils, anus and mouth (Sharma and Kumar, 2003).

Diagnosis- This is easy based on records and clinical signs. Peripheral blood or edema fluid smears can reveal the organism, a precipitation test (Ascoli test) from small pieces of ear or muzzle, can be performed. It should be differentiated from peracute black quarter, lead poisoning, acute leptospirosis and bacillary haemoglobinuria. All possible precautions should be observed when handling the carcass.

Therapy- There is not enough time to permit treatment in both forms. In animals of great value an anti-anthrax serum at 100-150ml intravenous associated to antibiotics (Streptomycin 8-10 g/day, oxytetracycline 5mg/kg BW for a minimum duration of five days) can be administered.

Prophylaxis- Spore vaccine works well and provides immunity for one year. The application of anthrax vaccine in risk situations can be helpful. After an outbreak, annual vaccination should be performed for at least three years.

PARASITIC DISEASES

Trypanosomiasis

Etiology- Trypanosoma protozoa are a large family belonging to the class of Mastigophora with a worldwide distribution. In the tropical regions some species are pathogen for animals and man and cause high mortality and morbidity rates. All Trypanosoma, but one (*T. equiperdum*), are transmitted by arthropodes as Glossina. In the water buffalo *T. evansi*, long and narrow (8-39 m), is the agent of Surra disease. It is widely prevalent in the Indian sub-continent and in a number of countries of south-east Asia.

Epidemiology- It is influenced by three factors: arthropodes distribution, protozoa virulence and host immune response. Surra disease is a chronic infection in water buffaloes, characterized by weight loss, infertility and abortion (Luckins, 1988; Davison et al. 1999; Lohr et al. 1986; Thu et al., 1998). It hardly shows clinical signs therefore these animals are often considered as reservoirs. Inapparent infections in buffaloes may develop into clinical conditions if they are stressed by inclement weather or by other infections including liver fluke, rinderpest, foot-and-mouth disease or piroplasm; they may also appear after vaccination. It is present in the blood and within vertebrates' tissues. In the water buffalo *T. evansi* has a high mortality rate as evidenced from different countries in Asia (Luckins, 1988; Lun et al. 1993). It is also widespread in North Africa, South America and throughout most of the livestock-producing areas of Indonesia. In a study carried out on water buffalo in Indonesia, Payne et al. (1991) have observed a prevalence rate of infections higher than for cattle. Furthermore an age-dependent prevalence rate was seen in buffalo and cattle with the highest rates seen in animals older than two years. *T. evansi*, in the water buffalo is incriminated for immunosuppression and may be the cause of vaccination failure against Pasteurella (Stephen, 1986; Holland et al., 2001). To survive in an immunocompetent host, *T. evansi* is able to regularly change the variable surface glycoproteins (VSGc) of the cell surface, a mechanism called antigenic variation. The appearance of new variable antigens types (VATs) results in sequential peaks of parasitaemia intermitted by periods during which the parasites are hardly detectable in the blood (Jones and McKinnell, 1985).

T. vivax is also known to parasitize river buffalo in Central and South America. The disease syndrome has a mortality of 22 percent in buffalo calves.

Clinical findings- There are three basic aspects of trypanosomiasis: Linphoadenopathy and spleen enlargement, haemolytic anaemia, as a main feature of the disease, mainly in cattle, and cell degeneration and inflammatory infiltrates in several organs and tissues such as muscles and the CNS. In buffaloes the disease is characterized by enlargement of the lymph nodes, bilateral mucous discharge from the eyes, emaciation, rough coat, weakness of the hindquarters and recumbency. Acute forms can be fatal (Bhatia, 1992).

Diagnosis- In the case of trypanosomiasis, in order to confirm a clinical suspect, parasites have to be detected in the blood by observing stained blood films.

The diagnosis of Surra can be problematic, particularly in chronic infections. Payne et al. (1991) have evidenced the infection with *T.evansi* by the microhaematocrit centrifugation technique (MHCT) and ELISA test for detection of antibodies to *T.evansi*. Several antibody detection assays based on a predominant VAT have been developed (Verloo et al. 2000). These tests include the CATT/*T.evansi*, which is a simple card agglutination test appropriate for antibody detection in blood or serum applicable under field conditions (Bajyana Songa and Hamers 1988; Davison et al., 1999; Holland et al. 2002).

Therapy- In sheep, cattle and goats diminazene (Berenil) and bromide (Ethidium and Novidium) are usually used. The first one has been used in buffaloes at a dose rate of 10 to 15mg/kg BW in intravenous injection (IV). Chemotherapy for trypanosomiasis in both cattle and buffaloes can be performed with suramin and antrycide methyl sulphate. The drug of choice for treating surra in buffaloes has been indicated to be a 10 percent solution of quinapyramine sulphate, 5mg/kg BW, subcutaneous injection (Bhatia, 1992).

Prophylaxis- In order to control the disease, two strategies are adopted: fighting the flies and a rational use of drugs (isometamidium-Samorin).

An important way to control Trypanosomiasis is through the protection of animals bound for endemic areas and coming from areas where Glossina is absent. A further suggestion involves the introduction, in endemic areas, of trypanotolerant breeds and a simultaneous use of drugs. Antrycide chloride is useful in prophylaxis. Finally, through genetic selection, it would be

possible to obtain trypanotolerant breeds, as the only possible solution to the disease.

Ascaridiosis

Etiology- Toxocara vitulorum is the larger worm of the small intestine of ruminants and it is prevalent in the buffalo population in a number of countries. It is considered a highly prevalent parasite of water buffalo calves between 15 and 120 days of age (Starke et al. 1983). Furthermore it is responsible for high morbidity and mortality rates resulting in serious economic losses.

Epidemiology- The severity of infestation varies from place to place, depending upon many factors such as management and nutrition. Buffalo calves are more susceptible to *T. vitulorum* than cattle calves under conditions of natural infection when they are raised together. This may have been due to differences in the natural immunity of each species (Lau, 2002). Griffiths quoted reports on high incidences of *T. vitulorum* infection in buffalo calves in India, the Amazon valley of Brazil, Malaysia, Sri Lanka and Pakistan. The usual routes of infection are transplacentally and transmammary. In the first route, during pregnancy, larvae become active and the foetus can be infected by ingestion of larvae present in the amniotic fluid. In the second route, the parasite is acquired by calves when they suckle colostrum/milk contaminated with infective larvae from infected cows. It is common to find buffalo calves highly infected between 15 and 90 days of age with the peak egg output occurring 31 to 45 days post-infection (Starke et al. 1983; Roberts, 1990).

After reaching the infection peaks, the parasites begin to be rejected by the hosts and, 120 days post-birth, eggs of *T.vitulorum* are no longer found in the faeces of the calves, suggesting a process of self-cure and immune protection against intestinal infection (Starke et al. 1983; Roberts, 1990). In addition to this, buffalo cows are also able to mount a significant specific antibody response against *T. vitulorum* and the antibodies are transferred through the colostrum to the young buffalo calves after the birth (Rajapakse et al., 1994; Starke-Buzetti et al. 2001).

These passively acquired antibodies do not protect the calves against the acquisition of *T. vitulorum* infection, but may have an important role in worm rejection by calves. However, the rejection is a complex process that involves not only humoral but also cellular immune response and little is known about the immune mechanism of *T. vitulorum* rejection in buffalo calves.

Clinical findings- Main clinical symptoms are due to the presence of adult parasites in the gut of six month old calves. Serious infestations cause growth reduction and diarrhoea in young buffaloes.

Diagnosis- The diagnosis is confirmed by checking faecal samples. Furthermore it is possible to detect serum and colostrum antibodies by indirect ELISA procedure (Starke-Buzetti et al. 2001).

Therapy- Adult worms are sensitive to a wide range of antihelminthics such as piperazine, levamisole and ivermectine.

While the adult parasites are relatively easy to remove from the intestines by anti helminthics, the larvae are difficult to kill, particularly larvae that can be hypobiotic in the musculature and the brain (Abo-Shehada and Herbert, 1984).

Prophylaxis- The diffusion of infestation can be successfully reduced by treating three to six week old calves in order to stop parasite development.

Fasciolosis

Etiology- Fasciolosis is a common disease of domestic ruminants worldwide. Infection by Fasciola hepatica, Fasciola gigantica and Paramphistomum spp. are important parasitic diseases of water buffalo and other livestock both in temperate and tropical climates.

Epidemiology- In some countries the disease has a huge economic importance since water buffalo is the main labour animal, used for work in rice fields and for meat and milk production. It is a serious disease of the liver measured in terms of lowered production and mortality. Young calves acquire infection readily during early winter and may suffer from an acute condition leading to death. It has been observed in many countries including India, Pakistan, Egypt, Turkey, Iraq and Europe. F. hepatica is widespread in Europe and in the higher altitude districts of India. Lymnea truncatula, a mud snail, is involved as the intermediate host for this species in these areas. F. gigantica is widely prevalent in the Indian sub-continent and south-east Asia. Lymnea rufescens, an aquatic snail, acts as an intermediate host in the Indian sub continent. L. rubiginosa is the intermediate host in south east Asia and L. natalensis in Egypt. Buffaloes also carry Dicrocoelium dendriticum infections in their livers in the hill districts of India, Italy and Turkey, and Eurytrema pancreaticum infections of pancreatic ducts in South-East Asia and Brazil (Griffith, 1974; FAO, 1977).

Clinical findings- Fasciolosis, in buffaloes, usually appears as a chronic infection, causing anorexia, weight loss, reducing labour and production capacities, similar symptoms to those in cattle.

Diagnosis- The diagnosis relies upon egg detection in faecal samples, clinical signs and two laboratory tests. The first one evaluates the serum level of hepatic enzymes GLDH (glutamate dehydrogenase), GGT and AST as a result of haepatic cell damage; the other one detects the presence of serum antibodies against some components of the parasite by ELISA method or passive haemoagglutination. Finally, nowadays, biotechnologies (PCR) allow a different and safer approach to the diagnosis.

Therapy- In cattle the antihelminthic treatment aims to reduce the parasite number during winter time when Fasciola is sensitive to drugs for adult parasites.

Prophylaxis- The control of fascioliasis can be dealt with in two ways: by reducing the number of intermediate hosts and by administering drugs. The most correct way to reduce mud population is the reclamation of land by elimination of water ponds in order to remove the intermediate host habitat. Otherwise it is possible to treat muddy areas with copper sulphate.

Babesiosis

Etiology- Babesiosis in cattle is a tick-borne haemoparasitic disease, which is the cause of livestock morbidity and mortality in all semi-tropical and tropical areas of the world. It is an acute and often fatal disease resulting in heavy economic losses. The aetiological agents belong to the genera Babesia and Theileria (Kjemtrup and Conrad, 2000). Babesia bovis is the main pathogen, killing more than half the susceptible cattle that it infects whereas Babesia bigemina, although it infects up to 40 percent of red cells, causes less severe infections (Brown, 2001).

Epidemiology- Griffith reviewed the status of this disease in buffalo in India, West Malaysia and Italy and concluded that babesiosis in buffaloes has rarely been reported.

Clinical findings- Usually B. bovis and B. bigemina invade and replicate exclusively within bovine erythrocytes, causing anaemia and in the case of B. bovis, a fatal cerebral disease associated with the adherence of infected erythrocytes to brain microcapillary endothelial cells (Wright, 1988; Clark et al. 1998).

The disease has a typical biphasic trend: an acute hemolitic crisis followed by a long convalescence in those animals that recover from the infection.

Diagnosis- For diagnosis, anamnesis and clinical signs are useful indications of infection. In order to confirm the diagnosis, it is necessary to examine a blood film stained by the Giemsa method.

Therapy- It depends upon the Babesia species and upon the availability of drugs in the different countries. The most known drugs are imidocarb, pentamidine and amicarbilide.

Prophylaxis-. The adoption of specific treatments for animals born in endemic areas is usually not necessary since the acquired immunity by colostrum is gradually strengthened following repeated babesia infections. On the contrary the main problem of babesiosis is due to difficulties in introducing new animals in endemic areas for genetic improvement. In Australia, selection and breeding of cattle resistant to tick infestations is practised. Control of babesiosis through the eradication of vector ticks is difficult because of the high prevalence rates of vector ticks, the high cost of modern acaricides and the development of resistance to acaricides in ticks. The use of vaccines against babesiosis is practised in tropical countries. In India the inoculation of B. bigemina exoantigens induced a protective immune response. Recently in Australia a live vaccine has been used. In addition a recombinant protein (Bm86 from intestinal cells of the tick Boophilus microplus) has been widely utilized as an anti-tick vaccine (Sharma et al. 2001).

Theileriosis

Etiology- Buffaloes are highly susceptible to the Theileria parva infection which occurs in East African cattle as East Coast Fever. This infection still represents a constraint to cattle breeding because of the large diffusion of the transmitting Rhipicephalus ticks, and the high mortality in animals introduced in endemic areas. In some regions of Africa, where cattle and buffaloes share the same pasture, the epidemiology is complicated by the presence of T. parva lawrenci, buffalo parasite and the natural reservoir of infection.

Epidemiology- Transmitted by Rhiphicephalus appendiculatus theileriosis, it causes illness and high mortality in cattle. Ticks can survive for more than two years on pastures and it is not necessary for buffaloes and cattle to be present together on the pasture for infection to occur. In India, Egypt and Asia, T. annulata infection affects both buffaloes and cattle, although less frequently in river buffaloes than in cattle; the disease is widely spread in tropical and subtropical areas including Portugal, East Europe, the Mediterranean countries (Mediterranean theileriosis), the Middle East, India and China. The infection is transmitted by ticks genera Hyalomma and contrary to T. parva the disease is not always lethal. T. mutans has been reported in swamp buffaloes from Indonesia, West Malaysia and a number of countries in south-east Asia (Griffith, 1974).

Clinical findings- The pathogenesis of the disease shows a first phase without clinical signs (incubation); a second phase characterized by a prominent limphoadenopathy starting from the lymph node involved in the tick bite; and a final phase with lymphoid depletion and lymphopoiesis depression. Usually death occurs after three weeks of infection.

Diagnosis- It is an endemic disease only in those areas where ticks are living. It is possible to observe parasites from spleen and lymph nodes film prepared with needle suction. Parasites may also be detected inside erythrocytes in blood films stained by the Giemsa method. IFAT (indirect immunofluorescence test) can be used to detect those animals that have overcome the infection.

Therapy- The first choice in Theileria treatment is represented by the group of naphthoquinone. Furthermore, tetracyclines can be effective but only when administered at the

beginning of the infection.

Prophylaxis- Traditionally the control of Theileriosis is based on a limitation of animal movements, enclosure of pastures in order to avoid contact with cattle and buffaloes bred extensively; ultimately, periodical use of acaricides is the practice most recommended. The use of an effective vaccine is not useful due to the immune mechanisms of Theileria and the presence of some T.parva strains immunologically different. Purified protein, recombinant protein and cell culture vaccines have been studied. The latter was evaluated on calves during extensive immunization trials and proved non-pathogenic, immunogenic and protective from the fifteenth day following administration up to six months, without passive transferrance from dams to offspring (Sharma et al. 2001).

Strongilosis

Etiology- In weaned buffaloes the main gastrointestinal parasites are the Trichostrongyles: Haemonchus, Cooperia, Ostertagia, Trichostrongylus, Oesophagostomum, Bunostomum, and Nematodirus. The most important gastrointestinal (GI) nematode responsible for considerable production losses in cattle is Ostertagia ostertagi and to a lesser extent, Cooperia oncophora, Nematodirus spp. and Trichostrongylus spp. (Armour, 1989).

Epidemiology- It is generally agreed that mainly first grazing season (FGS) calves may be heavily infected (Ostertagiosis type I) by larvae ingested three to four months previously, whereas, in yearling cattle infection by the Ostertagiosis type II is more common when the animals are turned out for their second grazing season. This is due to the maturation of ingested larvae during the previous autumn. In many cases yearling buffaloes may become immune. In general this is true for genera such as Cooperia and Nematodirus, which induce a rapid build up of protective immunity in their host (Armour, 1989). In contrast, during the FGS calves only develop a partial resistance to the highly pathogenic abomasal nematode O. ostertagi.

Clinical findings- In less developed agricultural systems the severity of the disease caused by these parasites may present the classical clinical signs of stunted growth, tissue oedema, and severe diarrhoea. In more affluent agricultural systems the extensive use of highly efficacious broad spectrum anthelmintics has resulted in a situation where clinical disease is not commonly encountered. But even in these intensively managed herds, the parasites hinder optimal growth and productivity of their hosts. Furthermore, numerous studies have shown that even in well-managed herds, with no signs of clinical parasitism, the presence of the parasites in the herds results in decreased growth in young animals, and decreased milk production in adult cows. Gastrointestinal nematode infections of cattle remain a constraint on the efficient raising of cattle on pasture throughout the world.

Diagnosis- Although sufficient morphological differences exist among adult and larval cattle GI nematodes to allow their accurate identification, the availability of similar techniques for nematode eggs remains an obstacle to reliable diagnosis. In some cases the structure and size of the egg can be diagnostic; however, in many instances, similarities among eggs from different species and even distinct genera require alternative methods for their differentiation. Presently, the method commonly utilized for GI nematodes of cattle involves in vitro cultivation of eggs up to infective, third stage larvae (L3), followed by recovery and morphological identification (Keith, 1953). This procedure is labour intensive, time-consuming and prone to errors due to the variation in egg viability and parasite development in culture. Other methods utilizing microscopic examination require measurements on as many as twenty different parameters per egg, followed by computer analysis and assimilation of the data (Georgi et al. 1989; Sommer, 1996). Clearly, these procedures are equally labour intensive, requiring expensive equipment and the creation of a considerably large data system to decrease the error rate. Christensen et al. (1994) identified and cloned, genus-specific repetitive DNA fragments from a number of important genera of strongylid nematodes infecting cattle and used these as

probes to screen parasite genomic DNA. While this method is sensitive, specific and adaptable to screening egg-derived DNA, as designed, the technique employs radioisotopes and requires multiple assays to distinguish mixtures of eggs. Zarlenga et al., (1998), utilizing a unique internal repeat within the first internal transcribed spacer (ITS 1) of O. ostertagi and Ostertagia lyrata, the most pathogenic of nematode species infecting cattle, developed, a polymerase chain reaction (PCR)-based technique that collectively differentiates and quantifies these species from other common bovine GI nematodes. This method, however, fails to discern one specific species among the other nematode species in the midst of a mixed population. A plethora of parasite-specific PCR primers have been generated for identifying individual nematode species some of which work at the level of a single egg. These and other similar tests require that each parasite DNA be analyzed through a matrix of primer sets and PCR reactions for identification.

Therapy- Ostertagia is sensitive to benzimidazole, levamisole and avermectins both in the adult and developing phase. Following treatment it would be advisable to move animals onto non-exploited pastures. The same therapy is applied for Hemonchus, Trichostrongylus and Cooperia.

Prophylaxis- Historically, control of GI nematodes was accomplished by complicated management programmes which kept stocking rates low and minimized the exposure of susceptible animals to heavily infected pastures. The development of broad spectrum, highly efficacious anthelmintic drugs changed the nature of parasite control programmes, and has resulted in a situation where parasite control now relies almost exclusively upon the repeated administration of drugs to a large percentage of herd members (Zarlenga et al. 2001). For livestock producers, it is important that they identify both the animals harbouring high numbers of parasites, as well as those individuals carrying the more pathogenic parasite species, such as Ostertagia ostertagi in the more temperate regions. Rapid parasite identification would greatly assist the development of control programmes and reduce the number of unnecessary drug treatments (Zarlenga et al., 2001). With regard to Ostertagia, it is kept under control through routine treatments in calves when the larvae number increases. Although animals are treated, they still remain exposed to re-infestations, affecting their productivity. Nowadays the risk of development of helminthic strains resistant to drugs recommends avoiding frequent treatments. The control of other strongylosis is quite similar to what has already been mentioned for Ostertagia.

An increasingly attractive adjunct or alternative for the control of GI nematodes in cattle is the identification of host genes that influence acquired or innate resistance to the parasites and the use of the vast potential of the host genome to reduce parasite transmission in cattle populations. On account of the type of disease caused by these parasites, the control of disease does not require absolute protection from infection. An optimal control programme should instead minimize both the impact of these parasites on productivity and the level of economic input into the production system, while maximizing utilization of renewable resources such as pastures.

Coccidiosis

Etiology- Coccidia are intracellular parasites of the epithelial cells of the intestine. They present a single host in which they undergo both asexual and sexual multiplication.

Epidemiology- Coccidiosis in buffaloes, as in cattle, is widely prevalent and usually affects calves below one year of age, but it may occasionally also occur in yearling calves. Older buffaloes are more resistant to clinical coccidiosis due to either age resistance or acquired immunity. Thirteen Eimeria species are common to both the hosts parasitizing their intestines. In 13 pathogenic species cattle have been detected. Main pathogens are E. zuernii and E. bovis. The first one is particularly severe, affecting the coecum and colon thus causing haemorragic diarrhoea. E. bovis affects the same part of the gut causing enteritis. The prevalence of the

disease may vary from place to place, depending on climatic conditions. The trend of the disease varies according to particular conditions such as overcrowding and lack of hygiene, that are liable to influence the number of oocistis ingested.

Clinical findings- The disease develops in acute, subacute and chronic forms and is responsible for exceedingly heavy economic losses to the dairy industry as its main adverse effect strikes young calves below six months of age. An acute form of coccidiosis in buffalo calves is characterized by bloody diarrhoea. The infected animals may show other clinical signs, such as anorexia, weakness, loss of body weight, anaemia, emaciation and dehydration. Most lesions are found in the small intestine while the large intestine manifests severe catarrhal enteritis and is full of fluids, with blood and fibrinous clots.

Diagnosis- Diagnosis is based on anamnesis, clinical signs and the presence of pathogen oocistis in faecal samples.

Therapy- The preferred drug is sulphamidine and amprolium.

Prophylaxis- Overcrowding and lack of hygiene present optimal conditions for the diffusion of Eimeria. As a result, disease control is based on good farm management, especially with regard to food and water recipients which must be clean as well as litters which should be dry.

Echinococcosis/hydatidosis

Etiology- The genus Echinococcus is very important in the Taeniidae family and is one of the smallest cestodes.

Epidemiology- E. granulosus and E. multilocularis play a significant role in veterinary medicine since their larvae, the hydatids, affect several intermediate hosts including man. E. granulosus is known to exist as biologically distinct subspecific variants or strains which may vary in their infectivity to domestic animals and man.

Clinical findings- Unilocular hydatid disease, caused by the metacestode of E. granulosus is widely recognized as an increasingly important disease in domestic animals in the developing countries. Shamsul (1994) carried out a study regarding lesions of the disease in Bangladeshi buffaloes and reported the higher prevalence of infection in the liver and lungs. Other authors (Munir, 1982; Prasad, 1980) reported the incidence of hydatid infection in different organs of buffaloes.

Diagnosis- Diagnosing hydatidosis is possible through scanning, radiology, serology and postmortem examination. The postmortem examination is usually an important component in monitoring the efficiency of control programmes.

Therapy- Several benzimidazole compounds have been shown to have efficacy against the hydatid cyst in the intermediate host. Long-term treatment with albendazole has a particularly marked effect on the cysts, while long-term treatment with praziquantel only has a limited effect with few changes in the germinal layer of the cyst.

Prophylaxis- Echinococcosis can be controlled through preventive measures that break the cycle between the definitive and the intermediate host. These measures include controlling dogs, inspecting meat, and educating the public regarding the risk to humans and avoiding feeding offal to dogs, as well as introducing legislation. However, none of these measures will work if not applied on a wide scale. Recently, a recombinant vaccine has been developed to be used on sheep.

Mange

Etiology- A serious skin disease in buffaloes, it is caused by Sarcoptes scabiei var. bubalis which may often become fatal in calves. Psoroptic mange in buffaloes is due to an unknown variety of Psoroptes communis.

Epidemiology- It is very common in Swamp buffaloes. The incidence of the disease is likely to increase during periods of drought when opportunities for wallowing become restricted. The disease has been reported in India and Thailand (Dissamaran, 1960). A wide prevalence of sarcoptic mange in buffalo in India has been reported in other works (Chakrabarti et al., 1981). Nowadays such pathology is often reported in cattle of some Northern European countries such as the UK even though the introduction of cattle from areas where this mange is particularly disseminated (Canada and the USA) is forbidden (Urquhart et al., 1996). Griffith (1974) suggested that the incidence of psoroptic mange in South East Asia was much lower than that of sarcoptic mange, from which it should be differentiated by identification of the mite involved. It frequently occurs in Egypt, Pakistan, India, Burma, Indonesia, the Philippines and Thailand (FAO, 1977). It is reported to be most prevalent in Egypt. Maske and Ruprah (1981) reported a high incidence of Psoroptic mange in buffaloes in India from June to September (rainy season) with the highest level of 71 percent being in July in Northern India.

Clinical findings- Early lesions are usually observed where the skin is thin, in particular parts of the body such as the neck and tail. Hair falls out and the skin becomes folding and scaly. Later, wrinkled crusts are formed containing numerous sarcoptic mites in their immature stages. In the beginning, small papules are formed which may turn into scabs. The affected animals try to relieve the irritation or itching by rubbing the lesions against various solid objects. As far as other kinds of sarcoptic mange are concerned, itching is very intense and the economic consequences are weight loss, reduced labour, and milk and meat production capacities, as well as the quality of the leather. In general, little effect is noticed regarding health status. Progressive emaciation, restlessness, weakness, and even death can be observed in heavy infestations. Psoroptic mange mainly affects the shoulder region and the root of the tail (Kassem and Soliman, 1966).

Diagnosis- In order to confirm a diagnosis a skin scraping is usually performed and the parasites are evidenced by microscopic examination, in the case of positivity.

Therapy- A 0.1 percent coumaphos water suspension is effective when applied four times at weekly intervals; a 10 percent sulphur suspension in liquid paraffin should be applied every one to two days (Sukhapesna, 1992). In addition, 0.03 percent water suspension of gamma-BHC, dieldrin, triclorphon, lindane, chlorpyriphos (0.012-0.025 percent), diazinon (0.025-0.05 percent), malathion (1 percent), carbaryl (1 percent) proved to be effective. The first choice is represented by avermectins/milbemycin. A further alternative is pyrethroids application (flumethrin) and amitraz.

Prophylaxis- Most control practices involve the use of insecticides or acaricides, but in some instances it may be necessary to replace chemical applications with accurate management and environmental manipulations.

FUNGAL DISEASES

Buffalo fungal diseases are mainly represented by mycotic mastitis, mycosis of the female reproductive system, mycotic abortion, pulmonary mycosis, mycotic gastroenteritis, cutaneous aspergillosis, keratomycosys, rhinosporidiosis and ringworm. Trichophyton verrucosum is the principal etiological agent of dermatophytosis in buffaloes (Refai, 1991; Adlakha and Sharma, 1992).

Deg Nala disease

Etiology- It is a mycotoxicosis most frequently caused by Aspergillus niger, Alternaria alternata, Fusarium avenaceum, Mucor heimalis, Fusarium oxysporum, Fusarium fusarioides, Cladosporium cladosporoides, Aspergillus flavus and Penicillium notatum (Maqbool et al.,1994).

Epidemiology- The disease affects cattle and buffaloes fed mouldy paddy straw (Sikdar et al., 2000). In buffaloes the disease is more severe than in cattle, due to the higher susceptibility of this species. Secondary bacterial infection of the lesions are partly responsible for the severity of the disease. Rice straw containing multiple dark specks is the main cause. In fact, the disease has been reported from rice-growing areas of India, Pakistan and Nepal and has been responsible for causing considerable economic losses.

Clinical findings- Also called gangrenous syndrome, affected buffaloes show lameness, edema, gangrenous ulceration of limbs, hooves, ears or tail that are cold to the touch. Sometimes the muzzle and tip of the tongue become gangrenous; there is emaciation, recumbency and eventually death. Sometimes gangrenous portions of the body drop off; in the case of hooves, bones can be exposed (Maqbool et al. 1994; Hokonohara et al. 2003). Usually lesions heal within a few weeks, but severe cases can last 1 to 32 months.

Diagnosis- Different fungi species can be isolated from rice straw in agar or liquid media.

Therapy- Lesions should be washed and dressed with nitroglycerine 2 percent ointment. In order to obtain a higher recovery rate, a therapeutic regimen can consist in oxytetracycline at 20 mg/kg BW in a single intramuscular injection or, better, in oral administration of penta-sulphate at 30 g daily for ten days (Maqbool et al., 1994).

Prophylaxis- It is certainly based on the control of straw quality. Maqbool et al. (1994) suggested the use of hydrated sodium calcium alumosilicate (HSCAS) or other sorbents to bind aflatoxins in the gastrointestinal tract.

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Chapter XIV

BUFFALO INTERNATIONAL ORGANIZATIONS

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THE FAO INTER-REGIONAL RESEARCH NETWORK ON BUFFALO FOR EUROPE AND THE NEAR EAST (BUFFALO NETWORK)

The Italian Animal Production Research Institute in Monterotondo (Rome) is the coordination centre for the FAO Buffalo Research Network. This Network is part of the FAO European System of Cooperative Research Networks for Agriculture which includes 13 networks, six of which include other regions in addition to Europe. In 1992 FAO decided to assign some funds in order to establish a Buffalo Research Network for countries where buffalo research occupied a secondary role compared to research on cattle.

OBJECTIVES OF THE NETWORK

The main objective of the Network is to develop a system of cooperation among research institutions from buffalo-producing countries of Europe and the Near East with a view to providing scientific and professional support to the buffalo production sector in general, and to small subsistence farmers in particular. The Network collects and analyses data on production systems, buffalo reproduction and marketing of buffalo products, and disseminates this information through meetings, workshops and the "Buffalo Newsletter". Short to medium-term objectives include the collection of data on animal genetic diversity, reproduction and the establishment of performance recording systems.

PRESENT STRUCTURE OF THE NETWORK

A. Structure

The Network consists of four working groups: with coordinators and vice-coordinators.
1. Reproduction and Biotechnology - A.H. Barkawi (Egypt) - G.M. Terzano (Italy)
2. Farming Systems - Y. Rouzbehan (Iran) - O. Sekerden (Turkey)
3. Products - D. Matassino (Italy) - A. Georgoudis (Greece)
4. Genetic resources - B. Moioli (Italy) - T. Peeva (Bulgaria)

The general coordinator is A. Borghese (Italy).

B. Objectives of the working groups

1. Reproduction and Biotechnology Working Group
The Group acts as a forum for the exchange of research results and as a coordinating body for research on buffalo reproduction and biotechnology. The Group has identified major issues affecting buffalo reproduction and the efficiency of buffalo production and adopted a programme of cooperative research in the following fields:
- puberty and ovarian activity maintenance;
- post partum anoestrus and interpartum period reduction;
- ovulation detection and improvements in artificial insemination efficiency;
- seasonality and oestrus induction;

- follicular dynamics;
- superovulation and embryo transfer;
- *in vitro* maturation and fertilization.

The main objective of the Group is to contribute to the efficiency of buffalo production through the attainment of better reproduction rates, early maturity and development and improvement of insemination efficiency.

The Group organized an International Symposium on Buffalo Reproduction (Sofia, Bulgaria, 6-8 October 1995) the proceedings of which were published in the Bulgarian Journal of Agricultural Sciences (1996), and a Satellite Symposium during the 54th Annual Meeting of the European Association for Animal Production (EAAP) "Recent Progress in Buffalo Reproduction" (Rome, Italy, 30 August 2003), the report of which was published in the Proceedings of the II° Congresso Nazionale sull'Allevamento del Bufalo (Monterotondo, Roma, 28-30 Agosto 2003).

2. Farming Systems Working Group
The objective of the Group is to improve the efficiency of buffalo production through enhanced research in the area of production systems, in particular for smallholders. The following two areas were considered as priorities for research and extension: (i) to ascertain and calculate buffalo nutritional requirements for growth, pregnancy and milk production; (ii) to improve the quality of crop residues as a base for buffalo nutrition in extensive production systems. The Group was responsible for organizing a Symposium on Buffalo Resources (Cairo, Egypt, October 1996) the proceedings of which were published by the Animal Production Research Institute of Cairo.

3. Buffalo products Working Group
The major objective of the Group is to provide a scientific base and guidelines for the production, control and protection of the quality of typical buffalo products in order to enhance consumption and thus contribute to the efficiency of the sector and to improving the standards of buffalo producers.

The Working Group coordinates an interchange of experience and information regarding ongoing research on buffalo products. This research covers consumption and marketing of buffalo products. The results of pertinent comparative studies on buffalo products: i.e. milk, meat and skin, were presented at the International Symposium on Buffalo Products held at Paestum, Italy (1 - 4 December, 1994) and published in the EAAP publication.

This Group will work towards regulating the different products from the developing countries that are very important for the economy of the Mediterranean area.

4. Genetic Resources Working Group
The Group has various objectives:
- The introduction of milk performance recording of buffalo in developing countries through the preparation of simplified guidelines in accordance with the standards of the International Committee for Animal Recording (ICAR), which promoted a Meeting in Slovenia (Bled, 16-17 May 2000).
- Comparative studies concerning genetic diversity of buffalo breeds. A research project aiming to evaluate the genetic diversity of three populations of buffalo has been carried out. This is the first research project which has been initiated as a result of the international links created by the Network. Participating countries are: Italy, Greece and Egypt. The Animal Genetics laboratory of the Animal Production Research Institute, Rome, provided the facilities, the researchers and the materials for the research, including DNA samples of the Italian buffalo population. The Greek and Egyptian partners have also provided DNA samples of the buffalo populations of their respective countries. This study revealed a genetic differentiation between the Italian and Greek buffalo of 0.031 ± 0.015; the differentiation between the Egyptian buffalo and each of the other two is 0.070 ± 0.020.

- The extension of the buffalo progeny testing trial to other countries since, at present, the Progeny testing trial is only beginning in Turkey.

ACTIVITIES OF THE NETWORK (1997-2004)

A. Joint programmes with other organizations

1. With the International Buffalo Federation (IBF)

The Coordination Centre of the Buffalo Network (the Animal Production Research Institute, Rome) actively participated in the organization of the Fifth World Buffalo Congress (Caserta, Italy, 13-16 October 1997) together with the International Buffalo Federation, through: (i) revision of the papers to be presented at the Congress; (ii) editing of the Proceedings; (iii) convening of a round table during the Congress regarding the status of buffalo research and future priorities in Europe and the Near East.

At the Sixth World Buffalo Congress (Maracaibo, Venezuela, 20-23 May 2001) the International Buffalo Federation nominated Professor Antonio Borghese as General Secretary: and this nomination is very important for the continuity of the relationship between the FAO Network and the IBF.

During the Seventh World Buffalo Congress (Manila, Philippines, 20-23 October, 2004) the IBF Constitution and By-laws, registered in Italy, were approved by the General Assembly of the meeting and Professor Luigi Zicarelli was elected as the new President of the IBF for the period 2004 to 2007. The Buffalo Newsletter reports on the activities of the IBF.

2. With animal nutrition scientists from Italian universities

The Coordination Centre of the Buffalo Network (the Animal Production Research Institute, Rome), with the participation of Italian experts on animal feeding and nutrition, convened a meeting in Turkey with buffalo farmers from several countries in order to discuss feeding strategies in intensive and extensive production systems.

3. With INTERBULL (the International Committee for the Standardization of Genetic Evaluation of Bulls)

The major accomplishment of the INTERBULL meeting (1997) was the decision to involve a few countries in a pilot multi-country project on bull evaluation, which began in 2002 as a Progeny testing trial in Turkey.

4. With the International Committee for Animal Recording (ICAR)

The joint goal of this cooperation activity is to promote buffalo recording in developing countries through the preparation of simplified standardized guidelines.
The major achievement of the cooperation with the International Committee for Animal Recording (ICAR) was the organization of the Joint FAO/ICAR Workshop on Animal Recording for Improved Buffalo Management Strategies in 2000, which was attended by 30 participants from 17 countries. As a result of this Workshop, simplified guidelines for milk recording in buffaloes in developing countries were prepared which are in agreement with the ICAR standards. These guidelines were drafted in order to promote animal recording at the country level in addition to encouraging the exchange of information on buffalo productivity in the world.
The Coordination Centre has conducted a survey on the extent of milk recording of buffalo and has published the results of this survey which includes data on production and reproductive parameters from 15 countries.

B. Other outcomes

The scientifical/technical journal of the Buffalo Network (the Buffalo Newsletter) is published regularly twice a year and has a circulation of 1 200 copies. This is an important and unique means of communication among developing and developed countries for the exchange of knowledge and research results.

A major achievement of the round table on the status/future of buffalo research was that it assembled not only participants from the traditional countries (Egypt, Bulgaria, Romania, Iraq, Syria, Greece, Italy, Turkey) but also from Azerbaijan and Iran. The round table therefore proved to be a first step in cooperation with these two more isolated countries.

Conclusions

1. The Buffalo Network is unique in the world.
2. The Buffalo Newsletter is a valuable instrument for communication and for the transfer of technical and scientific news.
3. The Network is the recognized centre for international research projects.

INTERNATIONAL BUFFALO FEDERATION

History

The International Buffalo Federation (IBF) was created during the First World Buffalo Congress, that took place from 27 to 31 December 1985, in Cairo, Egypt.

The initiators were the eminent scientists Professor Dr. M. R. Shalash, President of the Egyptian Veterinarian Buffalo Association and the American scientist Professor W. Cripe from the University of Florida, Gainsville.

Participants at this Congress also approved the organizational structure of the International Buffalo Federation and elected its managing body - the IBF Standing Committee.

The distinguished buffalo expert Dr. W. Ross Cockrill (England) was elected as Honorary President.

Professor Dr. M.R. Shalash was elected as President with three Vice-presidents and fourteen members of the Standing Committee, including scientists and experts from Australia, Brazil, Bulgaria, India, Italy, China, Pakistan, the USA, Singapore, Thailand, Trinidad and the Philippines.

The activities of the IBF to date have been undertaken in accordance with the Statutes and Rules, developed and approved by the Standing Committee.

The Second World Buffalo Congress was held from 12 to 16 December 1988, in New Delhi, India, under the Presidency of Dr. R.M. Acharya and with Professor V.D. Mudgal as Secretary-General.

The Third World Buffalo Congress was held from 13 to 17 May 1991, in Varna, Bulgaria, under the Presidency of Professor Dr. Tzeno Hinkovski and with Professor Dr. Aleko Alexiev as Secretary-General: ten volumes of Proceedings were published in addition to a special report on the FAO Workshop on the Biotechnology of Reproduction, which was the first link between FAO and the IBF, and which would prove to be the foundation of the FAO Inter-Regional Cooperative Research Network on Buffalo.

The Fourth World Buffalo Congress was held from 27 to 30 June 1994, in Sao Paolo, Brazil, under the Presidency of Professor Manoel Osorio Luzardo de Almeida and with Joao Ghasper de Almeida as Secretary-General. Three volumes of Proceedings were published.

During this Congress, the Italian scientist Professor Giovanni de Franciscis was elected President of the IBF. President de Franciscis went on to organize the Eighth Standing Committee Meeting of the IBF in Rome on 2 April 1996, where the following issues were on the Agenda: 1. A commemoration for Professor Shalash; 2. The organizational arrangements and the scientific programme for the Fifth World Buffalo Congress to be held in the Royal Palace in Caserta, Italy, were established; 3. A decision was taken regarding the preparation of an official letter to request the Breeders Associations to contribute US$100 each towards the cost of the organization of the Congress; 4. The transfer of the Secretariat to the Istituto Sperimentale per la Zootecnia (Animal Production Research Institute) was effected; 5. Professor Sayed Gharieb Hassan from Egypt and Dr. Hugh Popenoe from the USA were nominated to the Standing Committee.

The Fifth World Buffalo Congress was held from 13 to 18 October 1997, in Caserta, Italy; under the Presidency of Professor Giovanni de Franciscis and with Professor Antonio Borghese acting as Scientific Secretary. For the first time each paper to be communicated to the Congress was submitted for revision to two referee scientists from the specific field of competence. 189 papers

were published in the Proceedings, a book of 990 pages, that was distributed prior to the Congress and represented the State of the Art in buffalo sciences for many years.

During this Congress, the renowned Venezuelan buffalo breeder Mr. Pablo Moser Guera was elected the new President of the IBF.

It was decided that the Sixth World Buffalo Congress would take place in Venezuela in the year 2000 and the Seventh World Buffalo Congress in the Philippines, changing Continent each time (every three years) and appointing a new president to organize each Congress.

The Sixth World Buffalo Congress was held in Maracaibo (Venezuela) from 21 to 23 May 2001 and resulted in the first electronic version of the Proceedings on CD. During the business meeting of the IBF, Professor Borghese reported on the previous Congress, that had realized a profit of US$4 300, which had subsequently been transferred to the next Congress, and suggested establishing closer relations with the FAO Buffalo Network. The Standing Committee agreed to establish the Secretariat in Rome at the Istituto Sperimentale per la Zootecnia and Professor Antonio Borghese was appointed as General Secretary to be assisted by two Executive Officers: Aleko Alexiev and Hugh Popenoe. The next meeting was to be organized in the Philippines with Libertado Cruz as President assisted by two Vice-Presidents: S.K. Ranjhan and Jesus Reggeti. Libertado Cruz proposed that the changes to the Constitution could be presented to the General Assembly and put to a vote at the next Congress. The sub-committee, consisting of Libertado Cruz, Joao Gaspar, Antonio Borghese and Hugh Popenoe, would proceed with deliberations regarding the revised Constitution. In particular: 1. The Constitution needed to be more precise; 2. The membership could consist of two members from each country; country representatives could not miss more than two congresses or they would be dropped; the official language of the Congress would be English and that of the host country; the profit from any one Congress should go to the Secretariat to cover expenses and for the costs of future congresses. The Standing Committee approved the deliberations of the sub-committee.

There were several changes in the membership of the IBF Standing Committee members during the period 1984 to 2001.

The managing body of the IBF had the following membership at the Sixth World Buffalo Congress, in the period 1997 to 2001:
Honorary President: Dr. W. Ross Cockrill (England)
President: Mr Pablo Moser Guera (Venezuela)
Vice-President for Asia: Prof. Libertado C. Cruz (the Philippines)
For Europe : Prof. Dr. Tzeno Hinkovski (Bulgaria)
For America: Dr. Joao Gaspar de Almeida (Brazil)
Standing Committee members:
Eng. Marco Zava (Argentina)
Dr. Manoel Osorio de Almeida (Brazil)
Prof. Aleko Alexiev (Bulgaria)
Prof. K. H. Lu (China)
Prof. Wangzhen Quan (China)
Dr. L. Ricardo Bolero Jaramillo (Colombia)
Dr. L. Garcia Lopas (Cuba)
Prof. S. G. Hassan (Egypt)
Prof. Kamal Fouad (Egypt)
Prof. P. N. Bhat (India)
Prof. V. D. Mudgal (India)
Prof. Giovanni de Franciscis (Italy)
Mrs. Ingrid Caproni (Italy)
Prof. Antonio Borghese (Italy)
Dr. Abdul Rahman Khan (Pakistan)

Prof. Oswin Perera (Sri Lanka)
Dr.C. Devendra (Singapore)
Prof. Maneevan Kamonpatana (Thailand)
Prof. Charan Chantalakhana (Thailand)
Dr. Stephen P. Bennet (Trinidad)
Prof. Hugh Popenoe (USA)
Dr. Thomas J. Olson (USA)
Mr. Jesus Reggeti (Venezuela)

In 1992 the Asian Buffalo Association (ABA) was established under the Presidency of Dr. P.N. Bhat (India).

An IBF Council Meeting took place at the Congress Palace in Rome on 30 August 2003, organized by the General Secretary, Professor Antonio Borghese. The President Libertado Cruz, M. Zava from Argentina, W. Vale and J.G. de Almeida from Brazil, L. Zicarelli and R. Garofalo from Italy, H. Popenoe from USA, M. Larbier from FAO, A. Barkawi from Egypt, O. Sekerden from Turkey, Ruzbehean from Iran attended the meeting. The President Libertado Cruz thanked the organizer and recalled the Scientist Aleko Alexiev who had passed away. He then distributed the programme of the next Congress, to be held in Manila, from 20 to 23 October 2004. Gaspar de Almeida underlined the difficulties for Cuba to organize the next Congress following that to be held in Manila. Professor Borghese stressed the necessity of receiving funds from the Breeders Associations in different countries in order to run the Secretariat and to contribute to the Congress organization and he proposed the creation of a continuous link with the FAO Buffalo Network in order to locate researchers, exchange information, organize the congresses, and publish the Buffalo Newsletter with FAO and IBF sponsors and logos, distributing 1 200 copies free of charge worldwide and to promote TCP (Technical Cooperation Projects) in developing countries. In order to arrange for the legal Registration of the IBF in Rome and to create a website for the IBF, a contribution of US$100 was suggested from each country. A request was made for the financial report of the previous Congress from Mr Pablo Moser. Professor Zicarelli was elected Executive Officer and Representative for Europe in place of Dr Alexiev.

The President thanked the participants and asked for assistance in the organization of the Congress in Manila.

The organization of the IBF during the period 2001 to 2004 was the following:

International Buffalo Federation
Libertado Cruz, President (Philippines)
Antonio Borghese, General Secretary (Italy)
Hugh Popenoe, Executive Officer (USA)
Luigi Zicarelli, Executive Officer (Italy)

Honorary Committee
Pablo Moser G. (Venezuela)
Steve Bennet (USA)
Giovanni de Franciscis (Italy)

Vice Presidents
Jesus Reggeti, America (Venezuela)
S. Ranjhan, Asia (India)
Luigi Zicarelli, Europe (Italy)
Barry Lemcke, (Australia)
S.G. Hassan, Africa (Egypt)

Standing Committee

Brazil: Joao Gaspar de Almeida, William Vale.
Argentina: Marco Zava, Armando Rozenblum.
Colombia: Ricardo Botero, Berdugo J. A. Gutierrez, Alfonso Bernal.
Venezuela: Hector Scannone.
Italy: Raffaele Garofalo.
Trinidad: Leela Rastogi, Floyd Necles.
Cuba: Alina Mitat.
USA: Tom Olson.
Bulgaria: T. Hinkovski, T. Peeva.
India: Siran Uddin Qureshi.
Thailand: C. Chantalakana, M. Kamonpatana.
Sri Lanka: Oswin Perera, Abeygunawardena.
Vietnam: Julio Ly, Zao.
China: Yang Bing Zhuang, Xu Dianxin.
Philippines: Patricio Faylon.
Pakistan: R. Usmani.
England: Robert Palmer.
Egypt: A.H. Barkawi.
Germany: Henzi Heneton.
Australia: Barry Lemcke.
Turkey: O. Sekerden.
Iran: Y. Ruzbehean

The Seventh World Buffalo Congress took place in Manila, the Philippines, from 20 to 23 October 2004 and produced three volumes of Proceedings: Vol.I Invited Papers, Vol. II Contributed Papers, Vol. III Recent Developments in Animal Production, and one volume of Proceedings' Abstracts, plus one volume of "Abstracts of Researches on the Philippine Water Buffalo".

During the Congress the IBF Assembly Meeting took place at 19:00 hours on 21 October 2004 at the Makati Shangri-la Hotel in Manila.

The President of the IBF, Dr. Libertado C. Cruz opened the meeting, and thanked the delegates from sixteen countries.

The President recalled Professor Aleko Alexiev, an eminent scientist in the buffalo field, who had been Director of the Buffalo Research Institute in Shumen, Bulgaria, and President of the Bulgarian Buffalo Breeders Association. He had been involved with the IBF since its establishment in 1985 and had been elected Vice-President in 2001. He had passed away in 2002. The President also remembered Professor Giovanni de Franciscis, Professor at Naples University (Italy), Faculty of Veterinary Medicine, who had founded the School of Buffalo Sciences in Italy and had been the first President of the Italian Buffalo Breeders Association; he had also been involved with the IBF since its establishment and had been elected President in Sao Paolo, Brazil, (1994) and organized the Fifth World Buffalo Congress in Caserta, Italy, from 13-16 October 1997. He had passed away a few months earlier.

Following this all the delegates from the sixteen countries introduced themselves: A. Borghese, General Secretary, L. Zicarelli, A. Coletta, F. Infascelli, G.M. Terzano, V. L. Barile, from Italy; S. Ranhjan and O.P. Dhanda from India; H. Popenoe and T. Olson, from the USA; M. Zava, from Argentina, I. Soliman from Egypt; B. Lemcke, from Australia; T. Peeva and M. Alexieva, from Bulgaria; G. de Almeida, W. Vale, M. Almeida from Brazil; M. Eslami, from Iran; N. Ahmad, from Pakistan; L.C. Cruz, the President and A. del Barrio, from the Philippines; T. Seresinhe, from Sri Lanka; M. Wanapat, from Thailand; O. Sekerden, from Turkey; J. Reggeti, from Venezuela and Mai Van Sanh, from Vietnam.

Point 1. IBF Constitution.
The General Secretary of the IBF, Professor Antonio Borghese, submitted, for confirmation by the Assembly, the Legal Act of the IBF Constitution, registered in Monterotondo, Rome, on 11 October 2004, by the legal notary Dr. Francesco Di Pietro together with Professor A. Borghese, avv Raffaele Garofalo, Professor Luigi Zicarelli, Dr. Giuseppina Maria Terzano and Dr. Vittoria Lucia Barile; the legal address is the same as that of the General Secretariat: Istituto Sperimentale per la Zootecnia, Via Salaria 31, 00016 Monterotondo, Rome, Italy. The Constitution and By-laws are the same as those approved in Caserta on 16 October 1997, with the formal modifications proposed by President L. Cruz, and published in the Buffalo Newsletter, Number 20, dated September 2004, printed in 1 200 copies and distributed worldwide. The organization contained in the Legal Act is the same as that approved in Maracaibo, Venezuela from 21 to 23 May 2001, and published in the same issue of the Buffalo Newsletter. The IBF subscription is set at a minimum of US$100 for the years 2004 to 2007, and the IBF founder members will be the signers of the Application Form, which involves adherence to the IBF Constitution and By-laws, and payment of the minimum subscription.

The General Secretary emphasized the three priority goals for the efficient functioning of the IBF:
A. The legal constitution of the IBF, that was officially founded in 1985 but had not been bound by any legal documents in the preceding years;
B. The official publication of the IBF Constitution, By-laws, Organization, Representatives, Meetings and Activities in the Buffalo Newsletter, that originally was the bulletin of the FAO Inter-Regional Cooperative Research Network on buffalo and has now become also the bulletin of the IBF;
C. An economic foundation for the IBF activities, that clearly will be supported in part by subscriptions, and will allow participation in the IBF.

The Assembly approved points A and B, but there was some discussion regarding point C, concerning the subscription. Dr. Ranjhan proposed that the subscription be only made obligatory for Associations, and that there be free subscription for memberships. Many participants (Reggeti, Soliman, De Almeida, Zava, Popenoe, Cruz) commented on this point, and some proposed a referendum. The General Secretary replied that a referendum would be impossible since up until the present time it is difficult to know who really are the IBF members. Every year membership changes. At each congress some countries, who in the past were really interested in the IBF, have no representatives, or representatives change because there is no formal act of adhesion to the IBF. For the functionality of the IBF, there was a need for a Secretariat office, an economic foundation, but specifically a real act of adhesion to the IBF by members. Many participants agreed with the General Secretary's position.

Point 2. Appreciations.
Many participants expressed their appreciation for the reformatted IBF Constitution, as proposed by the President Libertado C. Cruz, and published in the Buffalo Newsletter, and congratulated the President on the excellent Congress organized in the Philippines, which had been an important success for the scientific community and for the buffalo breeders in the world.

Point 3. Next congress and President.
With regard to the next World Buffalo Congress in 2007, many people expressed their views (Cruz, Ranjhan, Vale and Zava) and the past willingness of Cuba and China to organize the Congress was reported. However, this proposed readiness was not confirmed by the presence of the respective representatives at the IBF Assembly, even if clearly invited. Professor Borghese proposed to change the Continent, as traditionally undertaken in the past: in 2001 the Congress had taken place in America, this year in Asia, the next would be in Europe and therefore he proposed Professor Zicarelli as President. Professor Sekerden also proposed Turkey as a host for the next Congress.

All the delegates voted for Italy, appreciating the past experiences in organizing congresses and the link with FAO (Peeva, Dhanda, De Almeida, Vale, Zava, Reggeti) and the economic possibilities. Professor Zicarelli thanked the delegates for the honour and declared his satisfaction to take up the legacy of his teacher Giovanni de Franciscis, requesting the assistance of the Italian Breeders Association, of the Agricultural Ministry, of Professor Borghese's Institute and of the other Italian organizations.

Point 4. Actual IBF organization.
The actual organization of the IBF for the period 2004 to 2007 was voted as follows:
President: Luigi Zicarelli (Italy) zicarell@unina.it
General Secretary: Antonio Borghese (Italy) antonio.borghese@isz.it
Executive Officer: Libertado C. Cruz (Philippines) pcc-oed@mozcom.com
Exexutive Officer: S. Ranjhan (India) sk_ranjhan@hotmail.com
Executive Officer: Hugh Popenoe (USA) hlp@ufl.edu
Vice-Presidents:
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Australia: Barry Lemcke barry.lemcke@nt.gov.au
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Standing Committee:

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Hand over of the IBF Flag from the Past President Libertado C. Cruz to the new elected President Luigi Zicarelli, Manila, the Philippines, 21 October 2004.

Participants of the IBF Assembly Meeting, Manila, the Philippines, 21 October 2004.

INTERNATIONAL BUFFALO FEDERATION
CONSTITUTION AND BY-LAWS

Section I. Name, Address and Nature

1. The International Buffalo Federation (IBF) was founded upon the unanimous recommendation of the General Assembly at the First World Buffalo Congress in Cairo, Egypt in 1985.

2. The Federation is an independent, non-political, non-religious and non-profit international organization.

3. The permanent headquarters of the International Buffalo Federation is Rome.

4. The seat of the Federation is the country which will be hosting the World Buffalo Congress.

Section II. Objectives and Activities

1. The Federation objectives are to promote the advancement of research and development regarding buffaloes worldwide.

2. The Federation organizes world congresses and roundtables; promotes the exchange and dissemination of scientific and technological knowledge; facilitates the spread of information on buffalo production and development; promotes internationally planned research; enhances contacts among scientists and extension personnel concerned with buffalo production; assists in strengthening the linkages between national, regional and international research; establishes and maintains relations with other organizations whose interests are related to the objectives of the Federation.

Section III. The Official Language

1. The official language at the World Congresses of the Federation is English.
2. The working language of the Secretariat is English.

Section IV. World Buffalo Congress

1. The World Congress shall be held every three years. Regional and National Congresses will not be held in conflict with the World Congress.

Section V. Organization and Institution and Election of Officers

1. The International Buffalo Federation is organized on a regional basis. For the purposes of the Federation, each continent shall be a region.

2. The Institutions of the International Buffalo Federation are the General Assembly, the Secretariat and the Executive Council.

3. The Executive Council of the International Buffalo Federation is composed of the elected Chairmen of the Regional Associations, a Secretary appointed by the President, and the President elected in the General Assembly by the representatives of the National Associations.

4. The Executive Council will be vested of its powers after having been elected (at the

end of the World Congress) and its tenure will be for three (3) years.

5. The Executive Council will meet as often as necessary. An absolute majority is the required quorum for the meetings. Resolutions will be approved by a two-thirds vote. The President does not vote except when the vote is a tie. His presence however counts for the quorum.

6. Members of the Regional Associations will elect their own Executive Councils. The Chairman of the Regional Association is by right a member of the International Executive Council.

7. The President of the International Buffalo Federation is elected at the World Congress.

8. Each National Association affiliated with the IBF nominates a representative to the World Congress for the purpose of electing the President.

9. National Associations enjoy equal voting rights, being allowed one vote each.

10. Nominations for the candidacy to the Presidency of the IBF is submitted to the Executive Council six months prior to the World Congress. The Executive Council is responsible for circulating information on the upcoming election and about nominees to all IBF members.

11. The committee organizing the World Congress allocates sufficient time in the official programme to hold the election of the President. The election shall be public. Only the official representatives of the National Association duly appointed by them, are considered electors of the President.

12. The election is to be held by secret ballot. The candidate who obtains the absolute majority, is proclaimed President by the outgoing Executive Council of the International Buffalo Federation.

12.1 If no winner can be proclaimed on the first turn a run-off election is held between the two highest vote getters.

12.2 The President serves for a maximum of two terms.

Section VI. Duties of the President

1. It is the duty of the President to represent the Federation at international meetings, and with International Organizations.

2. To convene the Executive Council as often as necessary or when at least two Regional Chairmen ask for it to be convened.

3 To promote the initiatives which will further the knowledge regarding buffaloes and pursue the objectives of the Federation.

4. To convene the Assembly to discuss the Federation's administration, general programme, policies and priorities.

Section VII. Membership

1. National, institutional and individual membership are recognized and encouraged by the International Buffalo Federation. However representation in the Executive

Council is accorded to Regional Officers who will be appointed according to the procedures set out in Article 6, Section V.

2. Membership of the International Buffalo Federation is renewable by submitting the application form and dues. Deadlines and fees shall be established by the Executive Council. Non payment of the fees, actually US$100/year, implies forfeiture of membership status.

3. Membership in the International Buffalo Federation falls into three categories: Collective, Associate and Individual membership.

3.1 National or Regional associations are Collective members.

3.2 Departments and Research Institutes are Associate members.

Section VIII. General Assembly Meeting

1. The General Assembly meets at the World Congresses every three (3) years.

Section IX. Amendments of the Constitution

1. In order to amend the By-laws of the International Buffalo Federation a written notice of the amendments must be circulated to all members in advance of the meeting at which they are to be considered.

2. If a meeting of the General Assembly cannot at that time be called, the membership is allowed to express its vote through a written ballot.

3. The amendment is approved if the majority of the members voting are in favour of it.

Section X. Transitory and Final Provisions

I. This Constitution and By-Laws adopted by the General Assembly of the Federation, in Caserta on 16 October 1997, promulgated by the President of the International Buffalo Federation, will render the former constitution null and void.

II. Elections of the Executive of the Regional Associations shall be called within one year of the implementation of this Constitution and By-Laws.

III. The first IBF Executive Council, under this Constitution and By-Laws, will be composed of the Presidents of the member National Associations until all the regional associations and their executives have been set up. The rules pertaining to the quorum and voting regulations shall be the same as those prescribed in Article 5 Section V of the Constitution and By-Laws

Finalized in Caserta, Italy on 16 October 1997.

LIST OF ACRONYMS

AACB	Asociacion Argentina de Criadores de Bufalos
ABA	Asian Buffalo Association
ACTH	adrenocorticotropic hormone
ADF	acid detergent fibre
ADL	acid detergent lignin
AETE	Association European Embryo Transfer
AGID	agarose immunodiffusion test
AHV-1	alcelaphine herpes virus-1
AHV-2	alcelaphine herpes virus-2
AI	artificial insemination
AIA	Italian Breeders' Association
ALP	alkaline phosphatase
ALT	alanine aminotransferase
ANASB	Italian Buffalo Breeders' Association
AOAC	Association of Official Analytical Chemists
APA	Associazione Provinciale Allevatori (Provincial Breeders' Association)
APRI	Animal Production Research Institute
ASPA	Associazione Scientifica Produzione Animale (Scientific Association of Animal Production)
AST	asparagine aminotransferase
ATP	adenosin triphosphatase
BCS	body condition score
BEF	bovine ephemeral fever
BES	buffalo oestrus serum
BLUP	best linear unbiased prediction
BO	Brackett & Oliphant medium
BOHB	beta-hydroxybutyrate
BRL	buffalo rat liver cells
BRV	bovine rotavirus
BUFF	buffalo follicular fluid
BVD	bovine viral diarrhoea
BW	body weight
CATT	card-type Testryp CATT agglutination test
CBC	cells blood count
CBG	corticosteroid binding globulin
CBPP	contagious bovine pleuropneumonia
CEIA	competitive enzyme immunoassay
CF	crude fibre
CFT	complement fixation test
CIDR	controlled internal drug releasing device
CIRB	Central Institute for Research on Buffaloes (India)
CIEP	counterimmunoelectrophoresis
CISE	Cattle Information System/Egypt
CL	corpus luteum
CNF	cytotoxic and necrotic factors
CNS	central nervous system
COCs	cumulus oocyte complexes
Co-EDTA	cobalt-ethylendiaminetetraacetic acid
COFA	Cooperativa Fecondazione Artificiale, Cremona, Italy
CP	crude protein
CPE	cytopathologic effects
Cr	Cr_2O_3, solid marker
CR	conception rate
CRESTAR	progestagen ear implant

CRF	corticotropin releasing factor
CRL	crown-rump length
CV	variability coefficient
DCP	digestive crude protein
DF	dominant follicles
DIPA	dairy herd improvement programme actions
DM	dry matter
DNA	deoxyribonucleic acid
D.O.P.	Denomination of Protected Origin
DWG	daily weight gain
E2	estradiol
EAAP	European Association for Animal Production
EBW	empty body weight
eCG	equine corionic gonadotrophin
ECM	equivalent correct milk
EDTA	etilendiamminicotetracetic acid
EE	ether extract
EGF	epidermal growth factor
ELISA	enzyme-linked immunosorbent assay
ESCORENA	European System of Cooperative Research Networks in Agriculture
ET	embryo transfer
ETEC	enterotoxigenic escherichia coli
EU	European Union
F1	first generation cross
F2	interbred of F1
FAO	Food and Agriculture Organization of the United Nations
FAT	fluorescent antibody test
FCM	fat corrected milk
FCS	fetal calf serum
FFA	free fatty acids
FGF	fibroblast growth factor
FGS	first grazing season
FL	femtolitre (10-15)
FMC	fat corrected milk
FMD	Foot and Mouth Disease
FSH	follicle stimulating hormone
FU	feed units
GGT	γ-glutamyltransferase
GI	gastrointestinal
g/l	grams/litre
GLDH	glutamate dehydrogenase
GnRH	gonadotrophin releasing hormone
GR	glutathione-reductase
GSH	glutathione
GSH-Px	glutathione peroxidase
GSH-S-t	GSH-S-transferase
HAU	Haryana Agricultural University
Hb	hemoglobin
hCG	human corionic gonadotrophin
HCT	hematocrit
HDL	high density lipoprotein
HGF	haematopoietic growth factor
Hly	hemolysin
HMG	human menopausal Gonadotrophin
HS	haemorrhagic septicaemia
HSCAS	hydrated sodium calcium alumosilicate

IBF	International Buffalo Federation
IBR/IPV	infectious bovine rhinotracheitis/infectious pustular vulvovaginitis
ICAR	International Committee for Animal Recording
ICAR	Indian Council of Agriculture Research
IETS	International Embryo Transfer Society
IFAT	indirect immunofluorescence test
IGF	insulin-like growth factor
IGFRI	Indian Grassland and Fodder Research Institute (India)
IGP	Indication of Protected Geographic Origin
INRA	Institut National de la Recherche Agronomique - France
INTERBULL	International Committee for Standardization of Genetic Evaluation of Bulls
ISZ	Istituto Sperimentale per la Zootecnia (Animal Production Research Institute)
ITS	internal transcribed spacer
IU	International Units
IV	intravenous
IVC	in vitro culture
IVEP	in vitro embryo production
IVF	in vitro fertilization
IVM	in vitro maturation
Keller	tank for urine and dung
KSOM	potassium simplex optimized medium
L3	third stage larvae
LDH	lactate dehydrogenase
LH	luteinizing hormone
l/l	litre/litre
LT	thermolabile toxins
MII	metaphase II
mAbs	monoclonal antibodies
MALR	Ministry of Agriculture and Land Reclamation (Egypt)
MAT	microagglutination test
MCF	malignant catarrhal fever
MCH	mean corpuscular hemoglobin
MCHC	mean corpuscular hemoglobin concentration
mcmol/l	micromoles/litre
mcU/l	microunit/litre
MCV	mean corpuscular volume
MD	mucosal disease
MDS	myelodysplastic syndrome
ME/MJ	milliequivalent/megajoule
MFU	milk feed unit
MHCT	microhaematocrit centrifugation technique
MHz	megahertz
ML	modified live
mmol/l	millimoles/litre
MOET	multiple ovulation and embryo transfer
MPUAT	Maharana Pratap University of Agriculture and Technology (India)
MPV	mean platelet volume
MRT	milk ring test
NARC	Nepal Agricultural Research Council
NDDB	National Dairy Development Board
NDF	neutral detergent fibre
NE	net energy
NE_L	net energy milk
NEFA	not esterified fatty acids
NEFDCCO	Nueva Ecija Federation of Dairy Carabao Cooperatives
ng	nanograms (10-9)

ng/lt	nanograms/litre
NH3	ammonia
NK	natural killer
NORs	nucleolus organizer regions
NPN	non proteic nitrogen
NSC	non-structural carbohydrates
NWFP	North West Frontier Provinces (Pakistan)
OHV-2	ovine herpes virus-2
OIE	Organisation Mondiale de la Santé Animale (World Organization for Animal Health)
OM	organic matter
OPU	ovum pick-up
P4	progesterone
PAGE	polyacrilamide gel electrophoresis
PAGs	pregnancy-associated glycoproteins
PCR	polymerase chain reaction
PCV	packed cell volume
PDI	intestinal digestible protein
PDGF	Plateled-derived growth factor
P/E	protein/energy
PFA	Prevention of Food Adulteration
PFDM	protein-free dry matter
PGF2α	prostaglandine F2α
PGFM	15 cheto-diidro PGF2α metabolite
PI	persistent infection
PMN	polymorphonuclear cells
PMSG	pregnant mare serum gonadotrophin
PPD	purified protein derivative
PRID	progesterone-releasing intravaginal device
PRL	prolactin
PSPB	pregnancy specific protein B
Px	peroxidase
RB	Rose Bengal
RBT	Rose Bengal Test
RDW	red cell distribution width
RFLP	restriction fragment length polymorphism
RIA	radioimmunoassay
RMSE	root mean square of error
RNA	ribonucleic acid
RP	rinderpest
SAR	rapid serum agglutination
SAS/NLIN	Statistical Analysis System/non linear regression
SAT	serum agglutination test
Sd	standard deviation
SDS	sodium dodecyl sulphate
SDTH	skin-delayed-type-hypersensitivity
SG	sub group
SO	superovulation
SOD	superoxide dismutase
SOF	synthetic oviductal fluid
S-phase	solid phase
ST	thermostable toxins
ST.E.	standard error
SVD	swine vesicular disease
T_3	triiodothyronine
T_4	thyroxine

TALP	Tyrode's modified medium
TCA	tricarboxylic acid
TCM	tissue culture medium
TCP	Technical Cooperation Project
TDN	total digestible nutrients
TE	transferable embryos
TGF	transforming growth factor
TH1	TH1 Lymphocytes
TH2	TH2 Lymphocytes
TNF	tumour necrosis factor
t.q.	tal quale (as fed)
TRH	thyrotropin releasing hormone
TSH	thyroid stimulating hormone
TTR	total retention time
U car.	Carratelli unity
UFC	meat feed unit
U/L	unit/litre
VAT	variable antigens type
VER	vaginal electrical resistance
VP1	viral capsid protein
VS	vesicular stomatitis
VSGc	variable surface glycoproteins
VT	vero cytotoxin
WBC	white blood count
WCY	warm carcass yield
WC1-N3	leucocytes cluster
WC1-N4	leucocytes cluster
$\bar{x}$	statistical mean

C.R.A.

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