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The Editorial Board of "Blue Cross Book", while delightfully bringing out its 31st volume, wishes all its readers a "Happy and Prosperous New Year 2015".

As the Blue Cross Book has been emphasizing in its earlier volumes the important issues in livestock health and productivity, the present volume also deals with the dearer issues like aetio-pathogenesis, diagnosis and therapeutics of diseases like ketosis and equine colic. Dealing with infertility in relation to nutritional and hormonal approach has become a regular feature of Blue Cross Book, which is being highly appreciated at readers' level. Treatment of Bovine Mastitis with available antibiotics requires understanding of pharmacodynamic and pharmacokinetic characteristics of a given antibiotic. An article in this issue provides guidelines for effective use of antibiotics in sub-clinical, clinical mastitis and dry cow therapy.

The clinical reports published in the present volume shall help widen the approach of a Veterinarian in the therapeutics of various diseases, using newer drugs and drug combinations.

Climate change and its effects on agriculture and livestock productivity are being debated extensively the World over. Apart from affecting the productivity of livestock, the emergence and re-emergence of zoonotic diseases due to climate change is also a matter of concern in livestock sector. This is new area of study in a country like India, which already has great zoonosis problems. When climate change is affecting agricultural productivity, the sustainable agriculture is possible only through the livestock rearing. This aspect needs greater attention from the planners as indicated in the article on sheep based integrated farming system.

It is hoped that readers appreciate the wide range of topics being covered by the 'Blue Cross Book'. A word in appreciation or otherwise is expected from the readers.

The 'Blue Cross Book' is changing the format of publication of articles from its next issue. The following changes need to be noted by the contributors. **Abstract of article and key words are mandatory. E-mail address of the corresponding author is a must, along with mobile phone number.**









Dr. Yash Goyal Managing Director, MSD Animal Health

Dear Professional Colleagues,

On behalf of MSD Animal Health family, I wish all esteemed readers and our professional fraternity a "Happy and Prosperous New Year 2015".

MSD, the world over, has accepted a lifelong obligation to continually improve the professional knowledge and competence of the Veterinarians working with the livestock owners. A publication of Blue Cross Book is one of the ways of our endeavor to get associated with the Indian Veterinary profession. MSD shall always strive to select such contributions in the 'Blue Cross Book' which are current and of interest to as wide a sector of our readership as possible.

The present issue (No. 31) is not an exception and covers all major concerns in livestock health and productivity like nutritional and hormonal management of infertility to metabolism and production related disorders, mastitis to worm infestation and so on.

We, at MSD, are working firmly for our mission to develop and market innovative, integrated animal health solutions; and we further assure that our rich science credentials would be fully utilized to fulfill this mission.

I personally appeal to those involved in Veterinarian Education, Research and Extension to contribute their experience and expertise to the benefit of those who have been directly working with the people engaged in livestock production.

Best Wishes,

Yash Goyal



OUALITY PROTEIN FOR A HEALTHY WORLD

BY 2050 IT IS ESTIMATED THERE WILL BE AN EXTRA 2 BILLION PEOPLE IN THE WORLD. TO FEED THEM, WE WILL NEED TO HELP ANIMAL PRODUCERS BECOME MORE EFFICIENT AND MORE SUSTAINABLE.

Animal diseases still cost farmers a significant proportion of their meat, fish and dairy yield. Preventing disease-related costs will be crucial if we are to meet the demand for protein, created by rising standards of living and population growth. In addition, the land and water available for agriculture will only decrease. So not only will our animals have to be healthier, they will have to be reared more efficiently too.

Our portfolio is already focused on helping farmers keep their livestock productive. Targeted intervention with vaccines, therapeutics and performance technologies ensures that animals reach their full weight in good health.

Our global reach also means few companies are better placed to limit production losses from epidemics. To tackle foot-and-mouth disease for example, Merck Animal Health has the resources, including antigen banks managed on behalf of governments, to better prevent and manage outbreaks across international borders.



SPOTLIGHT ON DIGITAL COWS

MERCK ANIMAL HEALTH IS DEVELOPING TECHNOLOGIES THAT HELP MAXIMIZE OPERATIONAL EFFICIENCY. TECHNOLOGIES LIKE TRI-MERIT – A SIMPLE EAR TAG SYSTEM THAT DIGITALLY VERIFIES AGE, SOURCE AND MOVEMENT OF ANIMALS. IT'S ANOTHER WAY THAT MERCK ANIMAL HEALTH IS HELPING FARMERS TO INCREASE THEIR PRODUCTION CAPABILITIES.

RUMINANTS

The lives of millions of people around the world depend on the health of their cattle, sheep and goat herds. But with fewer natural resources, and changes in our climate, we must rear them more efficiently, and continue to eradicate diseases that lower meat and milk yields.

Our portfolio for ruminants is organized into eight specific health platforms. Provided under the umbrella name EXPERTIS, our products and services have been developed to work together around our customers' greatest needs.

Our health platforms are:

- Herd Health
- Lung Health
- Neonatal Health
- Parasite Control
- Partners in Reproduction
- Performance Technology
- Sheep Health
- Udder Health

Tackling the world's most virulent infectious diseases is the first step in ensuring reliable meat and milk supplies.

Collaboration with veterinarians, farmers, regulators and scientists has helped us implement herd health programs in many regions of the world. BOVILIS BVD, for control of bovine viral diarrhea, is now one of the most important vaccines for dairy cattle in Europe. BOVILIS BTV8 was developed in less than two years, in rapid response to the spread of the bluetongue virus across Europe in 2007.

Controlling parasites is also vital to help farmers ensure maximum productivity. Our parasite control platform includes a full range of worm and fluke control products, including the anthelmintic PANAC UR/SAF E-GUARD (fenbendazole) and the antiprotozal drug HALOC UR (halofuginone lactate).

Our Lung Health platform provides customers with tailor made programs to tackle complex problems like bovine respiratory disease. It also includes antibiotics like NUFLOR (florfenicol) and RESFLOR (florfenicol plus flunixin). And vaccines that guard against infectious bovine rhinotracheitis (IBR) and parainfluenza-3 (Pi3).

Products that improve performance are key to efficient, sustainable production. Products like ZILMAX (zilpaterol), RALGRO (zeranol) and REVALOR (trenbolone/estradiol) are proven to improve feed conversion and aid weight gain.

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Ketosis in dairy cattle and its management

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Introduction:

Ketosis is a metabolic disorder of dairy cattle characterized by relatively high concentrations of the ketone bodies (acetoacetate, Beta hydroxy butyric acid and acetone) and a low to normal concentration of glucose in the blood (Brockman, 1979). Ketosis generally occurs 21 to 40 days after parturition and can occur both sub clinically and clinically. Clinically manifested ketosis is characterized by hypophagia, decreased milk productions, loss of body condition score, lethargy, hyperexcitability, hypoglycemia, hypoinsulenemia, hyperketonemia, hyperlipidemia and depleted hepatic glycogen (Drackley, et al., 2001; Bobe et al., 2004). Ketosis in dairy cattle is defined as the increase in concentrations of ketones such as BHBA, acetoacetate (AcAc), and acetone (Duffield et al., 1997). This increase in ketone concentrations in the serum of fresh cows has a negative impact on the health of the cow and is associated with a loss in milk production (Duffield et al., 1997). Fresh cows have a high risk for ketosis within the first 30 days in milk due to low dry matter intake and the rapid mobilization of fat after parturition. In addition, ketosis in fresh cows is most commonly associated with a negative energy balance postpartum (Oetzel, 2004).

Epidemiology:

Occurrence: Ketosis is a disease of high producing dairy cattle and is prevalent in most countries where intensive farming is practiced. Cross bred cattle are more susceptible to the disease.

Incidence :

Incidence of ketosis is high during the first 2 months of postpartum. In India, incidence of ketosis as recorded by various researchers varies from 4.22 to 50% and highest incidence is seen during first 30 days of calving. Emery *et al.* (1964) reported that about 50% of the cows in some high-producing herds had at least subclinical ketosis and that 20 to 30% of the subclinical cases developed into clinical ketosis. Death from lactation ketosis is not common.

Age : Cows of any age may be affected but mature cattle in their third to fourth lactation are highly susceptible as they attain maximum milk yield during this period.

Season : Higher risk is generally observed in cattle during the winter (Duffield, 2000). Probably during this period, animal is in need of extra energy to combat the stress of cold.

Economic significance : Clinical and subclinical ketosis is a major cause of loss to the dairy farmer (Duffield, 2000). Both clinical and sub clinical ketosis are accompanied by decreased milk yield, lower milk protein and milk lactose. There is increased risk of delayed estrous and lowered first conception rates with increased inter calving intervals.

Subclinical ketosis

Subclinical ketosis is defined as an excess of circulating ketone bodies without clinical signs of ketosis (Andersson, 1988). But potential of milk production is reduced to 1 to 9% (Dohoo





and Martin 1984). Subclinical ketosis also diminishes the fertility of the animal. The circulating ketone body most commonly used to diagnose subclinical ketosis is blood betahydroxy butyric acid (BHBA). Most new cases of subclinical ketosis occur within the first 2 to 3 weeks after calving in herds that manage cows in groups and feed a total mixed ration.

Aetio-Pathogenesis:

Specific biochemical and physiological causes of ketosis have not been proven. Baired et al., (1974) postulated that there is no single cause but an inadequate nutrient supply, especially of energy, is a major factor. Theories of ketosis development relate to glucose deficiency as a central theme; or glucose deficiency may be the primary theory; and various sub theories may deal with possible causes of glucose deficiency. This theory is logical because 60 to 85% of the available glucose is used in the mammary gland for milk synthesis (Bickerstaffe et al., 1974). As calving approaches, concentration of progesterone in blood decreases and that of estrogen remains high or actually increases (Grummer, 1995). High circulating estrogen is believed to be one major factor that contributes to decreased dry matter intake around calving (Grummer, 1993). During the last weeks of pregnancy, nutrient demands by the fetal calf and placenta are at their greatest (Bell, 1995), yet dry matter intake may be decreased by 10 to 30%, compared with intake during the early dry period. As lactation starts, glucose is essential for the formation of lactose (milk sugar) and milk fat. The requirement for glucose is at such high level that the blood becomes low in glucose (hypoglycaemia). Cows (and other ruminants) cannot be fed glucose in their diet; it has to be made in the rumen from suitable carbohydrates in the diet. If the amount of suitable carbohydrate in the diet is not enough to meet the glucose needs of the cow in full milk, the liver starts to manufacture glucose from other basic compounds in the body usually fat reserves. Amino acids from the diet or from breakdown of skeletal muscle as well as glycerol from mobilized body fat contribute to

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which promotes mobilization of long chain fatty acids from adipose tissue (body fat). Fatty acids released from adipose tissue circulate as nonesterified fatty acids (NEFA), which are a major source of energy to the cow during this period. The concentration of NEFA in blood reflects the degree of adipose tissue mobilization (Pullen et al., 1989); therefore, the greater the extent of negative energy balance, the more NEFA is released from body fat and the higher the concentration of NEFA in blood. The liver of cows takes up NEFA from the blood that flows through it. As the concentration of NEFA in blood increases around calving or in early lactation, more NEFA are taken up by the liver (Emerv, et al., 1992). Unfortunately, if the release of NEFA from body fat overwhelms the capacity of the liver to use the fatty acids as fuel, they are instead converted to ketone bodies such as acetone, aceto-acetic acid, and β hydroxybutyrate (BHBA). Ruminants have an inherently low capacity for synthesis and secretion of very-low density lipoproteins to export triglyceride from the liver (Kleppe et al., 1988; Pullen et al., 1989) yet the rate of production of triglycerides in the liver is increased at the time of calving (Grum et al., 1996). Cows fed typical diets during the dry period and transition period have an increased concentration of triglyceride in the liver 1 day after calving (Grum et al, 1996). If NEFA uptake by the liver becomes excessive, fatty liver may develop. Negative energy balance and carbohydrate insufficiency in the liver after calving leads to increased production of ketone bodies, which can result in ketosis. As the degree of fatty infiltration increases, normal functions of the liver are adversely affected. In particular, fat infiltration impairs the ability of the liver to detoxify ammonia to urea (Strang et al., 1998). Blood ammonia concentrations are positively correlated with the degree of fat accumulation in the liver of cows shortly after

glucose synthesis. The total intake of energy by

cows after calving usually is less than energy

requirements, even in healthy cows (Bell, 1995).

Negative energy balance results in a high ratio of

growth hormone to insulin in blood of cows,



calving (Zhu et al., 2000). In severe fatty liver, normal functions of the liver are severely depressed, which results in the condition of fatty liver syndrome or clinical fatty liver (Morrow, 1976). Feed intake and carbohydrate status of the cow are important in determining the extent of body fat mobilization, fatty liver, and ketone body production in the liver. The sudden start of milk synthesis in the udder results in a tremendous demand for calcium. As a result, blood calcium concentrations can drop precipitously at calving, leading to milk fever. Smaller decreases in blood calcium, called subclinical hypocalcemia, are believed to be contributing factors in disorders, such as displaced abomasum and ketosis, by decreasing smooth muscle function, which is critical for normal function of the digestive tract (Goff and Horst, 1997).

Types of bovine ketosis:

As per Chitkick (1969) ketosis has been classified as follows:

- 1. Bad season or starvation acetonemia
- 2. Good season or well-fed acetonaemia
- 3. Primary ketosis
- 4. Secondary ketosis
- 5. Clinical ketosis: It is divided into four types
 - a. Alimentary or digestive form of ketosis
 - b. Spontaneous or feeding ketosis
 - c. Nervous form of ketosis
 - d. Milk fever type of ketosis

Clinical finding:

Two major forms of bovine ketosis are described – wasting and nervous. The wasting form is more common of the two and manifests with a gradual but moderate decrease in appetite and milk yield over 2-4 days. The composition of visible signs shown by ketotic cows has been lucidly described by Fox (1971) and Schultz (1968; 1971). The cow first shows dullness. depression, a staring expression, loss of appetite, pick at her feed, and leave some grain. She may progress from leaving most of the grain to the stage of eating only small amounts of hay and preferring to eat bedding. The further the ketosis develops, the greater is the development of perverted appetite. Body weight is lost rapidly, usually a greater rate than one would expect from the decrease in appetite. Farmers usually describe affected cows has having a "woody "appearance due to the apparent wasting and loss of cutaneous elasticity due to presumably to disappearance of subcutaneous fat. The cow is moderately depressed and the hangdog appearance and disinclination to move. A characteristics odor of ketones is detectable on the breath and often in the milk (Radostits et al. 2000).

Hyperketonemia or hypoglycemia, or both, may lead to loss of appetite and perhaps also appearance of nervous signs. In general, the signs in clinical ketosis include sudden drop in milk yield (100%) followed by selective feeding (78.94%) wasting (73.68%), depression (63.15%) and smell in breath/milk (47.46%) and these seem to be the principal signs with potential for utility in diagnosis of bovine ketosis under field conditions, where laboratory facilities are limited or not available at all (Sharma et al., 2009). The nervous signs usually occur in short episode which last for 1 or 2 hours and may recur at intervals of about 8 to 12 hours. Affected cows may injure themselves during the nervous sign. The nervous signs which occur in some cases of bovine ketosis are thought to cause by the production of isopropyl alcohol, a break down product of acetoaceticacid in the rumen. The characteristic sign of nervous form include:

- 1. Walking in circle.
- 2. Straddling or crossing of the legs.
- 3. Head pushing or leaning in to stanchion.









- 4. Apparent blindness.
- 5. Aimless movements and wandering.
- 6. Vigorous licking of the skin and an inanimate objects.
- 7. Deprived appetite.
- 8. Chewing movements with salivation.

Nervous signs are seen in about 10% cases of primary ketosis.

Diagnosis :

Diagnosis of the ketosis is performed on the following basis-

1. History: History of recent calving and drastic decrease in milk production, change in feeding, generally the suffering animal first refuses to eat grain, then ensilage but may continue to eat hay and extra supply of energy source and minerals.

2. Clinical signs: Decreased milk production, woody appearance, temperature, pulse and respiratory rate are within normal range, a characteristics odor of ketone is detectable on the breath and often in the milk.

3. Laboratory diagnosis: Blood glucose: Blood glucose levels are reduced from the normal (50mg/dl) to 20 - 40 mg/dl. Ketosis secondary to other diseases is usually accompanied by blood glucose levels above 40mg/dl.

Blood ketone: Most commonly, plasma or serum beta-hydroxy butyrate (BHBA) is measured for the analysis of ketonemia. BHBA is the predominant circulating ketone body. This ketone body is more stable in blood than acetone or acetoacetate (Duffield, 2000). Clinical ketosis generally involves much higher levels of BHBA (25 mg/dl or more). Normal cows have plasma BHBA concentrations less than 1000 μ mol/L (10.4 mg/dl). Cows with sub clinical ketosis have concentration greater than 1400 μ mol/L (14.4 mg/dl) and the cow with

clinical ketosis have concentration often in excess of 2500 μ mol/L. Blood BHB concentrations typically increase after feeding (Kronfeld *et al.*, 1968). Consistent sampling at 4 to 5 hours after the start of feeding has been suggested in order to capture peak BHB concentrations (Eicher et al., 1998).

Urine and milk ketones: Rothera test is employed for detection of ketone bodies in the urine. Intensity of purple colour indirectly reflects the extent of ketoneuria. Normally urine contains less than 10 mg/dl ketone bodies but may contain up to 70 mg/dl. In case of primary or secondary ketosis, the value of urinary ketones ranges between 80 to 1300 mg/dl. More recently a milk strip test detecting BHBA in the milk is available and is graded on the concentration of BHBA µmol/L. In different studies, it has reported sensitivity and specificity of 73-96% and 69-96% (Driksen and Breitner, 1993) the normal value of milk ketone is less than 3mg/dL, but in case of ketosis this values reach up to 40 mg/dL.

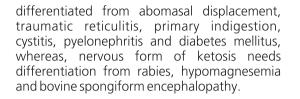
Milk fat to protein ratio: A fat to protein ratio > 1.5 in first day teat milk is indicative of a lack of energy supply in the feed and of risk for ketosis.

Clinical chemistry and hematology: White and differential cell counts are variable and not of diagnostic value for ketosis. There is usually elevation of liver enzymes but liver function tests are within normal range. Liver biopsy is the only accurate method to determine the degree of liver damage.

Necropsy finding : The disease is usually not fatal in cattle but fatty degeneration of liver and secondary changes in the anterior pituitary gland and adrenal cortex may be present.

Differential diagnosis :

Ketosis is differentiated from the following diseases on the basis of history, clinical picture, time of calving and biochemical estimation of hypoglyceamia, ketonemia, and ketoneuria. The wasting form of disease is to be



Treatment:

(a) **Replacement therapy:** The only rational treatment in ketosis is to relieve the need for glucose formations from tissue and allow ketone body utilization to continue normally. The intravenous injections of a 50% (500mL) solution of glucose results in transient hyperglycemia, increased insulin, decreased glucagon and reduced plasma concentration of non esterified fatty acids. A significant proportion of the administered glucose is lost to urinary excretion (Fox, 1971). To overcome the necessity for repeated injections, propylene glycol @ of 225gram twice daily for two days followed by 110gram daily for two days to cattle. 10 to 50% glucose @ 0.5 gram/ Kg body weight intravenous along with short acting insulin @ 0.5 unit Kg body weight injected subcutaneously after 10 to 15minute of initiations of intravenous glucose therapy is also fruitful treatment in case of ketosis (Sharma, et al., 2009).

(b) Hormonal therapy :

Injection with adrenal corticoids is dramatically successful in field cases. Betamethasone and dexamethasone are very much effective and can be given up to 30 mg intramuscularly. @ 10mL (10 mg per ml of Prednisolone Prednisolone) in large animal and 5 mL in small animal are also effective in case of ketosis. Glucocorticoids reduce ketone body formations by utilization of Acetyl CoA and raising blood glucose by making greater availability of glucose precursor in the liver (Chakrabarti, A., 2006). Anabolic steroids like trenboloneacetate @ 60 to 120mg is effective as single injections for the treatment of the ketosis. Insulin facilitates cellular uptakes of glucose, suppresses the fatty acids metabolism and stimulate hepatic gluconeogenesis. The dose of protamine zinc insulin is 200 to 300 IU per animal, administered subcutaneously every 24 to 48 hours as required.

Miscellaneous therapy :

Vit.B12 and Cobalt is recommended to promote propionate production. Nicotinic acid 12 g daily to promote gluconeogenesis, Chloral hydrate at an initial oral dose of 30 gram followed by 7 gram doses twice daily are also recommended.

Prevention and control:

Biochemical monitoring of herds for sub clinical ketosis and adequacy of peri-parturient feeding can be conducted using blood glucose estimations of cows in their second week of lactations. Blood glucose levels of below 35mg/dl suggest subclinical ketosis. Testing for ketones in urine or milk of cows in their first or second week of lactation is recommended for early detection of ketosis and early treatment to prevent milk loss and ketosis associated diseases (Radostits, et al. 2007). Propylene glycol can be used for the prevention of clinical and sub clinical ketosis. Propylene glycol is drenched to the cattle in the early lactation at doses varying from 350-1000ml daily for 10 days after calving (Nielsen and Ingvartsen, 2004). Glycerol can be substituted for propylene glycol at equivalent doses rates. Propionic acid 110gram/ day fed daily for 6 weeks, commencing at calving has given good result in the reducing the incidence of clinical bovine ketosis and improving the production. Monensin can be administered as a slow release capsule to cattle 2 to 4 weeks before calving. The capsule contain 32 gram monensin and releases approximately 335mg monensin for 95 days. It helps reduction in plasma levels of BHBA and reduces prevalence of clinical ketosis (Duffield and Bagg, 2000). Excessive fattening of the animal, abrupt changes in the feeding schedule, feeding large amounts of silage to the animal is to be avoided. Adequate amounts of good quality roughage and recommended amounts of protein,



vitamins, and minerals to the animals need to be provided.

Conclusions :

Ketosis is an important metabolic disorder of high producing dairy cattle that results from inadequate nutrient intake (especially energy) by the dairy cows in early lactations. Adequate and balanced nutrients in high producing dairy cattle to fulfill their metabolic demands is to be provided so that output (especially milk) of the animal does not exceed input (especially nutrients). Clinical and subclinical ketosis is a major cause of economic loss to the dairy farmer. Diagnosis of the ketosis is performed on the basis of history, characteristics clinical findings and laboratory findings such as estimation of blood glucose, estimation of ketone bodies in the serum and detection of ketone bodies in the urine and milk. Management of the dry cow near or at calving time plays an important role in the control of metabolic disorders like ketosis.

References

Andersson, L. (1988). Subclinical ketosis in dairy cows. *Vet. Clin. North Am. Food Anim. Pract.* **4**:233-251.

Baird, G. D. (1982). Primary Ketosis in the High-Producing Dairy Cow: Clinical and Subclinical Disorders, Treatment, Prevention, and Outlook. J. Dairy Sci., 65(1): 1-10. Baird, G.D., Heitzman, R. J., Hibbitt, K.G. and Hunter, G. D. (1974). Bovine ketosis: A review with recommendations for control and treatment. Part I. *Brit. Vet. J.*, 130: 214.

Bell, A.W. (1995). Regulation of organic nutrient metabolism during transition from late pregnancy to early lactation. *J. Anim. Sci.*, **73**: 2804-2819.

Bickerstaffe, R., Annison, E. F. and Linzell, J. L. (1974). The metabolism of glucose acetate, lipids, and amino acids in lactating dairy cows. *J. Agric. Sci.*, Camb., 82: 71.

Bobe, G., Young, J. W. and Beitz, D. C. (2004). Invited Review: pathology, etiology, prevention and treatment of fatty liver in dairy cow. *J. Dairy Sci.* **87**: 3105-3124.

Brockman, R. P. (1979). Roles of insulin and glucagon

in development of ruminant ketosis. *Can Vet J.* **20**(5): 121-126.

Chakrabarti, A., (2006). Metabolic Diseases. IN: Text Book of Clinical Veterinary Medicine. Third Edition, Kalyani Publishers, New Delhi- 110002. Pp.563-616.

Chitkick, A. J. (1969). Univ.Sydney Post.Graduate reference notes in Dis. Of Livestock Hungarford T. G. $8^{th}ed$. McGraw Hill Book Co.

Drackley, J. K., Overton, T. R. and Douglas, G. N. (2001). Adoptationof glucose and long chain fatty acid metabolism in the liver of dairy cow during peripartuient period. J. *Dairy Sci.* **84** (E supp): E-100-E-112.

Driksen, G. and Breitner, W. J. (1993). Vet Med Assoc, 40:779.

Duffield, T. F. (2004). Monitoring strategies for metabolic disease in transition dairy cows. In: Proc 23rd World Buiatrics Cong, Quebec, Quebec, Canada, July 11-16, pp. 34-35.

Dufflied, T. F. (2006). Epidemiology of subclinical production diseases in dairy cows with an emphasis on ketosis. In: Proc. of 12th Intl. Conf. on Production diseases in farm animals by Joshi, N. and Herdt, T.H., 126-135.

Eicher, R., Liesegang, A., Bouchard, E. (1998). Influence of concentrate feeding frequency and intrinsic factors on diurnal variations of blood metabolites in dairy cows. *Proc. Am. Assoc. Bov. Pract.*, **31**: 198-202.

Emery, R. S., Burg, N., Brown, L. D. and Blank, G. N. (1964). Detection, occurrence, and prophylactic treatment of borderline ketosis with propylene glycol feeding. *J. Dairy Sci.*, **47**: 1074.

Emery, R. S., Liesman, J. S. and Herdt, T. H. (1992). Metabolism of long chain fatty acids by ruminant liver. *J. Nutr.*, **122**: 832-837.

Fox, F. H. (1971). Clinical diagnosis and treatment of ketosis. *J. Dairy Sci.*, 54: 974.

Goff, J. P. and Horst, R. L. (1997). Effect of addition of potassium or sodium, but not calcium, to prepartum rations induces milk fever in dairy cows. *J. Dairy Sci.*, 80: 176.

Grum, D. E., Drackley, J. K., Younker, R. S., LaCount, D.W. and Veenhuizen, J. J. (1996). Nutrition during the dry period and hepatic lipid metabolism of periparturient dairy cows. *J. Dairy Sci.*, **79**: 1850-1864.







Grummer, R. R. (1993). Etiology of lipid-related metabolic disorders in periparturient dairy cows. *J. Dairy Sci.*, **76**: 3882-3896.

Grummer, R. R. (1995). Impact of changes in organic nutrient metabolism on feeding the transition dairy cow. *J. Anim. Sci.*, **73**: 2820-2833.

Hibbitt, K.G. (1979). Bovine ketosis and its prevention. *Vet. Rec.*, 105: 13.

Ingvartsen, K. L. (2006). Feeding and management related diseases in transition cow. Physiological adaptation around calving and strategies to reduces the feeding related diseases. *Animal Feed Science technology*. Vol. **128** pp.175-213.

Kleppe, B. B., Aiello, R. J., Grummer, R. R. and Armentano, L. E. (1988). Triglyceride accumulation and very low density lipoprotein secretion by rat and goat hepatocytes in vitro. *J. Dairy Sci.*, **71**: 1813-1822

Kronfeld, D. S., Raggi, F. and Ramberg, C. F. (1968). Mammary blood flow and ketone metabolism in normal, fasted, and ketotic cows. *Am. J. Physiol.*, **215**: 218-227.

Morrow, D. A. (1976). Fat cow syndrome. J. Dairy Sci., 59: 1625.

Oetzel, G. R. (2004). Monitoring and testing dairy herds for metabolic disease. Ontario. *Can. Vet. J.* **38**:713-718.

Pullen, D. L., Palmquist, D. L. and Emery, R. S. (1989). Effect on days of lactation and methionine hydroxy analog on incorporation of plasma fatty acids into plasma triglycerides. *J. Dairy Sci.*, **72**: 49-58. Radostits, O. M., Gay, C. C., Blood, D. C. and Hinchkliff, K. W. (2000). Veterinary Medicine. 9th edn. ELBS & Baillier Tindall. pp.563-618.

Radostits, O. M., Gay, C. C., Hinchcliff, K. W. and Constable, P. D. (2007). Veterinary Medicine. 10th edn. ELBS & Baillier Tindall. pp.1613-1690.

Schultz, L. H. (1968). Ketosis in dairy cattle. *J. Dairy Sci.*, 51: 1133.

Schultz, L.H. (1971). Management and nutritional aspects of ketosis. *J. Dairy Sci.*, 54:962.

Sharma, Neelesh, Upadhyay, S. R. and Srivastava, A. K. (2009). Bovine ketosis-An overview. *Agrovet Buzz*, **1**(6): 43-47.

Shaw, J. C. (1956). Ketosis in dairy cattle- A review. J. Dairy Sci., 39: 402.

Strang, B. D., Bertics, S. J., Grummer, R. R. and Armentano, L. E. (1998). Effect of long-chain fatty acids on triglyceride accumulation, gluconeogenesis, and ureagenesis in bovine hepatocytes. *J. Dairy Sci.*, **81**: 728-739.

Upadhya, S. R., Sharma, Neelesh and Pandey, V. (2007). Nervous ketosis in buffalo-A case report. *Intas Polivet*, 8(**2**): 404-406.

Zhu, L. H., Armentano, L. E., Bremmer, D. R., Grummer, R. R. and Bertics, S. J. (2000). Plasma concentration of urea, ammonia, glutamine around calving, and the relation of hepatic triglyceride, to plasma ammonia removal and blood acid-base balance. *J. Dairy Sci.*, **83**: 734-740











Colic in Horses: A Update

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Introduction

Colic, the most feared disease of horses, is potentially fatal (Traub-Dargatz et al., 2001). However, the majority of cases respond well to the treatment. The term 'colic' refers to abdominal or belly pain, and most of the horses experience an episode at some point of time in their life span. Provided that veterinary advice is sought early and that the appropriate treatments are given promptly, prognosis is generally favourable.

Predisposing factors

• Sex - Though stallions are stated to be at increased risk (Kaneene *et al.*, 1997), other

studies have not corroborated association of gender with colic.

- Age- Some reports indicated that horses aged <10 years are at increased risk (Tinker et al., 1997), but this remained to be substantiated.
- History- Horses with a previous history of colic are stated to be at increased risk (Hillyer *et al.*, 2002b).
- Feeding protocol Change in diet/feed, management:, type or quantum of concentrate in the ration has been associated with increased risk (Blikslager, 2008).





(2) nereas Aetiology

- Grazing Reports suggest that whereas increased access to grazing on pasture reduced the incidence of colic, improper housing/stable management was associated with higher incidence. Presumably, overcrowding with long hours in the stable increased the risk of parasite-induced colic (Hillyer *et al.*, 2002b).
- Exercise- Association between colic and the lack of exercise is well-established (Hillyer *et al.*, 2001).
- Drinking water- The importance of free access to water is corroborated by a study which revealed an increased risk in horses without access to water at will in outdoor enclosures (Reeves *et al.*, 1996).
- Owner care- Horses whose owners provide optimized care are at reduced risk of colic (Hillyer *et al.*, 2001).
- Parasites- GI parasites, especially Strongyle spp. that migrate in the gut blood vessels may induce severe, potentially fatal colic. Excessive worm load may cause thrombosis (clots) with markedly reduced blood supply culminating in infarction and necrobiosis in the affected section of gut obligating surgical intervention. With the current availability of effective worm pastes, the disease is rarely encountered in the regularly de-wormed horses. Tapeworms are also stated to inhibit the gut motility leading to colic. Round worms may completely obstruct the intestines of foals.
- Dental disorders- Presence of dental anomalies is a putative risk factor in certain forms of colic. It is reported that increased frequency of dental examination is associated with decreased incidence of simple colonic obstruction or distension of the colon (Hillyer et al., 2002b).

Migrating Strongyle spp. larvae damage the gut blood vessels, decreasing the blood supply markedly leading to necrosis, impaired intestinal motility and pain. Round worms, in large numbers, may cause impaction or obstruction of the intestines.

• Erratic/injudicious use of anthelminitics.



Colic of foals caused by round worm infestation

- Infectious diseases like rabies, pleurisy, dermatitis, or enteritis.
- Conditions affecting the locomotor system such as laminitis or other forms of lameness.
- Housing in sandy paddocks or overgrazed pastures predispose horses to 'sand colic'.
- Spoiled silage may obstruct the large intestine.
- Accidental ingestion of foreign bodies.
- Sudden change in the type or quantity of feed or moldy feed may cause fermentation and obstruction in the gut.
- Predominant concentrate diet with a gross deficit of long stem dietary rough ages.



- Bad eating habits like bolting and cribbing.
- Inadequate intake of wholesome water may cause impaction with resultant colic over a period of time.

Types of colic

- Pelvic flexure impaction: Following impaction of the food materials: grass, hay or gain, the left colon takes 180 degree turn to narrow down appreciably.
- Spasmodic colic: Evidenced by abnormally increased peristaltic contractions in the GIT, spasmodic colic may result from gas buildup, and the condition responds well to spasmolytic-cum-analgesic medication.
- Ileal impaction A condition caused by obstruction of the ileum with ingesta/ ascarids (*Parascaris equorum*) or tapeworms (*Anoplocephala perfoliata*).
- Sand colic End result of uneventful ingestion of sand or loamy soil over a prolonged period of time.
- Enterolithiasis Round balls of mineral deposits, entero-liths, formed around pieces of ingested foreign bodies such as gravel obstructing the intestine often mandate surgical remedy.



Massive enteroliths

- GI parasites- Mechanical obstruction may be produced by numerous round worms (*Parascaris equorum*), especially in the young horses. Sudden de-worming of the heavily infected horses may elicit a severe immune response to the dead worms, which damages the intestinal wall culminating in fatal peritonitis.
- Pathomorphological anamolies Left dorsal displacement of the colon occurs when entrapped above the spleen against the nephrosplenic ligament. Diagnosis is based on rectal examination/ultrasonography.
 Some times, right dorsal displacement of the bowel may be observed. Usually, surgery is the only available option.
- Torsion- Different portions of the horse's GI tract may intervine, most commonly involving the small intestine or part of the colon, often necessitating emergency surgery.
- Intussusception A pathoclinical condition wherein a portion of the intestine telescopes within itself, most commonly involving the small intestine of young horses, requiring urgent surgical intervention.
- Mesenteric entrapment Occasionally, a small hole may develop in the mesentery through which a segment of the bowel protrudes. In epiploic foramen entrapment, the bowel enlarges with associated marked oedema which precludes retraction from the site. This condition also calls for urgent surgical attention.
- Gastric ulceration In this fairly common pathoclinical state, the risk factors include confinement, infrequent feedings, high proportion of concentrate/ grain feeds, excessive NSAID use, and the stress associated with prolonged transportation and frequent equestrian events. Most cases







are amenable to medications that inhibit acidogenic cells in the gastric lining epithelium. Bleeding ulcers leading to stomach rupture are rare.

Clinical signs

- Pawing and/or scraping
- Stretching
- Frequent attempts to urinate
- Flank retraction: turning the head sideways towards the stomach/hind quarters
- Pacing
- Repeated prostration and rising up
- Rolling and groaning

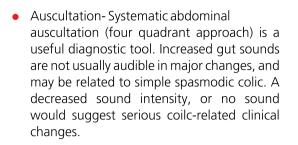
Diagnosis

- Anamnesis- The case history is carefully perused and recorded on presentation. This includes the patient's age, sex, recent activity profile, diet, any dietary changes, and the anthelmintic schedule.
- Cardiovascular parameters- The heart rate is known to accelerate with progress of colic. mainly because of decreased circulatory blood volume and endotoxaemia. The pulse rate that continues to rise despite adequate analgesic medication is an important surgical indication. Colour of the visible mucous membranes faithfully reflects the severity of impaired haemodynamics. Thus, perceptibly increased rubor (reddening) reflects the grossly deranged clinical state, and cyanotic membranes indicate poor prognosis. Laboratory tests may be employed to monitor the patient's cardiovascular status. PCV% is a dependable index of the *in vivo* cell hydration status; increasing values

>45% during repeated examination are considered clinically significant. The total protein (TP) concentration in circulating blood may aid in estimating the magnitude of protein loss through extrusion of blood into the intestinal lumen.

- Rectal examination- Repeated rectal examination is the cornerstone of colic diagnosis since several degenerative states can be definitively diagnosed. Demonstration of dilated small intestinal loops may help to decide on surgical remedy.
- Naso-gastric intubation Fluid is carefully refluxed from the stomach contents with a nasogastric tube, and > 2 litres fluid is considered significant. Increased fluid volume is generally the end result of back flow in the intestinal tract because of obstruction downstream. Representing relatively advanced stage of colic, it is often a surgical indication. Therapeutically, gastric decompression is important. If excessive fluid build-up occurs, fatal gastric rupture may follow.
- Abdominocentesis- Sampling of fluid from the peritoneum may be useful in assessing the pathoclinical state of the intestines. Sanguinous fluid represents infarction, and usually indicates surgery. Cloudy fluid is suggestive of increased number of white blood cells, highly suggestive of relatively advanced disease condition. The protein concentration in abdominal fluid is an indicator of patency of the intestinal blood vessels.
- Abdominal distension Abdominal distension is often indicative of abnormal condition affecting specifically the large intestines.





• Fecal examination-The amount of feces excreted and its nature may be helpful. In geographic areas where sand colic is common, faeces may be examined for the demonstration of sand by suspension in water, or simply by its texture.

Remedial therapy

I. Analgesic and spasmolytic regimens

Drug Classification	Name of the drug	Dose level			
NSAID	Flunixinmeglumine Ketoprofen	0.25-1mg kg-1iv or im, every 8-24 hr 2.2mg kg-1 iv every 12 hr			
Opiates Butorphanol Morphine sulphate		0.025-1mg kg-1iv or im 0.0501mg kg-1, slow iv			
α-2 Agonist	Xylazine	0.01-1 mg kg-1iv or im			
Spasmolytic	atropine	0.01-04mg kg-1iv or im			

II. Fluid and electrolytes/ Bowel lubricants

Horses with evidence of generalized tissue dehydration, compromised cardiovascular function or electrolyte imbalance should be administered (i) Ringer's lactate solution and/or (ii) Dextrose normal saline solution on case-tocase basis.

Intestinal lubricant and faecal softners: An effective intestinal lubricant, mineral oil should be given @ 10-15 ml kg⁻¹ only through a nasogastric tube (as accidental aspiration is associated with severe and usually fatal pneumonia) every 12-24 hr. Magnesium sulfate, an effective faecal softner is useful in the treatment of impaction colic @ 0.5-1.0 g kg⁻¹, suitably diluted with water using a nasogastric

tube. Sodium sulfate is a safe and effective faecal softner @ 1.0 g kg^{-1} (diluted in water) every 12 hr. with the nasogastric tube,

Control measures

- Avoid overgrazed pastures
- Ensure free access to adequate quantity of fresh water daily
- Follow proper daily feeding schedule
- Discard moldy or spoiled grain or hay
- Adequate quantity of long stem roughage should be provided in the daily ration.
- Stalls and paddock areas should be free from all foreign objects.
- Proper deworming programme rigidly followed.





References

Blikslager, A. (2008). Avoiding colic through management. The Horse, July, pp. 47-54.

Hillyer, M.H., Taylor, F.G.R. and French, N.P. (2001). A cross-sectional study of colic in horses on thoroughbred training premises in the British isles in 1997. Equine Vet. J. Ltd. Newmarket, **33**: 380-385.

Hillyer, L.L., Finn, N., le Pla, J., Lynch, A., Hillyer, M.H. and Coles, G.C. (2002a). Assessment of intestinal parasite control strategies on Thoroughbred studs in the UK. In: Proceedings of the 7th Equine Colic Research Symposium, Equine Veterinary Journal Ltd., Newmarket, **34**, p 73.

Hillyer, M.H., Taylor, F.G.R., Proudman, C.J., Edwards, G.B., Smith, J.E. and French, N.P. (2002b). Risk factors for simple colonic obstruction and distension colic in horses. In: Proceedings of the 7th Equine Colic Research Symposium, Equine Veterinary Journal Ltd., Newmarket, **34**, pp 99-100.

Kaneene, J.B., Miller, R., Ross, W.A., Gallagher, K., Marteniuk, J. and Rook, J. (1997). Risk factors for colic in the Michigan (USA) equine population. Prevent. Vet. Med., **30**, 23-36. Reeves, M.J., Salman, M.D. and Smith, G. (1996) Risk factors for equine acute abdominal disease (colic): results

from a multi-center case-control study. Prevent. Vet. Med., **26**: 285-301.

Schmid, A., Freeman, D.E. and Schaeffer, D. (2002). Risk by age, breed and gender for common forms of small intestinal strangulation obstruction in horses. In: Proceedings of the 7th Equine Colic Research Symposium, Equine Veterinary Journal., Newmarket. p 98.

Tinker, M.K., White, N.A., Lessard, P., Thatcher, C.D., Pelzer, K.D., Davis, B. and Carmel, D.K. (1997 b) Prospective study of equine colic risk factors. Equine Vet.J. Ltd. Newmarket, **29**: 454-458.

Traub-Dargatz, J.L., Kopral, C.A., Seitzinger, A.H., Garber, L.P., Forde, K. and White, N.A. (2001) Estimate of the national incidence of and operation-level risk factors for colic among horses in the United States, spring 1998 to spring 1999. J. Am. Vet. Med. Ass. **219**: 67-71.











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Role of Nutrients in Animal Reproduction

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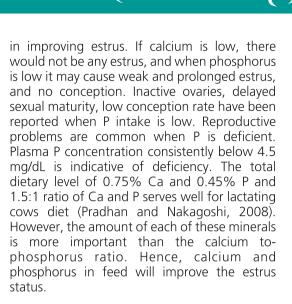
Introduction

With the introduction of Artificial Insemination (AI) during 1950s, the interest in bovine infertility increased and now the factors involved in infertility have become known to farmers and scientists (Roberts, 1956). The causes of infertility are many and can be complex (Arthur, 1982). The infertility or reduced fertility may be due to many reasons such as diseases, poor nutrition, inadequate herd management and hereditary. Physiological and reproductive disorders related to Graafian follicle development and maturation, oestrus onset, successful insemination, ovulation, fertilization, implantation, and the development and delivery of the foetus are also cause of infertility. The infertility and reduced fertility causes reduction in number of lactations that may account for 10 to 30% (Erb and Martin, 1980) loss in total production. In India, 40% cattle and buffaloes are infertile due to poor nutrition. The market is flooded with various feeds and feed supplements and most of them claim improvement of fertility in bovines. As per the provisional figures of 2007 livestock census, India has 199.1 million cattle which are 15 per cent of the world cattle population. Total buffalo population is 105.3 million that is about 56% of the world buffalo population (Annual report of GOI-2011-12). We have more than 25 indigenous cattle breeds and 10 buffalo breeds, which are well characterized. Recent work in dairy animals suggests the borderline nutrient deficiencies may be manifested by impaired fertility (Hurley and Doane, 1989). The article describes the role of major and minor nutrients in improving reproduction efficiency.

Calcium and Phosphorus

Calcium (Ca) and phosphorus (P) are two of the most abundant minerals in the body which are vital in ration balancing for cattle. The main function of both calcium and phosphorus is in formation of skeleton. Nearly 99% of the calcium is found in the skeleton, while 80% of the phosphorus is in bones and teeth. The remaining Ca is extracellular and plays a role in nerve conduction, muscle contraction, blood clotting and immune system activation. The remaining P is involved in energy utilization, and for cattle is required by ruminal microbes for growth and cellular metabolism.

The general symptoms of calcium deficiency include stunted growth, delayed maturity, reduced fertility, lowered milk yield, fragile bones and paralytic condition. Vitamin D is best known for its role in maintaining Ca homeostasis by increasing intestinal Ca absorption and by regulating Ca metabolism in bone. In addition to the role of Ca in skeletal growth and lactation, Ca is involved in control of many intracellular processes mediated through Ca-binding proteins. Reduced blood Ca may delay uterine involution and increase incidence of dystocia, retained placenta, and prolapsed uterus (Morrow, 1980; Risco et al, 1984). Excess Ca may impair the function of reproduction by causing a secondary deficiency of Phosphorus (P), Magnesium (Mg), Zinc (Zn) and Copper (Cu) and other microelements by inhibiting their absorption in the intestine (King, 1971). Calcium-dependent mechanisms are involved in steroid biosynthesis in the testes, adrenal glands, and ovaries (Veldhuis and Klase, 1982). Calcium (Ca) and Phosphate (P) play major role



Zinc

Zinc sulfate is used to supply zinc in animal feeds, fertilizers, and agricultural sprays and it is the inorganic compound. It is a colorless solid that is a common source of soluble zinc ions. Reproduction failure due to Zn deficiency has been documented extensively (Apagar, 1985; Chesters, 1979; Hidiroglou, 1979). Zinc is an essential component of a wide variety of enzymes and proteins that support metabolism, growth, production, reproduction, protein synthesis, nucleic acid metabolism, epithelial tissue integrity, cell repair and division, and transport and utilization of vitamin A and E (Cousins and Hempe, 1990). Zinc is essential for sexual maturity (puberty), reproductive efficiency, onset of estrus, repair and maintenance of uterine linings following parturition, return to normal reproductive cycle and estrus (Green et al., 1998). Precisely, all phases of reproductive process in females from estrous to pregnancy and lactation, may be affected by Zinc deficiency (Underwood 1981). Zinc helps in improving semen guality and libido in males. A recent study (Bindari et al., 2013) investigating level and source of zinc on a limited number of crossbred bulls (n=16) demonstrated that zinc supplementation increased mean ejaculate volume, sperm concentration, percent live sperms and percent

motility. Studying fertile and infertile males, it was observed that seminal zinc levels were lower in infertile male than fertile male and researchers suggested that poor zinc nutrition may be a risk factor for infertility in male. Zinc supplementation was shown to reduce asthenozoospermia in male by reducing oxidative stress, DNA fragmentation and apoptosis.

Copper

Reproductive problems that relate to copper deficiency manifest themselves in inhibited conception rate even though estrus may be normal. Symptoms of a copper deficiency include early embryonic death, resorption of embryo, increased retained placenta and necrosis of the placenta. Weak and silent heats have been reported due to deficiency of copper. Dairy cows with higher serum copper levels had significantly less days to first service, fewer services per conception and fewer days to open (Jousan et al., 2002). Copper deficiency is associated with early embryonic death, reduced ovarian activity, delayed or reduced estrus, reduced estrus activity, decreased conception rate, increased incidence of retained placenta and increased difficulty in calving. The availability of copper is reduced by excesses of calcium, sulfur, iron, zinc, and molybdenum in the diet or water. Copper deficiency is mainly manifested in reproductive disorders in the female. Early embryonic death is particularly common (Hidiroglou, 1997), and Cu requirements at specific periods of fetal and neonatal development have been demonstrated in mouse strains (Hurley et al., 1989). Administration of Cu sulfate has induced normal breeding patterns in heifers with low fertility associated with Cu deficiency, and supplemental Cu has increased conception rate in cows with marginally low blood Cu (Hunter, 1977).

Ferrous (iron)

This is usually supplemented in the form of ferrous sulfate. It is required for the synthesis of



haemoglobin, myoglobin, enzymes and cytochrome enzymes of electron transport chain. Iron functions in transport of oxygen to tissues, maintenance of oxidative enzyme system and is concerned with ferretin formation (Khillare et al, 2007). The reproductive performance of Iron deficient animals may be badly affected due to anaemia, reduced appetite and lower body condition. A deficient animal becomes repeat breeder and requires increased number of inseminations per conception and occasionally may abort.

Ascorbic Acid (Vit C)

The ovaries, and other endocrine tissues, accumulate large amounts of ascorbic acid. Within the ovaries, ascorbic acid accumulates in the granulosa, thecal and luteal cells and it has long been associated with fertility (Luck et al., 1995). Studies on luteinizing granulosa cells have shown that ascorbic acid stimulates production of progesterone (Byrd et al., 1993) and that increasing progesterone concentrations block the uptake of ascorbic acid (Stansfield and Flint, 1967). Therefore, the action of LH may indirectly control the fluctuations in ascorbic acid concentration observed throughout the ovarian cycle. In addition, ascorbic acid acts as a co-factor in the amidation of some proteins and has been implicated in the regulation of oxytocin secretion by ovaries (Luck and Junglas, 1987). The role of ascorbic acid in promoting collagen biosynthesis has been studied extensively (Pinnell, 1985). During follicular growth, ovulation and formation of corpora lutea, basement membranes and the extracellular matrix are undergoing constant remodeling and, therefore, have high requirements for collagen. Earlier studies implicated ascorbic acid in the regulation of the Graafian follicular basement membrane and showed that lack of ascorbic acid causes degeneration of follicle membranes and high doses inhibit collagenolytic activity in mature follicles (Espey and Coons, 1976). Vitamin C deficiency has been associated with premature rupture of placental membranes.

Vitamin E

Vitamin F functions as an intra-cellular antioxidant scavenging for free reactive oxygen and lipid hydroperoxidases, and converting them to non-reactive forms, thus maintaining the integrity of membrane phospholipids against oxidative damage and peroxidation (Sinclair et al. 2000: Wichtell et al. 1996). The process whereby free radicals "steal" electrons from the lipids in cell membranes, resulting in cell damage and increased production of free radicals is called peroxidation. In deficiency of vitamin E with selenium, these free radicals accumulate and not only damage cell membranes, but also disrupt several processes linked to the synthesis of steroids (Seagerson and Libby, 1982), prostaglandins (Harrison and Conrad, 1984), sperm motility and the development of the embryo (Goff, 1999). Negative impacts of vitamin E and selenium deficiencies have been observed on various components of the reproductive events, including ovulation rate (Goto et al., 1992), uterine motility, sperm motility and transport (Robinson, 1996), conception rate and postpartum activities (Jie et al., 2004), fetal membrane expulsion (Talavera et al., 1985), embryo survival, milk production and post natal growth (Garcia et al., 2001). Vitamin E is important for immune function and works closely with selenium. Vitamin E reduces the incidence of retained placenta in fresh cows.

lodine (I)

lodine is required for synthesis of thyroid hormone, thyroxin, which regulates the rate of metabolism (NRC, 2001). lodine has an indirect effect on reproduction through its action on the thyroid gland. Inadequate thyroid function reduces conception rate and ovarian activity. Thus, iodine deficiency impairs reproduction. Recently, the effects of excessive iodine intake have been recognized. Excessive iodine intake has been associated with various health problems including abortion and decreased resistance to infection and disease. Signs of subclinical iodine deficiency in breeding females





include suppressed estrus, abortions, still births, increased frequency of retained placentas and extended gestation periods (Hess et al., 2008). Calves born to cows that are marginally deficient in iodine are weak and may be hairless (Patterson, 2003). Furthermore, animals that have a subclinical iodine deficiency will also have increased incidence of foot rot and respiratory disease due to suppressed immune responses. One notable characteristic of a clinical iodine deficiency is an enlargement of the thyroid gland, often termed as goiter.

Potassium (K)

Potassium is a macromineral required for reproductive efficiency. The limited research which has been done suggests that feeding required levels of potassium help in onset of puberty, ovulation, corpus luteum development and regulates estrous in heifers. However, Smith and Chase (2010) reports lower fertility in cows fed with high levels of potassium or diets in which the potassium-sodium ratio was too wide.

Conclusion

Nutrition is directly related to reproduction in the dairy animals. Nutrient deficiency has been shown to be capable of altering reproduction. The best recommendation at present is to provide a feeding program for dairy animals which is balanced for all nutrients and meets all known nutrient requirements.

References

Apagar J. (1985). Zinc and reproduction. Ann. Rev. Nutr. **5**:43.

Arthur GH. (1982). Veterinary reproduction and obstetrics. 5th edition. Bailleire Tindall, London, UK.

Bindari Y R , Shrestha S, Shrestha N and Gaire TN. (2013). Effects of nutrition on reproduction- A review. Advances in Applied Science Research. **4**(1):421-429.

Byrd JA, Pardue SL and Hargis BM. (1993). Effect of ascorbate on luteinizing hormone stimulated

progesterone biosynthesis in chicken granulose cells in vitro. Comparative Biochemistry and Physiology C. **104**:279281.

Chesters JK. (1978). Biochemical functions of zinc in animals. World Rev. Nutr. Diet. **32**:135.

Cousins RJ and Hempe JM. (1990). Zinc, Present knowledge in nutrition. M.L Brown ed. International Life Sciences Institute Nutrition Foundation, Washington DC: 251-260.

Erb HN and Martin SW. (1980). Interrelationships between production and reproduction diseases in Holstein cows. Journal of Dairy Science. **63**: 1911-1917.

Espey LL and Coons PJ. (1976) Factors which influence ovulatory degradation of rabbit ovarian follicles. Biology of Reproduction. **14**: 233-245.

Garcia G, Cavellaro L, Broussalis A, Ferraro G and Martino V. (2001). J Pharmacol Exp Therap. **297**: 1.

Goff JP. (1999). Dry cow nutrition and metabolic disease in parturient cows. Proceeding Western Canadian Dairy Seminar Red Deer.

Goto Y, Noda Y, Narimoto K, Umaoka Y and Mori T. (1992). Oxidative stress on mouse embryonic development in vitro, Free Radical Biology Research. **13**: 47-53.

Green LW, Johnson AB, Paterson J. and Ansotegui R. (1988). Feedstuff. **70**(34).

Harrison JH and Conrad HR. (1984). Effect of selenium intake on selenium utilization by the non-lactating dairy cow. Journal of dairy science. **67**: 219-223.

Hess BW, Moss GE and Rule DC. (2008). A decade of developments in the area of fat supplementation research with beef cattle and sheep. Journal of Animal Science. **86**: 188-204.

Hidiroglou M. (1997). Trace element deficiencies and fertility in ruminants: a review. J. Dairy Sci. **62**:1195.

Hunter AP. (1977). Some nutritional factors affecting the fertility of dairy cattle. N. Z. Vet. J. **25**:305.

Hurley WL and Doane RM. (1989). Recent Developments in the Roles of Vitamins and Minerals in Reproduction. J Dairy Sci. **72**:784-804.



Jie JL, Douglas SJ, Drago R and Bruce DR. (2004). Contemporary Drug Synthesis, John Wile & Sons Inc Hoboken, New Jersey, pp 397.

Jousan FD, Utt MD and Beal WE. (2002). Effects of differences in dietary protein on the production and quality of bovine embryos collected from superovulated donors. J Animal Science. **8**: 1.

Khillare KP, Sahatpure SK, Vanlalpeka K, Bombatkar RS and Tijare G S. (2007) Trace Minerals and Reproduction in Animals. Intas Polivet., **8**(2):308-314.

King JOL. (1971). Nutrition and fertility in dairy cows. Vet. Rec. **89**:320.

Luck MR and Junglas B. (1987). Catecholamines and ascorbic acid as stimulators of bovine ovarian oxytocin secretion. Journal of Endocrinology. **114**: 423-430.

Luck MR, Jeyaseelan I and Scholes RA. (1995). Ascorbic acid and fertility. Biology of Reproduction. **52**: 262-266.

Morrow DA. (1980). The role of nutrition in dairy cattle reproduction. Page 449 in Current therapy in Theriogenology. D. A. Morrow ed WB Saunders Co., Philadelphia, PA.

National Research Council (2001). Nutrients requirement of dairy cattle, 7th rev. ed. Natl. Acad. Sci, Washington D. C.

Patterson HH, Adams DC, Klopfenstein TJ, Clark RT and Teichert B. (2003). Supplementation to meet metabolizable protein requirements of primiparous beef heifers: II. Pregnancy and Economics. J. Anim. Sci. **81**: 503-570.

Pinnell SR. (1985) Regulation of collagen synthesis by ascorbic acid: a review. Yale Journal of Biology and Medicine. **58**: 553-55

Pradhan R and Nagakoshi N. (2008). Reproductive disorders in cattle due to Nutritional status. J Inter. Develop. Cooperation. **14**(1):45-66.

Pradhan, R. (2008). Reproductive disorders in cattle due to Nutritional Status. J Intern. Develop. and Cooperation. **14**:45-66.

Risco CA, Reynolds JP and Hird D. (1984). Uterine prolapse and hypocalcemia in dairy cows. J. Am. Vet. Med. Assoc. **185**:1517.

Roberts SJ. (1956). Veterinary obstetrics and genital diseases. 1st edition. Edwards Brothers, Ann Arbour, Michigan, USA.

Robinson JJ. (1996). Animal Reproductive Science. **42**: 25-34.

Seagerson EC and Libby DW. (1982). Ova fertilization and sperm number per fertilized ovum for selenium and vitamin E treated Charolais cattle. Theriogenology. 17: 333-341.

Sinclair KD, Kuran M, Gebbie FE, Webb R and McEvoy TG. (2000). Nitrogen metabolism and fertility in cattle: Development of oocytes recovered from heifers offered diets differing in their rate of nitrogen release in the rumen. J. Anim.Sci, **78**: 2670-2680.

Smith RD and Chase LE. (2010). Nutrition and Reproduction, Dairy Integrated Reproductive Management.

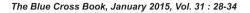
Stansfield DA and Flint AP. (1967). The entry of ascorbic acid into the corpus luteum in-vivo and in-vitro and the effect of luteinizing hormone. Journal of Endocrinology. **39**: 27-35.

Talavera E, Park CS and Williams GL. (1985). Relationships among dietary lipid intake, serum cholesterol and ovarian' function in holstein heifers. Journal of Animal Science. 60: 10-45.

Underwood EJ. (1981). The mineral nutrition of livestock. Common Wealth Agricultural Bureaux, Slough (Englang) **189**.

Veldhuis JD and Klase PA. (1982).Mechanism by which calcium ions regulate the steroidogenic actions of luteinizing hormone in isolated ovarian ceils in vitro. Endocrinology **111** : 1.

Wichtell JJ, Craigie AL, Thompson KG and William NB. (1996). Effect of selenium and A-tocopherol supplementation on postpartum reproductive function of dairy heifers at pasture. Theriogenology. **46**: 491-502.



Use of MID-cycle $PGF_2\alpha$ and GnRH at breeding to improve conception rate in repeat breeding Cows and Buffaloes under field conditions

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Introduction

Dairy farming in India is one of the most important agricultural activities, but the repeat breeding syndrome in animals leads to loss of economy of dairy farms (Modi et al., 2011). High reproductive performance is an essential requirement to ensure maximum livestock production and satisfactory economic return (Baruselli et al., 2012). There are apparently several reasons for the repeat breeder syndrome and no single treatment is likely to alleviate the condition in every herd or animal. Several innovative ideas and management technologies have come up recently to improve reproductive efficiency of dairy animals. Many field veterinarians are now adopting the same in routine farm practices to optimize and maintain a high degree of breeding efficiency in dairy herds.

PGF₂ α and its analogues have a luteolytic effect between day 5 and 17 (luteal phase) of bovine estrous cycle (Rowson *et al.*, 1972), and by employing these agents subsequent endocrine events closely resemble those of the normal cycle. Prostaglandins were reported to increase the conception rate in repeat breeder bovines (Goley and Kadu, 1995; Kharche and Srivastava, 2001; Savalia *et al.*, 2013; Patel *et al.*, 2014). Similarly GnRH, hCG and/or progesterone analogues have been used successfully to sustain early pregnancy and improve conception rate in repeat breeding bovines (Sreenan and Diskin, 1983; Kim *et al.*, 2007; Patel *et al.*, 2014). The present study was, therefore, planned to evaluate whether midcycle PGF₂á and AI + GnRH treatments influence the conception rate in repeat breeding cows and buffaloes under field conditions.

Material and Methods

The animals owned by farmers were selected through organizing sexual health control camps in villages of milk shed areas of AMUL (Anand) and Panchamrut Dairy (Dahod) of Gujarat from September 2012 to February 2013. The animals bred 3 or more time previously at regular intervals with good quality frozen-thawed semen and yet failed to conceive were initially screened gynaeco-clinically for their reproductive status and genital health. In all 56 and 52 pluriparous postpartum cows and buffaloes, respectively, of average body condition score were included in this study. These included 40 and 35 repeat breeders and 18 and 17 normal cyclic (control) cows and buffaloes, respectively. These were regularly followed for a period of at least 3 months posttreatment. The animals in spontaneous or induced estrus were inseminated using good quality frozen-thawed semen by the trained inseminators of the concerned Milk Unions.



All the selected animals were initially dewormed by providing 1 kg medicated concentrate mixture of Amul or 3 g fenbebdazole bolus (Fendikind, Mankind) and administering ivermectin 70 mg s/c (Inj. Ivectin 7 ml, Indian Immunologicals Ltd). They were also treated once with i/m injection of 3.0 g enrofloxacin (Inj. Conflox 15 ml, Concept Pharma) to check invisible genital infection, if any. Owners of the ear-marked animals were supplied with mineral mixtures (Amul brand) or multi-minerals bolus (Minotas, Intas Pharma) for supplementing to their animals for 7 to 10 days. Problem breeders were confirmed by rectal palpation twice 10 days apart, and were subjected to the following therapeutic regimes.

Twenty repeat breeding crossbred cows and 18 buffaloes (Gr.-I) with mature palpable mid-cycle CL on either of the ovaries were treated with i/m injection of PGF₂ α 25 mg (Inj. Lutalyse 5 ml, Pfizer Animal Health) and fix timed AI (FTAI) was done twice at 72 and 96 hours later. The other 20 repeat breeding cows and 17 buffaloes (Gr.-II) with clear standing estrus were inseminated and simultaneously administered with i/m Inj. of GnRH-Buserelin acetate 20 ig (Inj. Receptal 5 ml, MSD/Intervet Indian Pvt. Ltd.). Moreover, 16 cows and 17 buffaloes (Gr.-III) detected in estrus first time spontaneously within 90 days postpartum and inseminated using good guality frozenthawed semen without any treatment, served as normal cyclic/fertile controls.

Animals in all the groups once inseminated were followed for recurrence of estrus and inseminations regularly at least for 3 cycles and in non-return cases, pregnancy was confirmed per rectum 60 days post-AI. Estrus response and conception rate as well as interval from PG treatment to induced estrus/ conception were calculated and compared between groups.

Results and Discussion

Repeat breeding in dairy animals is associated with estrus detection errors, endocrine dysfunctions, ovulatory defects, uterine infection, poor gamete quality etc, and thereby poor fertilization rates and/or early embryonic deaths. The endocrine or hormonal therapies give good results in classical repeat breeders in absence of other causes of conception failure.

Effect of Mid-cycle PGF₂α **Treatment**:

Out of 20 repeat breeding crossbred cows and 18 buffaloes treated with mid-cycle $PGF_2\alpha$ injection, 18 (90.00%) and 17 (94.44%) animals responded with behavioural estrus within 71.25±3.57 and 82.67±4.22 hr, respectively, due to rapid luteolysis. These findings were also authenticated and correlated earlier with the plasma progesterone profile evaluated by RIA on the day of mid-cycle PG treatment, day of AI/FTAI and on day 21 post-AI (Savalia et al., 2013; Patel et al., 2014). Of the induced animals, 12 cows exhibited prominent estrus and 6 showed moderate estrus signs. while among buffaloes 8, 6 and 3 animals were observed in prominent, moderate and weak estrus, respectively. The nature of estrus induced with mid-cycle PG injection was almost similar to that in normal cyclic buffaloes, but in cows only 66.67 % animals expressed prominent estrus and remaining 33.33 % showed moderate signs as compared to 90 and 10 % in normal cyclic control cows (Table 1).

A comparable estrus response of 85 to 95 % has been documented following mid cycle PG injection in cows and buffaloes by Rao and Rao (1979), Totewad *et al.* (2007), Savalia *et al.* (2013) and Patel *et al.* (2014), while little lower response of 75 to 85 % has been reported by others (Kharche and Srivastava, 2001; Khasatiya *et al.*, 2008), and still lower estrus response of







around 65 % (Sathiamoorthy et al., 2007; Patel *et al.*, 2009) was found by some researchers. Khasatiya *et al.* (2004) and Butani *et al.* (2009) found 100.00 per cent estrus response following PGF₂ α treatment in suboestrus buffaloes. Prostaglandin F₂ α (25 to 30 mg) and its analogues (500 µg) induce luteolysis and were found to induce ovulatory estrus and improve reproductive efficiency in subfertile cows and buffaloes (Rao and Rao, 1979; Khasatiya *et al.*, 2004; Patel *et al.*, 2014). The luteolytic action is most potent between day 5 and 17 of the bovine cycle and most of the animal show ovulatory estrus within 3-4 days of treatment (Rowson *et al.*, 1972).

The estrus induction intervals recorded following mid-cycle $PGF_2\alpha$ injection in present study are comparable to those reported earlier

ranging from 54.4 ± 7.6 to 71.33 ± 6.38 hr in subestrus and/or repeat breeding bovines by Sathiamoorthy *et al.* (2007), Savalia *et al.* (2013) and Patel *et al.* (2014), while delayed estrus response between 3 and 4 days has been noted by others (Kharche and Srivastava, 2001; Totewad *et al.*, 2007). In early postpartum Surti buffaloes, Khasatiya *et al.* (2004) observed this interval as 3.40 ± 0.40 days, while in five months postpartum suboestrus buffaloes they (Khasatiya *et al.*, 2008) found this interval as 4.07 ± 0.53 days.

The conception rates in cows under mid-cycle $PGF_2\alpha$ protocol were 40.00, 41.66, 42.88 and 80.00 (16/20) per cent at induced/first estrus, second, third cycle and overall of 3 cycles, respectively. The corresponding figures in buffaloes under similar treatment were 42.78,

Animal	Treatment Protocols	No. of Animals	Per cent Estrus Induction Response	Estrus	PG Inj. to Estrus		
Species				Prominent	Moderate	Weak	Interval (hrs)
	Mid-cycle PGF ₂ α	20	90.00 (18/20)	66.67 (12)	33.33 (6)	0.00 (0)	71.25±3.57 (n=18)
Crossbred cows	Al+ GnRH	20	100.00 (20/20)	80.00 (16)	10.00 (2)	10.00 (2)	_
	Normal Cyclic Control	16	100.00 (16/16)	90.00 (14)	10.00 (2)	0.00 (0)	-
	Mid-cycle PGF₂α	18	94.44 (17/18)	47.05 (8)	35.29 (6)	17.65 (3)	82.67±4.22 (n=17)
Buffaloes	Al+ GnRH	17	100.00 (17/17)	41.18 (7)	41.18 (7)	17.65 (3)	_
	Normal Cyclic Control	17	100.00% (17/17)	47.05 (8)	41.18 (7)	11.17 (2)	_

Table 1: Effect of mid cycle $PGF_2\alpha$ on estrus induction and estrus intensity in repeat breeding and in normal cycling cows and buffaloes

Figures in parenthesis indicate number of animals.



27.27, 25.25 and 66.66 (12/18) per cent. These results were very much parallel to the conception rates obtained in normal cyclic controls in the respective species (Table 2, Fig. 1), and were achieved within mean intervals of 17.55 ±5.96 and 16.12±6.74 days from PG injection among total conceived animals. This 40 % first service conception rate found following PG induced estrus in the present study corroborated well with earlier reports of Rao and Rao (1979). Kharche and Srivastava (2001) and Sathiamoorthy et al. (2007). Similarly, 80 and 67 % overall conception rates obtained in repeat breeding cows and buffaloes with PG treatment compared favourably with reports of Patel et al. (2014) and Patel et al. (2009), respectively, while others noted much lower conception rates of 40 to 57 % (Totewad et al., 2007; Butani et al., 2009). In contrast to these, Khasativa et al. (2004, 2008) observed 100.00 per cent overall conception rate among PG treated suboestrus Surti buffaloes. The beneficial effect of mid-cycle PG injection could be due to better synchrony of endocrine events leading to timely ovulation and strengthening of luteal function in repeat breeding crossbred cows and buffaloes.

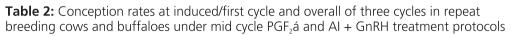
Effect of GnRH Treatment at Breeding:

The pattern of estrus intensity expressed, whether prominent, moderate or weak was identical in both AI + GnRH injected and normal cyclic control groups, suggesting that estrus behaviour of normal cyclic and repeat breeding cows and buffaloes was not distinctly different (Table 1). The conception rates in repeat breeding cows with AI + GnRH protocol were 30.00, 35.71, 33.33 and 70.00 (14/20) per cent, at induced/first estrus, second, third cycle and overall of 3 cycles, respectively. The corresponding figures in buffaloes were 29.41, 25.25, 11.11 and 52.94 (9/17) per cent. These results were around 10 per cent lower

particularly at first AI and overall of three cycles in both the species as compared to the conception rates obtained in normal cyclic controls and mid-cycle PG treated groups in specific (Table 2, Fig. 1).

Around 30.00 per cent first service conception rates obtained with GnRH treatment at the time of AI in repeat breeder cows and buffaloes compared favourably with the report of Patel et al. (2014), but were lower than the earlier report of 54 per cent by Rao (1991). Further, the first service and overall conceptions obtained in crossbred cows under GnRH treatment at AI corroborated with the reports of Rayos (1995), Rangekar et al. (2002), Kumar et al. (2009), Parmar et al. (2013) and Patel et al. (2014). The present 52.94 % overall conception rate obtained in GnRH treated buffaloes was. however, comparable with the previous reports of Butani et al. (2009) as 57.14 % and Savalia et al. (2013) as 50 %, while Vijayarajan et al. (2007) found it as only 40.00 % in repeat breeders. However, Sharma and Dhami (2008) observed 90.00 % overall conception rate in suboestrus buffaloes, while Rao (1991) observed it as 71.4 %, which is much higher than the present result.

The variations observed in estrus induction response with PG injection and fertility with both PG and GnRH treatment in different studies are due to many factors such as stage of estrus/estrous cycle at the time of treatment, product potency, estrus detection efficiency, nutritional status, general and genital health, breeding time and quality of semen used, season/climate, and luteal activity or sustainability leading to embryonic mortality post-breeding etc. The present findings and those of many of the above researchers clearly support that PGF₂ α analogues have definite standing in successful management of suboestrus and repeat



A i	Treatment Protocols	No. of Animals	Per cent Conception Rate at				PG Inj. to
Animal Species			Induced Estrus (FTAI)	Second Cycle	Third Cycle	Overall of 3 Cycles	Fertile Estrus Interval (days)
	Mid-cycle PGF ₂ α	20	40.00 (8/20)	41.66 (5/12)	42.88 (3/7)	80.00 (16/20)	17.55±5.96 (n=16)
Crossbred	Al+ GnRH	20	30.00 (6/20)	35.71 (5/14)	33.33 (3/9)	70.00 (14/20)	-
COWS	Normal Cyclic Control	16	43.75 (7/16)	44.44 (4/9)	20.00 (1/5)	75.00 (12/16)	87.28±4.78* (n=12)
	Mid-cycle PGF₂α	18	42.78/ (7/18)	27.27 (3/11)	25.25 (2/8)	66.66 (12/18)	16.12±6.74 (n=12)
	Al+ GnRH	17	29.41 (5/17)	25.25 (3/12)	11.11 (1/9)	52.94 (9/17)	-
Buffaloes	Normal Cyclic Control	17	35.29 (6/17)	27.28 (3/11)	25.00 (2/8)	64.71 (11/17)	99.87±6.57* (n=11)

Figures in parenthesis indicate number of animals, * Service period/days open.

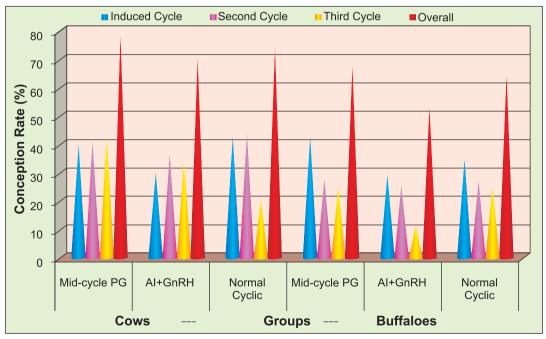


Fig. 1: Conception rates in repeat breeding crossbred cows and buffaloes under mid cycle $PGF_2\alpha$ and AI + GnRH treatment protocols in relation to normal cyclic ones 32



breeding conditions in cows and buffaloes, since these drugs induce ovulatory estrus following luteolysis. The results obtained in the present study using PGF₂á injection were better than with GnRH injection at the time of Al, at par with normal cyclic control groups. Thus, the application of mid-cycle PGF₂α injection can be used as a good tool for induction of fertile estrus as well as enhancement of conception rate in repeat breeding crossbred cows and buffaloes under field conditions.

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References

Baruselli, P. S., Sales, J. N. S., Sala, R. V., Vieira, L. M. and Sá Filho, M. F. (2012). History, evolution and perspectives of timed artificial insemination programs in Brazil. *Anim. Reprod.*, **9**(3): 139-152.

Butani, M. G., Dhami, A. J., Rajesh Kumar., Hirani, N. D., Ramani, V. P. and Patel, K. P. (2009). Influence of hormonal and antibiotic therapy on fertility and trace minerals profile in a repeat breeding buffaloes. *Indian J. Field Vets.*, **3**(3): 12-16.

Goley, R. R. and Kadu, M. S. (1995). Efficacy of $PGF_2\alpha$ (Lutalyse), GnRH analogue (Receptal) and hCG (Chorulon) in treatment of repeat breeder cows. *Indian Vet. J.*, **72**: 472-475.

Kharche, S. D. and Srivastava, S. K. (2001). Fertility following treatment of suboestrus buffaloes with PGF_2 alpha. *Indian J. Anim. Reprod.*, **22**(2): 170-171.

Khasatiya, C. T., Desai, P. M., Dhami, A. J., Dugwekar, Y. G., Panchal, M. T. and Kavani, F. S. (2004). Effect of GnRH and PGF₂ alpha on productive performance and progesterone profile in postpartum true anoestrus and suboestrus Surti buffaloes. *Indian J. Dairy Sci.*, **57** (5): 324-328.

Khasatiya, C. T., Kavani, F. S., Dhami, A. J., Derashri, H. J., Panchal, M. T. and Desai, P. M. (2008). Studies on puerperal events and reproductive efficiency following hormone therapy at day 42 postpartum in Surti buffaloes. *Int. J. Agri. Biol.*, **1**: 132-137.

Kim, U., Suh, G., Hur, T., Kang, S., Kang, H., Park, S., Kim, H. and Kim, I. (2007). Comparison of two types of CIDR-based timed artificial insemination protocols for repeat breeder dairy cows. *J. Reprod. Dev.*, **53**(3): 639-645.

Kumar Rajesh, Butani, M. G., Dhami, A. J., Kavani, F. S. and Shah, R. G. (2009). Effects of different therapies on fertility and serum progesterone, metabolites and minerals profile in repeat breeding crossbred cows. *Indian J. Field Vets.*, **5**(2): 1-8.

Modi, L. C., Suthar, B. N., Nakhashi, H. C., Sharma, V. K. and Panchasara, H. H. (2011). Physical characteristics of oestrual cervical mucus and conception rate in repeat breeder Kankrej cattle. *Indian J. Vet. Med. Sci.*, **5**(4): 416-423.

Parmar, Sachin V., Patel, J. A. and Dhami, A. J. (2013). Effect of hormone therapy on fertility and plasma minerals profile in repeat breeding Gir cows. *Indian J. Field Vets.*, **8**(4):18-25.

Patel, K. R., Dhami, A. J., Hadiya, K. K., Savalia, K. K., Killedar, A. and Patel, S. B. (2014). Effect of mid-cycle PGF2á and GnRH at AI on conception rates, plasma progesterone and biochemical profile in repeat breeding crossbred cows. *Indian J. Field Vets.*, **9**(3): 5-11.

Patel, P. P., Panchal, M. T., Patel, B. N. and Kavani, F. S. (2009). Effect of supplementation of concentrate and minerals, GnRH and $PGF_2 \acute{a}$ on postpartum reproduction in buffaloes. *Indian J. Anim. Reprod.*, **30**(1): 73-77.

Ranganekar, M. N., Dhoble, R. L., Sawale, A. G., Gacche, M. G., Ingawale, M. V. and Jadhav, J. M. (2002). Effect of gonadotropin releasing hormone (GnRH, Fertagyl), administration on the conception







rate of repeat breeding cows. *The Blue Cross Book*, **18**: 20-21.

Rao, A. R. and Rao, S. V. (1979). Treatment of suboestrus in buffaloes with cloprostenol. *Vet. Rec.*, **105**: 168-169.

Rao, A. V. N. (1991). Gonadotrophin releasing hormone therapy in anoestrus, repeat breeding and follicular cystic cows. *Indian Vet. J.*, **68**(3): 267-270.

Rayos, A. A. (1995). Conception rate in repeat breeder cows after treatment with GnRH analogue (Buserelin) during estrus. *Philippine J. Vet. Med.*, **32**: 10-13.

Rowson, L. E. A., Teruit, H. R. and Brand, A. (1972). The use of prostaglandin for synchronization of estrus in cattle. *J. Reprod. Fertil.*, **29**: 145-154.

Sathiamoorthy, T., Parthasarathy, R., and Kathirchelvan, M. (2007). Efficacy of PGF₂ alpha, CIDR and Ovsynch treatment on estrus induction and fertility in postpartum buffaloes. *Indian J. Anim. Reprod.*, **28**(1): 8-11.

Savalia, K. K., Dhami, A. J., Patel, K. R. and Hadiya, K.

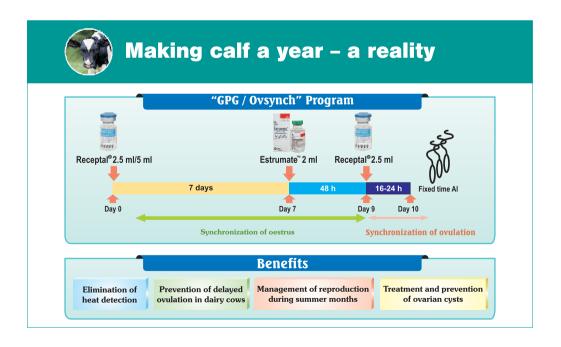
K. (2013). Influence of controlled breeding technologies on fertility and plasma macro-minerals profile in conceiving and non-conceiving anoestrus and repeat breeding buffaloes. *Indian J. Field Vets.*, **9**(2): 28-35.

Sharma, S. K. and Dhami, A. J. (2008). Effect of postinsemination antibiotics and hormone therapy on fertility in relation to macro-minerals profile in repeat breeding animals. *Indian J. Field Vets.*, **3**(3): 1-6.

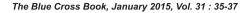
Sreenan, A. M. and Diskin, M. G. (1983). Early embryonic mortality in the cow: its relationship with progesterone concentration. *Vet. Rec.*, **112**: 517-521.

Totewad, G. D., Dhoble, R. L., Sawale, A. G., Naik, P. M. and Mane, P. M. (2007). Efficacy of cloprostenol in buffaloes by intramuscular route. *XXIII Annual Convention of ISSAR and National Symposium*, 7-9 Dec., QUAT, Bhubaneswar, Orissa, India.

Vijayarajan, A., Chandrahasan, C. and Napolian, E. (2007). Effect of pre- and post-insemination substitution of GnRH in repeat breeding buffaloes. *Indian Vet. J.*, **84**: 940-943.





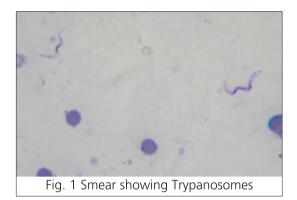


Therapeutic Management of Trypanosomiasis in a buffalo

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Introduction:

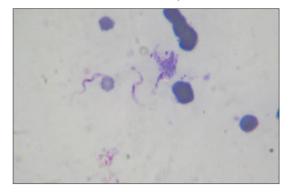
'Trypanosomiasis' or "Surra' is one of the most widely distributed pathogenic, mechanically transmitted vector borne haemoprotozoan diseases of livestock and wild animals in India. It is transmitted by biting flies viz., Tabanus and Stomoxys (Gill, 1991). It is caused by Trypanosoma evansi resulting in hypoglycemia, anaemia, pyrexia, progressive emaciation, nervous signs and finally death (Radostits et al, 2000). In India, T. evansi infection is widely prevalent in different parts. It has significant economic importance in livestock production as it causes huge economic losses to the farmers in terms of morbidity, mortality, abortion, infertility and reduced milk yield etc (Juyal, 2011). The economic losses due to this disease are underestimated in cattle and buffaloes, mainly because of its sub-clinical nature. The present paper reports the diagnosis and therapeutic management of trypanosomiasis in a buffalo.

Case History and Clinical Examination:

A five year old non descript buffalo was presented to Teaching Veterinary Clinical Complex, Post Graduate Institute of Veterinary and Animal Sciences, Akola with the history of drop in milk production and inappetance. The clinical examination of buffalo revealed rise in temperature (104.6 °F), serous nasal discharge and congested mucous membrane. The wet blood film sample examination of buffalo revealed presence of number of vigorously motile trypanosome parasites. The microscopic examination of blood smear stained with Giemsa's showed the presence of extracellular flagellated *Trypanosoma spp.* (Fig.1). Based on history, clinical examination and laboratory findings the case was diagnosed as Trypanosomiasis.

Treatment and Discussion:

The animal was treated with a single dose of Inj. Surral (Isometamedium hydrochloride) @





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0.5mg/kg body weight intramuscularly. The supportive treatment given to the animal included Inj. Dextrose 25%, 500 ml intravenously, Inj. Avilin vet (Pheniramine maleate) 10 ml intramuscularly, Inj. Melonex plus (Meloxicam and Paracetamol) @ 0.5 mg/kg body weight I/M and Inj Bivinal plus (B complex with liver extract) 10 ml I/M for three consecutive days. The animal responded to treatment and showed marked improvement on second day of treatment.

Isometamidium Chloride is a latest and most effective synthetic trypanocidal drug used for chemotherapy and chemoprophylaxis in trypanosomiasis in livestock (Anene et al., 2001, Shweta Anand et al., 2013) and showed higher clinical efficacy against Typanosoma evansi on single dose I/M administration (Sinha et al., 2013). Several workers reported efficacy of Isometamidium as remedial measure against trypanosomiasis in animals (Magona et al., 2004; Karaye, 2012). The mode of action of Isometamidium is not fully understood, but there is an evidence that kinetoplastic topoisomerase type II of trypanosoma is selectively inhibited by the drug (Mehlhorn, 2008).

As trypanosomes consume large quantity of blood sugar resulting in the breakdown of the liver function, leads to hypoglycaemia and fatal intoxication (Pathak and Narendra Singh, 2005). Therefore, in the present case, supportive therapy such as Dextrose, antihistaminic and vitamin B complex with liver extract were administered for 3 days (Kumar et al., 2011, Lakshmi and Padmaja, 2012). Meloxicam and paracetamol combination was given for reducing inflammatory changes and fever (Sahu, 2008). The blood smear examination after 24 hours was found negative for trypanosome parasite. The animal showed complete recovery after 3 days with normal appetite, rectal temperature, absence of nasal discharge and improvement in milk production.

The present case of trypanosomiasis in buffalo was diagnosed and successfully treated with the latest trypanocidal drug Inj Isometamedium hydrochloride along with supportive treatment of fluids, antihistaminics, analgesic and antipyretic and B complex. The recovery was complete within three days of treatment.

References:

Anene B. M., Onah D. N. and Nawa Y. (2001) Drug resistance in pathogenic African trypanosomes: What hopes for the future? *Vet. Parasitol*, **96**:83100.

Gill B. S. (1991) Trypanosomes and trypanosomiasis of Indian livestock, Indian Council of Agriculture Research, Publication, Krishi Anusandhan Bhawan, Pusa, New Delhi.

Radostits O. M., Gay C. C., Blood D. C. and Hinchcliff K. W. (2000) Veterinary Medicine. A text book of the diseases of cattle, Sheep, pigs, goat and horses, **9th** Edition, *Saunders Elsevier Publication, Philadelphia*, USA, pp. 1329-1339.

Kumar R., Singh R. K. and Singh J. B. (2011) Trypanosomiasis in buffaloes and its clinical management, *Indian J. Vet. Med.* **31**(1): 61-62.

Sahu B. D. (2008) Trypanosomiasis in dairy buffaloes Clinico haematological and therapeutic findings, *Intas Polivet*, **9**(11):272-273

Lakshmi K. and Padmaja K. (2012) Therapeutic management of Trypanosomiasis in a buffalo, *The Blue Cross Book*, **27**:65-66

Juyal P. D. (2011). "Newer Perspectives in the Diagnosis and Control of Trypanosomosis (Surra) in Domestic Livestock in India", TROPMED -Internationale Wissenschaftliche Publikationen, pp. 1-13.

Karaye G. P. (2012) The Efficacy of Isometamidium Chloride in the Treatment of Trypanosomosis in Red Sokoto Bucks Experimentally Infected with *Trypanosoma Congolense and Trypanosoma Brucei* Single and Mixed Infection of the Two. M.Sc. (Veterinary sciences) thesis submitted to the School of Postgraduate Studies, Ahmadu Bello University,







Magona J. W., Mayende J. S. P., Okiria R., Okuna N. M. (2004) Protective efficacy of isometamidium chloride and diminazene aceturate against natural Trypanosoma brucei, Trypanosoma congolense and Trypanosoma vivax infections in cattle under a suppressed tsetse population in Uganda, Onderstepoort J. Vet. Res., 71(3):231-237

Mehlhorn H. (2008) Encyclopedia of parasitology, 1-2:381 Volume 1 (3rd edn). Springer. Verlag, Heidelberg, Germany.

Sinha S., Anand S. and Mandal T. K. (2013) Study of plasma protein binding activity of isometamidium

and its impact on anthelmintic activity using trypanosoma induced calf model, Vet World, 6 (7): 444-448

Shweta Anand, Mandal T. K. and Suprita Sinha (2013) Effect of dose on disposition kinetics of isometamidium chloride/hydrochloride in trypanosomiasis induced calves, African Journal of Pharmacy and Pharmacolo, 7 (15): 801-808

Pathak K. M. L. and Narendra Singh (2005) Animal Trypanosomiasis, Intas Polivet, 6 (2):194-199

Population Dynamics of Indian Livestock (1951 to 2012)

							Figures in millions
Category	1951	1970	1991	2003	2007	2012	% chang over 200
Cattle							
Indigenous	155	178	189	160	166	151	-8.94
CB/Exotic	-	-	15	27	33	40	+20.18
Total Cattle	155	178	204	187	199	191	-4.10
Buffaloes	43	57	84	98	105.34	109	+3.19
Goats	47	67	115	124	140.53	135	-3.82
Sheep	39	40	50	61	71.55	65	-9.07
Pigs	4	7	12	13	11.13	10	-7.54
Equines	2.86	1.98	1.96	1.79	1.18	1.14	-3.39
Camel	0.60	0.10	1.03	1.13	0.51	0.40	-22.63
Total Livestock including all other	288.46	352.08	452.99	505.92	529.70	512	-3.33
Poultry (including ducks, turkeys)	24	138	300	489	649	729	+12.39

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The Blue Cross Book, January 2015, Vol. 31 : 38-39

A case of Jone's Disease in a Pandharpuri buffalo

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Introduction :

Johnes disease (JD) is a sporadic but fatal, chronic, infectious, gastrointestinal disease encountered in animals, mainly in cattle and is caused by obligate, pathogenic bacilli, Mycobacterium avium sub species It also affects paratuberculosis (MAP). buffaloes, sheep, goats, wild ruminants and humans (Harris and Barletta, 2001). Clinically, the disease is characterized by chronic intermittent or persistent watery diarrhea and progressive emaciation. The disease therefore, is sometimes labeled as chronic specific enteritis or chronic bacillary dysentery (OIE Terrestrial Manual, 2014). The incubation period ranges from 2 to 5 years. Generally, calves acquire the infection through milk of infected mother or during their uterine life and exhibit the symptoms after they become an adult (Chauhan et al., 2011). Taking into consideration, the diminishing productivity, heavy economic losses, treatment cost, and the zoonotic potential, the disease has become a matter of concern worldwide.

Case history and observations:

A six years old Pandharpuri buffalo with the history of progressive emaciation and intermittent diarrhea for 40-50 days was presented to the Veterinary Dispensary, Manivali, Thane. On clinical examination, animal appeared very weak, dehydrated and emaciated to hide and bone condition (Figure 1). Skin appeared rough, scaly with absence of normal luster. The animal showed intermittent watery and foamy diarrhea. Anal and adjoining



Fig. 1 Clinical case

area was soiled with fecal material. Animal had continued to become weak in spite of normal dietary intake. On hematological examination, all parameters were within reference range except marginal relative increase in lymphocytes and monocytes. Fecal examination ruled out the parasitic etiology but when stained with modified acid fast method, fecal smear revealed

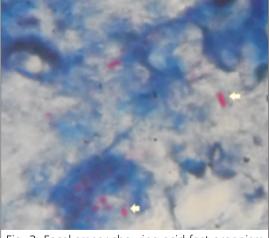


Fig. 2: Fecal smear showing acid fast organism



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acid fast bacterial rods similar to *Mycobacterium paratuberculosis* (Figure 2). Rectal pinch examination revealed epitheloid cells. These findings were suggestive of Johne's disease.

Treatment and discussion

Initially, the animal was treated with Streptomycin @ 10 mg/kg IM and Metronidazole @ 20 mg/kg IV, once a day for 5 davs. Atropine sulphate @ 0.02 mg/kg IM, Meloxicam @ 0.3 mg/kg SC, Chlorpheniramine maleate @ 30 mg IM (once a day) was administered for 7 days. The animal was given supportive therapy using Ringer Lactate solution @ 3 lit/day to maintain fluid status and Neblon powder @ 30 gm for 7 days. Even after intensive treatment with antibiotics, antiinflammatory drugs, anthelmentics, the animal continued to show the symptoms, though the severity was guite less than that of pretreatment period. After a week, the treatment was discontinued at the owner's request.

Generally, till the symptoms become visible, the

animal remains in carrier or subclinical stage. However, once the immunity gets down the bacteria overcome cellular defense and establishes the clinical disease. Ruminants once infected, generally remain infected throughout life, though the early treatment may have some positive response. Regular check up for every 6 months and culling of infected animals is suggested.

References:

Harris N. B., Barletta R. G. (2001). Mycobacterium avium subsp. paratuberculosis in Veterinary Medicine. *Clin Microbiol* Rev. **14**: 489512.

Chauhan H. C., Dadawala A.I., Patel S.S., Ranaware P. B., Jadhav K. M., Patel K. G., Shah N. M. and Chandel B.S. (2011). Paratuberculosis (Johne's disease) in a buffalo: a case report. *Buffalo Bulletin.* **30**: 111-12.

OIE Terrestrial Manual. (2014). Chapter 2.1.11.: Paratuberculosis (Johne's disease). (Obtained from h t t p : // w w w . o i e . i n t / f i l e a d m i n /Home/eng/Health_standards/tahm/2.01.11_ PARATB.pdf).

19th Livestick Census 2012;

Some observations over 18th livestock Census (2007)

- Livestock population decreased by 3.33%
- Poultry population increased by 12.39%
- Cattle population decreased by 4.10%
- Indigenous cattle population decreased by 8.94%
- Crossbred/exotic cattle population increased by 20.18%
- Buffalo population increased by 3.19%
- Goat population decreased by 3.82%
- Sheep population decreased by 9.07%
- Pigs/Equines/Camel population decreased by 7.54%, 3.39% and 22.63% respectively





Dystocia and its Surgical Management in a Siamese queen

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Introduction

Dystocia is the inability of the dam to expel the fetus at parturition through the birth canal without assistance. It is the most important obstetrical condition and needs immediate attention. Dystocia in the cat has been poorly described due to a wide variation in the kittening process over the various breeds. In the cats, the incidence is around 5 per cent overall, but reaches 20 per cent in some breeds of cats, with the highest incidence in Persian cat (Widmann, 1992, Linde-Forsberg and Eneroth, 2000) followed by the Siamese type and Devon Rex cats (Gunn Moore and Thrushfield, 1995). In the present communication, a case of dystocia in Siamese gueen is reported and its clinical and surgical management is described and discussed

Materials And Methods

A Siamese queen weighing 4.5kg and age about three and half years was presented with the history of delivering 3 dead kittens and straining from the past 24 hours with foul smelling vaginal discharge (Figure 1), which was unresponsive to the medical therapy. On clinical examination, cat appeared normal and alert. The temperature, pulse and respiration were in normal ranges. Per-vaginal examination revealed dead fetus in uterus with relatively large size. The case was diagnosed as dystocia. Animal was given epidosin and PGF2 alpha but even after 24 hours cervix did not open. In this case, surgery was



Figure 1: a case before the treatment (straining from the past 24 hours with foul smelling vaginal discharge).

warranted, hence it was decided to perform caesarean section.

The animal was premedicated with atropine sulphate at the rate of 0.04 mg/kg and xylazine at the rate of 1mg/kg intramuscularly. Induction was done with a combination of diazepam at the dose rate of 0.27mg/kg plus ketamine at the dose rate of 5.5mg/kg intravenously. Maintenance of anesthesia was done with reduced doses of diazepam at the dose rate of 0.27 mg/kg plus ketamine at the dose rate of 5.5mg/kg. The site of operation was prepared for aseptic surgery, secured and draped. After restraining the animal in dorsal recumbency, a ventral midline incision just caudal to the umblicus was given (Figure 2). Uterus was incised and one dead fetus inside was removed. (Figure 3). The foetal membrane was also removed. The uterus was flushed with normal saline and Betadine solution. Antibiotic pessaries







Figure 2: Positioning of the animal during surgery

Furea boli (Pfizer Limited, Mumbai) were kept intrauterine. Uterine incision line was closed with double row of Lambert sutures using catgut no. 01 while pushing back the uterus into the abdominal cavity a superficial uterine rupture was seen on the body of uterus which was also closed with double row of Lembert sutures using catgut no. 01. Neosporine powder was then sprinkled in the abdomen and the abdominal incision was closed in routine manner using three layer suturing technique. Adequate fluid therapy was given with 5% DNS and D5 intravenously during and after surgery. After recovery from anesthesia, Elizabeth collar 10 no. was fitted to prevent her from licking the site. Following hysterectomy, the animal was treated with injection enrofloxacin (Floxidin 10%, MSD Animal Health, Intervet India Pvt Ltd.) dosed at 5 mg/kg body weight intramuscularly once daily along with injection meloxicam (Melonex, Intas Pharmaceutical Ltd, Ahmedabad) dosed at 1 ml /10 kg body weight intramuscularly, injection B-complex (Tribivet, Intas Pharmaceutical Ltd, Ahmedabad) dosed at 2 ml intramuscularly and injection pheniramine maleate (Avilin, MSD Animal Health, Intervet India Pvt Ltd.) dosed at 1 ml intramuscularly were administered for 5 days. Injection Oxytocin (Novertis, Mumbai) 10 IU was injected in the uterine musculature. Postoperatively, intravenous dextrose saline solution was continued for 1 hour to ensure adequate rehydration and detoxification. The animal was maintained with intravenous fluid for first three



Figure 3: Dead fetus inside the uterus was removed.

days. Daily dressing of wound was done with silver sulfadiazine ointment for 8 days. The animal was given liquid diet for 5 days and advised abdominal bandage for 10 days. The skin sutures were removed on 10th postoperative day.

Results And Discussion

The gueen recovered uneventfully and wounds were completely healed without any complication. Dystocia has been classified into maternal and fetal types (Sloss and Dufty, 1980). Maternal causes include uterine inertia, narrow bony birth canal, uterine torsion, vaginal septum formation and hydroallantois. Among maternal causes of dystocia, uterine inertia constitutes the biggest maternal cause of dystocia in cat (Ekstrand, 1993). The foetal causes are less common and include foetal maldisposition, foetal oversize, foetal malformation and foetal death (Jackson, 1995). Although there are many causes of dystocia but mostly it is usually the combination of different causes that leads to dystocia.

Dystocia is an emergency condition, if not treated immediately, can result in death of the queen as well as kittens. Usually kittening should complete within 2 to 6 hours but may take 10 to 12 hours in older females (Laliberte, 1986). If it takes more than 12 hours, it should be taken as a case of dystocia. For dystocia, medical therapy is indicated if foetuses can be expulsed through birth canal without delay,







otherwise surgical intervention is mandatory. The mortality of queens can be significantly reduced by timely diagnosis and prompt veterinary medical care. With the scientific progress in veterinary surgery, especially in veterinary anesthesia, a caesarean section is the treatment of choice in a wide variety of dystocia (Reichler and Michel, 2009). Therefore it can be concluded that the best treatment for dystocia with dead foetus should be conservative caesarean section without further delay, as it was effective in the present case.

Refrences

Ekstrand, C. and Linde-Linde-Forsberg, C.1994. Dystocia in the cat: a retrospective study of 155 cases. *J. Small Anim. Pract.* **35:** 459-464.

Gunn-Moore, D.A. and Thrushfield, M.V.1995. Feline dystocia : Prevalence and association with cranial conformation and breed. *Vet. Record.* **136**: 350-353.

Jackson, P.G.G.1995. *Handbook of Veterinary Obstetrics*, W.B. Saunders Co., Philadelphia, USA.

Laliberte, L.1986. Pregnancy, Obstetrics and Postpartum Management of the Queen in: Morrow D.A., *Current therapy in Theriogenology*. W.B. Saunders Company, pp: 813-816

Linde-Forsberg, C. and Eneroth, A. 2000. Abnormalities in pregnancy, parturition, and the periparturient period. In: Ettinger SJ, Feldman CE, editors. *Textbook of Veterinary medicine*. Philadelphia: Saunders, W.B. pp: 1527-38.

Reichler, I M. and Michel, E. 2009. Dystocia: recognition and management. Europ. J. Comp. *Anim.Pract.* **19:** 165-173.

Sloss, V. and Dufty, J.H.1980. *Handbook of bovine obstetrics.* Williams and Wilkins, Baltimore, USA. pp: 208.

Widmann, A.B.1992. Effects of breeds on reproduction and pup mortality in dogs with regard to the susceptibility of some dog and cat breeds to dystocia. Thesis, Tierarztliche Hochschule Hannover, Germany: pp:104-108.

Peri-parturient Disorders : A hurdle in Dairy Development

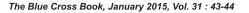
The transition period of dairy animal from 3 weeks before parturition to 3 weeks after parturition is crucial in its production cycle, in the sense that, no other period can affect subsequent health, productivity and reproductive capability of the animal, so greatly. The profitability of a



dairy cow or a buffalo during lactation is determined solely by effective nutritional management during this period. Inadequate nutrition, in particular, during this period, not only impedes the production capacity but also the health of the animal. The greatest challenge faced by the animal during this period is the sudden and marked increase of nutrient requirements for fetal development and ensuing milk production, at a time when dry matter intake and the nutrient supply lags far behind the requirement.

This transition period of 6 weeks is marked as "**Peri-parturient Period**", wherein dairy animals undergo large metabolic adaptations in glucose, fatty acids and mineral metabolism. Inadequate nutritional management fails to support these metabolic adaptations, culminating into metabolic disorders like milk fever, ketosis, hypomagnesimia and hypokalemia, collectively known as "**Peri-parturition Disorders**"





Postpartum Mummification of a Co-Twin Fetus in a Bakarwal Doe

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Introduction

Mummification has been reported to occur frequently in swine, occasionally in dogs, uncommonly in sheep, cattle and horses and rarely in goats (Roberts, 1971). Fetal mummification associated with a persistent corpus luteum is observed mainly in cattle and rarely in goats, both species being dependent on progesterone produced by the corpus luteum for the maintenance of pregnancy (Mathew et. al., 1980). Several authors have described fetal mummies in goats (other than Bakarwal goat doe) with dystocia (Markandeya et al., 1991; Javanth et al., 1993; Tutt, 1997). Postpartum retention and mummification of a co-twin fetus in a Bakarwal dwarf doe has not been reported. The present paper puts on record a case of postpartum mummification of a co-twin fetus in a Bakarwal doe and its surgical management.

Case history and observations

A 22-month-old Bakarwal doe delivered a kid normally. After parturition, the doe continued with straining and was treated for retention of placenta by a field veterinarian but without success. The doe was further examined by another field veterinarian and was misdiagnosed as a case of retention of urine because of distended abdomen due to accumulation of fluid. The doe was subjected to tube cystostomy. The cystostomy tube had got retrieved back into the abdominal cavity of the doe due to continuous straining. Finally the doe was presented to Division of Veterinary Surgery and Radiology for confirmative diagnosis and further surgical management. At the time of presentation, the doe was weak, listless and febrile (42°C). The goat would stand for short periods after being assisted to rise but was ataxic. A large, firm mass was palpated in the caudo-dorsal abdomen which appeared to be associated with the uterus because it protruded cranially from the pelvis. A radio-opague mass was visualized in the caudal abdomen on the radiograph. The caudal abdomen was examined ultrasonographically, but it was not possible to penetrate the mass and multiple acoustic shadows were present. From physical. radiographic and ultrasonographic examination, presence of second fetus was diagnosed. Considering the exigency of the condition, emergency surgery was decided.

Surgical management

The animal was restrained in right lateral recumbency. Slow intravenous infusion of 1.5 liters of normal saline and 2 liters of dextrose normal saline solution was started before surgical operation. Left para-lumbar fossa was prepared for aseptic surgery. Local infiltration of surgical site with 2% lignocaine hydrochloride (Astra IDL, Bangaluru) was achieved. A 6 inch long left ventrolateral oblique incision was made on the skin. Muscles and peritoneum were incised. On incising the peritoneum, approximately 4 to 5 liters clear straw coloured fluid escaped from the peritoneal cavity. The

gravid uterus was exteriorized through abdominal wound. A longitudinal incision was placed on the uterine body. A mummified kid was taken out. The uterus was lavaged with normal saline and betadine solution. The uterine incision was closed by double row of lambert suture using chromic catgut No. 2. The abdominal wound was closed as per standard procedure. Postoperatively, intravenous dextrose saline solution was continued for 1 hour to ensure adequate rehydration and detoxification. Injection enrofloxacin (Floxidin 10%, MSD Animal Health, Intervet India Pvt Ltd.) dosed at 5 mg/kg body weight intramuscularly once daily along with injection meloxicam (Melonex, Intas Pharmaceutical Ltd, Ahmedabad) dosed at 1 ml/10 kg body weight intramuscularly, injection B-complex (Tribivet, Intas Pharmaceutical Ltd, Ahmedabad) dosed at 2 ml intramuscularly and injection pheniramine maleate (Avilin, MSD Animal Health, Intervet India Pvt Ltd.) dosed at 1 ml intramuscularly were administered for 5 days. The doe was maintained with intravenous fluid for first three days. Daily dressing of wound was done with silver sulfadiazine ointment for 8 days. The skin sutures were removed on 10th postoperative day.

Results and discussion

The doe recovered uneventfully after the surgical management. Successful handling of uterine cases requires skill and correct clinical judgment so that the life of either dam or fetus or both is not jeopardized. Fetal mummification usually occurs after the death of the fetus, during the second or third trimester of gestation in cattle. In contrast to maceration, during which a fetus undergoes bacterial digestion within the uterus with an open cervix, mummification occurs when the cervix is closed and the fetus undergoes autolysis (Mathew *et al.*, 1980). After its death, while the cervix is still closed, the fetal, amniotic and allantoic fluids are resorbed and, in time, the fetus becomes

mummified and moulded into a tight contorted mass by the involuting uterus, usually while it remains sterile. The dead fetus is retained owing to the failure of normal parturition or abortion mechanisms. Mathew et al. (1980) described two cases involving the birth of a normal kid together with mummified fetuses. In cattle, both fetuses of a twin pregnancy may become mummified owing to the anastomoses of the allantoic blood vessels, the causative agent thus affecting both fetuses (Arthur et al., 1989). Fetal death during the first trimester of pregnancy results in the total resorption of the conceptus and a return to oestrus (Arthur et al., 1989). In the present case, the uterus and the retained fetus was dry. The fetus was tightly wrapped by the involuted uterus. This indicates that the death of the fetus had occurred during late gestation. At the time of parturition when one kid was delivered normally, because of absorption of fluids and tight wrapping of the uterine wall, the mummified foetus could not be expelled out.

References

Arthur, G. H., Noakes, D. E. and Pearson, H. (1989).Veterinary Reproduction and Obstetrics.6th edn. London, BailliereTindall. pp:114.

Jayanth, R. S., Balasubramanian, S., George, R. S., Ayyappan, S. and Dhanapalan, P. 1993. *Cheiron.* **22**: 254.

Markandeya, N. M., Pargaonkar, D. R., Bakshi, S. A. andDoijode, S. V. 1991. *Indian Journal of Agricultural Research*.**12**: 107.

Mathew, J., Madhavan, E. and Iyar, C. P. N. 1980. *Livestock Adviser.* **80**: 61.

Roberts, S.J. 1971. Veterinary Obstetrics and Genital Disorders (Theriogenology). Ithaca, New York. pp: 170.

Tutt, C.L.C. 1997. Postpartum mummification of a co-twin fetus in a Cameroon dwarf goat doe *Veterinary Record*. **140**: 229-231.





Clinical Management of Dermatophilosis In A Dairy Farm

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Introduction

Dermatophilosis is a skin infection of domestic, aquatic and wild animals caused by Dermatophilus. congolensis. It is reported worldwide but mainly recorded in tropical and subtropical areas, where it results in considerable economic loss. Animals of all ages are affected, but higher prevalence has been reported in young animals (Gitao et al., 1998; Nath et al., 2010). In contrast, higher prevalence is also reported among adult animals by Admassu and Alemu (2011). Recently, dermatophilosis was identified as the cause of widespread lower leg dermatitis, locally called as pododermatitis, among dairy cattle in Kerala and majority of cases were noticed in adult cows compared to calves (Tresamol, 2012).

Materials & Methods

Fourteen adult lactating cross bred cows at the Instructional Livestock Farm, Kerala Veterinary and Animal Sciences University, Wayanad, were presented with complaint of recurrent lower



Fig 1. First day

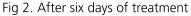
leg dermatitis. Clinical features of the condition included pustules, matting of hairs, scab formation and cracks or fissures on the skin of lower limb, udder, hindquarters and inner region of thigh. Skin scrapings were collected and sent for microbiological examination. From the lesions and microbiological examination, the condition was identified as dermatophilosis and chemotherapy was initiated.

The affected area was washed thoroughly to remove the crusts in lukewarm water containing sodium bicarbonate and sodium chloride (10 g each in one litre lukewarm water) and topical application of Terramycin spray. All the animals were treated with oxytetracyline long acting injection IM at the rate of 20 mg/kg body weight at 3 day interval for 3 times (Aning 1996, Tresamol, 2013).

Result & Discussion

Thirteen animals in the group showed marked clinical cure after two weeks. One cow was retreated with ciprofloxacin at the rate of





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Figure 3. After fourteen days of treatment

5mg/kg body weight IM for three consecutive days after which it showed improvement. Clinical cure resulted in disappearance of crusts, scab and growth of new hairs. There are several reports of *D. congolensis* infection in farm animals of different species of animals. Lesions of bovine dermatophilosis in Kerala, India were more frequently seen on distal part of forelimbs and hind limbs, udder and/or perineum of affected animals (Tresamol, 2012). In the present case also, lesions were found to be mainly on the lower limbs and the udder.

The most important predisposing factors for dermatophilosis are skin trauma, moist environment and humid climate. The wet skin of the lower limbs facilitates transmission of motile zoospores. The incubation period of dermatophilosis ranges from 2-14 days .The present paper supports use of Oxytetracycline as an antibiotic of choice for the successful treatment of Dermatophilosis in cattle without clinical recurrence.

References

Aning KG1, Koney EB.(1996). Chemotherapy of dermatophilosis-a preliminary study. *Tropic. Anim. Health.*(**2 suppl)**, 38S-43S; discussion 74S-86S.

Admassu, M. and Alemu, S. (2011). Study on clinical bovine dermatophilosis and its potential risk factors in North Western Ethiopia. *Intl. J. Anim. Vet. Adv.* **3**:33-36.

Gitao, C.G., Agab, H. and Khalifalla, A.J. (1998).Outbreaks of *Dermatophilus congolensis* infection in camels (Camelus dromedarius) from the Butana region in Eastern Sudan. *Rev. Sci. Tech.* **17**: 743-48.

Nath, B.D., Ahasa, M.S., Rehman, M.S. and Haque, A.K.M.F. (2010). Prevalence and therapeutic management of bovine dermatophilosis. Bangaladesh *.Res. Pub. J.* **4**: 198-207.

Tresamol, P.V. (2012). Studies on pododermatitis in cattle. PhD Thesis submitted to Kerala Veterinary and Animal Sciences University, Pookode, Wayanad

Tresamol P. V. and Saseendranath, M. R. (2013). Antibiogram of Dermatophilus congolensis isolates from cattle. *Int. J. Liv. Res.* **3**(2),117-12

Peri-parturient Disorders : A hurdle in Dairy Development Peri-parturient disorders have a special significance in Indian dairy scenario as Indian Dairy Production System has been rapidly transforming from traditional 'low input - low output' system to large scale commercial venture with introduction of high yielding crossbred cows and buffaloes. These high producing dairy animals are likely to suffer more from peri-parturient disorders, if their peri-partuient period is not effectively managed through nutritional inputs.



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Therapeutic efficacy of *Calcarea fluorica* in the treatment of the Frontal Bone Exostosis in a dog

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Introduction:

Exostosis is defined as benign growths of bone extending outwards from the surface of a bone. Exostosis has been reported in dinosaur fossils from several species (Molnar, 2001). It appears as a localized round or oval hard bony mass with or without pain. The reason for the formation still remains obscure. Frontal bone exostosis occurs seldom in the dogs. There is no well defined protocol of the treatment and surgical intervention with cranioplasty is a recommended medical intervention. Homeopathic medicine Calcarea fluorica is a known treatment of fibrosis and exoxtosis of the bone. Calcarea fluorica is the chemical union of the lime and fluoric acid. It is a powerful tissue remedy for hard, stony glands, varicose and malnutrition of the bones. There is, however, no report of its use in frontal bone exoxtosis in dogs. The present paper presents the therapeutic use of homeopathic remedy Calcarea fluorica in the treatment of frontal bone exostosis in dogs.

Case History and Clinical Observations:

A Doberman male dog, about 1 year of age was brought to the Teaching Veterinary Clinical Complex (TVCC), College of Veterinary Sciences & Animal Husbandry N.D.U.A. &T. Kumarganj Faizabad with the history of swelling on the head region since last 15 days. Clinical

examinations revealed that swelling is hard to touch, having no pain, of about the size of table tennis ball (Fig.1). Animal was active and alert and all the physiological parameters were within the normal range. The rectal temperature was 101.8°F, respiration rate was 22/min and pulse was 86/minutes, mucus membrane was pink in color and any abnormal ocular and nasal discharges were absent. Feed and water intake was normal. Defecation and urination was also normal. Blood parameters were also within the normal range. Blood examination revealed 12.4gm/dl hemoglobin. Radiography confirmed the exoxtosis of the frontal bones. Calcarea fluorica 30 C (SBL Industries Pvt. Ltd., Haridwar, Uttarakhand) was given @ 1 tab orally BID for 30 days.



Fig.1. Frontal bone exostosis in a dog before the treatment

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Results and Discussions:

Animal responded well to the treatment. The size of the swelling reduced from tennis ball size to the size of the pea in first 30 days of the treatment. The treatment was further continued for next 30 days and complete recovery was observed after 50 days (Fig. 2). No recurrence was noticed after discontinuation of therapy. In the course of the treatment, animal remained active and alert, suggesting no unwanted reaction and absence of toxicity. The drug proved effective and safe in treating frontal bone exostosis in dogs. Trivedi, N., (2012) reported successful treatment of exostosis of mid clavicular region in a girl using calcarea fluorica 3 X. Calcarea fluorica is Schussler's "bone salt". It is found in surface of bones, enamel of the teeth in elastic fibres and in the cells of epidermis. It is principally used for dispersing bony growth, ulceration of the bone and for fistula. It is postulated that a disturbance of the equilibrium of the molecules of calcarea fluorica causes a continued dilation. or chronically relaxed condition of the implicated fibers. If the elastic fibers of any portion of the vessels of the connective tissue or the lymphatic



Fig.2. Dog after the treatment

system have arrived at such a condition of relaxation, the absorption of the solid exudation in such a part cannot take place and thus indurations of the parts set in., seen as a hard knotty exudation on the surface of bone. There are two possibilities as regards the reabsorption of such indurations i.e. a) the elastic fibers near the indurations have lost their functional ability on account of the pressure exerted. Molecules of calcarea fluorica administered, restore their functional integrity and thus are capable to throw off indurations, which will then be reabsorbed by the lymphatic vessels. B) by means of volumetric force carbonic acid contained in the blood as a part of the fluorine is split off the fluoride of lime, this combines with nascent hydrogen forming hydrofluoric acid which gradually dissolves the molecule of morbid product and these are taken by the lymphatic's (Vithoulkas, G., 2011).

Conclusion:

Homeopathy offers a successful treatment protocol for exostosis. This paper presents the therapeutic use of homeopathic remedy *calcarea fluorica* in the treatment of frontal bone exostosis in dogs. Homeopathy therapy is economical and easier in administration. This therapy is also helpful in avoiding the surgical intervention (cranioplasty).

References:

Trivedi, N. (2012). From abnormal to normal with homeopathy - the process of restoration. Homeopathy4 everyone. **9** (8).

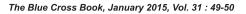
Vithoulkas, G. (2011). Tissue salts by Schussler calcarea fluorica. International Academy of Classical homeopathy, Alonissos, Northern Sporades, Greece.

Molnar, R. E., (2001). Theropod paleopathology: a literature survey: In: Mesozoic Vertebrate Life, editedby Tanke, D. H., and Carpenter, K., Indiana University Press, pp. 337-363.









Acute Theileriosis in a cross bred Cow

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Figure 1: Punched out ulcers and hemorrhages in abomasum.

Theileriosis is a tick-borne protozoan disease in cattle, sheep, and goats as well as in wild and captive ungulates. The genus Theileria belongs to the Amplicomplexa group. The life cycle of Theileria spp. involves cyclical development in ticks to form sporozoites which, on being injected with tick saliva into the mammalian host, develop into schizonts in leukocytes and then piroplasms (merozoites) in erythrocytes. The diseases in ruminants are characterized by high fever and lymphadenopathies and are associated with varying degrees of leukopenia and/or anemia (Radostits, et al., 2000). The disease occurs when there is much tick activity, mainly in summer and the rainy seasons. A single tick can cause fatal infection since its salivary glands usually contain numerous sporozoites. Theileria spp. such as T. annulata, T. Parva, T. mutans, T. hirci, T. orientalis and T. buffeli causes theileriosis in different parts of world. Among these, T. annulata is most prevalent and enzootic in India.

Case History and Observations

A cross bred cow of 5.5 years age was presented with the history of high fever and anorexia. On clinical examination, animal appeared dull, mucus membranes were icteric and eves were watery. Examination of peripheral lymphnodes revealed enlargement of pre-scapular lymph nodes. On auscultation of upper chest area. a loud thumping heart beats were noticed. Hematological examination revealed anemia, neutropenia, and lymphocytosis. Neumerous lymphoblasts containg schizonts (Kochs Blue bodies) and hypochromic RBCs containing merozoites were observed on blood smear examination. These findings were suggestive of Theileriosis. Thrombocytopenia and prolonged prothrombin time have also been reported in cattle infected with T. annulata (Radostits, et al., 2000).

Treatment and Discussion

Animal was treated with Buparvaquone @ 2.5 mg/kg IM, B-complex, Iron Dextran 1 gm and Chlorpheniramine maleate @ 30 mg IM. As the treatment was delayed, the animal continued to show the symptoms and died, even after intensive therapy. At necropsy, lymadenopathy, icterus, punched out ulcers on abomasal mucosa were noticed. High fever, lymphadenopathy, hematological examination and necropsy findings were confirmative of theileriosis. Theileriosis can be acute, sub-acute, or chronic, depending on the resistance of animal and prevalence of ticks and has seasonal occurrence. The acute disease is characterized by fever and very high mortality in 3-6 days. In







sub-acute and chronic cases, signs are generally less marked except for anemia and emaciation. It has been reported that, *T. annulata*, and *T. hirci* produce numerous schizonts and piroplasms and are very pathogenic than other spp. of Theileria (Radostits, *et al.*, 2000). Regular destruction of ecto-parasites is needed to prevent disease.

Vaccine to calves is available and can be given in endemic areas.

References:

Radostits O. M. , Gay C.C., Hinchcliff K. W. , and Constable P. D. 2000. Diseases associated with protozoa. In: Veterinary medicine $(10^{th}$ Edn.), Saunders publication, pp. 1526-30.

Peri-parturient Disorders : A hurdle in Dairy Development



Milk Fever

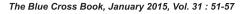
Milk Fever or hypocalcemia is an acute / per acute, afebrile, flaccid paralysis of mature dairy animal (cow, buffalo) that occurs more commonly at or soon after parturition, characterized by weakness, recumbency, ultimately leading to shock and death.

Sudden drop in ionic calcium (Ca⁺⁺) levels in tissue fluids is the basic biochemical event in milk fever. The physiological levels of calcium (8.5 to 10 mg /dl) may drop down to 2-7 mg/dl. Concurrently, serum phosphorus levels may also be decreased. The onset of lactation results in sudden large demand on the calcium homeostasis as colostrum contains 2.3 g of calcium per kg of milk production in a single milking. Thus, a animal giving 10 kg of colostrum losses 23 g of calcium and this quantity is 9 times as much calcium as that present in the entire plasma calcium pool of the animal. This calcium lost from the plasma pool must be replaced by increasing intestinal absorption and bone resorption of calcium. The dry period requirement of calcium (10-12 g/day) increases to 30 g/day at the onset of lactation. If this requirement is not fulfilled, hypocalcemia or milk fever ensues.

Occurrence of milk fever is related to :

- Age : Milk fever occurs most commonly in high producing adult lactating dairy animal. Young ones or heifers are rarely affected. Mature dairy cattle are most commonly affected in 5-10 years age group (3rd lactation and onwards to 7th lactation). Very rarely, hypocalcemia is seen in 1st/2nd lactation.
- **Breeds :** All breeds of Indian dairy animals, including non-descript cattle and buffaloes are susceptible. Among exotic breeds, Jerseys and their cross-breds are more affected.
- **Individual animals :** Complete milking within 1st 48 hours pre-disposes the animal, rather than the animals in which suckling by calf is allowed.
- **Starvation :** Hypocalcemic paresis during a period other than post-parturient period may occur in conditions of starvation, particularly in pregnant animals.





Antibacterial Therapy of Mastitis - A Practical Approach

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Introduction:

The response to the treatment of clinical mastitis in cattle is influenced by a variety of factors, including the effectiveness of the cow's immune response, the severity of mastitis at the onset of treatment, the duration of infection at the onset of treatment, the causative pathogen, and the drug regimens and ancillary measures used.

Antibiotic treatment is most likely to be effective if initiated early in the course of clinical mastitis. Delaying treatment for several days can allow potentially susceptible pathogens, such as *Streptococcus uberis* and *Staphylococcus aureus*, to become well established and evade treatment and host defenses.

The targeted compartment (milk, mammary tissue, blood) should be considered when choosing an antibiotic treatment regimen. In streptococcal mastitis, organisms reside mainly in the milk compartment and intramammary antibiotic therapy is appropriate; there is little benefit to using parenteral antibiotics. On the other hand, cows with severe coliform mastitis are often bacteremic, and parenteral antibiotic administration may improve the outcome.

Intramammary route

The most common route of administration of antimicrobials in mastitis is the intramammary (IMM) route.

• The advantages of this route are high concentrations of antibiotics, achieved in the milk compartment of the mammary gland and

low consumption of the antimicrobial substances as the drug is administered straight to the infection site.

- Disadvantages could be the uneven distribution of many substances throughout the udder, risk for contamination when infusing the drug via the teat canal, and possible irritation of the mammary tissue caused by the drug. In addition, some *in vitro* studies have shown that antibiotics may disturb phagocytosis when given by intramammary route. Numerous intramammary products have appeared in the market but, without supportive/sufficient scientific data on their efficacy.
- The drugs currently available as intramammary preparations are time dependent antimicrobials and hence need to be administered at periodic regular intervals (8-12 hours). Extending the duration of therapy is expected to be more effective than giving a higher dose at each treatment without extending the duration.
- Intramammary preparations with combinations of two or even three antibiotics are introduced in mastitis therapy due to suggested synergistic action and to cover all pathogens, gram-negative bacteria included. However, the fixed combinations have shown no superiority over single components in controlled clinical trials.
- Broad-spectrum intramammaries such as 3rd or 4th generation cephalosporins may enhance emergence of wide-spectrum beta-

lactamase production among bacteria because, they are less efficient than narrow spectrum preparations against gram-positive mastitis pathogens, as they are more targeted towards gram-negative bacteria.

Parenteral route

A novel approach always has been to apply parenteral (systemic) administration as an adjunct to intramammary therapy.

- Systemic use of antimicrobials has been successful for increasing cure rates for chronic *S. aureus* intramammary infections in dry cows and lactating cows, with antimicrobials such as fluorquinolones, macrolides, and tetracyclines which were selected as good pharmacokinetic candidates because of good volume of distribution (lipophilic), relatively long half-life, and high bioavailability (low serum protein binding).
- Because of a high degree of resistance to antimicrobials in commercial intramammary products, systemic antimicrobial therapy for the treatment of acute gram-negative mastitis has been attempted.
- It could be interpreted on the basis of pharmacokinetics, that intramammry route is preferred in subclinical, chronic and mild clinical mastitis, whereas, in acute clinical mastitis with swollen parenchyma and inflamed, blocked milk duct system, parenteral route is preferred together with IMM route.

Antibacterial Agents

The antibacterials approved for bovine mastitis treatment include: pirlimycin, hetacillin, cloxacillin, amoxicillin, novobiocin, penicillin G, dihydrostreptomycin, cephapirin, erythromycin, novobiocin, and sulfa preparations.

Other agents used on extralabel use are : fluoroquinolones, penicillins, aminopenicillins, cephalosporins, chloramphenicol, tetracyclines, aminoglycosides and macrolides.

- Milk antibiotic concentrations after systemic treatment are highest for macrolides, fluoroquinolones and trimethoprim and lowest for beta-lactams (penicillins and cephalosporins) and aminoglycosides (eg: gentamicin, amikacin).
- The use of fluoroquinolones (eg: . ciprofloxacin, moxifloxacin, orbifloxacin, difloxacin or enrofloxacin) and tetracyclines is better avoided in animals that are still nursing young ones as these antibiotics can be harmful to the growing animal
- Macrolides (eg: erythromycin), florfenicol, oxytetracycline, some fluoroquinolones and rifampin have good distribution to the udder following systemic administration.
- Sulfa drugs, penicillin G, ampicillin, ticarcillin and cephalosporins have intermediate or limited distribution following systemic administration.
- Ceftiofur, aminoglycosides, spectinomycin, colistin and polymixin B have poor distribution to the udder on systemic use.
- The dosage of beta- lactam drugs, but not the aminoglycosides may be greatly increased without the fear of toxicity to force a higher blood/mammary concentration gradient.
- Third generation cephalosporins (eg: cefotaxime, ceftriaxone, cefixime, ceftazidime, ceftizoxime,cefoperazone etc) though have high antibacterial activity against gram negative organisms, are less effective than 1st (eg: cephapirin, cefadroxil



etc) and 2nd generation (eg: cefaclor, cefuroxime axetil, cefprozil, cefuroxime etc) cephalosporins against gram positive bacteria.

• Third generation cephalosporins have moderate activity against gram positive bacteria and are inferior in activity against staphylococci thus are not to be preferred as the first choice antimicrobial agents in routine mastitis cases as the continous use of these wide spectrum antibiotics may lead to enhanced development of resistance by the organisms; resulting in failure of therapy.

Cephapirin: A first -generation cephalosporin that has a wide spectrum of activity against gram positive and moderate activity against gram-negative organisms; cephapirin is more resistant to beta-lactamases than are the penicillins and is indicated (300mg/quarter, b.i.d) in the treatment of mastitis caused by susceptible bacteria, such as *Staphylococcus aureus* and *Streptococcus agalactiae*. However, cows with acute or peracute mastitis are often given other medications, such as systemic antibiotics and/or supportive therapy, concurrently with intramammary therapy.

Ceftiofur: Intramuscular administration of ceftiofur, a structural analog of cefotaxime, significantly reduces the bacteremia associated with coliform mastitis, though may not completely eliminate the udder infection.

Cefquinome: 1 mg/kg, IM, q24h (intramammary preparation in India) has proved to be more efficacious compared to ampicillin, amoxicillin-clavulanic acid, tetracycline treatment.

Tilmicosin: is a macrolide closely related to tylosin, is a narrow spectrum (gram positive)

antibiotic, administered intramammarily (300mg) and is effective in staphylococcal and streptococcal infections.

Pirlimycin: a lincosamide antibiotic, active only against gram positive bacteria, is effective in eliminating prepartum infections; not effective aginst coliforms.

Erythromycin is active primarily against gram positive bacteria, such as *Staphylococcus aureus* and *Streptococcus agalactiae*, *Strep*. *dysgalactiae* species, including penicillin resistant ones. Intramammary therapy (300mg/quarter, bid-lactating animals; 600mg/quarter, b.i.d –nonlactating animals) alone is indicated only in the treatment of subacute or subclinical mastitis manifested by mild changes in the milk or udder. Cows with acute or peracute mastitis, should be administered systemic antibiotics and supportive therapy.

Amoxicillin and clavulanic acid (potassium clavulanate) is effective against beta-lactamase producing Escherichia coli, Klebsiella spp., Neisseria spp. The combination has a spectrum of activity similar to that of a first or second generation cephalosporin. Amoxicillinclavulanic acid injection is not compatible with and should not be reconstituted or mixed with dextrose solution or sodium bicarbonate solution for injection and also it should not be mixed with any other medication. Although amoxicillin and clavulanate potasssium may also be effective against nonbeta lactamase producing organisms susceptible to amoxicillin alone, the combination drug should be reserved for use in the treatment of infections caused by or suspected of being caused by beta-lactamase producing organisms when amoxicillin alone would be ineffective.



Therapy Strategies

• The mastitic cows have lower concentrations of antioxidants viz: vitamin E, selenium, vitamin A, vitamin C and the minerals: zinc and copper, which are essential for maintaining and protecting udder health.

- Anti-oxidant supplementation of dietary rations improves anti-bacterial function of neutrophils and decreases incidence and severity of clinical mastitis
- The most effective therapeutic method for treating intramammary infections may be via systemic administration of antibiotic combined with intramammary infusion.
- Combination of multiple intramuscular injections with intramammary infusions over a three-day period results in highest tissue antibiotic concentrations.

Subacute clinical mastitis

- Intramammary infusion with an approved product for a minimum of three days, accompanied by frequent hand stripping to remove secretion and debris, is often adequate.
- Treatments should be continued until at least 24 hours after the disappearance of clinical symptoms.
- Otherwise, the infection may only be suppressed back to the subclinical level.
- A true cure, whereby all infecting microorganisms are eliminated from the affected quarter, occurs in only in 10 to 50% of cases. The cure rate is dependent on how long the infection has been present, age of the cow and type of organism involved.

Acute mastitis

Acute, or systemic, mastitis is most often caused by coliform and other gram-negative organisms. However, numerous other pathogens including gram-positive cocci and mycotic organisms can all result in severe mastitis.

The treatment recommended for coliform mastitis can be as follows.

- Suggested therapeutic regimens for coliform mastitis include antimicrobials, supportive fluids, stripping out of infected quarters, antiinflammatory agents, glucose, bicarbonate, and calcium. However, the efficacy of therapy, particularly for antimicrobials, is unproven
- Antimicrobials such as aminoglycosides and cephalosporins, that have a high proportion of bacterial isolates susceptible *in vitro*, are often selected for use
- Typically, intramammary therapy to inhibit gram-positive growth in addition to parenteral (systemic) antimicrobials that have broad spectrum of activity are administered
- Macrolides such as erythromycin and tilmicosin are not effective against coliform bacteria.
- Penicillin, oxytetracycline (IV), ceftiofur, cephapirin and florfenicol offer some choices, although penicillin and ceftiofur do not penetrate udder tissue well.
- The efficacy of systemic ceftiofur as a treatment of clinical mastitis remains unproven and caution should be exercised in continuing antimicrobial therapy in cows with grossly abnormal milk, but with improved appetite, attitude, and milk production.



- Milking out the affected quarter every 2 -3 hours; oxytocin may be used to facilitate milk evacuation and remove toxic materials and debris; based on the fact that many mild cases of clinical mastitis are self-limiting and that the animals own defense mechanisms can successfully clear the infection. However the oxytocin treated cows may have more relapses and additional infections due to environmental streptococci.
- Corticosteroids are recommended only in peracute toxic cases, but should never be used in other mastitis cases.
- Aqueous dexamethasone sodium sulfate, a single dose one time treatment is recommended and high dose or continued treatment is contraindicated. They suppress the natural defense mechanisms of the cow and administration during the last three months of pregnancy may also induce premature calving, followed by retained placenta and infection of the uterus.

They do, however, aid in reducing swelling and pain and enhance the removal of toxic secretions as well as promote better diffusion of intramammary infusions

- Non-steroidal anti-inflammatory drugs (NSAIDS) such as flunixin meglumine, aspirin, phenylbutazone, meloxicam and ketoprofen may be used to reduce signs of inflammation and endotoxemia.Flunixin meglumine, though the logical drug of choice in most cases.
- Fluid replacement is necessary for those animals showing signs of dehydration. A severely affected cow may require 40 to 60 liters of fluid IV in the first 24 hr. Large volumes of balanced electrolyte solutions are administered intravenously (oral fluids are not absorbed in such cases.) 20 litres in the first

one to two hours, and up to 60 liters over a 12-hour period.

Although this is admittedly difficult and time consuming in a practical situation, convenient methods of fluid therapy administration should be developed. Commercial distilled water can be bought in large economical quantities and mixed with pre-weighed amounts of salt to provide the fluids needed.

- Alternatively, treatment with hypertonic (7.5%) saline solution (4ml/kg) will provide immediate expansion of extracellular fluid volume and temporarily counter some of the effects associated with endotoxemia. Cows should either voluntarily drink or be administered per os 5 to 10 gallons of water following hypersaline use.
- Caution should be exercised in administering hypersaline to cows with marked dehydration (diarrhoea, heat stress) or shock precipitated by causes other than endotoxin.
- If the cow cannot stand, administering 150 to 250 grams of sodium bicarbonate with the first three to 5 liters of electrolyte solution and adding 500 mL of 50% glucose to the first few liters of electrolyte restores vital body fluids, dilutes toxins, and counteracts acidosis.
- Because of the likelihood of clinical or subclinical hypocalcemia and hypokalemia associated with acute coliform mastitis , calcium needs to be administered; more safely administered subcutaneously or diluted in 5-10 litres of IV fluids or or orally and oral administration of potassium chloride in anorexic cattle.
- Dietary vitamin E and selenium reduce the severity and frequency of coliform mastitis. Thus, herd dietary selenium and vitamin E

supplementation, particularly that for dry cows and heifers, should be reviewed periodically

Chronic mastitis

Many intramammary infections that are chronic or are observed as mild clinical cases offer a more voluntary approach to therapy.

- Elimination of infections can result in increased production and, in the case of contagious pathogens, remove the reservoir of infection for non-infected cows. However, many of these infections are of long duration, frequently recur with mild clinical mastitis despite previous therapy, and can add substantial costs and risks associated with treatment
- Given the slow onset of infection, identification of the pathogen should be performed before any extensive therapy is instituted. Drug distribution, although theoretically available in the mammary gland, may not be efficacious because of extensive fibrosis and micro-abscess formation in the gland.
- In herds with a high prevalence of *S. aureus* infections, the emphasis should be placed on teat dipping, culling, milking machine maintenance, milking hygiene, and segregation of infected cows to gradually reduce prevalence of infection.
- Antibiotic treatment may reduce shedding of bacteria by clinical cows, and thus help reduce the spread, but it will not reduce overall prevalence in the herd.
- Heifers calving with mastitis can be dry treated 50 to 60 days with approved dry cow therapy. Heifers can be treated 7 to 14 days prior to calving with one lactating tube per

quarter. The quarters should not be stripped out prior to treatment.

The teats should be sealed with an external teat sealant after treatment.

Another management program that reduces new infections and calms heifers down significantly after calving is to run the heifers through the parlor once a day and dip their teats for approximately 7 days prior to calving.

Failure of therapy

The frequent therapy failures during acute mastitis are due, in part, to poor or uneven distribution of the drug throughout the intensely swollen udder parenchyma in which the duct system is either compressed or blocked by inflammatory products. Other reasons include: lack of contact between bacteria and antibiotics due to scar tissue formation, poor drug diffusion, and inactivation by milk and tissue proteins; microbial resistance to antibiotics; development of bacterial L-forms; metabolically inactive organisms; and improper treatment procedures like stopping the therapy too soon.

Certain mastitis-causing pathogens, such as *Mycoplasma*, *Prototheca*, *Nocardia*, *Pseudomonas*, and yeast are non-responsive or poorly responsive to antibiotics. Antibiotic therapy would be expected to fail and better to be avoided in these confirmed cases. Mildly affected cows with no bacterial growth or a low concentration of *E. coli* in milk also are unlikely to benefit from antibiotic therapy.

On the other hand, resolution of mastitis caused by Streptococcus species is enhanced when intramammary antibiotics are used, and antibiotic therapy should not be avoided in such cases.



Literature cited:

Constable, P. D. and Morin, D.E. (2003). Treatment of clinical mastitis: Using antimicrobial susceptibility profiles for treatment decisions. Vet.Clin.N.Amer.Food Anim. Pract. **19**:139-155.

Dopfer, D., Barkema, H.W. and Lam, T.J. G. M. (1999). Recurrent clinical mastitis caused by Escherichia coli in dairy cows. J.Dairy Sci. **82**:80-85.

Erskine, R.J., Bartlett, P.C. and VanLente, J.L.(2002). Efficacy of systemic ceftiofur as a therapy for severe clinical mastitis in dairy cattle. J Dairy Sci.**85**:2571-2575.

Erskine, R.J., Wagner, S.A. and DeGraves, F.J. (2003). Mastitis therapy and pharmacology. Vet. Clin. N. Amer. Food Anim. Pract. **19**:109-138.

Hess, J. L., Neuder, L.M. and Sears, P.M. (2003).Rethinking clinical mastitis therapy. In: Proceedings Natl. Mastitis Counc. **42**:372-373.

Hillerton, J.E. and Kliem, K.E.(2002). Effective treatment of Streptococcus uberis clinical mastitis to minimize the use of antibiotics. J.Dairy Sci.**85**:1009-1014.

Morin, D.E., Shanks , R.D. and McCoy, G.C.(1998). Comparison of antibiotic administration in conjunction with supportive measures versus supportive measures alone for treatment of dairy cows with clinical mastitis. J. Am. Vet. Med. Assoc. **213**:676-684.

Shpigel, N., Chen, R. and Winkler, M.(1994). Antiinflammatory ketoprofen in the treatment of field cases of bovine mastitis. Res.Vet.Sci.**56**:62-68.

Sol, J., Sampimon, O.C. and Snoep, J.J.(1997). Factors associated with bacteriologic cure during lactation after therapy for subclinical mastitis caused by Staphylococcus aureus. J. Dairy Sci. **80**: 2803-2808.

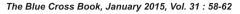
Sol, J., Sampimon, O.C. and Barkema, H.W. (2000). Factors associated with cure after therapy of clinical mastitis caused by Staphylococcus aureus. J. Dairy Sci. **83**:278-284.

Wenz, J.R., Barrington, G.M. and Garry, F.B. (2001). Use of systemic disease signs to assess disease severity in dairy cows with acute coliform mastitis. J.Am.Vet. Med.Assoc. **218**:567-572.

Peri-parturient Disorders : A hurdle in Dairy Development



Though, systematic study of prevalence and economic losses due to milk fever are not available at individual States or National level, one study in Tamil Nadu reports that prevalence of milk fever is to the extent of 13.67% in cows and 11.99% in buffaloes. Considering this as a clue, around 2.65 million cross-bred cows and 6.12 million buffaloes out of total 19.42 million cross-bred cows and 51.05 million buffaloes (19th Livestock Census, 2012) are at risk in India.



Impact of Climate Change and Agricultural Intensification linked to emergence of Zoonosis

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Environmental changes have always been associated with the emergence of new diseases or the arrival of old diseases in new places. India is at a greater risk of infectious diseases associated with extreme weather changes. In the last century, improved nutrition and hygiene and the use of vaccines and antimicrobials reduced the infectious disease burden. However, in recent decades, increasing global travel and trade, expanding human and livestock populations, and changing of climate behaviour have been linked to a rise in disease emergence risk and the potential for pandemics. Interactions at the animal-human interface are increasingly recognized as the source for potential epidemics and the generation of novel pathogens.

Emerging infectious diseases pose a growing threat to human populations. Human pathogens of all taxa contain zoonotic species. Nearly 80% of viruses, 50% of bacteria, 40% of fungi, 70% of protozoa, and 95% of helminthes that infect human beings are zoonotic. Most of the identified reservoirs are mammalian (about 80%) or to a lesser extent. avian species. There are more than 1600 species of human pathogens responsible for transmitting the disease in human being, from that about 1040 pathogen are zoonotic in nature in which 325 spp. of Bacteria, 184 spp. of viruses, 145 spp. of fungi, 54 spp. of protozoa and 341 spp. of Helminths are directly as well as indirectly responsible for transmitting infection in various age of peoples. Although poorly documented zoonoses is a major public health

problem in India, zoonotic diseases assume a great public health importance as approximately 80% population in India lives in close contact with domesticated animals and poultry and there is also an abundance of vectors.

Most of the scientists have found the strong evidence that modern farming practices and intensified systems can be linked to disease emergence and amplification. However, the evidence is not sufficient to judge whether the net effect of intensified agricultural production is more or less propitious to disease emergence and amplification than if it was not used. Expansion of agriculture promotes encroachment into wildlife habitats, leading to ecosystem changes and bringing humans and livestock into closer proximity to wildlife and vectors, and the sylvatic cycles of potential zoonotic pathogens. This greater intensity of interaction creates opportunities for spillover of previously unknown pathogens into livestock or humans and establishment of new transmission cycles. Anthropogenic environmental changes arising from settlement and agriculture include habitat fragmentation, deforestation and replacement of natural vegetation by crops. These modify wildlife population structure and migration and reduce biodiversity by creating environment that favours particular hosts, vectors, and/or pathogens.

The impact of climate change on the emergence and re-emergence of animal diseases has been confirmed in a worldwide study conducted by the World Health Organization for animal



health, (OIE) in its 77th general assembly. The three main animal diseases notified were blue tongue, Rift Valley Fever and West Nile Fever. Climate change alters the boundaries between different species and when these boundaries break down; it becomes possible for pathogen to switch between hosts. Most of the zoonotic parasites display three distinct life cycles; sylvatic, zoonotic, and anthroponotic. In adapting to changed environmental conditions, including reduced non-human population and increased human population, some vectors display conversion from a primarily zoophilic to primarily anthrophyllic orientation. The influence of geoclimatic change on zoonotic disease epidemiology is evident by changes in reservoir and vector dynamics. Climatic variation creates new ecological niches for vectors, hence altering temporal and spatial distribution of disease.

Although history shows the cascade of events leading to emergence of disease is different each time, several factors are known to favour such emergence. There are various anthropogenic factors leading to the emergence, persistence and spread of zoonotic diseases

Human factors:

Travel and tourism:

The ease of transmission of emerging pathogens is facilitated by intercontinental travel. Efficient air and land travel links make disease containment difficult as illustrated by the SARS corona virus outbreak

Trade:

Globalization has seen goods transported via air, rail, sea and land to any, virtually any, destination on the planet. The trade in used tyres provided the breeding ground for mosquitoes adapted to urban environments and small-container breeding (*Aedes aegypti* and *Aedes albopictus*). This allowed the rapid invasion of the Asian tiger mosquito and thus a shift from the historical sylvatic transmission of chikunguniya to cause widespread human epidemics

Pets:

Zoonotic transmission of diseases harboured by companion animals is relating to the close contact with their owners. Human salmonellosis is associated with keeping exotic reptile pets. Petting zoos are popular in urban areas and animal contact has lead to *Escherichia coli* O157:H7 outbreaks

Agricultural practices and Intensification of Livestock Farming

The human population explosion creates increased demand for food and increased land use for agriculture and livestock farming which disrupts natural ecosystems. Dams and canal built for agricultural practices are potentially new breeding sites for mosquito vectors. Intensification of livestock production, especially pigs and poultry, facilitates disease transmission by increasing population size and density. Although effective management and biosecurity measures may mitigate the between-herd spread of zoonotic diseases, such as brucellosis and tuberculosis. Large scale animal husbandry is rising to compensate for consumer demand. The large numbers of animals or multiple species being farmed together facilitate infection across species barriers. Antibiotics used in livestock farming to improve growth and treat infection apply selective pressure leading to the emergence of antimicrobial resistant strains in livestock and poultry. Domestic animals themselves are reservoirs of zoonotic diseases. The first case of pig-farming related methicillin-resistant Staphylococcus aureus (MRSA) was in the Netherlands. These porcine ST398 strains were non-typeable by conventional methods and are

spreading throughout Europe. Farming is now considered a risk factor for Livestock-associated MRSA.

Influenza A virus emergence is linked to poultry farming practices. Influenza A viruses are segmented RNA viruses that evolve constantly by reassortment and mutation to create new strains of varying pathogenicity and host range. They are found in birds, humans, pigs, horses, cats, dogs, and other animals. The expansion of intensive livestock production in the last few decades, particularly for short generation interval species such as poultry and pigs, creates large high density populations in which there is an increased probability of adaptation of an introduced influenza virus and amplification for transmission between farms, to humans, and to wild animals. Farming systems that allow contact between wild and domestic birds and pigs and have large high density populations that facilitate transmission, adaption and amplification are increasing the risk that such a pandemic variant will emerge.

Deforestation and Urban expansion:

Medical anthropologist have identified anthropogenic changes in land use and agriculture, suburbanisation, habitat destruction, increased demand for animal protein, use of bushmeat, live animal transport and domestication of animal around 1000 years ago and exotic pet trade as biggest conduits for zoonotic disease transmission. This transmission maybe bi-directional with molecular typed confirmations observed in bovine and human tuberculosis.

The loss of biodiversity due to deforestation has an impact on the transmission of zoonotic diseases by the "dilution effect". It is reasoned that in areas of high biodiversity, more species sustain vectors and the disease is diluted. If there are fewer species, the burden of disease is higher. New ecological niches are created for certain vectors e.g. water puddles in deforested areas have decreased acidity and are better breeding sites for mosquitoes. Urbanization also attracts foreign settlers. Human migration for employment or asylum has the possibility of importation of new vectors or diseases. In developing countries, these settlements are generally informal with poor infrastructure and create opportunities for rodent and tick borne zoonoses.

Role of wildlife populations:

Biological impoverishment, habitat fragmentation, climate change, increased toxification and the rapid global movement of people and other living organisms have worked synergistically to diminish ecosystem function. This has resulted in unprecedented levels of disease emergence driven by human induced environmental degradation, which poses a threat to survival and health of biodiversity. Opportunities for the emergence of zoonotic diseases depend on the frequency of contacts between wildlife species and humans. Critically endangered wildlife species are at grave risk of extinction by disease outbreaks.

Health experts from Wildlife Conservation Society released a report that lists 12 pathogens that could spread into new regions as a result of climate change, with potential impact to both human and wildlife health and global economies. These 12 pathogens include Avian influenza, Babesiosis, Cholera, Ebola, intestinal and external parasites, Lyme disease, Plague, Red tides, Rift Valley Fever, Sleeping sickness, Tuberculosis and Yellow fever.

Pathogen adaptation and Host susceptibility:

Zoonotic pathogens may acquire novel virulence traits that offer survival advantages. The



causative agent itself, undergoing mechanism at molecular level such as genetic drift and shift enhancing the virulence or acquisition of multidrug resistance. Antibiotic-resistance may emerge due to the selective pressure from antibiotic usage as seen in multi-drug resistant Salmonella spp. Changes in human susceptibility to infections in general are increased with the advent of organ transplantation, immunosuppressive drugs, chemotherapy and the emergence of HIV/ AIDS. Patients with HIV/AIDS have increased coinfections with leishmaniasis. Bacillary angiomatosis, peliosis and cryptosporidium.

Socioeconomic conditions in India and spread of zoonoses:

Depending on social customs, economic status, habits, hobbies and occupation, different sections of society are likely to be exposed to different sets of zoonotic diseases. Although the risk of exposure to zoonoses is equal to all members of society, certain occupational groups are at special risk of contracting zoonoses. In India, agriculture and animal husbandry workers such as farmers, livestock owners, animal handlers, veterinary extension workers and veterinarians have been found to commonly contact approx 40 zoonotic diseases, whereas people engaged in production and processing of livestock products such as personnel working in abattoir, dairy, poultry enterprises and piggery suffer frequently with about 22 zoonotic diseases.

Conclusions

Human population growth and associated changes and increase in demand for food and other commodities are drivers of environmental change, such as urbanization, agricultural expansion and intensification, and habitat alteration. These play an important role in the emergence and re-emergence of infectious diseases by affecting ecological systems at

landscape and community levels, as well as host and pathogen population dynamics. Climate variability interacts with these environmental changes to contribute to disease emergence. Changes in the ecosystem can lead to increased pathogen transmission between hosts or greater contact with new host populations or host species. This occurs against a background of pathogen evolution and selection pressure, leading to emergence of pathogen strains that are adapted to the new conditions. The intensity of the interface between wildlife, humans, and domestic animal species has never been static, and all biological systems have an inherent capacity for both resilience and adaptation, but the current pace of anthropogenic change could be too fast to allow system adaptation and overwhelm resilience.

The likelihood of climate change impacts on various diseases, and vulnerability of human and animal population to them, need to be identified, improving our knowledge base and our ability to respond at local, regional and national levels is important to maintain our current levels of health.

West Nile fever, Chikengunya fever and Lyme disease are excellent examples of how climate change, anthropogenic and natural factors converge to result in the emergence of zoonoses. A paucity of adequate healthcare and collapse of public health measures are common due to poverty and conflict. Cross-border mobility and immigration is another contributing factor in developing countries with poor boundary controls. However, the effects of climate change are predicted to be worse for the developing world where challenging socioeconomic and political environments are exacerbated by a lack of epidemiological studies on zoonotic diseases.

The Centre for Diseases Control (CDC) has outlined 11 priority actions that address research, prediction, training, communication,







preparedness, and prevention to prepare the nation and the world for confronting potential health problems associated with climate change, including identifying populations at greatest risk. The establishment of baseline data in the vulnerable developing countries is imperative to enable further tracking and predictive models. Multidisciplinary approaches and a concerted global effort are necessary to predict and prevent outbreaks and emerging zoonoses.

Literature cited:

CDC Policy on Climate Change and Public Health. Available:http://www.cdc.gov/climatechange/policy. htm Accessed on 19 March 2014

Jonesa, B. A., Graceb, D., Kockc, R., Alonsoa, S., Rushtona, J., Saidb, M.Y., McKeeverc, D., Youngb, J., McDermottb, J. and Udo Pfeiffera, D. (2013). Zoonosis emergence linked to agricultural intensification and environmental change, PNAS **110**(2): 8399-8404 Malik, S.V.S. (2008). Global and national perspective of zoonotic diseases: Impact, concerns and strategies In; National workshop on "Veterinary and medical perspective on Emerging and important zoonotic diseases in India, March 29, Division of Vet Public Health, Indian Veterinary Research Institute, Izatnagar, UP, pp 10-17

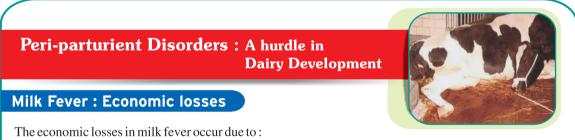
Mills, J.N., Gage, K. L. and Khan, A.S. (2010). Potential Influence of Climate Change on Vector-Borne and Zoonotic Diseases: A Review and Proposed Research Plan, Environmental Health Perspectives **118** (11): 1507-14

Morse SS (1995) Factors in the emergence of infectious diseases. Emerg Infect Dis **1**(1): 715.

Naicker, P.R. (2011). The impact of climate change and other factors on zoonotic diseases, Archives of clinical microbiology,**2** (2): 3823-26

Singh, B.B. Sharma, R. Gill, J.P.S., Aulakh, R.S and Banga, H.S. (2011). Climate change, zoonoses and India, Rev. sci. tech. Off. int. Epiz., **30** (3): 779-788

Taylor L.H., Latham S.M. and Woolhouse M.E.J. (2001). Risk factors for human disease emergence. Phil Trans R Soc Lond B., **356**: 983-89.



• Expenditure on the treatment of affected animal

- Mortality and / or culling of unproductive animal
- Reduction in the quantity of milk produced

A study in Tamil Nadu shows that total economic losses due to milk fever episode in a cow is to the tune of Rs. 1068/- (58% on treatment cost, 32% due to reduction in milk production and about 10% due to mortality and culling) whereas, in buffaloes it is Rs. 665/- (73% treatment cost and 27% due to milk reduction.

Considering the above figures, the national level loss due to milk fever may amount to **Rs. 6900 crores** (Rs. 2830 crores in a milking population of 2.65 million cross-bred cows and Rs. 4070 crores in 6.12 million buffaloes, suffering from milk fever).





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Brucellosis and its zoonotic implications

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Introduction:

Animal and human health is inextricably linked to each other. Although, domestic animals play a significant role in the human lives in terms of providing nutrition, socio-economic development and companionship, still these animals can transmit certain diseases to humans termed as 'Anthropozoonosis'. Brucellosis is an anthropozoonosis of both public health and economic significance in most developing countries (WHO, 2006). According to OIE, it is the second most important zoonotic disease in the world after rabies, causing extensive economic losses. Brucella has been listed by CDC as a possible bio-terrorist agent (CDC, 2002). Brucellosis, also known as "undulant fever", "Mediterranean fever" or "Malta fever", affects people of all age groups and of both sexes. Although there has been great progress in controlling the disease in many countries, there still remain regions where the infection persists in domestic animals and, consequently, transmission to the human population frequently occurs. There are only a few countries in the world that are officially free of the disease although cases still occur in people returning from endemic countries.

Microbiology and virulence : Brucella species are small, nonmotile, gram-negative coccobacilli. Four Brucella spp. can cause infection in humans:

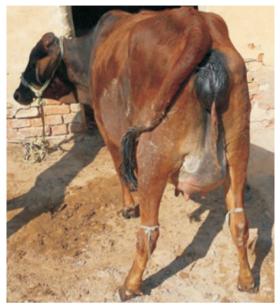
- 1. *Brucella melitensis*, which is found in goats, sheep and camels, is the most widespread and is the most virulent.
- 2. *Brucella abortus*, which is found in cattle and camels, is less virulent
- **3.** *Brucella suis,* which is found in pigs, is also less virulent
- **4.** *Brucella canis,* which is found in dogs, is the least common.

Other animals, including wildlife, may provide a reservoir for brucellae. Brucellosis is transmitted to humans by contact with tissues, blood, urine, vaginal discharges, and especially aborted fetuses the placenta of infected animals and also by ingestion of unpasteurized milk or milk products from infected animals. The disease is predominantly an occupational illness in persons such as farm and workers, veterinarians, livestock slaughterhouse employees, meat inspectors and lab personnel. Isolated cases of infection with Brucella canis also occur in animal handlers from contact with dogs, especially beagles. B. canis is the species of Brucella species that can infect dogs. This species has occasionally been transmitted to humans, but the vast majority of dog infections do not result in human illness. Although veterinarians exposed to blood of infected animals are at risk, pet owners are not considered to be at risk for infection. This is partly because it is

unlikely that they will come in contact with blood, semen, or placenta of the dog. The bacteria may be cleared from the animal within a few days of treatment; however reinfection is common and some animal body fluids may be infectious for weeks. Immunocompromised persons (cancer patients. HIV-infected individuals. or transplantation patients) should not handle dogs known to be infected with B. canis. Infection is transmitted by inoculation through cuts and abrasions in the skin, by inhalation of contaminated aerosols, by contact with the conjunctival mucosa, or by oral ingestion. There is no danger from eating cooked meat products because the diseasecausing bacteria are not normally found in muscle tissue and they are killed by normal cooking temperatures. The disease may be transmitted to humans when slaughtering infected animals or when processing contaminated organs from freshly killed animals. Human to human transmission has been also documented (during sexual intercourse).



Aborted foetus (still birth condition) in a case of brucellosis



Recently aborted cattle, condition showing retention of placenta (found positive for brucellosis)

Disease in animals:

In animals, the main symptoms in all breeds suffering from the four main zoonotic strains previously mentioned are focal necrosis of the placenta, abortion and future infertility. The birth fluids and afterbirth are highly infective, and grazing cattle are infected by ingesting contaminated material from pasture. The disease is not apparent before the heifer aborts. Bulls may also be infected and can sexually transmit the pathogen, until ultimately becoming sterile. Cattle may be infected with any of the zoonotic strains, whereas horses appear to be resistant to all of the known zoonotic strains. A new species of Brucella (tentatively named Brucella maris) has been identified cetaceans (whales, in seals, dolphins and porpoises) off the northern coast of England, in an otter from the south-western coasts of England, and in a bottle-nosed dolphin from California. It is unknown yet if this strain is zoonotic.



Disease in humans :

Human disease presents with lymph node swelling, enlargement of the spleen, fever, testicular swelling, influenza-like symptoms, and lethargy, nausea and weight loss. Endocarditis or meningitis may follow, sometimes with fatal results. There is also a chronic undulant form that has been often seen in people who work with cows and veterinary surgeons. Periodic bouts of high fever and clinical symptoms are interspersed with periods of remission with no clinical signs. This can persist for years or decades. The use of



Hygroma of the knee joint in case of human brucellosis

antibiotics quickly resolves most clinical cases; however, prolonged therapy may be necessary in refractory cases. A septicaemic form is also occasionally seen. There is a evidence that this is caused by the inhalation of infected aerosols in abattoirs and meat-processing plants where infected animals or their tissues are processed. It is characterized by an acute systemic disease with high fever.



Orchitis condition in case of human brucellosis



Patient suffering with brucellosis, showing unilateral swelling on ankle

Diagnosis:

Diagnosis follows RBPT, STAT, MRT, blood culture or using polymerase chain reaction (PCR) testing. The bacteria are relatively slow growing and successful culture in laboratories may prove difficult. Complement fixation is also now used indiagnosis.

Treatment

Treatment relies on the use of antimicrobials. usually in combination to prevent resistance. The British National Formulary (BNF) and the WHO recommend the use of doxycycline plus rifampicin or streptomycin. In the past, cotrimoxazole was often used. Associated toxicity has led to its replacement by more suitable agents. Therapy is usually prolonged; the WHO recommends 6 weeks as a minimum duration. The BNF recommends rifampicin 600 mg to 1.2 g daily in two to four divided doses with doxy-cycline 100–200 mg/day; the WHO recommendation is similar: rifampicin600–900 mg/day plus doxycycline 200 mg. In severe cases, streptomycin may be used in place of or in addition to rifampicin. Longer-term therapy may be required in the undulant form of the disease. Quinolones in combination with rifampicin have undergone trials and been demonstrated to be as effective. Currently no effective vaccine for human brucellosis is available.

Prevention

Suitable protective clothing will reduce the risk from occupational exposure. The use of disinfectants, especially chlorinated or iodine- or ammonia-based products, can prevent environmental hazards. The mainstay of prevention is eradication by animal vaccination or slaughter programmes. On a personal basis, travelers to areas where the disease is endemic should be encouraged to avoid unpasteurized dairy products and undercooked meat. Following the discovery of sea mammals infected with a variety of Brucella, which is not currently known to be zoonotic, the following advice has been offered to them: People who handle or work with seals or small cetaceans are advised to take suitable precautions to avoid any risk of infection although the new species of Brucella is not known to present any risk to human or animal health. In domesticated animals, once Brucella is detected, often during routine testing and carcass screening, any infected beasts shall be culled, and herds to be subject to a strict testing regime. Wild animals, such as deer that maybe infected in the local area, will also be monitored post mortem.

Literature cited

Anon. (2000).Brucellosis in Control of communicable diseases manual. American Public Health Association, Washington. **17**:75-78.

Apan, T. Z.; Yldrm, M. and Istanbulluoglu, E. (2007).Seroprevalence of brucellosis in human, sheep and cattle populations in Krkkale (Turkey).Turkish Journal of Veterinary and Animal Sciences.**31**(1):75-78.

Cadmus, S. I. B.; Ijagbone, I. F.; Oputa,H. E.; Adesokan, H. K. and Stack , J. A. (2006). Serological survey of brucellosis in livestock animals and workers in Ibadan, Nigeria. African Journal of Biomedical Research.**9**(3):163-168.

Centers for Disease Control (2002).Public Health Fact Sheet–Brucellosis, Massachusetts, USA.

Dhand, N. K.; Gumber, S.; Singh, B. B.; Aradhanabal, M.S.; kumar, H.; Sharma, D. R.; Singh, J. and Sandhu, K.S. (2005). A study on the epidemiology of brucellosis in punjab(india) using survey toolbox. Revue ScientifiqueEt Technique De L Office International Des Epizooties. **24**(3):879-885.

Hesterberg, U. W.; Bagnall, R.; Perrett, K.; Bosch, B.; Horner, R. and Gummow, B. (2008). A serological prevalence survey of Brucellaabortus in cattle of rural communities in the province of KwaZulu-Natal, South Africa.Journal of the South African Veterinary Association.**79**(1):15-18.

W.H.O. (1971). Technical report series on brucellosis. 464.

W.H.O. (2006).Brucellosis in humans and animals.WHO/ CDS/ EPR/ 2006. 7. Geneva.



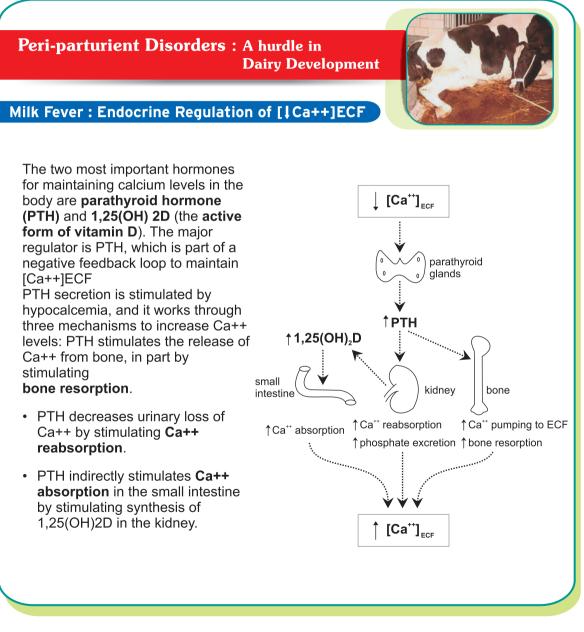






Araj, G. F. (1999). Human brucellosis: a classical infectious disease with persistent diagnostic challenges. Clinical and Laboratory Science.**12**:207-212.

Young, E. J.; (2005).Brucella species. In: Mandell, Douglas and Bennett's Principles and Practice of Infectious Diseases. Elsevier, Churchill, Livingstone, Philadelphia, USA. 2669–2674.







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Toxocara vitulorum : an enemy of dairy calves in early stage of life

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Introduction

The parasitic infections are severe constraints in livestock productivity and cause considerable economic losses, particularly in rural areas where farmers are completely ignorant about losses caused by the parasites. The youngest groups of cattle and buffalo suffer mostly from the large intestinal round worms. *Toxocara vitulorum* is one important round worm, which is also responsible for mortality in growing calves.

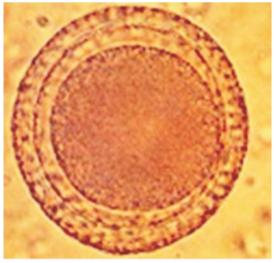
Toxocara vitulorum is a large round worm that infects cattle, buffalo, bision and other bovids. It is also called *Neoascaris vitulorum*. It is found world wide ,including Europe but it is more abundant in region, with humid tropical and subtropical climate as in Asia and Africa. In endemic region up to 100% of the calves can be infected. The disease caused by *Toxocara vitulorum* is called toxocariasis or toxocariosis.

Etiology

Adult *Toxocara vitulorum* are the largest worms that infect cattle, they can be up to 40 cm long and 7mm thick and females are larger than males. They are whitish color, translucent, and look very much like cooked spaghetti. The eggs are almost spherical, about 70x80 micrometer, contain a single cell with a thick and pitted membrane. Final location of *Toxocara vitulorum* is the small intestine, migration larvae can be found in numerous organs like lungs, trachea, bronchi, liver, kidney and mammary glands.

Life Cycle

The life cycle of *Toxocara vitulorum* is relatively short and involves visceral larvae migrans. The worms are found in small intestine. Larvae hatching from ingested egg pass to the tissue and in pregnant cows are mobilized late in pregnancy and pass via the milk to calves from 3 week of age and are easily recognized by their pitted shells. Tissue migration of larvae occur in



Toxocara egg

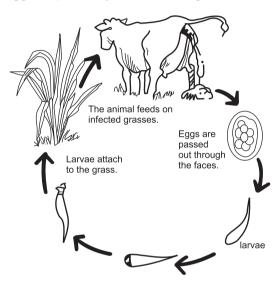
the liver and some time in the brain. An infected calf can shed up to 8 million egg daily. Larvae development inside the egg take about 7 to 15 days at 27 to 30° c which is the ideal temperature. The eggs then become infective and contaminate the pasture. They can survive for months and possibly for years, but are sensitive to light. Maximum egg output occurs in calves up to 3 months of age.



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Epidimiology

Prevalence of ascariasis is more common in buffalo calves as compared to cow calves (under 1 year of age). *Toxocara vitulorum* larvae are present in greatest numbers in the colostrum 2-5 days after calving and few are present after days 9. Mature worms are present in the intestine of the calves by 10th day of age and eggs are passed by 3rd week. Indigenous calves



Life Cycle of Toxocara

are highly susceptible due to difference in climate condition and management practices. In Chhattisgarh state, the disease is more prevalent in buffalo calves after postmansoon season (Sharma et al; 2005). Exposure to cold weather and habits of calves of licking earth due to mineral deficiency, seems to aggravate the condition. High rate of infection was recorded during rainy season, when temperature and relative humidity were high, while, it was low during winter months.

Notably, temperature and moisture or rainfall are required for rapid embryonation and

development of egg to the infective stage(larvaeted egg). Both, the higher temperature and humidity favor early and higher percentage of eggs to develop to the infective stage (Devi *et.al.* 1999)

Pathogenesis

Migration of larvae through the liver results in haemorrhage and fibrosis. In heavy infestation, diffuse fibrosis may occur. The most serious damage occurs in the lungs, where the larvae provoke alveolar injury with edema and consolidation.

Clinical findings

Clinical signs associated with the parasite are diarrhoea and anemia, which is macrocytic, hypochromic and most frequently follows acute blood loss. The blood loss is due to hemorrhages caused by migrating *Toxocara vitulorum* larvae through lung parenchyma or due to suppression of erythropoitic activity of bone marrow, caused by some parasitic toxin (udall, 1954) released in ascariasis.

Toxocara vitulorum is usually non pathogenic in adult cattle, but it can be harmful to calves in tropical and subtropical region with high mortality rates, if left untreated.

Migrating larvae can seriously damage numerous organs in adult cattle, particularly lungs, where they can induce secondary bacterial infections.

In calves, adult worms in small intestine compete for nutrition with the host and cause diarrhea, (often putrid), colic, enteritis, loss of appetite and weight loss. The large size of worm in massive infection can obstruct gut and even perforate it. Occasionally, worms may also migrate through the bile duct and obstruct it causing cholangitis.



Treatment and Control

Various broad spectrum anthelmintics like Fenbendazole, albendazole, levamisole, ivermectin, moxidectin, doramectin, eprinomectin, piperazine etc; can be used, but not all of them are effective against both immature & adult worms. Pyrantel @ 250 mg for a calf have efficacy against both larvae and mature worms of Toxocara vitulorum. Since calves are most susceptible to these worms and infections are acquired prenatally from infected dams, it is essential to prevent the infection in pregnant cows. Since most eggs are shed by young calves, pasture occupied by these calves may often be highly contaminated. In areas with a history of Toxocara vitulorum, prevention through manure removal & disinfection of the calf boxes is recommended. Toxocara vitulorum being cattle specific, alternate grazing with sheep and or horse may be considered. The longer the absence of cattle, the higher will be the reduction of the Toxocara vitulorum population in the pasture. Emphasis must be placed on preventing the environment from becoming contaminated.

Literature cited-

Devi, H. U., Ansar I, M. Z., Singh, S. K., and Kumar, A., (1999) - clinicohaematological studies in cow and buffalo calves with natural infection of ToxocaraVitulorum J. Vet. parasitol 14(2):155-157.

Jones, T. C. and Hunt, D. C. (1983)- Veterinary Pathology 5 edn. Lea and Febiger. BailliereTindall, London.

Rajkhowa, S., and Hazarika, G.C., (2001)-Prevalence of intestinal nematode in female calves of greater Guwahati of Assam, Indian Vet. J 78 (5): 449-451.

Sharma, K., Roy., N. and Sharma, M., (2005) Prevalence of Bovine and Bubaline Toxocariosis in Chhattishgarh . IntasPolivet vol, 6 No. 11 : 274 277

Soulsby, EJL., (1986) - Helminth, Arthropod and protozoa of domestic animals. 6thedn. Bailliere, Tindall and Cassell, London.

Udall, D.H. (1954) The practice of Veterinary Medicine 6thedn. Oxford and IBH publishing Co. New Delhi.

Kumar, M., Banerjee, P. S., and Singh, H., (2002) Incidence of different helminthic infection in tarai and Western plains of U.P. Indian Vet. J. 26 (6) : 105-108

Peri-parturient Disorders : A hurdle in Dairy Development



Milk Fever : Clinical findings

There are three discernible stages of parturient paresis

Stage 1 : Animal ambulatory, but shows signs of hypersensitivity and excitability, mild ataxia, fine tremors over the flank & triceps, ear twitching & head bobbing may be noticed. Restlessness with shuffling of hind feet and bellowing may also occur. If untreated at this stage, next stage ensues.

Stage 2 : Unable to stand, but maintains sternal recumbency, dry muzzle, anorectic, subnormal body temperature and cold extremities are the prominent signs. Weak peripheral pulse, tachycardia and decreased intensity of heart sounds. Smooth muscles paralysis induces GI stasis, bloat, defecation failure and lose of anal sphincter tone. Animal assumes typical "kink in the neck" posture

Stage 3 : Coma. Unable to maintain sternal recumbency, complete muscle flaccidity, unresponsive to stimuli, severe bloat, peripheral pulse undetectable. Death within few hours if untreated









HACCP Apply In Milk Plant

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(received 5/4/2014 - accepted 24/11/2014)

Most dairy products have an excellent safety record, due to well-controlled processing conditions. The main potential hazards are microbiological. Pasteurization, however, has proved to be successful as a CCP to control classical zoonoses as well as newer food borne pathogens.

Introduction

The dairy industry throughout the world has recognized the importance of Hazard Analysis and Critical Control Point system in controlling hazards in dairy products, thereby, not only increasing the safety of consumers but winning their confidence as well as of HACCP system, which is scientific systematic and identifies a specific hazard throughout the food chain.

The Hazard Analysis Critical Control Point (HACCP) program is a system which identifies and controls the critical steps in producing safe and wholesome dairy products. The ultimate goal of a HACCP program in the dairy plant is to eliminate all public health risks.

Principles of HACCP

- 1. Identification of potential hazards
- 2. Identification of critical control points
- 3. Establish target levels and tolerances
- 4. Establish monitoring system
- 5. Establish corrective action
- 6. Verification
- 7. Establish documentation

What Is Hazard

Hazard is any aspect of the production chain that is unacceptable because it is a potential cause of harm. It may be due to any of the following reasons:-

- 1. Unacceptable presence of biological, chemical or physical contaminants in raw materials, semi-finished or finished products.
- 2. Unacceptable growth or survival of pathogenic micro-organisms and the release of toxic metabolites in semi- finished or finished products.
- 3. Unacceptable recontamination of semifinished or finished products with microorganisms and residual chemicals.

Critical Control Points (CCP)

Critical control points in food production starting from the raw state through processing to consumption by consumer at which the potential hazard can be controlled or eliminated.

HACCP Apply In Milk Plant

Dairy Plant Universal Control Points

1. Equipment Inspection : CIP Effectiveness:

After pasteurization, proper cleaning of equipment is the most critical process in ensuring a finished product, free from contamination. As such, an adequate in-house inspection program must be implemented to



ensure that all equipments involved in the processing, storage, and packaging of dairy products are properly cleaned.

2. General Environment : Sanitary Condition:

A contaminated environment increases the likelihood of finished product contamination. Non-essential personnel and the public must not be allowed to access the production areas. Essential personnel must change their outer wear, including shoes prior to entering the production area. Further, condensation, insects and rodents, plugged drains, personnel practices, dust, are other examples of potential sources of environmental contamination. As such, a regular cleaning and inspection program of the general environment must be implemented in any dairy processing plant. As well, the general design of the dairy plant must be examined with respect to design faults that may lead to contamination. Examples of poor design include mezzanines, stairs, ladders, case conveyors, or dripping overhead lines being over or adjacent to exposed product. Special preventative measures must be implemented if design flaws exist in the dairy plant.

3. Personnel Practices:

Dairy plant personnel are potential carriers of pathogenic organisms. As a result, every effort must be done to ensure that hands are adequately washed and sanitized prior to touching product contact surfaces or postpasteurization ingredients. Workers suspected or known to be carriers of a communicable disease must be excluded until receiving medical clearance. Good work habits are as well essential in preventing product contamination. For instance, careless use of high-pressure hoses can lead to aerosols or splash that can contaminate equipment and product. Inappropriate handling of chemicals, raw milk, and post-pasteurization ingredients could lead to product contamination. The plant and equipment must be designed to minimize poor practices, while encouraging good practices. As well, personnel must be adequately trained to ensure that procedures for equipment and product handling, clean up, and personnel hygiene are properly performed.

4. Pest Control:

Insects or rodents present in the dairy plant can lead to contamination of finished products. This can be caused by deposition of pest feces, transfer of raw product to pasteurized product via the pests, etc. As such, an adequate pest control program must be in place not only for eliminating pests from the dairy plant, but also for excluding them as well.

5. Cross-Connection Control:

Cross-connections have been known to cause food poisoning outbreaks in dairy plants. Valves, check valves, etc. cannot be relied upon to separate pasteurized from raw product or CIP systems. Because of the extreme vulnerability of pasteurized product, an on going program must be in place to ensure no cross connections exist in the dairy plant.

6. Ingredients Listing - Allergen Control:

A significant proportion of the general population is allergic to a wide variety of ingredients and products. In some instances, consumption of even minute amounts of allergenic substance can result in the death of the individual. As such, all ingredients used must be listed on the package label in accordance with legislation.





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Extrapulmonary Tuberculosis

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Introduction

Tuberculosis is the major public health problem affecting almost 2-3 million human population annually in India. According to World Health Organisation (WHO) statistics, India is rated as highest Tuberculosis burden country accounting for more than 1/5 of the global incidence, out of 9.4million cases of tuberculosis worldwide.

More multidrug resistant strains of Tuberculous bacilli have emerged i.e. extremely drug resistant and totally drug resistant cases are more in India, rather reported cases accounted in WHO. In India, the microbial pathogens, like Mycobacterium tuberculosis and Mycobacterium bovis both involved in classical and extrapulmonary forms of TB, are not confined to their susceptible host i.e. in bovine or human population. They cross the species barrier as they rely on same habitat conditions. Mycobacterium bovis is more associated with HIV positive patients rather than Mycobacterium tuberculosis (Pitchenik et al., 1984). The disease caused by Mycobacterium *bovis* is indistinguishable to that caused by Mycobacterium tuberculosis (Collen and Granze, 1983). Both organisms are genetically and culturally indistinguishable and M. bovis seems to predominate in Extrapulmonary form of tuberculosis (Grang, 1994; Cosivi et al., 1998).

Clinically, Tuberculosis has been manifested in two forms, pulmonary and extrapulmonary form of tuberculosis (EPTB) of which the former being the commonest form (WHO). EPTB means the organs other than lungs are affected, such as lymph nodes, pleura, abdominal organs like liver, kidney, genitourinary tract and other organs like skin, joints bones, brain etc. EPTB cases are increasing at alarming rates in India accounting for 15 - 20% of infections seen in immunocompetent patients, while 50% accounted for HIV positive patients. The infection is usually manifested to occur in presence or absence of pulmonary disease.

Transmission

The classical form of disease is transmitted through respiratory tract only.

EPTB (Non- Pulmonary)forms seen are cervical lymphadenopathy, intestinal and lupus vulgaris are commonly seen in the Agricultural workers. The incidence of non-pulmonary form of tuberculosis caused by *M. bovis* is little known due to limited laboratory facilities (Cosivi et al., 1998). The major route of transmission of EPTB is swallowing of contaminated food or sputum followed by haematogenous spread, extension from adjacent affected organs and inhalation.

Common forms of EPTB

- 1. Lymph node Tuberculosis
- 2. Pleural Tuberculosis
- 3. Abdominal Tuberculosis
- 4. Pericardial Tuberculosis
- 5. Neurological Tuberculosis
- 6. Skeletal Tuberculosis



- 7. Genitourinary Tuberculosis
- 8. Cutaneous Tuberculosis
- 9. Ocular tuberculosis
- 10. Breast tuberculosis
- 11. Disseminated/Milliary Tuberculosis

Common Symptoms

Patients with EPTB generally suffer from fever, anorexia, weight loss, malaise, extreme night sweats and fatigue. If the effected organ resides in an inaccessible areas, the case may be presented with pyrexia of unknown origin(PUO) and the symptoms and signs are related to the organ system involved.

- 1. Lymph node tuberculosis : Synonyms are King's Evil and Scrofula, This is the most common form of EPTB seen in India and other developing countries (Dandapat, *et al.*,1990). Children and young adults are mostly affected with enlargement of cervical lymphnode followed by auxillary and inguinal lymph nodes.
- 2. Pleural Tuberculosis : Pleural effusion is categorized under EPTB despite close association with lungs and pleura (Ferrer, 1997). It is characterized by chest pain, nonproductive cough and dyspnea. The empyema condition only arises, when there is presence of caseous material in pleural spaces.
- 3. Abdominal Tuberculosis : Mostly Mycobacterium tuberculosis involves more in Gastrointestinal Tuberculosis rather than Mycobacterium bovis, due to wide spread pasteurization of milk. Tuberculosis of gastrointestinal tract, omentum, messentric lymph nodes and other organs like liver, spleen and pancreas, often

associated with disseminated or milliary tuberculosis that commonly occurs in immunocompromised patients. Gatrointestinal tuberculosis as chronic form shows abdominal pain, diarrhea, anorexia, weight loss and mild fever, sensation of moving lump in the abdomen, melena, nausea and vomiting. Peritoneal tuberculosis mostly seen in the persons undergone emergency surgeries. It occurs as chronic disease in different forms, i.e. Ascitic form and Encysted or localized fibrous forms. The constitutional symptoms seen are like fever, anorexia and abdominal distension, with classic tenderness due to ascitis or by partial obstruction.

- **4. Neurological Tuberculosis:** It is categorized into clinicopathological categories, i.e. Tuberculous Meningitis, Tuberculoma and Arachnoiditis
 - a) Tuberculous meningitis is mostly caused by *Mycobacterium tuberculosis* and non-tuberculous mycobacteria (NTB). This is mostly seen in the young children of 1-3 years of age and characterized by irritability, behavioural changes due to onset of meningitis. The important sequelae is complete or partial loss of vision. Neurological tuberculosis is five times more frequent in HIV+ patients than HIV -ve patients (Berenguer *et al.*, 1992).
 - b) Tuberculoma are firm, avascular, spherical and granulomatous masses measuring about 2-8 cms in diameter. These masses consists of necrotic masses composed of caseous material, thick and purulent, where the bacilli can be demonstrated. The intracranial tuberculomas can occur at any age and they may be single or multiple, common in frontal and parietal lobes



(Ravindra, 1999). The disease is characterized by general neurological s y m p t o m s a l o n g w i t h continuous severe headache and neck rigidity.

- c) Arachnoiditis is a pain disorder caused by the inflammation of the arachnoid. one of the membranes that surround and protect the nerves arising from the spinal cord. The arachnoid can become inflamed due to irritation from chemicals and infection. Symptoms are numbness, tingling and a characteristic stinging and burning pain in the lower back or legs. Some people with arachnoiditis will have debilitating muscle cramps, twitches, or spasms. It may also affect bladder, bowel, and sexual function (National Institute of Neurological Disorder and stroke, information).
- 5. Pericardial tuberculosis : The Involvement of pericardium may result in acute pericarditis, chronic pericardial effusion and cardiac tamponade, mostly seen in the persons with pulmonary tuberculosis. The disease is characterized by high fever, chest pain, orthroponea and ankle oedema seen in 40% to 70% of patients (Fowler, 1991).

6. Skeletal Tuberculosis

It is a kind of tuberculous arthritis of the intervertebral joints. Scientifically, it is called tuberculous spondylitis and commonly localized in the thoracic and lumbar portion of the spine followed by hip, foot and elbow joints (Bhan and Nag 2001).The disease results from haematogenous spread of tuberculosis from other sites. The infection spreads from two adjacent vertebrae into the adjoining intervertebral

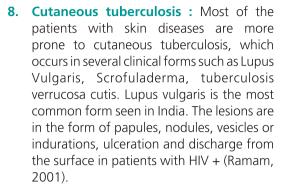


Fig. 1 Skeletal Tuberculosis

disc space. If only one vertebra is affected, the disc is normal, but if two are involved, the disc, which is avascular, cannot receive nutrients and collapses. The disc tissue dies and is broken down by caseation, leading to vertebral narrowing and eventually to vertebral collapse i.e Kyphosis and spinal damage. The disease is characterized by constitutional symptoms along with spinal mass, sometimes associated with numbness, paraesthesia, or muscle weakness of the legs. The vertebral body becomes soft, easily get compressed and produces anterior wedging of the thoracic spine, leading to kyphotic curve called pott's paraplegia, a serious complication of spinal tuberculosis. This complication is reported very high, as 30% of spinal tuberculosis(Malaviya and Kotwal, 2003).

7. Genitourinary Tuberculosis : Generally occurs in patients already affected with pulmonary tuberculosis, mostly transmitted through the haematogenous route, from active infective sites(Hemal, 2001). The symptoms are characterized by dysuria, haematuria without pain, renal mass, sterile pyuria and recurrent urinary tract infection. It is the important cause of infertility (Kumar, 2001).





- **9.** Ocular tuberculosis : In this type, the part of eye, Choroid, is the most commonly affected. Primary ocular tuberculosis is extremely rare condition that arises in patients with primary TB infections (Bouza *et al.*, 1997).
- 10. Breast Tuberculosis : It is usually caused by *M. tuberculosis* and entry of infection is through skin abrasions on the nipple or through duct openings in the nipple. Commonly affects the reproductive age group individuals between 21-30 years in females (Shinde *et al.*, 1995). The disease is characterized by hard lump situated in the central or upper outer quadrants (Jalai *et al.*, 2005). The lesion may progress to tuberculous ulcer over breast skin and abscess with or without discharges.
- **11. Dessiminated/Miliary tuberculosis :** It is a form of tuberculosis involving two or more non - contagious sites. This form is due to dissemination of small numbers of tubercles into the blood circulation during the primary infection or reactivation of latent focus spreading to the visceral sites, which are rich in vascular supplies, such as liver, spleen, bone marrow and the brain(Hill *et al.*, 1991).The diagnosis is based on the symptoms like fever, inanition, cough, dyspnea and

organomegaly. The condition exhibited will be dependent on the organ affects i.e. Hepatomegaly and icterus for liver and meningitis, tuberculomas for brain.

12. Other forms of Tuberculosis

Laryngeal tuberculosis is seen in the endemic areas and occurs as secondary infection to pulmonary tuberculosis, a effected person shows hoarseness of voice (Soni and Chetterjee, 1978).**Tuberculosis** of ear is usually seen when organism attacks the Eustachian tube usually occurs through haematogenous route and accompanied with pale granulation on tympanic membrane with perforations on it (Sachdeva et al., 1978).

Diagnosis

Definitive diagnosis of tuberculosis involves the demonstration of *Mycobacterium tuberculosis*



Fig. 2 Tuberculous bacilli

by using Ziehl Neelson stain by processing the various clinical samples. The demonstration of bacilli in deep seated tissues and body fluids is very difficult, so the clinicians will rely more on radiological and endoscopic approach to diagnose extrapulmonary tuberculosis. Specimens or samples collected on the basis of organs affected like lymph node, cerebrospinal fluid, ascitic fluid etc. and various clinical specimens like sputum, urine, faeces, blood and tissue biopsies etc. should be transported as soon as possible, keeping in refrigerated



condition not more than over night and collected in sterile wide mouthed jars. Specimens like stools and blood from persons affected with AIDS will be collected and processed by employing Isolator Lysis Centrifugation system and directly inoculated into BACTEC13A medium for early detection of Mycobacterium sps. Tissue and other body fluids like Cerebrospinal fluid (2-3ml), exudates (3-5ml) and synovial and pericardial fluid (10 15ml) will be subjected for staining methods by using Ziehl neelson and kinyoun stain. TheAuramin phenol and Auramin rhodamine flouruchrome stains are used to visualize reddish yellow fluorescent bacilli under fluorescent microscopy.

2. Solid and Liquid medias

By using traditional media, the bacilli need long incubation periods for growth but now, growth detection of bacteria is shortened by use of automated or semiautomated liquid culture system, where fully automated non-radiometric detection of growth developed in MB/BacT, BACTEC 9000 and mycobacterium growth indicator tube(MGIT)(Becton, Dickinson). These systems measure the changes in gas pressure, CO_2 production and oxygen consumption either fluorometrically or colorimetrically. The level of flourescense corresponds to the amount of oxygen consumption by the organisms and when certain level of flourescense is reached, the instrument indicates the tube is positive. The mean time of detection of these is minimum 9 days, while traditional media takes 21 -25 days (Drobniewski et al., 2003).

Conventional medias like Egg based media, Lowenstein Jensen media, Gruft modiication of LJ media, Petragnani and American thoraic society media are the traditional media used for isolation of *Mycobacterium spp*.

Colony morphology

The Colonies on conventional media are either smooth and soft or rough and friable exhibited by Mycobacterium spp. The colonies of *M. tuberculosis* are often exhibit patterned texture referred to as cording phenomenon. Basic biochemical tests conducted in the laboratories for preliminary identification of *Mycobacterium tuberculosis* and *M. bovis*.

Biochemical properties	M. tuberculosis	M. bovis
Nitrate reduction	+	-
Niacin production	+	-
Catalase	+	-
Colony identification on egg based medias	Thin, flat, spreading and friable with rough, irregular and wrinkled appearance, buff coloured	Small, granular, rounded white colonies with irregular margins after 21 days of growth.
Emulsification of colony	Difficult	Easy
Growth characteristics with 0.5%glycerol.	Grows luxuriantly(eugonic)	Grows sparsely(dysgonic)

Biochemical Identification

Serum based media like Serum Albumin agar media (middle brook 7H10 & 7H11) are used for isolation of isoniazid resistant strain of *Mycobacterium tuberculosis*. Liquid media like Middle brook 7H9 broth are used for maintenance of stock cultures and the primary isolation of bacilli is done on middlebrook 7H11 media, where the microcolonies are detected by use of conventional microscopes within 9 days (Idigoras et al., 1995).

3. The precise Anatomical localisation of lesions in EPTB, is done by using ultrasound scaning Computed Tomography scan, MRI, endoscopy, colposcopy cystoscopy. are used.

4. Tuberculin Skin test is highly used in most of the endemic areas of India, Positive reaction is not sufficient to diagnose infection. Percentage of positive reaction of TST in different forms of Tuberculosis is given below (Sharma and Mohan, 2004).

Lymph node TB	74-98%
Pleural effusion	73-93%
Abdominal TB	58 100%
Pericardial TB	75 200%
Cutaneous TB	67%
Disseminated TB	21 62%

5. Radiologically guided, fine needle aspiration cytopathology is useful for excision biopsy of accessible peripheral lymph node. In pleural tuberculosis, intra thoracic lymph adenopathy condition can be surgically operated by Video Assisted Thoracoscopic Surgery.(VATS).

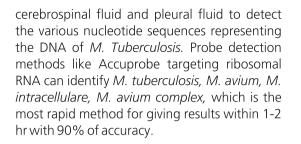
6. In diagnosis of miliary tuberculosis/ Dessiminated tuberculosis, various invasive methods are employed to ascertain the person suffering from dessiminated form of tuberculosis. More than one tests are performed for confirmatory diagnosis like sputum examination, bronchoscopy, cerebrospinal fluid examination, liver biopsy, lymph node biopsy etc. Ultrasonography and computerized tomography is useful for diagnosis of encysted and multiloculated pleural effusions. Contrast enhanced (CF) CT scan is useful for detection of pulmonary and pleural affections of tuberculosis. Abdominal CT scan is useful for detecting high density ascitis and also granulomas or abscesses in the liver, pancreas and spleen. Crodolinium enhanced MRI is superior to CT scan in detection of small tuberculomas. Later, contrast Enhanced CT scan is useful in detection of diffuse and local Meningeal Granulomatous lesions. The spinal tuberculosis MRI is widely used.

7. Immunodiagnostic Technique like ELISA are conducted for neurological and pleural tuberculosis in the body fluids(Samuel et al., 1984)

8. Nonconventional methods like the enzyme like Adenosine deaminase (ADA) of purine metabolism are found more useful in the lymphoid tissues of humans. The estimation of high levels of ADA found to be useful in the diagnosis of TB of pleural effusions and ascitis. Another enzyme, Lysozyme is found helpful in identifying the pleural tuberculosis. (Aggarwal et al., 1999). High levels of interferon(IFN-ã), a cytokine produced by activated T-lymphocytes, have been reported more in pleural effusions (Sharma et al., 2002). The latest Chromographic technique, High performance Liquid Chromatography, is used for detection of tuberculostearic acid in cerebrospinal fluid for detection of tuberculous meningitis condition (Muranishi etal., 1990)

9. Molecular Methods like Polymerase Chain Reaction (PCR) are often applied to the





In latest diagnostic technique, the disease causing bacterium i.e. *M. tuberculosis* can be detected within 12 minutes by adopting Surface

Plasmon Resonance, this is a Malhotra's sensor which detects the DNA of *M. tuberculosis* when the infected person sera is passed through the sensors, the DNA binds to complementary nucleotide sequences fixed to a gold surface, this causes change in optical properties (Nirmal et al.,2008).

10. The common diagnostic approaches adopted to identify the bacilli in different forms of tuberculosis are shown in Table. II.

Forms of EPTB	or various forms of Tuberculosis Different diagnostic methods	
1. TB lymphadenitis	Fine needle Aspiration Cytopathology, later cultural examination and histopathology. Surgical removal of affected lymphnode by mediastinoscopy or thoracoscopy.	
2. Pleural Tuberculosis	Chest X ray, Sputum Culture examination, detection of Adenosine Deaminase level, histopathological examination of tissues, thoracoscopy or thoracotomy	
3. Skeletal Tuberculosis	Tuberculin Skin Test, Tissue biopsy, MRI, CT scan, Synovial biopsy(increase in WBC count)	
4. CNS Tuberculosis	Cerebrospinal fluid examination(increase in lymphocyte count and protein level and decreased level of glucose, MRI and CT scan	
5. Abdominal Tuberculosis	Tuberculin skin tests (+ for 70%), Chest X- rays, Cultural examination of ascitic fluid or laproscopy or laprotomy or colonoscopy	
6. Genitourinary Tuberculosis	Tuberculin Skin test (+ for 90% of cases), cultural examination of urine, biopsy of lesion, chest X-ray.	
7. Pericardial Tuberculosis	CT and MRI scans, Fine Needle aspiration of pericardial fluid, pericardial fluid biopsy	
8. Dessiminated TB	Chest X-ray, Sputum culture examination, blood culture examination, biopsy material examination of superficial lymphnode, Bone marrow, liver.	
9. Peritoneal tuberculosis	Laproscopy with direct biopsy, ultrasonography	

Table II. Various diagnostics for various forms of Tuberculosis







Treatment

The treatment of Extrapulmonary TB follows standard Revised National Tuberculosis Control Programme (RNTCP) treatment guidelines depending on categorization, is consistent with guidelines given by WHO and the International Union Against Tuberculosis and Lung Diseases(IUATLD).Categorisation is done on the basis of history, clinical and diagnostic criterion(Fraser et al.,2000)

Table.III. Tractment guidelines for TB as Revised National Tuberculosis Control Programme.

Category of treatment	Type of patient	Regimen
Category I	 New sputum smear positive pulmonary TB Seriously ill** new sputum smear negative PTB Seriously ill** new EPTB 	$2 H_{3}R_{3}Z_{3}E_{3} + 4H_{3}R_{3}$
Category II	 sputum smear positive relapse sputum smear positive failure Sputum smear positive treatment after default others*** 	2S ₃ H ₃ R ₃ Z ₃ E ₃ + 1H ₃ R ₃ Z ₃ E ₃ + 5H ₃ R ₃ E ₃
Category III	New sputum smear – negative PTBNew EPTB, not seriously ill	$2H_{3}R_{3}Z_{3} + 4H_{3}R_{3}$

Numbers before letters indicates months of treatment, subscript refers to number of doses per week. Dosage strength are H isoniazid (600mg), R Rifampicin (450mg), Z pyrazinamide (1500mg) E ethambutol (1200mg) S streptomycin (750mg), Patients with more than 60 kgs of weight will receive 150mg extra of Rifampicin and patients which less than 50 kgs receive 500mg Streptomycin and if body weight is less than 30 kgs then drugs given on the basis of their body weights.

Literature Cited

Aggarwal, A. N., Gupta, D. and Jindal, S. K. (1999). Diagnosis of tuberculous pleural effusion. Indian J. Chest Dis allied Sci, **41**:89-100.

Berenguer, J., Moreno, S., Laguna, F., Vincente, T, Adrados, M. Ortega, A. (1992). Tuberculous meningitis in patients infected with human immunodeficiency virus. N Engl J Med, **326**:668-72. Bhan, S. and Nag, V. Skeletal tuberculosis. (2001). In: Sharma, S. K. and Mohan, A. editors. Tuberculosis, New Delhi, Jaypee Brothers Medical Publishers, 237-60.

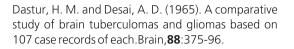
Bouza, E., Merino, P., munoz, p., Sanchez-carrillo, C., Yenez, j. and Cortes, C. (1997). Ocular tuberculosis. Prospective study in a general hospital Medicine (Baltimore),**76**:53-61.

Collins, C. H. and Granje, J. M. (1983). A Review: The bovine tubercle bacillus. J Appl Bacteriol, **55**: 13-29

Cosivi, O., Meslin, F-X., Daborn, C. J. and Grange, J. M. (1995). The epidemiology of Mycobacterium bovis infection in animals and humans with particular reference to Africa. Scientific and Technical Review, **14**: 733-46.

Dandapat, M. C., Mishra, B. M., dash, S. P., and Kar, P. K. (1990). Perpheral lymphnode tuberculosis: a review of 80 cases. Br J Surg, **77**:911-7.





Drobniewski, F. A., caws, M., Gibson, A. and Young, D. (2003). Modern laboratory diagnosis of tuberculosis. The Lancet infectious diseases,**3**:141-147

Ferrer, J. (1997) Pleural Tuberculosis Eur Respir. J,**10**: 942-7.

Fowler, N. O. (1991). Tuberculosis pericarditis. JAMA, **266**:199-203

FraserWares, Balasubramanian, R., Mohan, A., and Sharma, A. K. (2005). Extrapulmonary Tuberculosis: Management and control. Tuberculosis control in India, Directorate General Health services, Ministry of health and family Welfare, New Delhi.

Granje ,J., Daborn, C. and Cosivi. (1994). HIV related tuberculosis due to Mycobacterium bovis. Eur.Respir J; **7:1564**-6

Hemal, A. K. (2001). Genitourinary Tuberculosis. In: Sharma, S. K. and Mohan, A. editors. Tuberculosis. New Delhi: JaypeeBrothers Medical Publishers, 325-37.

Hill, A. R., Premkumar, S., Brustein, S., Vaidya, K., Powell, S. and LiP-W (1992). Disseminated tuberculosis in the acquired immunodeficiency syndrome era. Am Rev Respir Dis, **144:1164**-70.

Idigoras, p., Perez-Tralluro, E., allorta, M., Gutierrez, C., and Munoz Baroja, I. (1995). Rapid detection of tuberculous and non tuberculous Mycobacteria by microscopic observation of growth on Middlebrook 7H11 agar. Eur. J. Clin. Microbiol. Infect. Dis, **14**(1):6-10.

Jalai, U., Rasul, S., Khan, A., Baig, N., Khan, A., and Akhter, R. (2005). Tuberculosis mastitis. J. coll. Physician surg Pak, **15**: 234-7.

Kumar, S. (2001). Female genital tuberculosis. In: Sharma, S. K. and Mohan, A. editors. Tuberculosis. New Delhi: Jaypee Brothers Medical Publishers,311-24. Malaviya, A. N. and Kotwal, P. P. (2003). Arthritis associated with tuberculosis. Best prac res Clin Rheumatol, **17**:319-43.

Muranishi, H., Nakashima, M., Isobe, R., Ando, T. and Shigematsu, N. (1990). Measurement of tuberculostearic acid in a sputa, pleural effusions and bronchial washings: A clinical evaluation of pulmonary tuberculosis. Diagn. Microbiol Infect. Dis, **13**: 235-40.

Nirmal prabhakar, Kavita, A., Sunil, K. Arya, Pratima, R., Solanki, M., Iwamoto, Harpal Singh and Malhotra, B. D. (2008).Nucleic Acid sensor for M. tuberculosis detection based on surface plasmon resonance. Analyst,**133**:1587.

Pichenik, A. E., Belmatoung, N., and Fantin, B.(1984). Tuberculosis, atypical mycobacteriosis and acquired immunodeficiency Syndrome among Haitian and non-Haitian patients in south Florida. AnnIntern Med, **101**:641-5

Ravindra Kumar Garg. (1999). Tuberculosis of Central Nervous System. Post. Grad. Med. J,**75**: 133-40.

Ramam, M. (2001). Cutaneous tuberculosis. In: Sharma, S. K. and Mohan, A. editors. Tuberculosis. New Delhi: Jaypee Brothers Medical Publishers: 261-71.

Sachdeva, O. P., Kukreja, S. M. and Mohan, C.(1978). Lupus vulgaris of external ear. Indian j otolaryngol, **30**:136-7.

Samuel, A. M., Kadirval, G. V., Stehlekar, M. D., Ganata, R. D. (1984). Evaluation of Tubercular antigen and antitubercular antibodies in pleural ascitic effections . Ind. J. Med Res, **80**: 563-565.

Sharma, S. K., Mohan, A. (2004) Extrapulmonary tuberculosis. Indian J Med Res, **120**: 316-53

Sharma, S. K., Mitra, D. K., Balamurugan A., Pandey R. M., Mehra, N. K.(2002). Cytokine polarization in military and pleural tuberculosis. J. Clin. Immunol, **22**: 345-52.

Soni N. K. and Chatterjee, P. (1978). Laryngeal tuberculosis. Indian J. Otolaryngol, **30**:115-7.







Shinde, S. R., Chandawarkar, R. Y. and Desamukh, S.P.(1995). Tuberculosis of the breast masquerading as carcinoma: A study of 100 patients. World J. Surg, **19**: 379-81

Stager, C. E., Libonath, J. P., Siddique, S. H., Davis, J. R., Hoopa, N. M., Baker, J. F., and Carter, M. E. (1991). Role of Solid media when used in conjunction with BACTEC system for mycobacterial isolation and identification. *Journal of Clinical Microbiology*, 154-57.

Peri-parturient Disorders : A hurdle in Dairy Development



Milk Fever : Treatment

Treatment is directed towards restoring normal serum calcium levels as soon as possible to avoid muscle and nerve damage; and recumbency.

Recommended treatment is i/v. inj of a calcium gluconate salt, although s/c or i/p routes are also used. A general rule for dosage is 1 gm calcium / 45 kg body wt. Most of the available solutions are contained in a 450-500 ml single dose bottles (8 to 11 gm calcium). In large, heavily lactating cows/buffous, a second bottle given s/c may be helpful, as it provides a prolonged release of calcium into circulation.

S/C calcium treatment alone may not be adequately absorbed due to poor peripheral perfusion and should not be the sole route of therapy

Many solutions contain Magnesium in addition to calcium, which may protect against myocardial irritation produced by the administration of calcium. Magnesium is also necessary for appropriate PTH secretion and activities in response to hypocalcemia.

Administration of oral calcium may be useful in mild cases of hypocalcemia, however, it is not recommended as the sole approach for clinical milk fever cases. Calcium propionate in propylene glycol gel or powdered calcium propionate (0.5 kg dissolved in 8-16 lit of water administered as a drench) is effective, less injurious to tissues and supples the gluconeogenic precursor propionate. It is important to note that cows/buffaloes with hypocalcemia often have poor swallowing and gag reflexes. Care most be exercised during administration of calcium containing solutions to avoid aspiration pneumonia.









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Sheep Based Integrated Farming System

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Introduction

Productivity, efficiency, and income stabilization are key issues for the sustainability of small farmers in India. Small and fragmented land holdings do not allow farmers to have independent farm resources like draught animals, tractors, bore wells/tube wells and other sophisticated farm machineries for various cultural operations. While crop production is the primary farming enterprise in the country, livestock raising serves as an auxiliary activity to crop production. About 90% of livestock population are in the hands of the small holders where adequate supply of high quality forage is a common problem. So investigation on possible diversified production systems may provide helpful information for improving productivity and stabilizing the small farmer's income. Different farming systems have been evolved independently and being practiced by the farmers without any rationale for utilizing the wastes and residues arising out of cropping/animals and other associated enterprises at farm, resulting into wastage of resources. To fulfil the basic needs of household including food (cereal, pulses, oilseeds, milk, fruit, honey, fish, meat, etc.) for human, feed and fodder for animals and fuel & fibre for general use warrant an attention about Integrated Farming System.

What is integrated farming system?

Farming system is a resource management strategy to achieve economic and sustained production to meet diverse requirements of farm households, while preserving resource base and maintaining a high level environmental quality (Lal and Millu, 1990). Integrated farming system models developed in different parts of the country involving dairy, duckery, poultry , horticulture, apiary, pisciculture and plantation crop sviz; coconut, cocoa, nutmeg, banana, pineapple etc. along with crops, have been found to increase net profit significantly as compared to cropping alone. These IFS systems were also found more sustainable and employment generative. In Telangana zone of Andhra Pradesh, the major crops grown are rice, maize, jowar, groundnut, sugarcane and cotton and other components include buffalo, goat, sheep and poultry. The results of a study (Radha et al; 2000), conducted on survey based with three agricultural and livestock based farming systems viz., dairy, poultry and sheep rearing clearly revealed that all the integrated farming systems generated more than 3 times additional employment over arable farming. The net returns were higher in agriculture + dairy (Rs. 35293) followed by agriculture + poultry (Rs. 26830) and agriculture + sheep rearing (Rs. 14665).

Integrated farming system (IFS), a component of farming system research (FSR), introduces a change in farming techniques for maximum production through optimal utilization of resources. The farm base is better recycling for productive purposes in the integrated farming system. Integrated farming system involves the utilization of primary produce and secondary produce of one system as basic input of other system, thus making the mutually integrated as one whole unit. There is a need to effective linkages and complementarities of various components to develop holistic farming system.

Sheep integrated farming system

Raising of small ruminants is now getting popular among smallholders due to the relatively lower capital investment and risk involved as compared to cattle. Like goats, sheep can be easily raised by smallholders due to its relatively lower capital investment and risk involved compared to cattle. Sheep is small in size and can be easily attended to by women and children. They have early maturity, shorter reproductive cycle and can be fed with different forages including weeds. The rice growing areas offer some potential for raising sheep. Aside from the straw, stubbles after rice harvest, weeds growing in rice paddies are available. To introduce sheep raising and demonstrate fodder tree supplementation in a rice cropping system, on-farm study is needed.

In sheep production, intertemporal decisions on capital and labour investments, and inventory adjustments such as the number of sheep sold and the number retained in each time period, determine the farmers' projected profit in subsequent periods. These intertemporal decisions are influenced by farmers' expectations toward market conditions as well as biological characteristics of sheep such as lambing interval, reproduction rate, and survival rate, which contribute partially to the optimal flock size for a given level of family resources.

Integration of sheep and utilization of fodder trees In rice-based cropping system

An experiment was done to know the prospects and limitations of integrating sheep into the rice based farming system in a Philippine village. Integration of sheep with rice crop based farming system needed an on field experiment.

Selection of farmer : Farmer co-operators were selected based on their willingness to raise sheep under a repayment- in-kind distribution scheme. All farmer-co-operators practised semi confinement system, wherein they tether the sheep on roadsides, rice fields, fallow lands and even in park in the morning and get them back in the afternoon.

Sheep distribution scheme : The project's distribution scheme involves repayment of two female lambs for every ewe credited. Ram belongs to the government, wherein, they are rotated annually among farmer co-operators to avoid inbreeding. Repayment sheep were redistributed to new co-operators. According to this scheme in Indonesia by 1993, the number of participant farmers had increased from 12 to 77 (Soedjana; Sirait et al. 1993).

Feeding practice : The farmers recorded their daily activities on sheep and it showed that farmers practice semi confinement feeding wherein they tether the sheep on roadsides during the rice growing period and on rice fields and fallow land after rice harvest. During the rice growing period, farmers feed the sheep with rice straw and weeds from rice paddies and paddy bunds. These weeds include Echinocloa crusgalli (40%), Cyperus rotundus (26.25%), Oplisimus compositus (11.5%), Ipomea aquatica and other species (8.75%) and Brachiaria mutica (13.5%). After rice harvest (early part of the dry season), sheep are tethered with other ruminants in rice fields and feed on stubbles. Later in some areas, standing hay of mung bean is available and in some occasions peanut hay and other crop residues are given.

Live weight performance: The live weight performance of sheep can be studied for the 166-day period. Sheep raised in the traditional



way with fodder tree supplementation had the highest live weight and average daily gains of 11.35 kg and 74 g, respectively, which is significantly higher than those animals without supplementation. On the other hand, sheep tethered in park with fresh *Leucaena* supplementation have an average daily gain of 24g. This data are according to the study of E.E. Victorio and F.A. Moog in Philippines from October 1994 to February 1995.

Advantages:

- Raising sheep in a lowland rainfed farming system requires at least one and two hours per day after rice harvest and during the rice growing period, respectively. The time is spent on tethering and provision of water during the former and gathering of weeds and feeding during the latter.
- Aside from the savings on the cost of labour in weeding the rice paddies and bunds during the rice cropping period, it generated approximately 2,640 man-days valued at 162,570 for four years.
- In rice-based farming system, raising of large ruminants is not commonly practiced as feed supply is limited and fear of crop destruction and source of stock posed problems. Nevertheless, sheep production has great potential as mutton is becoming a popular dish in the Philippines.
- In a village in Tarlac, initial study shows that raising of sheep has very bright prospects due to the following reasons:
 - 1. Sheep are less destructive to crops than goats.
 - 2. Market is not a problem as mutton is becoming a favorite dish.
 - 3. Presence of fodder trees and initial awareness of farmers on its feeding value.

Integrated Hill Sheep Production

In Ireland Mountain, hill and peat lands above 150 m account for 1.5 m ha and sheep farming is a very important enterprise in these regions. The physical and physiographic characteristics of the hill have been described in detail by Little (1995) and Walsh et al. (2000). The options to improve the physical performance of the sheep enterprise on hill farms are limited by the proportion of lowland available. Sustainability must also be assured. Thus the objective of this project was to exploit the integrated use of hill and lowland in a sustainable manner. The project was carried out during 4 years (1998/99 to 2001/02) at the Teagasc Hill Sheep Farm at Leenane, Co. Mayo. The project involved two integrated systems aimed at maximising the use of hill (Hill) and lowland (Lowland) grazing respectively. The Hill and Lowland systems consisted of 200 Blackface ewes mated to Blackface rams and 150 Blackface ewes crossed with Belclare rams respectively. Hill ewes spend their first three breeding seasons on the hill and are then transferred to the Lowland system.

The flock performance targets were,

- a) 0.9 (Hill) and 1.2 (Lowland) lambs reared per ewe joined,
- b) Male Hill and Lowland lambs finished to carcass weights of 12 and 18 kg, respectively
- c) Hill and Lowland female lambs to weigh 40 kg at first mating (18 months) and 36 kg at about 6 months old, respectively.

The production targets were to finish Hill and Lowland male lambs to carcass weights of 12 and 18 kg respectively, that the Hill flock would produce its own replacements and that the Lowland females would be suitable as replacements in lowland meat lamb systems. This would overcome the problem of traditional unsteady trading markets to lowland farmers and give improved producer independence and

control. (Hanrahan and O'Malley (1999), Walsh et al. (2000), Guinan et al. (2002) and Walsh and Feinstein (2003))

Project objective

Main objective of the project was

- a) ewe replacements of 40 kg at first mating within the Hill system and cross-bred females of 36 kg in September of their first grazing season for sale as replacements for lowland flocks, and
- b) Hill and Lowland male lambs of 12 and 18 kg carcass weights respectively.

Management detail

Mating to lambing : In September 1998, 389 Blackface ewes which had been run as one flock (Hanrahan and O'Malley, 1999) were divided so as to transfer all 4th crop and older ewes into the Lowland flock and retain all younger ewes in the Hill flock. Ewes grazed on their respective lowland areas during mating, which commenced in late October for the Lowland flock and mid-November for the Hill flock. All ewes were mated, in groups of 40 to 45, with individual rams, to generate pedigree information. All matings were recorded and was subsequently used to facilitate management through segregation into groups according to expected lambing date. After a 5-week mating period, rams were withdrawn and all ewes were put to graze on the hill. The Lowland flock was offered silage ad libitum supplemented over the final 6 weeks of pregnancy with concentrates. The Hill flock continued to graze on the hill until pregnancy scanning in mid-February. All housed Lowland ewes were moved outdoor into the appropriate lowland area for lambing in cohorts according to mating date. During this period they were offered concentrate supplement at 450 g/head/day.

Lambing to Weaning : the Hill flock, ewes rearing single female lambs were allocated a separate part of their lowland grazing at lambing to facilitate an early return to the hill, where they remained until weaning. All ewes and lambs in the Lowland flock were grazed on lowland until weaning when lambs were 14 weeks old.

Rearing Hill lamb replacements : In the Hill system about 80 females were retained as replacements each year. The objective was to ensure that they would be 40 kg at first mating at 19 months of age.

Measurements:

- Lambs were weighed and tagged within 24 hr of birth. Hill lambs were weighed again at about 3 weeks of age when female singles were moved to hill grazing. Lowland lambs were weighed at 5 weeks of age. Further weighing was carried out for all Hill and Lowland lambs at weaning, for all females in September and for all males before slaughter. Carcass weight was recorded for all lambs slaughtered.
- The number of eggs per gram of faeces (EPG) was determined weekly for ewes and lambs on the lowland areas of both systems from April to October during the 2001 and 2002 grazing seasons. Eggs from Nematodirus and other Trichostrongyles were enumerated separately.

Ewe performance:

- In general the liveweight and body condition score declined from mid-pregnancy to lambing and increased during the 5 weeks after lambing.
- Fertility rate of ewes were also increased.



- Overall average litter size was increased to 1.12 in that area.
- Lamb mortality at 5 weeks old was very low i.e 1.8%. About 5% of ewes were barren at pregnancy scanning, which is slightly higher than the 4% recorded by Nolan and McNamara (2000) in a high output lowland system.
- Overall ewe mortality was also decreased.
- Overall Average daily Gain was satisfactory in Hill Imbs. But subsequently decreased in successive years.

The results show that a mixture of Hill and Lowland systems can be effectively exploited with proportions of ewes in each reflecting the proportion of low to hill land available. The target sheep performances were achieved under a grazing management which led to improved hill vegetation cover and in this context are sustainable.

Literature cited

Guinan, L. M. et al., 2002. Response of vegetation to the exclusion of herbivores on a western hill farm. Agricultural Research Forum, 6p

Hanrahan. J.P. and O'Malley, L. 1997. Performance of Scottish Blackface sheep at Leenane hill farm. Teagasc Research Centre, Athenry, 13p Hanrahan. J.P. and O'Malley, L. 1999. Hill sheep production system. ISBN 1841700404

Lal, R., and Millu, F.P. (1990). Sustainable farming for tropics. In: Sustainable Agriculture: Issues and Prospective. Indian Society of Agronomy, IARI, New Delhi.Vol.I (Ed.) R.P.Singh, pp 69-89.

Little, E. M. 1995. A study of mixed cattle and sheep grazing on hill and lowland vegetation in the west of Ireland. M. Sc., Thesis, Department of Botany, UCG, Galway, pp. 75

Nolan, T. and McNamara, N. 2000. High output midseason lamb meat system. ISBN 1 841 70 309 5

Radha,Y., Eshwaraprasad,Y. and Vijayabhinandana, B.(2000). Study on income and employment generation on agricultural based livestock farming system. Paper presented at VIII Annual Conference of AERA at TNUASU, Chennai, 28-29 Dec., 2000.

Soedjana, T. D. (1993). "Economics of raising small ruminants." Draft report, Small-Ruminant Collaborative Research Support Program (SR-CRSP), North Sumatra, Indonesia.

Walsh et al. 2000. Evaluation of the impact of livestock on the hill environment. ISBN 184170 1343

Walsh, M. and Feinstein, M. H. 2003. Grazing preferences of hill sheep in relation to physiography, soils and semi-natural vegetation using field observations and satellite tracking. Agricultural Research Forum, p26.







Reproductive management of dairy cattle during summer

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Introduction

Heat stress can be defined as the forces external to the animal that act to displace body temperature from set point temperature (Hansen 2009). Body temperature is closely regulated by matching heat production with heat loss to the environment via conduction, radiation and evoperation. At its most, severe heat stress induces heat stroke and death in domestic animals. It also reduces feed intake. productivity and reproduction (Collier et al 2006; Hansen 2009). The negative effects of heat stress on dairy cows are multifaceted and have been studied for several years. This article highlights some of the reproductive challenges faced by cows during heat stress, reducing a dairy's profit. Several solutions to minimize the negative impact of heat stress are also reviewed.

Effects of heat stress on reproduction

Various studies (Jordan 2003; Rensis,2003; West, 2004) have shown that heat stress challenges the reproductive performance of dairy cows through a variety of altered physiologic means, including altered follicular development , lowered estrus activity and impaired embryonic development.

The first reproductive challenge facing the heat stressed cow is altered follicular development. Heat stressed cows decrease feed intake causing less frequent pulses of luteitinizing hormone (LH) resulting in longer follicular waves. This lenghthening of follicular wave leads to the selection and ovulation of multiple,

smaller dominant follicles (Sartori, 2002), thus reducing the guality of oocytes and modulating follicular steroidogenesis (Roth et al; 2000). Follicles are responsible for producing estrogen, a hormone that causes cows to show signs of heat .Smaller follicles will produce less estrogen than larger ones, therefore resulting in less estrus activity. Estrus activity is also lowered due to the cow's reduced motor activity, a means of trying to decrease endogenous heat out put. Another possible reason for reduced estrus expression is from suppressed endocrine hormones such as luteinizing hormone and estradiol, important for follicle growth and initiation of estrous behaviour (Rensis and Scaramuzzi.2003). Thus the occurance of silent ovulation or silent heat increases which will ultimately reduce heat detection efficiency even in well performing heat detection programs. The high uterine temperature of heat stressed

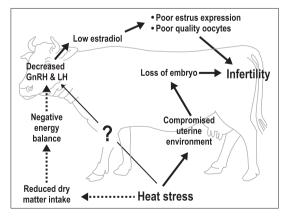


Fig 1: Depiction of heat stress on dairy cows (Rensis and scaramuzzi, 2003)



cow can impair embryonic development, resulting in poor embryo implantation and increased embryo mortality(Jordan, 2003 and West, 2004). Fig I illustrates the challenges heat stress poses for dairy cows.

Heat stress not only depends on temperature, but also depends on humidity of the surroundings. This is known as the temperature humidity index or THI. Heat stress occurs when the THI exceeds 22.2°C(72°F) which is when the cow's body is unable to cool itself adequately. Some common signs of heat stress include increased body temperature 39.2°C (>102.6°F) and panting >80 breaths/minute. Reduced physical activity , feed intake >10-15% and milk yield >10-20% are also effects of heat stress (West ,2004).

Measures to reduce the heat stress in dairy cattle

The following are the recommendations followed to reduce the negative reproductive effects of heat stress in dairy cattle.

a) Implementation of the aggressive breeding programs

As cows do not display heats under heat stress as much as they do in cool temperature, a good heat detection program should be followed with the help of a professional technician, use of timed breeding protocols be followed. Timed breeding program is helpful when animals are not showing the signs of heat. There are several well researched timed breeding programs to pick from. Some dairy owners make a mistake by discontinuing A.I. breeding and use bulls for natural breeding, because they believe A.I. performance declines in the summer. This is a big mistake because fertility of bulls is also lowered just as much or more than in cows due to heat stress. However, heat stress does not affect the fertility of frozen semen when handled and administered properly.

b) Implementation of cow cooling program

Cooling is one of the most effective ways to manage heat stress and minimize economic losses. Sprinkling or soaking with water, along with supplemental airflow has been shown to reduce respiration rates by 17.6 40.6%, improve dry matter intake by 7-9% and increase milk vield by 8.6 15.8% (Buckline, 1991 and West ,2004). This is also known as evaporative cooling. Many producers try soaking or sprinkling cows without having fans for supplemental air flow. This is not recommended. With increased levels of water vapor in the air, water alone raises relative humidity. Consequently, THI will rise because nothing will dissipate the humid air around the cow .Fans in combination with water provide the best cow cooling as demonstrated in several studies (Brouk, 2005; Brouk, 2003; West, 2004).

c) Feed high quality rations

Early lactation cows exposed to heat stress may go even further into negative energy balance because they aren't consuming as much as feed needed. Consequently, they are more likely to have lower reproductive performance due to altered follicle development and lower estrus activity. Feeding high quality forages and balanced rations will decrease some of the effects of heat stress.

Conclusion

Heat stress challenges dairy cattle reproductive performance by altering follicular dynamics , lowering the display of estrus and changing the uterine environment leading to increased embryo mortality . Implementing aggressive breeding programs , cow cooling strategies and top notch feeding programs can help minimize some of negative effects of heat stress.







References

Brouk, Michael J., Joseph P. Harner and John F. Smith. March (2003). Effectiveness of cow cooling strategies under different environmental conditions .pro 6th western dairy management conference .

Brouk, Michael J., Joseph P. Harner and John F. Smith. March (2005). Evaluating and selecting cooling systems for different climates .pro 7th western dairy management conference .

Bucklin , R. A., L. W. Turner, D. K. Beede, D. R. Bray and R. W . Hemken. (1991).Methods to relieve heat stress for dairy cows in hot, humid climates . Apple. Eng. Agric. **7**: 241-247.

Collier RJ, Dahl G. E. Van baale MJ. (2006). Major advances associated with environmental effects on dairy cattle . journal of dairy science **89**, 1244-1253.

Hansen PJ. (2009). Effects of heat stress on mammalian reproduction . philosophical transaction of the Royal society of London biology society **364**, 3341-3350.

Harner III, J. P., M. j. Brouk, J. P. Murphy and J. F. Smith. September (2000).Reducing heat stress in the holding pens. http://www.oznet.ksu.edu/library/lvstk2/mf 2468.pdf.

Jordan, E. R. (2003). Effects of heat stress on reproduction. J. Dairy Sci. **86** : E. supple.:E 104 E 114.

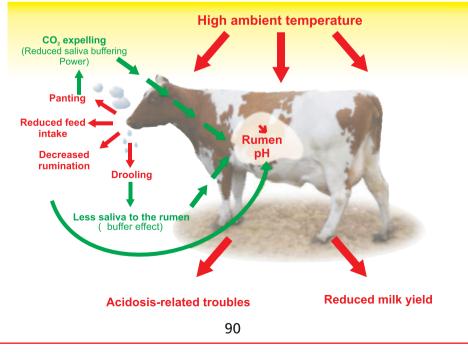
Rensis , FD., and R. J. Scaramuzzi. (2003). Heat stress and seasonal effects on reproduction in the dairy cows. a review . Theriogenology **60** : 1139-1151.

Roth, Z. A. Bor, R. Braw Tal and D. Wolfenson. (2000). Immediate and delayed effects of heat stress on follicular development and its association with plasma FSH and inhibin concentration in cows. J. Reporod. Fertil. **120** : 83-90.

Sartori, R., G. J. Rosa and M. C. Wiltbank. (2002). ovarian structures and circulating steroids in heifers and lactating cows in summer and lactating and lactating and dry cows in winter . J. Dairy sci.**85** : 2813 -22.

Techline. ABS Global Technical services news letter managing reproduction during times of heat stress.

West, Joe W. November (2004). Heat stress affects how dairy cows produce and reproduce. Pro. Southeast Dairy Herd Management Conference.











Chicken Infectious Anemia

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Introduction:

Chicken Infectious Anemia (CIA) is an emerging disease, primarily affecting young chickens and characterized by lymphoid atrophy, poor weight gain, aplastic anaemia, aplasia of the bone marrow, subcutaneous and muscular hemorrhages and immunosuppression. Mortality in natural outbreaks ranges from 5-20 % but can reach up to 60 percent. It causes severe immunosuppression as the virus multiplies in the lymphocytes and causes destruction of lymphocytes from primary (Thymus and Bursa) and secondary lymphoid organs (Spleen, Caecal tonsil, Harderian gland, etc.). Due to immunosuppression, the affected birds suffer from high incidence of secondary opportunistic infection like Gangrenous dermaitis, due to Staphylococcus aureus, Clostridium perfringenes, (Engstrom and Luthman, 1984; Vacchani, 2005; Goryo et al, 1987) and colibacillosis (Gorvo et al, 1987) coccidiosis, cryptosporidiosis (Schat, 2003) and pulmonary aspergillosis Goryo et al, 1987). It also enhances pathogenicity of co-infecting pathogens such as New Castle disease virus (NDV), Marek's disease virus (MDV), Adenovirus and Reovirus (McNulty, 1999; Schat, 2003).

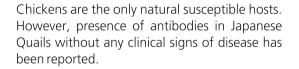
In India, Chicken Infectious Anemia (CIA) was reported from young growing chickens at Namakkal, Tamil Nadu by Venugopalan et al. (1994). They identified CAV through Immunoperoxidase test. Kataria et al. (1999) reported CAV from chicks at Tamil Nadu, Maharashtra and Utter Pradesh. They confirmed the existence of CAV by PCR. CIA has also been named as Chicken anaemia agent (CAA), pale bird syndrome, Hemorrhagic syndrome, Hemorrhagic anemia syndrome, Infectious aplastic anemia, Anemia-dermatitis syndrome, Gangrenous dermatitis, Blue wing disease (Yuasa et al., 1979; Engstrom and Luthman,1984; Schat, 2003). However, CIA is most common and widely used name for the disease (Schat, 2003).

Etiology:

The chicken anemia virus (CAV) was first described by Yuasa et al. (1979) from contaminated vaccine in Japan. Chicken anaemia virus (CAV) belongs to genus Gyrovirus of Circoviridae family. Circoviruses are small, non enveloped ico-sahedral viruses having circular, single stranded (ss) DNA genomes. Their genomes are the smallest possessed by animal viruses. Till now, complete nucleotide sequences for 7 CAV isolates have been identified. All isolates belong to a single serotype (Schat, 2003).

Resistance and hosts:

CAV showed resistance to 5% solutions of many commercial disinfectants at 37°C for 2 hr viz. quaternary ammonium compound, soap and orthodichlorobenzene (Yuasa et al., 1983). The virus is inactivated by 1% gluteraldehyde for 10 min at room temperature (R.T.), 0.4% βpropiolactone for 24 hr at 4°C, 5% formaldehyde for 24 hr at R.T, 1% iodine and sodium hypochlorite, heating at 100°C for 15 min (Schat, 2003). The various studies indicated that the CAV is very sturdy virus.



Epidemiology:

CIA is ubiquitous in all the chicken producing countries of the world. The majorities of the natural outbreaks of CIA have been reported in broilers. The clinical signs develop at the end of second week of age with fewer incidences in pullets. Interestingly, in India, majority of the outbreaks of CIA (based on serology and lesions) are seen in pullets (layer) of 4-12 week age (personal observation of the third author).

Transmission:

CIA is transmitted by horizontal and vertical route. Vertical transmission occurs when breeder layers (Broiler/ Layer parents) with no antibody to CAV or no previous exposure to virus become infected as they come into egg production. The transmission of the virus through eggs occurs until seronegative hen become infected/ vaccinated and continues to shed virus through egg till neutralizing antibodies develop. No clinical disease is seen in breeders-but the virus is transferred to offspring which in turn develop clinical disease. It starts at 3-12 weeks after exposure-usually at the start of egg laying.

Horizontal transmission of CAV occurs by the feco-oral route and perhaps by respiratory route.

It occurs in chickens lacking maternal antibodies to CAV. The chicks hatched from the eggs of CIA infected parents are viremic and spread of the virus to susceptible chicks occurs by horizontal route.

Susceptibility:

Chickens of all age are susceptible. Chicks of 2 week age are more susceptible. Susceptibility to clinical disease decreases rapidly due to development of immune competence to produce effective humoral response. Chicks less than 3 weeks of age (lack maternal antibodies) are prone to get the infection. Most of the breeder flocks will seroconvert as a result of horizontal infection to develop humoral immunity. Both sexes are susceptible. Other infections like IBDV, IB, MDV and REV at early age as well as production and transportation stress increases susceptibility to CAV.

Pathogenesis:

Incubation period is 10-14 days. CAV enter body vertically through egg to chick or / horizontally by feco-oral route. Viraemia results by entry of CAV into various organs including lymphoid organs viz. hemocytoblasts in bone marrow and precursor lymphocytes in thymus, gonads and embroyonal tissues (latent infection). The virus reaches and multiplies in any organ with highest concentration found in liver. Anaemia results due to direct cytolytic effect of the virus on the erythroblastoid precursor cells in bone marrow (VP3 Protein). Destruction of thrombocytes leads to impaired clotting and results into hemorrhages throughout the body. Destruction of precursor T cells in thymic cortex, depletion of T-Helper and T-cytotoxic cells alone or other immunosuppressive agents like MDV, IBDV, REV, Adenovirus leads to immunosuppression.

Secondary complications with gangrenous dermatitis (Engstrom and Luthman, 1984; Goryo et al, 1987), colibacillosis (Goryo et al, 1987) coccidiosis, cryptosporidiosis results in death (Adair, 2000; Daniel, 2000; Daren, 2000; Schat, 2003; Ezzi et al., 2012).



Significance of Disease:

Clinical disease generally occurs at 2-4 weeks of age in vertically transmitted/ maternal antibody negative broiler chickens. However, the chicken having maternal antibody resist infection.

Subclinical infection: The breeder flock when seroconvert from 8-14 week of age, as a result of horizontal spread, the infection appear to be subclinical in these flock.

Immunosuppression occurs at 2-3 weeks of age. Depletion of thymocytes, lymphocytes, myeloid progenitor cells results in immunosuppression. Virus impairs T-cell and macrophage functions which leads to reduction in phagocytosis, bactericidal activity and increased susceptibility to secondary bacterial, viral and fungal infections. It also leads to vaccine failure.

Clinical Signs:

Depression, paleness of comb (anemia) that peak at 14-16 days post infection (DPI), weakness, anorexia, ruffled feathers and stunted growth (10-20 DPI) is seen in affected birds. Bluish discolorations of skin is seen due to accumulation of serosanguineous exudates at subcutaneous areas of wing and hence the name blue wing disease. Morbidity is variable and ranges from 20-60 % and mortality ranges from 5-20 % which occasionally may go up to 60 % (Engstrom and Luthman,1984; Schat, 2003).

Hematology: Blood appears watery, pale color and shows increased clotting time. Blood smear shows immature erythrocytes that may exceed 30%. Immature forms of white blood cells and thrombocytes may be seen. Hematocrit values decreases by 8-10 DPI and are generally less than 27 % (Fig. 1). PCV drops to 10-20% at 14-20 DPI with average PCV of 15%. Blood sample

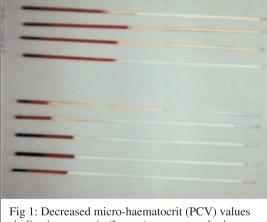


Fig 1: Decreased micro-haematocrit (PCV) values indicating anaemia (Lower) as compared microhaematocrit values of healthy chicken (Upper)

analysis shows aplastic anaemia, leucopenia and thrombocytopenia.

Gross lesions:

Prominent Lesions are seen in bone marrow (BM) and lymphoid tissues. In addition to BM and lymphoid tissue, the lesions are also observed in muscle, liver, gizzard and proventriculus These lesions noted here have been described by various authors in natural outbreaks and experimental studies on CIA (Toro et al., 1997; McNulty, 1999; Vacchani, 2002; Schat, 2003).

Thymus: Moderate to severe atrophy of thymus is one of the characteristic and consistent lesions seen in CIA (Fig. 2). The other lymphoid organs viz. bursa, Spleen, caecal tonsil show varied degree of atrophy.

Bone marrow: Bone marrow of the long bone viz. femur becomes fatty and yellow or white in color (Fig 3).

Muscles: Muscles appear pale with petechial to ecchymotic hemorrhages on breast and thigh muscle. Gangrenous dermatitis is most common concurrent infection seen along with CIA (Fig 4).



Fig. 2: Severely atrophied thymus in a pullet

Occasionally haemorrhages on heart are noticed due to secondary bacterial infection.

Gizzard and proventriculus (PV): Gizzard erosions (Fig. 5) are also commonly observed in CIA. Proventricular musosa shows haemorrhages.

Liver: Liver shows enlargement with petechial haemorrhags. Focal areas of necrosis on liver are noticed due to secondary bacterial infection.

Kidney: Kidneys are pale and swollen.

Other lesions: Occasionally, grey-whitish fungal nodules/ greenish patches on air sac, lung or peritoneum are seen in CIA complicated with fungal infection.

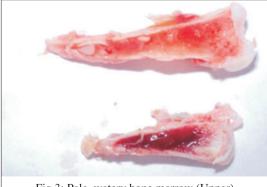


Fig 3: Pale, watery bone marrow (Upper) with normal bone marrow (below)

Microscopic lesions:

Thymus: Thymic lobes are severely atrophied and some time even they are difficult to find due to severe atrophy of the organ. Severe lymphoid depletion in cortex and medulla leads to loss of demarcation between cortex and medulla (Fig. 6).

Bone marrow: Bone marrow shows atrophy and hypoplasia of all compartments.

Haematopoietic tissues are replacement by adipose tissue.

Bursa of Fabricius shows atrophy of lymphoid follicles, infolded epithelium, with cystic follicle and hyperplasia of reticular cells and connective tissue.

Spleen shows atrophy of lymphoid tissue and hyperplasia of reticular cells. Presence of numerous bacterial colonies may be seen.

Cecal tonsils (CT) show lymphoid depletion with overall atrophy of CT.

Liver shows swelling of hepatocytes, dilatation and congestion of sinusoids and focal necrosis with numerous bacterial colonies.

Kidney shows degenerative changes in tubular epithelium with vascular congestion and haemorrhages.

Proventriculus, duodenum do not show any specific lesion except congestion and hemorrhage.

Eosinophilic intranuclear inclusions have been described in hemocytoblasts and lymphoblasts in thymus and bone marrow (Toro et al., 1997) but practically these are very difficult to find in tissue sections (Personal observations).





Fig 4: CIA- Gangrenous dermatitis

Diagnosis:

Tentative diagnosis:

Tentative diagnosis of CIA can be done on the basis of flock history, clinical signs, gross and histopathological lesion as well as hematological alterations (PCV less than 27 %, blood smear evaluation- immature erythrocytes, thrombocytopenia and leucopenia).

Confirmatory diagnosis:

Isolation of CAV: One day old chicks, embryonated eggs and cell culture i.e MDCC-MSB1 are used for isolation of CAV.

Serological Assays: Serum neutralization test and ELISA are commonly used for large scale screening of unvaccinated flocks.

Detection of CAV antigens:

Indirect Immunofluorescent staining: Fluorescence staining will reveal small and irregular granules in nucleus.

Immunoperoxidase staining: Brown staining particulate deposition in nucleus and cell membrane are observed.

Detection of CAV at DNA level:

PCR- has been found to be reliable and one of the most commonly employed tests for diagnosis of CIA. The organs like liver, bone marrow and thymus are preferred sample for diagnosis of disease (Daniel et al., 1992).

Differential Diagnosis:

Marek's disease, Infectious bursal disease, Inclusion body hepatitis, and Aflatoxin and sulphonamide intoxications need to be differentiated from CIA.

Prevention and Control:

Vertical transmission and clinical disease can be controlled by proper management and live attenuated, oil emulsified formalin inactivated and genetically engineered vaccines (Schat, 2003).

Conclusions:

CAV is ubiquitous in all major chicken producing countries in the world. Horizontal infection in breeder flocks during egg production causes a subclinical infection, which may have an impact on their performance. Vertical infection of CAV results in clinical disease in young chickens. Problems with vertical transmission appear when breeders are reared in strict isolation before start of egg production. Vaccinations of breeders during rearing protect the progeny from vertical infection and give them protective maternal antibodies. CAV has an immunesuppressive effect on young chickens. Dual infection with CAV and other immunosuppressive viruses enhance the pathogenicity of both the viruses. Exposure of litter material containing CAV between 10 and 16 weeks (natural challenge with virulent virus at this age has no adverse effects) is although crude method but has been suggested to control CIA in progeny.







References:

Adair, B. M. (2000). Immunopathogenesis of chicken anemia virus infection. Develop. Comp. Immunol. **24**: 247-255.

Daniel. T. (2000). Circoviruses: Immuno suppressive threats to avian species: a review. Avian Pathol., **29**: 373-394.

Daniel T., Karen A.M., Mc. Nulty M.S. (1992). Detection and differentiation of Chicken Anemia Virus isolation by using the Polymerase chain reaction. J. Clinical Microbiol. 1661-1666.

Daren .C.N., Kant .A., Van Roozelaar .D.J., Hartog. L., Noteborn. M.H., Koch. G. (2000). Studies on the pathogenesis of chicken infectious anemia virus infection in six week old SPF chickens. Acta Veterinaria Hungarica. **48**: 455-467

Engstrom, B. and Luthman, M. (1984). Blue wing disease of chickens: signs, pathology and natural transmission. Avian Pathol. **13**: 1-12.

Ezzi, A., Shoushtari. A., Marjanmehr. H., Torogi. R., Tavasoly. A., Bahmaninejad. M. A. (2012). Experimental studies of pathogenicity of chicken infectious anemia virus (3 isolates) in Iran. Archives of Razi Institute, **67**: 13-19.

Kataria, J.M., Suresh, R.P., Verma, K.C., Toroghi, R., Kumar, N.S., Kataria, R.S. and Sah, R.L. (1999). CIA in

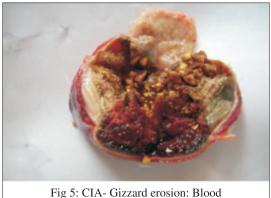


Fig 5: CIA- Gizzard erosion: Blood tinged gizzard content

India: detection of the agent by PCR and transmission study. Indian J. Comp. Microbiol. Immunol. Infec. Dis. **20**: 91-95.

Vacchani K. V. (2002). Etio-Immuno-pathological studies on chicken infectious anemia -gangrenous dermatitis syndrome in Pullets. M.V.Sc thesis, Submitted to Department of Veterinary Pathology, AAU, Anand.

McNulty, M. S. (1991). Chicken anaemia agent: A review. Avian Pathol. **20**:2: 187-203

Schat K. A. (2003). Chicken Infectious Anemia. In Diseases of Poultry. 11th edition. Edited by Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE. Ames, Iowa: Iowa. State University Press, pp. 182–202.

Toro, H., Ramirez, A. M. and Larenas, J. (1997). Pathogenicity of chicken anaemia virus (isolate 10343) for young and older chickens. Avian pathol. **26**: 485-499.

Yuasa N., Taniguchi T., and Yoshida I. (1979). Isolation and some characteristics of an agent inducing anemia in chicks. Avian Dise., **23**: 366-338.

Venugopalan, A.T.; Elankumaran, S.; Dhinakar Raj, G.; Murali Manohar, B., Thangavelu, A.; Ravikumar, G.; Koteeswaran, A. and Sundara Raj, A. (1994). Isolation of chicken anemia virus in Tamilnadu. Indian Vet. J. **71**: 411-412

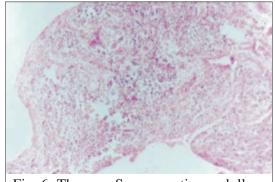
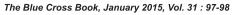


Fig. 6: Thymus: Severe corticomedullary lymphoid depletion





Occurrence and histopathology of sarcosporidiosis in esophagus of sheep(Ovisaries)

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Introduction

Sarcosporidiosis is caused by protozoa of the genus *Sarcocystis*, which is a member of the family Sarcocystidae. These parasites commonly and characteristically cause a chronic, subclinical infection in the esophagus, tongue and skeletal muscle of livestock by forming macrosarcocysts or microcyst in the striated muscle of sheep. This layer is called secondary cyst wall, or may be also formed by defense cells of the host and may later induce the calcification of the muscle fiber containing the parasite.

Material and methods

Sixteen samples of sheep esophagus naturally infected with macrosarcocysts of *S. gigantea were* collected from abattoir. Twelve of them were calcified, their grunting sound was heard after cutting by knife, fixed in 10% neutral buffered formalin, then after 48-72 hr dehydrated by ethyl alcohol 70-100%, cleaning by xylol and embedded in paraffin wax by acetone and benzene technique (Lillie, 1965).The microtones tissue sections at 4-6 im thickness were stained with H&E tech.

Result and Discussion

In the present study, esophageal sarcosporidiosis was found in 75 per cent cases.

Grossly; samples of infected esophagi showed white rice-grain sized nodules including calcified cheesy content and after cutting by knife, grunting sound was heard (Fig;1).Similar finding also described by Al-Hyali et al (2011).

Microscopically; In esophageal sarcosporidiosis, histopathology revealed typical granuloma. Calcified myofibers were locally surrounded by macrophage and numerous inflammatory cells and multinucleated giant cells (Fig; 2). Histological section of esophagi infected with *S. gigantea showed* merozoites (bradyzoites) releasing between muscle bundles. (Fig;3). Similar finding also have been described by Al-Hoot et al (2005) and Munday et al (1984).

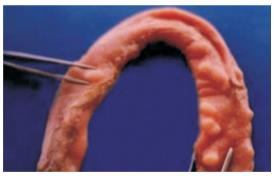


Fig 1: Gross photograph of esophagus showing white rice-grain sized nodules includingcalcified cheesy content.

References

Al-Hoot, A. S.; Al-Qureishy, S. A.; Al-Rashid, K. and Bashtar, A. R. (2005): Microscopic study on *Sarcocystismoulei* from sheep and goats in Saudi Arabia. J. Egypt. Soc. Parasitol. **35**: 295-312.



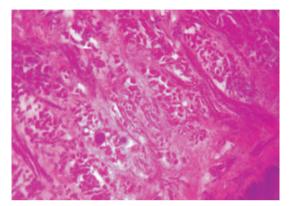




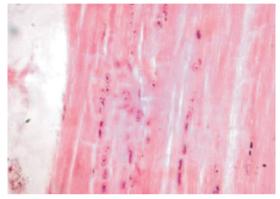
Al-Hyali, N. S.; Kennany, E. R. and Khalil, L. Y. (2011): Fate of macrosarcocyst of *Sarcocystisgiganteain* sheep. Iraqi J. of Vet. Sci.**25**: 87-91.

Lillie, R. D. (1965): Histopathological technique and Practical histochemistry, Mc Graw Hill Book co., New York and London.

Munday, B. L. and Obendorf, D. L. (1984): Morphology of *Sarcocystisgigantea* in experimentally infected sheep. Vet. Parasitol. **16**: 193-199.



Fig;2 Microphotograph of esophagus showing typical granuloma(blue dot) locally surrounded by macrophage and multinucleated giant cells.H&E 10x.



Fig; 3 Microphotograph of esophagus showing merozoites (bradyzoites) release in muscle bundles. H&E10x.

Peri-parturient Disorders : A hurdle in Dairy Development

Milk Fever : General suggestions for prevention

Enlightening dairy farmers to make them aware of the importance of nutritional management of their animals, particularly during pre-partum and peri-partum period. This could be a major initiative for preventing milk fever.

Prophylactic treatment of susceptible cows and buffaloes at calving, with either S/C calcium on the day of calving or oral calcium gels at calving and 12 hr later.

Since high potassium diets usually induce milk fever, pre-calving potassium levels be kept at as low as possible. As dry fodder contains more potassium, feeding dairy animals wholly on dry fodder be discoraged. Inclusion of silage and succulent green fodder should be a major portion of dry cow's diet.





News.... National...

India declared free of two important diseases -African Horse Sickness (AHS) and Contagious Bovine Pleuro Pneumonia



New Delhi - July 14, 2014 - India has been endowed with the official disease free status from a deadly and highly infectious arthropod borne viral disease of equines, namely, African Horse Sickness (AHS) on 25th May 2014 as per the resolution No. 19 (82nd General Session), according to the provisions of Chapter 12.1 of the Terrestrial Code by the World Health Organization for Animals (82 GS/FR/Paris, May 2014). India is now declared as a Member Country recognized free from African Horse Sickness. This proud and extra-ordinary achievement is a result of dedicated research involving virus isolation,

purification, diagnostic techniques and vaccine development at Indian Veterinary Research Institute, so also the efforts by National Research Center on Equines and Department of AHD, Government of India in obtaining the disease free status.

The AHS epizootic started in 1959 in Saudi Arabia and spread across Lebenon, Syria, Jordon, Iraq, Turkey, Cyprus, Iran, Afganistan, Pakistan to India and by 1961 killed almost 90% of affected equines, the toll reacting to 300,000 equines.

In India, AHS started in Jaipur in April 1960, wherein, 16 000 horses died out of 17845 affected. The disease affected horse population in several States, Maharashtra suffering the maximum loss of almost 28000 equines.



New Delhi - As per OIE resolution No. 17 (General Session 82 of May 2014), India, Argentina, Australia, Botswana, Canada, China, Portugal, Singapore, Switzerland and USA have CBPP (Contagious Bovine Pleuro Pneumonia) free status.

CBPP was recorded first time in India in 1942 in Asom (Golapara district). The disease was responsible for very heavy mortality among cattle in North-East region. The responsible micro-organism was *Micoplasma mycoides* subspp. mycoides. The eradication programme was

initiated in 2001. The country was declared provisionally free from CBPP in 2003. In 2007, OIE declared India free from CBPP and now this has been confirm in May 2014.





The Blue Cross Book, January 2015, Volume 31 : 100-101

Know the prestigious institute



ICAR-Directorate of Poultry Research Rajendranagar, Hyderabad-500030, Telangana, India





The Directorate of Poultry Research is one among the premier institutions in the field of poultry science research and extension in India. This institute was established on 1st March 1988 at Hyderabad, Andhra Pradesh under the aegies of Indian Council of Agricultural Research. Besides to coordinate and monitor AICRP on Poultry Breeding and other ICAR sponsored research programmes, the Directorate has been entrusted the task of developing germplasm suitable for rural poultry production and maintenance and improvement of elite broiler and layer pure lines; maintenance of random bred control populations; and two gene lines (Naked neck and Dwarf) for augmenting productivity under tropical climate. The institute has been elevated from its erstwhile position of Project Directorate on Poultry to Directorate of Poultry Research on 18th September 2013. In 2014, Directorate of Poultry Research has been certified with ISO 9001:2008 for the quality management system. Recently, the Institute has been awarded with the most prestigious Sardar Patel outstanding ICAR Institution award (Small Institution Category) for the year 2013 for its contribution in the field of Poultry research and extension.

With the **vision** of enhancing productivity of chicken for household nutritional security, income and employment generation and the **mission** of developing and propagating improved varieties of chicken for sustainable production under intensive and extensive systems, the mandate of the Institute has been as follows.



- To coordinate and monitor ICAR-sponsored network research programmes
- To undertake applied research on genetics and breeding, and conservation of improved chicken germplasm with supportive research on nutrition, disease control and management
- To lay emphasis on development of chicken varieties to meet the needs of rural/tribal and other underprivileged sections of the society

DPR has developed Vanaraja, Gramapriya and Srinidhi varieties for free range farming in rural and tribal areas, and Krishibro (coloured broiler) and Krishi layer (commercial layer) suitable for extensive and semi-intensive systems of rearing. White Gramapriya (Swethapriya), a layer for small scale intensive and rural poultry was also developed. In addition, the Directorate has been conserving native chicken germplasm such as Aseel, Ghagus and Nicobari. In addition, several high performing layer and broiler varieties were developed under AICRP on Poultry Breeding at different centres.

The Directorate has undertaken molecular characterization of chicken lines and molecular typing of birds. The whole sequence assembly of Aseel bird has been explored for the first time. Silencing of gene in chicken is in progress for improving productivity in birds.

Standardization of nutritional requirements of the varieties/ strains of broilers, layers and germplasm developed for rural/backyard poultry farming developed/maintained at this Directorate, Identification of novel/alternate feed ingredients, Improving efficiency of nutrient utilization and production of functional foods through nutritional manipulations were achieved.

PCR based assays for detection and differentiation of Mareks disease virus and Avian Leukosis virus; duplex PCR for detection of *Mycoplasma gallisepticum* and *Mycoplasma synoviae*; PCR-RFLP method for differentiation of field strains of *M. gallisepticum* from vaccine strains of *Mycoplasma gallisepticum* have been developed. The prevalence of mycoplasma and nephropathogenic IBV has been determined.

Trademarks have been obtained for the Gramapriya and Krishibro varieties, and two patents have been filed. The following Recognitions have been received for the contributions made by DPR and its scientists.

- Sardar Patel Outstanding ICAR Institution Award (Small Institute category)
- ICAR Tearm research Award
- ICAR Hari Om Ashram Trust Award
- ICAR Young Scientist Award (twice)
- ICAR Jawaharlal Nehru Award
- Hindi Implementation Award from Ministry of Home Affairs, Govt. of India
- CLFMA Award

So far, a total of 65.25 lakhs of germplasm were supplied by the Directorate in the form of fertile eggs, day old chicks and grown up chicks covering almost all the states in the country. The revenues generated during last 5 years are equivalent to 35-37% of the planned budget of the Institute.

The Directorate has also been actively involved in providing training, contract research, consultancy and contract services to the industry in the areas of poultry health and nutrition through public-private partnership mode.



The contributions to the journal are accepted in the form of review articles, research articles (clinical / field studies), case reports, other information pertaining to animal health and production. The decision of the Editorial Board members will be final regarding acceptance of the article for publication. The manuscript should be typed on one side of the paper with double spacing except for footnotes and references for which single spacing be used. The style of reference citing should be followed as shown below.

The manuscript should be arranged in the following order:

The manageripe should be any		
Title:		
Name/s of author/s:		
Place of work :		
Abstract :		
Key words :		
Introduction :		
Material and Methods :	(In details)	
Results and Discussions :		
Summary / Conclusions :		
Acknowledgment :	(If necessary)	
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Books :	Name/s of author/s., year of publication in parenthesis, title of the book, edition (Bold), name of publishers (Italics) and place.	
Tables and Figures:	Tables are to be numbered in Roman numbers (1 II and so on). Each table should have a clear title. Figures should be of good quality and numbered in Arabic numbers (1,2,3 and so on).	
Clinical articles and short communications:	Not exceeding 3 to 4 typed pages. In case reports, history, observation, tentative and confirmatory diagnosis, line of treatment and follow up on the case should be given. Trade names of drugs should be given in the Material & Methods and their details like composition, manufacturer etc. as a footnote.	
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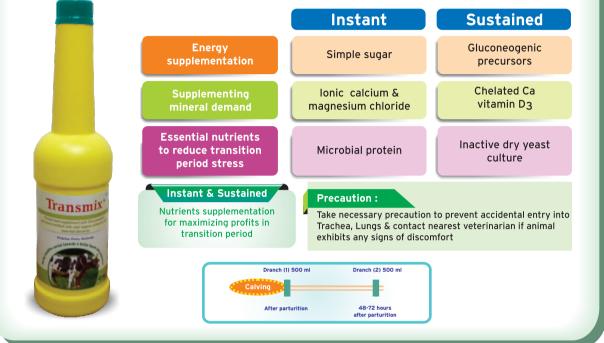
NEW INTRODUCTION

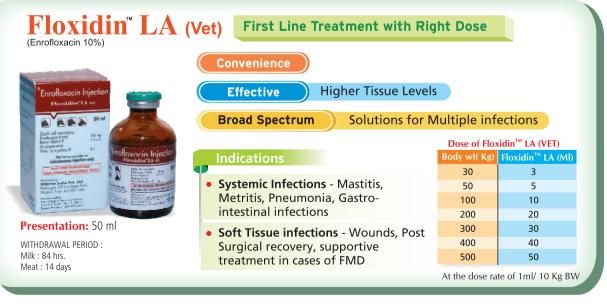


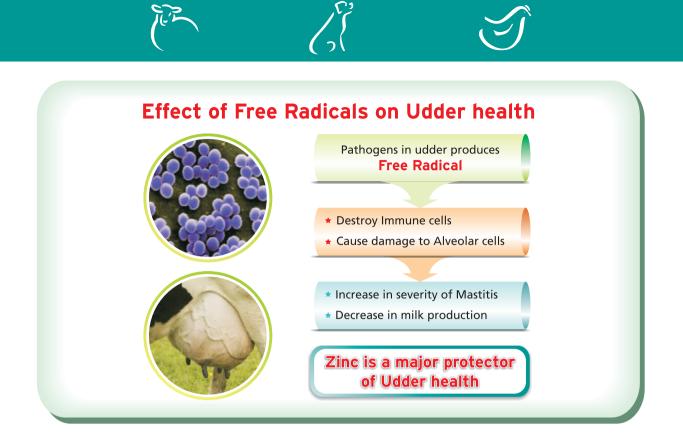




Transmix™



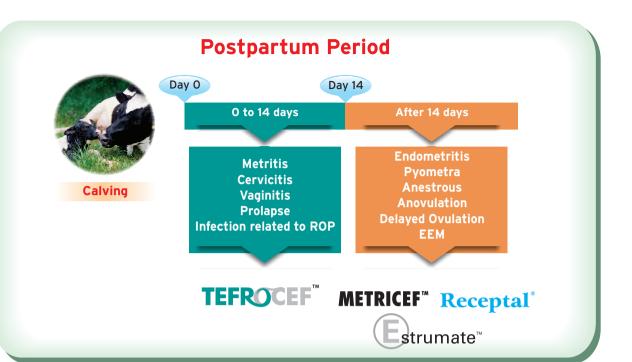












TEFROCEF A Solution to Many Problems...

1g vial with 20ml sterile water for injection, disposable syringe and needle.



WITHDRAWAL PERIOD Milk : 0 (Zero) days Meat : 4 days

COMPOSITION

Ceftiofur Sodium sterile powder equivalent to Ceftiofur.....1g One ampoule of sterile water for Inj. IP20ml

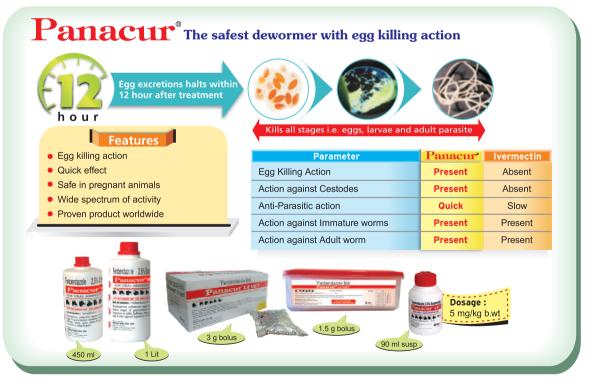
INDICATIONS

- Genital infections of bovine (acute metritis, cervicitis, vaginitis, prolapse related to ROP cases ets) associated with *Arcanobacterium pyogens, Fusobacterium necrophorum and Bacteroides spp.*
- Respiratory diseases of cattle, buffalo, sheep and goat (shipping fever, pneumonia) associated with *Pasteurella haemolytica, Pasteurella multocida* and *Haemophilussomnus*.
- Acute interdigital necrobacillosis (Foot rot, Pododermatitis) caused by *Fusobacterium & Bacteroides.*

DOSAGE AND ADMINSTRATION

Cattle, Buffalo, Sheep & Goat : 1.1 to 2.2 mg ceftiofur per kg body weight by IM route for 3 to 5 days.













Taktic[®] 12.5% EC (25ml) The Brand Advantage

Features	Advantages			
Brand Advantage	Quality , consistency and reliability			
	 Effective even after prolonged application and against all life cycle stages of ectoparasite. Ease of using specially in large and medium sized dogs. 			
Amitraz (12.5%) Advantage (amitraz-125 mg)	 Dose Rate- Demodectic mange-4ml/ltr of water Sarcoptic mange- 2ml/ltr of water Tick and lice: 2ml/ltr of water Weekly bath or dip to the dog till clinical symptoms subside. 			
Broad Spectrum	Effective against ticks , mange and lice			
Safety	• Not a organophosphorous compund, very safe . Rarely toxic and recovery is spontaneous			



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strumate[™] The Global Brand...



	Rx Discound Safer Sector Estrumate ^W
Annual Salar	Minist Michael and Minist Michael and The Annual Annual Annual The Annual Annual Annual The Annual Annual Annual The Annual Annual Annual The Annual Annual Annual Annual The Annual Ann
Strumate	€ MSD

WITHDRAWAL PERIOD

Milk : 0 (Zero) days Meat : 0 (Zero) days

COMPOSITION

Each ml of Estrumate contains 263 mcg of cloprostenol sodium, equivalent to 250 mcg of cloprostenol.

INDICATIONS

Induction of luteolysis in dairy cattle and horses-

- Anestrous,
- Subestrous, Luteal Cyst, Pyometra,
- Persistant Corpus Luteum (PCL),
 Expulsion of Mummified Foetus,
 Termination of Pregnancy,
- Induction of parturition Synchronization of Estrous

DOSAGE AND ADMINSTRATION

Cattle - 2.0 ml by IM route Ponies - 0.5-1.0 ml by IM route Thoroughbreds, hunters and heavy horses 1.0-2.0 ml by IM route





Ovilis[®] PPR



COMPOSITION

Live attenuated PPR virus with NLT $10^{2.5}\,\rm TCID_{50}$ per dose with suitable freeze drying stabilizer.

INDICATIONS

For the active immunization of sheep and goats in the control of PPR infection.

DOSAGE AND ADMINSTRATION

1 ml per animal by subcutaneous route.

PRESENTATION

Vials of 100/50/25 doses.

RECENT INTRODUCTION

Nobivac[®] KC

COMPOSITION

Contains both Brodetella bronchiseptica (Bb) and canine parainfluenza virus (CPiV)

INDICATIONS

Vaccination against "Kennel Cough"

DOSAGE AND ADMINISTRATION

Nobivac KC aims to make administration as easy as possible:

- Low 0.4 ml dose Single nostril only
- Can be used with or without applicator

PRESENTATION

One box contains 5 vials of dose and 5 vials of diluent along with one applicator



COMPOSITION

Scalibor P B 65 cm contains 1 gm of deltamethrin Scalibor P B 48 cm contains 0.76 gm of deltamethrin

INDICATIONS

• Anti tick, anti flea, anti sandfly and anti mosquitoes

DOSAGE AND ADMINISTRATION

One collar for six months. 65 cm (medium to large dogs) and 48 cm (smaller dogs)

PRESENTATION

6 x 65 cm and 6 x 48 cm.



KC







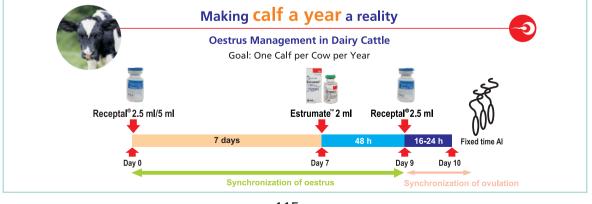


HORMONES

		Receptal [®] VET.		
	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
And the second s	Each ml contains : Buserelin acetate 0.0042 mg equivalent to 0.004 mg buserelin.	 True anoestrus Improvement of conception rate (at the time of AI) Ovarian cyst (Follicular), Irregular oestrus, Nymphomania Delayed ovulation & Anovulation Improvement of pregnancy rate (11-12 days post AI) Improvement of post partum fertility (10-15 days post-calving) 	5 ml, IM 2.5 ml, IM 5 ml, IM 2.5 ml, IM 2.5 ml, IM 5ml, IM	Vial of 10 ml and 2.5 ml WITHDRAWAL PERIOD Milk : 0 (Zero) days Meat : 0 (Zero) days

		CHORULON®				
	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION		
	Each vial contains human Chorionic Gonadotrophin	 Improvement of conception rate (cows/buffaloes) 	1500 IU at AI or mating, IM or IV	Box containing 5 vials (1500 IU		
124(Nami)	(hCG) as a white freeze-	 Enhancement of luteal function post AI 	1500 IU, 4-6 days post AI, IM	each) with 5 vials		
	dried crysta ll ine powder (1500 IU)	 Cystic Ovarian Disease (anoestrus, prolonged estrus, nymphomania) 	3000 IU, IV	of solvent		
		 Induction of ovulation (mares) 	1500-3000 IU, IM or IV, 24 hours before Al/mating	WITHDRAWAL PERIOD Milk : 0 (Zero) days Meat : 0 (Zero) days		

		FO	LLIGON®	
	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
And	Each vial contains Pregnant Mare Serum Gonadotrophin (PMSG) as a white freeze-dried crystalline powder (1000 IU)	Females: • Anoestrus • Super ovulation	Cow/Buffalo Anoestrus : 500 - 1000 IU IM Super ovulation: 1,500-3,000 IU, IM between day 8-13 of cycle	Box containing 5 vials (1000 IU each) with 5 vials of solvent
		 Increase of fertility rate after progestagen pre-treatment 	300-750 IU, IM, at the end of a progestagen treatment	Milk : 0 (Zero) days Meat : 0 (Zero) days







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ANTI-INFECTIVE

	COBACTAN [®] 2.5%		
COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each ml of suspension contains 29.64 mg Cefquinome Sulphate (equivalent to 25 mg Cefquinome).	 Cattle Respiratory disease caused by <i>Pasteurella multocida</i> and <i>Mannheimia haemolytica</i> Digital dermatitis, infectious bulbar necrosis and acute interdigital necrobacillosis (foul in the foot) Acute <i>E. coli</i> mastitis with signs of systemic involvement Calf <i>E. coli</i> septicaemia 	1 mg cefquinome/kg bw MI (2ml/50 kg bw) 1 mg cefquinome/kg bw MI (2ml/50 kg bw) 1 mg cefquinome/kg bw MI (2ml/50 kg bw) 2 mg cefquinome/kg bw MI (4ml/50 kg bw)	50 ml multidose vial. WITHDRAWAL PERIOD Milk : 1 day Meat : 5 days

COMPOSITION	INDICATIONS	DOSAGE PRE	SENTATION
Each syringe contains 75 mg Cefquinome sulphate as active ingredient.	For the treatment of clinical mastitis in lactating cows caused by Staphylococcus aureus, Streptococcus uberis, Streptococcus dysgalactiae, Escherichia coli & other entero-bacteria susceptible to cefquinome.	the infected quarter every 12 hours after each of 3 successive milkings. Milk out the affected quarter (s). After thoroughly cleaning & disinfecting the teat & teat orifice, gently infuse the contents of one syringe into affected quarter. Disperse the product by gently massaging the teat & udder of the fift and prime the contents.	of 3 injectors 3 isopropyl nol soaked els DRAWAL PERIOD *84 hours 2 days

		Floxidin [™] vet			
alarma a second alarma a second a secon	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION	
	injection : Each ml contains -	 Alimentary canal e.g. Enteritis, calf scours. Respiratory tract e.g. Pneumonia Urogenital system e.g. Metritis, cystitis Skin e.g. Bacterial dermatitis, pyodemra. Mastitis, & Haemorrhagic Septicaemia. 	Floxidin can be given once daily, for 3-5 days. Cattle, Sheep & Goat 2.5-5 mg/kg body weight IM Dog/Cat (adult) 5 mg/kg body weight IM Camel 2.5 mg/kg body weight IM	15 ml, 50 ml also available too mi WITHDRAWAL PERIOD Milk : 3.5 days Meet : 14 days	

		Tetracycline WSP VET				
* Treese ration de fais blands Water faiteride VET	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION		
	Each gm contains	In Sheep & Goat : Pneumonia, Joint ill, Anthrax, Septicaemia, Contagious Caprine Pleuro-Pneumonia, Scours, Acute Mastitis, Acute Metritis,	Sheep & Goat: 1 gm/kg body weight	Sachet of 100 grams		
A second	Tetracycline Hydrochloride WS I.P. 50 mg	In Cattle : Infectious diseases like Haemorrhagic septicaemia, Anthrax, Black Quarter, Leptospirosis, Foot Rot & Contagious Bovine Pleuro- Pneumonia, Calf Scours, Calf Diphtheria, Pneumonia, Septicaemia, Acute Metritis, Acute Mastitis.	Cattle : 2.5-5 gm/15kg body weight for 5 days	WITHDRAWAL PERIOD Milk : 7 days Meat : Cattle-15-22 days, Poultry-5 Days		

1		METRICEF™		
MARTIRIA	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
₽	Each single dose syringe of 19 g contains: Cephapirin - 500 mg (as benzathine) Excipient to - 19 g	 Subacute/chronic endometritis in cows over 14 days postpartum Repeat breeders (3 or more unsuccessful inseminations). 	Single dose syringe to be administered intra-uterinely	Single dose (19 g) syringe provided with a separate disposable catheter and a glove.

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PARASITE CONTROL

Ideally sui	but Highly effective & safe ector ted for control of ticks, mites, lice		
COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each ml contains : Deltamethrin BP 12.5mg	To control the ectoparasites in cattle, sheep, goats, horses, camels, dogs & farm houses.	Spray or dip: Ticks : 2 ml/lit Mites : 4 ml/lit Flies : 2 ml/lit Lice : 1 ml/lit	Aluminium container of 5 ml, 15ml, 50 ml, 250 ml and 1 lit with plastic measuring cup WITHDRAWAL PERIOD Milk : 0 (Zeno) day Meat : 20 days

		Tak	t ic ® 12.5% EC	
Conception of the		Broad spectrum ectoparasit	icide against ticks, mites, lice & keds	
Martin F	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
And the set of the set	Each ml contains : Amitraz I.P. (Vet) 125 mg	 For prevention & control of ectoparasitic infestation like ticks, mites, lice & keds in cattle, sheep, goat, camel & pig. Taktic kills tick, mite and lice. Taktic kills organochlorine, organophosphate & pyrethroid resistant strains of ectoparasites. 	Taktic 12.5%/lit of water for ticks : Cattle/Buffaloes/Camel: 2.0 ml Sheep/Goat : 4.0 ml Pigs : 4.0 ml Taktic 12.5%/L of water for mites and keds : Cattle / Camel : 2.0 ml Sheep/Goat : 4.0 ml Pigs : 4.0 ml Sheep/Goat : 4.0 ml Pigs : 4.0 ml	Tin Container of 15 ml, 50 ml & 250 ml with plastic measuring cup WITHDRAWAL PERIOD Milk : 7 hrs after applications Meat : 1 day for Cattle & Goats & 7 days for Pigs & Sheep

All and a second s		Panacur	[®] VET	
	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Ferbendazor Bol	The active ingredient of Panacur is Fenbendazole which is the research product of Intervet/Schering-Plough Animal Health. Each 1.5 g Bolus contains 1.5 g of active Fenbendazole. Each 150 mg tablet contains 150 mg of active Fenbendazole.	Infestation of cattle, buffaloes, sheep, goat & horses with gastro-intestinal nematodes, lungworms & tapeworms such as Haemonchus spp., Ostertagia spp., Trichostrongylus spp., Cooperia spp. and Nematodirus spp.	Recommended for cattle, sheep, goat, horses & pigs. Panacur 150 mg table per 30 kg body weight & Panacur 1.5 gm bolus per 300 kg body weight (5 mg Fenbendazole per kg body weight). Dose for horses : 7.5mg/kg bw	Box of 5 x 2'- 1.5 gm bolus Box of 5 x 2'- 3 gm bolus Box of 5 x 10'- 150 mg tablets. WITHDRAWAL PERIOD Mik: 0 (Zero) day Meat : 8 days

Panacur [®] VET Powder					
COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION		
Each gram contains Fenbendazole I.P (Vet) 250 mg	Infestations of cattle, buffaloes, Sheep & goats with gastro- intestinal nematodes, lungworms & tapeworms such as Haemonchus spp., Ostertagia spp., Trichostrongylus spp., Cooperia spp., Nematodirus spp., Neoascaris vitulorum, Oesophagostomum spp., Chabertia spp., Bunostomum spp., Gaigeria pachyscelis, Capillaria, Trichuris spp., Strongyloides spp., Dictyocaulus filaria, Dictyocaulus viviparus, Moniezia spp., Infestation of dogs with Ancylostoma spp., Infestation of horses with strongyles, Ascarids, Ascarids (Parascaris), Oxyuris & Strongyloides Infestation of pigs with Hyostrongylus rubidus, Oesophagostomum spp., Ascaris suum, Trichuris suis & Metastrongylus spp.	Recommended for cattle, sheep, goat & pigs. Infestation with gastrointestinal nematodes & lungworms : (5 mg Fenbendazole per kg body weight) Suspension to be made by mixing dean water as: 6 g with 100 ml 60 g with 1 lit. 120 g with 2 lit.	6 g sachet, 60 g & 120 g container WITHDRAWAL PERIOD Milk : 0 (Zero) day Meat : 8 days		







PARASITE CONTROL

		Panacur [®] VET	Suspension	
	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Contracts 255 Sector	Each ml of Panacur 2.5% suspension contains 25 mg Fenbendazole in 90 ml 450 ml and 1 lit pack.	Infestation of cattle, buffaloes, sheep & goats with gastrointestinal nematodes lungworms & tape worms such as Hoemonchus spp., Ostertagia spp., Trichostrongylus spp., Cooperia spp., Nematodyrus spp.,	Dose recommended for cattle, buffaloes, sheep, goats & pigs' infestation with gastrointestinal nematodes & lungworms: (5 mg Fenbendazole per kg body weight)	90 ml 450 ml and 1 lit HDPE bottle pack of Panacur 2.5% suspension. WITHDRAWAL PERIOD Mik : 0 (Zero) day Meat : 8 days

	Tolzan [®] Plus - L		
COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Oxyclozanide3.4% Levamisole Hydrochloride2.5%	 Tolzan Plus-L treats the round worms and liver flukes in cattle, sheep and goats Tolzan Plus-L controls adult and immature stages of conical flukes also (Paramphistomum spp.) Tolzan Plus-L can be used safely in pregnant animals during all stages of pregnancy. Tolzan Plus-L can safely be given to all cattle, sheep and goats without any pre-dosing, starving or change of diet. 	Cattle: 90 ml for 300 kg live mass PO Sheep and goats: 9 ml for 30 kg live mass PO	120 ml HDPE bottle, 1 Ltr can WITHDRAWAL PERIOD Milk : 0 (Zero) days Meat : 14 days

		Tolzan [®] F VET		
	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Oxycitzanie Bit Tolzavi v sus Partine Alexandrowie Company Alexandrowie	Each ml of suspension contains Oxyclozanide I.P (Vet) suspension of 34 mg	 Tolzan -F is used in the treatment of acute & chronic Fascioliasis in cattle, buffaloes, sheep & goats. The important species are : Fasciola hepatica Fasciola gigantica Tolzan -F is also used to treat paramphistomiasis. The species involved are : P. microbrothriodes, P. microbrothridium, P. gotal, P. orthocoelium Tolzan -F also acts on Monezia tapeworm in sheep. 	Cattle & Buffalo : Orally 10-15 mg/kg body weight Sheep & Goat: Orally 15 mg/kg body weight	90 ml HDPE bottle & 1 ltr jerry can. Also available as 1 gm bolus 1x3x10 strip pack. WITHDRAWAL PERIOD Milk : 0 (Zero) days Meat : 14 days

			nil [®] VET 7% RTU and the states of the sta	
And	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
	Each ml contains : Diminazine Aceturate 70 mg Phenazone B. P. 375 mg	Babesiosis & Trypanosomiasis, Tenacious Trypanosomiasis, Theileriosis & mixed infections, Pyrexia of Unknown Origin	Babesiosis and Trypanosomiasis at 5-10 ml per 100 kg b.w. Resistant strains of Trypanosomiasis at 10 ml per 100 kg b.w. Theileriosis & Mixed infections at 5 -10 per ml 100 kg b.w. along with antibiotic (3-4 antibiotic injections on alternate days)	Amber coloured vials of 20 ml, 30 ml and 90 ml WITHDRAWAL PERIOD Milk : 3 days Meat : 20 days









SUPPORTIVES

Tonophosphan[®] VET

Injectable phosphorus preparation for improving metabolism, milk production & fertility in livestock. Its content of organically bound phosphorus is 20%.

And the second se	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
	Each ml contains : Sodium salt of 4-dimethylamine, 2-methylphenyl- phosphinic acid 0.2 g	As a tonic in general metabolic disorders, debility, exhaustion, repeat breeding & infertility due to phosphorus deficiency. For disorders of bone formation as in rickets & osteomalacia. To promote callus formation in fractures in combination with calcium & vitamin D. For treatment of tetany & paresis resulting from calcium, magnesium & phosphorus imbalance (as in milk fever).	Large Animals : 5-20 ml. Small Animals : 1-3 ml. In chronic conditions- Large Animals : 2.5-5 ml Small Animals : 1-2 ml.	Vial of 10 ml and 30 ml also Now also available 100 ml vial

		VM	all [™]	
	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
VM ^{all}	Each Kg contains a nutritional value of : Cobalt 120mg, Copper 1000mg, Magnesium 5000mg, Iron 2500mg, Potassium 100mg, Manganese 2000mg, Flourine 60mg, Calcium 150g, Selenium 10mg, Vit A 1200000 IU, Vit D3 120000 IU, Sulphur 0.70%, Vit E 1200 IU, Iodine 300mg, Zinc 5000mg, Phosphorus 60g, Niacinamide 4g, Vit K 200mg, Sodium 8mg.	To improve on fertility. To safeguard health and growth. To optimize milk yield and fat.	Ruminants Mix 100-200 g per 10 kg of feed depending on the availability of other fodder/feed. For direct feeding, Cow and Buffalo: 25-30 g/head/day Calf, Sheep and Goat: 15-20 g/head/day Aqua: Mix 100g to 10 kg of fish feed.	1 kg Zip- Locked pouch with measuring spoon. 5 Kg & 25 Kg bag

					$\mathbf{V}\mathbf{M}^{\mathbf{all}^{\scriptscriptstyle{M}}}\mathbf{P}$		
		COMP	NOITICO		INDICATIONS	DOSAGE	PRESENTATION
VM all P A and a second secon	Each KG cor (When pack Cobalt Copper Iodine Iron Magnesium Manganese Potassium Sodium Sulphur Zinc	ed): 150 mg 2200 mg	Vit D3 Vit K Vit E Calcium	200000 IU 120000 IU 200 mg 500 IU 225 g 90 g	 To improve on fertility To safeguard health and growth. To optimize milk yield and fat. 	Ruminants Mix 100-200 g per 10 kg of feed depending on the availability of other fodder/feed. For direct feeding, Cow and Buffalo: 25-30 g/head/day Calf, Sheep and Goat: 15-20 g/head/day Aqua: Mix 100g to 10 kg of fish feed.	25 kg Sealed bag also available 5 Kg bag







SUPPORTIVES

Rumicare[®] Vet

		Normalises milk production by restoring rum	inal activity.	
Rumicare Vet	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
A standard of the standard of	Each gm powder contains : Calcium Propionate 480.00 mg Methionine 40.00 mg Picrorhiza Dry Extract 2.00 mg Cobalt Gluconate 0.32 mg Vitamin B6 IP 0.32 mg Dextrose Anhydrous IP 428.00 mg	Bloat, digestive disorders caused by decreased activity of reticulum & rumen or sudden dietary changes &/ or intoxication. As a supportive therapy in diseases caused by foreign bodies & hypo-glycaemic conditions in cattle, calves, sheep & goats.	Adult Cattle : 125 gm sachet twice daily, (once in 12 hours Young Animals : 65 gm (approx) once or twice daily Sheep & Goat : 32 gm once or twice daily	125 g & 500 g sachet

		Avilin® vet For quick relief from allergic manifest	ations.	
Personal Address of Assistance of	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
	Each ml contains: Pheniramine maleate IP 22.75 mg.	Itching due to eczema, dermatitis, urticaria, skin oedema, insect bites, photo-dermatitis, rhinitis, tail eczema in horses, stomatitis & inflammation of the hooves of cattle, serum sickness, paresis during pregnancy, toxaemia & retention of placenta, pulmonary oedema in cattle, pulmonary emphysema in horses.	Large animals : 5-10 ml. Small animals : 0.5-1 ml. or more. By IM or IV route	Amber coloured vial of Avil 10 ml and 33 ml WITHDRAWAL PERIOD Milk : 2 days Meat : 7 days

			Prednisolone Ace	tate Injection	
and here and			For quick relief from k	etosis.	
*****	-	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
a li sur		Each ml contains : Prednisolone acetate I.P. 10 mg	Prednisolone is indicated in ketosis in dairy cattle, shock, inflammations (especially rheumatic arthritis, dermatitis, bursitis) and	Cattle, horses : 5-20 ml. Calves, pigs : 2.5-5ml. Piglets, dogs, cats :1-3 ml. or as	Vial of 10 ml
-			allergic conditions of livestock	recommended by Veterinarian.	WITHDRAWAL PERIO Milk : 3 days Meat : 5 days

		Vetal	gin [®] vet		
		Highly effective analgesic, antispasn	nodic, antirheumatic	& antipyretic agent.	
	COMPOSITION	INDICATIONS	D	OSAGE	PRESENTATION
Californ of Analytic Veralgin veral Analytic Veralgin ver	Each ml contains : Analgin I.P. 0.5 g Chlorbutol (as bacteriostat) 0.4% w/v	For relief from pain, fever, labour, spastic condition of cervix during parturition, rheumatic conditions, neuritis, neuralgia, retention of placenta, dysentry, bloat & gastritis in domestic animals.	injection. Horse : 2 Cattle : 2 Foal, Calf : 5 Sheep, Goat : 2 Pig : 1	nous, otherwise combination of IV/IM 20-60 ml 20-40 ml 5-15 ml 2-8 ml 10-30 ml 1-5 ml	Vial of 33 ml WITHDRAWAL PERIOD Milk : 2 days Meat : Cattle 12 days/Pig 3 days & Horse IV 5 days









RUMINANT BIOLOGICALS



BU	VILIS Clovax		
COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Trivalent FMD vaccine contains inactivated and concentrated antigens of Foot and Mouth Disease virus serotypes O, A and Asia 1, adjuvanted with mineral oil sufficient to elicit > 3 PD _{so} as per Indian Pharmacopoeia regulations.	For the active immunization of cattle, buffalo, sheep and goats against Foot and Mouth Disease.	Cattle, Buffalo & Calves: 2 ml, Sheep & Goat: 1 ml by deep intramuscular route	Vials of 25 doses (50 ml).



	BOVILIS [™] HSBQ		
COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each vaccine dose contains inactivated anacultures of Pasteurella multocida and Clostridium chauvoei as water in oil emulsion sufficient to induce protective levels of antibodies against HS and BQ diseases	For the prophylaxis against Haemorrhagic septicaemia and Black quarter disease in cattle and buffaloes	2 ml of vaccine per animal by deep intra- muscular route	Vials of 100 ml(50 dose)



BRUCELLA ABORTUS STRAIN 19 VACCINE LIVE IP				
COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION	
Each vaccine dose contains 40 X 10° of live attenuated Brucella abortus strain 19 organisms in freeze dried form	For the active immunization of female calves of cattle and buffaloes against Brucella abortus infection	2 ml of reconstituted vaccine per animal by subcutaneous route only	Vials of 5 doses with sterile diluent	

	BOVILIS [™] ET		
COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
The vaccine contains highly immunogenic toxoids of <i>Clostridium perfringens</i> type D adsorbed on aluminium hydroxide gel as an adjuvant sufficient to induced protective levels of epsilon antitoxin titres in vaccinated animals.	For active immunization of sheep and goats against Pulpy kidney disease (Enterotoxaemia) caused by <i>Clostridium</i> <i>perfringens</i> type D	Sheep/Goats - 2 ml by subcutaneous injection only.	Vial of 50 doses (100 m l)

	Clostridium Pe	rfringens Vaccine Inac	tivated IP	
	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Controller Portion	Each vaccine dose contains inactivated anacultures of <i>Clostridium perfringens</i> types-B,C & D adsorbed on aluminium hydroxide gel sufficient to induce protective levels of beta and epsilon antitoxin titres in vaccinated animals.	For active immunization of sheep and goats against infections due to <i>Clostridium perfringens</i> types-B, C & D.	2 ml per animal by subcutaneous route	Vials of 25 doses (50 ml).







COMPANION ANIMAL



 INC			
COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each dose contains live attenuated strains of : Canine Parvo virus (strain CPV 154) $\geq 10^{7}$ TCID ₅₀ Canine Distemper virus (strain Onderstepoort) $\geq 10^{5}$ TCID ₅₀	Vaccination against CDV and CPV. Efficacious in puppies with maternal antibodies.	Reconstitute one vial of Nobivac Puppy DP in one vial of Nobivac Solvent & inject subcutaneously.	One box contains 10 vials of 1 dose.



	Nobivac [®] :DHPPi		
COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each dose contains live attenuated strains of : Canine Parvo virus (strain CPV 154) $\geq 10^7 \text{ TCID}_{s_0}$ Canine Distemper virus (strain Onderstepoort) $\geq 10^4 \text{ TCID}_{s_0}$ Canine Adeno virus type 2 (strain Manhattan LPV3) $\geq 10^4 \text{ TCID}_{s_0}$ Canine Para-influenza virus (strain Cornell) $\geq 10^{5.5} \text{ TCID}_{s_0}$	Vaccination against CDV, CAV2, CPV & CPi. Besides providing protection against CAV2 disease entities such as respiratory tract infections, the vaccine also protects against infectious canine hepatitis (ICH) caused by CAV1.	Reconstitute the contents of one vial of Nobivac DHPPi in one vial of Nobivac Solvent, Nobivac Lepto, Nobivac Rabies or Nobivac RL immediately prior to use & inject subcutaneously.	

		Nobivac®:Lept	0	
	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Notice LEPIO	Each dose contains inactivated strains of : Leptospira canicola (strain Ca- 12-000) \geq 40 hamster PD ₈₀ Leptospira icterohaemorrhagiae (strain 820k) \geq 40 hamster PD ₈₀	Active immunisation against Leptospirosis caused by <i>L.icterohaemorrhagiae</i> & <i>L.canicola of Leptospira interrogans.</i> Animals are protected against dinical disease, & also against becoming renal carriers after challenge.	Inject 1 ml of Nobivac Lepto subcutaneously. Nobivac Lepto can also be used to reconstitute Intervet's freeze dried vaccines Nobivac Puppy DP & Nobivac DHPPi.	One box contains 10 vials of 1 dose

	No			
NUMBER RABIES	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
	Each 1 ml contains inactivated Rabies strain Pasteur RIVM with potency ≥ 2 IU. The virus is grown on the BHK-21 clone CT cell line inactivated with β -propiolactone, and adsorbed on aluminium phosphate.	For the active immunisation of healthy dogs, cats, cattle, sheep, goats, horses and in principle all healthy mammals against Rabies.	1 ml by subcutaneous or intramuscular injection. Shake well before use.	One box contains 1 ml x 10 vials or one box contains 10 ml x 10 vials

		Nobivac®: RL		
Nobivace RL	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
	Each dose contains Rabies strain Pasteur RIV inducing more than 3 IU in the potency test, and inactivated strains of <i>Leptospira canicola</i> (strain Ca-12-000) \geq 40 hamster PD ₈₀ and <i>Leptospira icterohaemorrhagiae</i> (strain 820k) \geq 40 hamster PD ₈₀	For the active immunisation of dogs against rabies, and canine leptospirosis caused by <i>L.interrogans</i> serogroups <i>canicola</i> and <i>icterohaemorrhagiae</i> .	1 ml by subcutaneous injection. Can be used to reconstitute Nobivac DHPPi. Intended for dogs from 8 weeks of age onwards.	One box contains 1 ml x 10 vials.











COMPANION ANIMAL

		Ta	ktic° 5% EC	
tati Takiici	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
	Each ml contains : Amitraz I.P. (Vet) 50 mg	It is indicated for the topical treatment of Demodectic & Sarcoptic Mange, ticks & lice in dogs.	Mixing Rate / lit of water: Ticks & lice - 6 ml Mites - 10 ml 3-5 applications for mange and 2 applications for ticks and lice at weekly intervals. Taktic to be used as dip or spray	Glass bottle of 25 ml with plastic measuring cup

		San [®] Coat®		
	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Sorticar The state of the state	Essential Fatty Acids (Linoleic Acid, Alpha Linolenic Acid, Gamma Linolenic Acid, Eicosapentaenoic Acid and Docosahexaenoic Acid) Vitamins (Vitamin A and E, Biotin and Pyridoxine) Zinc and Inositol Omega 6 and Omega 3 fatty acids in 6:1 ratio	San Coat is indicated as an aid in the management of allergic and inflammatory skin conditions like alopecia, dull and dry hair coat, pruritis, atopic dermatitis, <i>Malassezia pachydermatis</i> , pyoderma, mange etc. in dogs.	Pour measured dose on food once daily according to the following schedule. 0.3 to 1.0 ml per kg body weight. Under 7 kg - 3.75 ml 7 - 23 kg - 7.5 ml Over 23 kg - 15.0 ml	Container of 150 ml (bettix shape)

	VM®365®			
(====	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
OT.	Vitamins and minerals	Beneficial for all dogs as a daily vitamin- mineral supplement, and especially during periods of stress, convalescence, growth, pregnancy and lactation.	For oral administration to dogs. Puppies and dogs under 10 lbs/4.54 kg – ½ tablet daily Dogs over 10 lbs/4.54 kg – 1 tablet daily	Container of 60 tablets

		DELVOSTERON [™]		
B	COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
DELVOSITION" DELVOSITION" DELVOSITION DEL	Each ml contains 100 mg proligestone	Suppression & postponement of oestrus in the bitch, treatment of pseudo pregnancy in the bitch, suppression and postponement of oestrus in the queen and suppression and postponement of oestrus in the ferret.	Dogs Body weight Dosage < 3 kg	20 ml Vials







POULTRY PRODUCTS

Live Vaccine

W		Nobilis [®] Gumboro 228	BE	
	COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
	The vaccine contains : Live I.B.D. virus strain 228E: >=2.0 $\log_{10} EID_{50}$	The vaccine is recommended for active immunization of chickens against Gumboro Disease (IBD)	One dose per bird through drinking water	1000 ds 2500 ds

	Nobilis [®] Gumboro D7	8	
 COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains : Live I.B.D. virus strain D78: >=4.0 log ₁₀ TCID ₅₀	The vaccine is recommended for active immunization of chickens against Gumboro Disease (IBD)	One dose per bird through drinking water	1000 ds 2500 ds

		Nobilis [®] ND Clone 30		
	COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
90.04E 30	The vaccine contains : Live ND strain Clone 30: $> = 6.0 \log_{10} ELD_{50}$	The vaccine is recommended for active immunization of chickens against Newcastle Disease	One dose per bird through drinking water, spray, intranasal/intra ocular	1000 ds 2500 ds 5000 ds

-			Nobilis [®] IB H120		
		COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
	The vaccine contains : Live IB strain H120: $> = 3.0 \log_{10} \text{EID}_{50}$	The vaccine is recommended for active Immunization of chickens against Infectious Bronchitis	One dose per bird through drinking water, spray, intra- nasal / intra-ocular	1000 ds 2500 ds 5000 ds	

		Nobilis [®] MG 6/85		
	COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
Rids J	The vaccine contains : Live M gallisepticum strain $6/85: > = 10^{69}$ CFU	The vaccine is recommended for active immunization of chickens to reduce the clinical signs of Mycoplasma gallisepticum infection.	One dose per bird through intraocular	1000 ds

Cell Associated Vaccine

Λ		INTOVAX [®] ND-SB1		
	COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The Grant State of State	Each ampoule contains per dose at least 1534 PFU of live HVT strain/NDV-F and 1514 PFU of live chicken Herpes virus strain SB-1 in the cell associated form	The vaccine is recommended for active immunization of chickens against Marek's Disease (MD) and Newcastle Disease (ND)	0.2 ml injection subcutaneously per chick in the neck	2000 ds 4000 ds







Inactivated Vaccine

		Nobilis [®] MG inac		
	COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
Colores and the second	The vaccine contains: Inactivated Mycoplasma gallisepticum	The vaccine is recommended for active immunization of chickens against infections caused by Mycoplasma gallisepticum.	0.5 ml S/C	500 ml (1000 ds)

-		Nobilis [®] E. coli inac		
THE REAL PROPERTY OF	COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
	<i>The vaccine contains:</i> E. coli fimbrial antigen (F11) E. coli flagellar antigen (FT)	The vaccine is recommended for passive immunization of broilers against colibacillosis by vaccination of broiler breeders	0.5 ml S/C or I/M	500 ml (1000 ds)

		Nobilis [®] Salenvac T		
-	COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The second secon	The vaccine contains: Inactivated Salmonella enteritidis PT4 and Inactivated Salmonella typhimurium DT104	The vaccine is recommended for active immunization of chickens against S. enteritidis and S. typhimurium and to give passive immunity against these agents in the progeny	0.1 ml for day-old chicks and 0.5 ml for older birds I/M	500 ml (1000 ds)

		Nobilis [®] Newcavac		
	COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
A CONTRACTOR	The vaccine contains: Inactivated ND Clone 30 virus	The vaccine is recommended for booster vaccination of layers and breeding stock for protection against Newcastle Disease throughout the laying period	0.5 ml S/C or I/M	500 ml (1000 ds)

	Nobilis [®] ND Broiler		
COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains: ND virus Clone 30	The vaccine is recommended for the vaccination of Newcastle Disease in day-old chicks in areas where ND is endemic	0.1 ml S/C or I/M	200 ml (2000 ds)

		Nobilis [®] Corvac		
	COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
CONTRACT	The vaccine contains: Inactivated Avibacterium paragallinarum strain 083 (serotype A), strain Spross (serotype B), strain H-18 (serotype C) in oil base adjuvant	The vaccine is recommended for protection against Avibacterium paragallinarum infections in chicken	0.5 ml S/C	500 ml (1000 ds)







<u> </u>		Nobilis [®] Coryza		
	COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
	The vaccine contains : Inactivated Avibacterium paragallinarum strain 083 (serotype A), strain Spross (serotype B), strain H-18 (serotype C) in saponin base adjuvant	The vaccine is recommended for protection against Avibacterium paragallinarum infections in chicken.	0.25 ml I/M or S/C	250 ml (1000 ds)

		Nobilis [®] Reo inac		
	COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
HO HO	The vaccine contains : Inactivated Reovirus strains 1733 and 2408	The vaccine is recommended for booster vaccination of breeding stock against Avian Reovirus to protect their offspring against Avian Reovirus infections	0.5 ml S/C or I/M	500 ml (1000 ds)

		Nobilis [®] G + ND		
	COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The set	The vaccine contains : Inactivated ND virus Clone 30 Inactivated Gumboro virus strain D78	The vaccine is recommended for booster vaccination of future breeders to protect against Newcastle Disease throughout the laying period, and to induce high maternal antibody levels against infectious Bursal Disease in their offspring.	0.5 ml S/C or I/M	500 ml (1000 ds)

 Nobilis [®] IB + ND			
COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains: Inactivated ND Virus Clone30, Inactivated Infectious Bronchitis virus strain M41	The vaccine is recommended for the booster vaccination of layers and breeding stock for protection against Newcastle Disease and the Massachusetts type of Infectious Bronchitis.	0.5 ml S/C or I/M	500 ml (1000 ds)

		Nobilis [®] IB multi + ND		
	COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
Hone Hone	The vaccine contains : Inactivated IB strain M41 Inactivated IB strain D274 Inactivated ND Clone 30	The vaccine is recommended for booster vaccination of layers and breeding stock for protection against the Massachusetts and D207/D274 (and related nephropathic) serotype of Infectious Bronchitis and Newcastle Disease.	0.5 ml S/C or I/M	500 ml (1000 ds)

		Nobilis [®] IB + G + ND		
	COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
A C 4 HO The second se	The vaccine contains : Inactivated IB strain M41 Inactivated Gumboro strain D78 Inactivated ND Clone 30	The vaccine is recommended for breeding stock: as a booster vaccination to protect against Newcastle Disease and the Massachusetts serotype of Infectious Bronchitis, and to induce high maternal antibody levels against Infectious Bursal Disease in their offspring	0.5 ml S/C or I/M	500 ml (1000 ds)









DOSE & ROUTE

0.5 ml

S/C or I/M

PRESENTATION

500 ml

(1000 ds)



COMPOSITION The vaccine contains : Inactivated IBV strain M41 Inactivated NDV virus Clone 30 Inactivated IBDV strain D78 Inactivated Reo virus strains 1733 and 2408

INDICATIONS
The vaccine is recommended for booster vaccination of breeding stock for protection against the Massachusetts serotype of Infectious Bronchitis and for protection against Newcastle Disease; and for immunization against Reovirus infection and Infectious Bursal Disease virus, in order to protect the offspring of the vaccinated birds against Reovirus infections and Gumboro Disease by maternal antibodies for at least the first weeks of life

Nobilis[®] Reo + IB + G + ND

Feed Supplement

		Enradin		
CURANU CIT	COMPOSITION	BENEFITS	INCLUSION RATE	PRESENTATION
	Each 1 Kg of Enradin contains 80 gm of Enramycine HCL	Helps in reducing incidence of sub- clinical necrotic entritis in chicken	5-10 ppm (63-125 gm) per ton of feed	20 Kg Withdrawal period - 7 days Avoid use in laying hens

		Amnovit [®]		
	COMPOSITION	BENEFITS	INCLUSION RATE	PRESENTATION
Annovic Allow and a second	Scientifically Balance formulation of vitamins and amino acids	Helps in relieving the stress conditions by supporting vitamins and minerals	Through water 1gm/lit for 5-7 days Through feed 500gm/ton for 5-7 days	1 Kg

Pharma Product

	Floxidin [™]			
	COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
	Enrofloxacin 10% oral solution	The product is recommended for treatment of the common infections caused by gram- positive, gram-negative, anaerobes and mycoplasma species	10 mg per kg BW for 3-5 days	5 Lt Withdrawal period - Meat - 8 days Eggs - Stop using 14 days before laying

VAC-SAFE	VAC-SAFE [®]					
	COMPOSITION	BENEFITS	INCLUSION RATE	PRESENTATION		
	An effervescent tablet that dilutes easily and neutralizes the chlorine in the water	Helps in improving the quality of drinking water during vaccination	1 tablet /100 Lt water	Box of 30 tablet		







The Science of Healthier Animals[™]

Animal Health

INTRODUCING

4



CANINE PLUS »

An advanced formula of 30 synergistic nutrients to support health in dogs.





CARDIO STRENGTH »

Unique formulation with L taurine, L Carnithine, DMG, Coenzyme Q10, Folate, Mg, EPA, GLA, Mg, K and Se to support specific cardiac function and ensure healthy heart.





DERMA STRENGTH»

Highly advanced formula with MSM, DMG, Methionine, Cysteine, Ascorbic acid, Proline, essential fatty acids, Zinc citrate, Hyaluronic acid, Vitamin B3, Vitamin A, and Perilla seed to support skin and coat condition.





GLYCOFLEX »

Balanced formula with Glucosamine, Perna canaliculus, MSM, DMG and Manganese to support joint heath in working , athletic and exercising dogs at all ages .





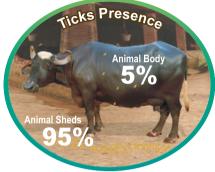
RENAL ESSENTIALS »

Highly superior formula with unique ingredients like Astragalus root powder, Rehmannia(chinese herb), Nettle(Urtica dioica), Cordyceps Sinensis extract to support specific kidney function and healthy and functional kidney.





Tick Eradication Program





For Application on Animal Shed

DUTOX[®]Vet Power

WITHDRAWAL PERIOD

Do You Know ?

Milk : 0 (Zero) day Meat : 20 days

For Application on Animal Body

Taktic[®]12.5% EC

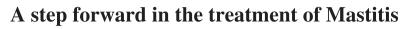
WITHDRAWAL PERIOD

Milk : 7 hrs after applications Meat : 1 day for Cattle & Goats & 7 days for Pigs & Sheep



Advantages

- Reduced tick load in animal shed
- Low incidences of tick born diseases
- Increased interval between two consecutive spray on animal body
- Better herd health





Control Measures for Mastitis

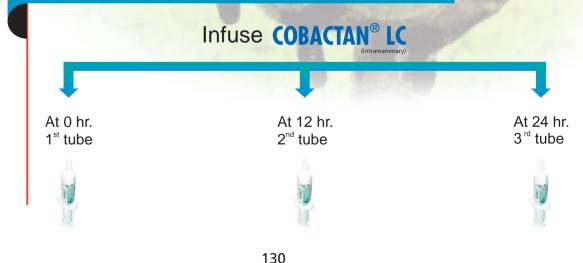
- Wash the hands with soap and water before hand milking.
- Clean the udder with antiseptic solution before & after milking.
- Use full-hand milking instead of knuckling.
- Allow animals to stand for 30 minutes after milking by providing feed or grass.
- Identify the chronic mastitic cow and milk them at last.

Advantages of Using Cobactan LC in Early Stages:

- Stops the Progression of Mastitis.
- Faster Recovery.
- Symptoms disappear quickly.
- Quick return to normal Milk production.

Withdrawal Period: Milk- 84 hrs. (7 milking) Meat- 2 days









A trusted source for comprehensive animal health solutions

Today's Merck is a global healthcare leader working to help the world be well. MSD Animal Health, known as Merck Animal Health in the United States and Canada, is the global animal Health business unit of Merck. MSD Animal Health offers veterinarians, farmers, pet owners and Governments the widest range of veterinary pharmaceuticals, vaccines, health management solutions and services. MSD Animal Health is dedicated to preserving and improving the health, well being and performance of animals. It invests extensively in dynamic and comprehensive R & D resources and a modern, global supply chain. MSD Animal Health is present in more than 50 countries, while its products are available in some 150 markets.

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