Nutritional & Health Management of Dairy Animals

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This e-book is a compilation of resource text obtained from various subject experts of GADVASU, Ludhiana & MANAGE, Hyderabad, on "Nutritional and Health Management of Dairy Animals".This e-book is designed to educate extension workers, students, research scholars, academicians related to veterinary science and animal husbandry about the nutritional and health management in dairy animals. Neither the publisher nor the contributors, authors and editors assume any liability forany damage or injury to persons or property from any use of methods, instructions, orideas contained in the e-book. No part of this publication may be reproduced or transmitted without prior permission of the publisher/editors/authors. Publisher and editors do not give warranty for any error or omissions regarding the materials in this e-book.

Published for Dr. P. Chandra Shekara, Director General, National Institute of Agricultural Extension Management (MANAGE), Hyderabad, India by Dr. Srinivasacharyulu Attaluri, Program Officer, MANAGE and printed at MANAGE, Hyderabad as e-publication.

MESSAGE

National Institute of Agricultural Extension Management (MANAGE), Hyderabad is an autonomous organization under the Ministry of Agriculture & Farmers Welfare, Government of India. The policies of liberalization and globalization of the economy and the level of agricultural technology becoming more sophisticated and complex, calls for major initiatives towards reorientation and modernization of the agricultural extension system. Effective ways of managing the extension system needed to be evolved and extension organizations enabled to transform the existing set up through professional guidance and training of critical manpower. MANAGE is the response to this imperative need. Agricultural extension to be effective, demands sound technological knowledge to the extension functionaries and therefore MANAGE has focused on training program on technological aspect in collaboration with ICAR institutions and state agriculture/veterinary universities, having expertise and facilities to organize technical training program for extension functionaries of state department.

Feeding dairy animals utilize the sciences of nutrition, biochemistry, and microbiology and combine them with animal husbandry. Scientific feeding of dairy animal is vital for their good health as well as to exploit their production and reproduction potential. Imbalance feed, and improper health management, hamper the profitability directly and indirectly. Proper understanding about feeding at different physiological stages of animals, their digestion, and their role helps in maintaining animal health and thereby good milk yield. Some of the specific strategies in feeding practice offer additional advantages in prevention of nutrition-related metabolic diseases. Other diseases can be controlled by adopting the scientific practices and biosecurity measures.

It is a pleasure to note that, SAU- Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana, Punjab and MANAGE, Hyderabad, Telangana is organizing a collaborative training program on on “Nutritional and Health Management of Dairy Animals” from 03-05 August, 2022 and coming up with a joint publication as e-book on “Health and Feed Management of Dairy Animals” as immediate outcome of the training program.

I wish the program be very purposeful and meaningful to the participants and also the e-book will be useful for stakeholders across the country. I extend my best wishes for success of the program and also I wish SAU- Guru Angad Dev Veterinary and Animal Sciences University (GADVASU), Ludhiana, Punjab many more glorious years in service of Indian agriculture and allied sector ultimately benefitting the farmers. I would like to compliment the efforts of Dr. Shahaji Phand, Center Head-EAAS, MANAGE, Hyderabad and the Director Extension Education, SAU-GADVASU, Ludhiana for this valuable publication.

Dr. P. Chandra Shekara
Director General, MANAGE

FOREWORD

Owing to decreasing cultivable land under population pressure coupled with its fragmentation over generations, plateau in production, increasing input cost and ever increasing demands of animal based food among society shifted the farmer focus from agriculture to livestock farming especially toward dairy. Milk and its products are the commodities whose demands will never subside in India as they are now an integral part of nutrition in almost all households in the country. Dairy farming is not new for Indians, they are doing it since the time of yore, but to reap its fullest potential cum profit, it should be done on scientific ground. Many success stories are there across the country where educated youth started the dairy farming on scientific basis and are now not only earning good income but also providing the livelihood to many others. Profitability of dairy industry revolved around feeding and health management of dairy animals. GADVASU - the leading veterinary university of the country is playing a pivotal role in upliftment of this sector through developing and disseminating, new and farmer friendly technologies to the end users by all possible means.

I am really delighted that our university is conducting a free online training program on "Nutritional and Health Management of Dairy Animals" sponsored by the National Institute of Agricultural Extension Management (MANAGE), Hyderabad for the Extension officials of state/central animal husbandry departments, veterinarians, faculty of SAUs/KVKs/ICAR institutes, etc. during 03-05 August, 2022 through Cisco Webex Online Platform. Both Nutritional followed by health management are pillars of success and ensure profitability. The lectures of this online course are exactly designed to expose the participants to various aspects of health and feed management of dairy animals. I hope that the participants from different parts of the country would be immensely benefitted from this online course by interactions with the expert resource persons selected for this training. I have no doubt that the course will be intellectually rewarding the participants.

The compendium for the above said training programme has been designed to provide first hand knowledge to the readers. I extend a warm welcome to all the trainees and wish them to have a fruitful and informative interaction.

Dr. Parkash Singh Brar
Director Extension Education,
GADVASU, Ludhiana

PREFACE

India has the largest population of livestock in the world. Cattle and buffalo constitute over half of the livestock population. As such, dairy farming is the backbone of livestock sector and agricultural GDP. Evidence suggests that distribution of livestock among farmers is more equitable as compared to land. Besides, it presents opportunity for entrepreneurship in processing, value addition and feed manufacturing. The demand for milk and milk products is expected to grow further in the future with the demand for milk and milk products in the domestic as well as international market. However, the sector is faced with challenges that threaten to derail the growth in dairy sector. Nutrition and animal health are interrelated and demand utmost attention to reap the maximum profit from this venture. Topics have been carefully selected to offer possible solutions to common problems/ issues encountered in a dairy farm. This e-book was made possible by the sincere efforts of the contributing authors.

This e-book is an outcome of collaborative online training program on "Nutritional and Health Management of Dairy Animals" conducted from 03-05 August, 2022. This book will be highly useful to field functionaries as well as extension workers who are working at the ground level. A myriad of topics from nutrition to reproduction and management of major diseases has been covered for the benefit of the readers.

The editors express sincere thanks to Dr. Inderjeet Singh, Hon'ble Vice-Chancellor, GADVASU Ludhiana, for encouragement in publishing this e-book. The financial aid provided by MANAGE, Hyderabad for this training program is duly acknowledged. We hope and believe that the suggestions made in this e-book will help to improve the ability of all the stakeholders to improve health and nutritional management of dairy animals.

August, 2022

Dr. Parkash Singh Brar
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Chapter 1

FEEDING STRATEGIES TO RAISE A HEALTHY HEIFER

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"The ultimate measure of success of the heifer rearing program is how well heifers perform once they are in the milking herd."

Today's female calves are futures cows. Future's of dairy industry depends upon these female claves, their health and upbringing. The goals for raising heifers have changed over the years as weight at first calving has taken precedence over age. A heifer that is well-grown and calves a little later is preferable to one who calves at 24 months but is undersized. In the milking herd, an undersized heifer would probably have difficulty during the first lactation and divert nutrients from production to growth. Calf management needs to be appropriate and adequate for the dairy sector to succeed. By enhancing farm-level management measures that aim to minimize calving intervals, boost calving and fertility rates, reduce stillbirth and pre-weaned calf mortalities, and minimize unhealthy heifers in the herd, it is possible to efficiently and economically replace the dairy herd. Growth begins during fertilization and occurs mostly (by about 70%) in the third trimester of pregnancy. Because birth weight and growth rate are strongly correlated, the intrauterine growth of the fetus has a significant impact on postpartum growth. Calves born to indigenous cows usually weigh 20 to 25 kg, whereas those born to crossbred and Jersey cows often weigh 30 to 35 kg and 18 to 25 kg respectively, while buffalo weigh 25 to 30 kg.

Heifer health starts before birth by making sure that dry cows are fed appropriately, are in good physical condition, have had vaccinations, and calve in a comfortable environment. The care and management of cows in late pregnancy are crucial for the birth of a healthy calf. The critical period, when the fetus grows at its fastest rate, is the final two months of gestation. A pregnant cow's diet should be adequate to support both the growth of the fetus and its own maintenance needs. The nutrients should be sufficient in terms of calories, protein, vitamins, and

minerals. Neglected feeding or underfeeding slow down te growth of fetus. One of the major obstacles to a dairy enterprise's success continues to be calf mortality, which causes a significant economic loss. Worldwide, newborn calf mortality ranges from 8.70 to 65.00 percent, making up 84.00 percent of all deaths in the first month of life. It is highest in the third week following birth (Jenny et al., 1982; Umoh, 1982). Cattle and buffalo calf mortality typically varies from 29.10 percent to 39.80 percent (Afzal et al., 1983). The dairy farm typically accepts a minimum mortality rate of 5.00% under standard management settings. Therefore, nutritional control of calves lowers mortality, and the farm's herd health status remains favorable.

General calf management

There are three stages to consider while managing the calves;

Prenatal management

Adequate nutritional management at last quarter of pregnancy ensure rapid growth of calf as well as attaining puberty in female calves at an early age. To compensate the decreased DMI during this stage, high quality and densified feed should be used to ensure proper nutrient intake. Bypass fat (calcium salt of fatty acids) and bypass protein (rumen undegraded protein) can be used in the ration of pregnant animals at this stage. Adequate quantity of good quality green fodder (20-25Kg) and concentrate (1-2.5Kg) should be offered to animal, along with 24X7 to clean water. The animal needs to be immunized at the proper period and safeguarded from contagious diseases. Lactating animals should be dried 60 days before calving so that nutrients can be channelized for foetus and maternal growth.

Parturition (Calving) Care

One week before calving, the animal should be shifted to a separate and clean calving pen with provision of adequate bedding material.During this time animal should be provided with bran mash with one kg of boiled millet/grains and a handful of jaggery to avoid constipation.This will also help in increasing milk production but will also help in expulsion of the fetal membranes.

Postnatal Care

The mucus from the mouth and nose of the calf needs to be cleaned after birth.Artificial respiration should be performed if the calves do not begin to breathe on their own.It is necessary

to clean the calf, ligate the navel cord, and take precautions against the common cold and infectious infections.The calf's navel cord is cut about 1 cm below the ligature and knotted about 2.5 cm away from the body.To combat infection, 7 percent iodine tincture is applied to the site.Reticulo-rumen in calves is non-functional from birth; as a result, calves should be fed throughout the first three months of life as a non-ruminant animal.A calf's life is most vulnerable during the first two to three weeks; at this time, the digestive system is not mature enough and still growing in terms of digestive secretion and enzyme activity

Phases of digestive system development of calves:

The calves' feeding system is separated into three stages. Diet requirement for different stage is mentioned below:

1. Liquid feeding Phase (Birth to 2 or 3 weeks): Milk or high-quality milk replacer and water

2. Transition Phase (2 or 3 weeks to weaning): Milk or high-quality milk replacer, calf starter, and water

3. Ruminant Phase (From weaning onwards): Dry feed and water

Colostrum feeding

Colostrum is the first secretion of a cow after calving. It is thick and yellow in color. It contains 4 to 5 times more protein and 10 to 15 times more vitamin-A than normal milk. Colostrum is also rich in minerals like Cu, Fe, Mg, and Mn. It also contains several other vitamins like Riboflavin, choline, Thiamine, Pantothenic acid, etc., which are important for the growth of calf. Large amount of gamma globulins (antibodies) present in colostrum provides the calf with an umbrella of passive immunity. Colostrum provides a laxative effect which is helpful in the expulsion of meconium (first faeces). The quantity of colostrum to be fed is 1/10th of body weight. Quality, quantity and quickly are three pillars of colostrum feeding. A bottle with nipple is a easy way to ensure every calf has the recommended amount of colostrum quickly after birth. Excess feeding can results in calf scour. It can be stored by refrigeration and can be used for other calves or orphan calves.

IgG, IgA, and IgM are the primary colostrum antibodies. IgG constitutes 80 to 85 percent of all IGs in colostrum. Half-life of IgG is 21 days. IgA comprises 8 to 10 percent. Half-life of

IgA is 4 days. IgM makes up 5 to 12 percent. Half-life of IgM is 2 days. Gama globulins must be absorbed as such across the intestinal wall into blood stream without being broken down into the constituent peptides or amino acids. This permeability is rapidly lost after the first few hours of life. These globulins pass across the gut wall at the most rapid rates during the first 1-2 hours of life. So highly useful to feed colostrum in the first 15-30 minutes followed by a second dose in approximately 10-12 hours. As the calf grows older hour by hour, there is a transition of epithelial cells of small intestine from immature type to mature type which cannot allow large protein molecules. As more and more cells mature, the capacity of the calf to absorb immunoglobulins diminishes proportionately until 'closure' when no more absorption can take place. This phenomenon is called 'gut closure'. The non-nutritive substances insulin-like growth factor I and II, epidermal growth factor, transforming growth factor, and nerve growth factor, as well as insulin, cortisol, and thyroxin, are all abundant in colostrum. Colostrum also reported to have bactericidal capabilities as well.

Effects of early colostrum feeding on intestinal *E.coli* attachment in calf

Feeding	Results
E.coli fed alone	Bacterial attachment to intestine and level of *E.coli* in circulation high
Colostrum and *E.coli* fed together	No bacterial attachment to intestine
Colostrum fed alone, *E.coli* fed after one-hour later	No bacteria attached to the intestine and no *E.coli* in circulation. High level of circulating antibodies

Source: J Dairy Science 60: 1416-1421

In addition to its nutritional value, colostrum has a laxative effect that aids in eliminating faeces that have accumulated in the intestines (Meconium).

Composition of colostrum and milk

Items	Colostrum			Milk
	1st Milking	2nd Milking	3rd Milking	
Solid (%)	23.9	17.9	14.1	12.9
Protein (%)	14.0	8.4	5.1	3.1
IgG(mg/ml)	32.0	25.0	15.0	0.6
Fat (%)	6.7	5.4	3.9	4.0
Lactose (%)	2.7	3.9	4.4	5.0
Minerals (%)	1.1	1.0	0.8	0.7
Vitamin A (ug/dl)	295.0	190.0	113.0	34.0

Source: J Dairy science 61:1033-1060

In case of non-availability of colostrum due to accidental death of mother or agalactia, colostrum substitute can be used. It can be prepared by mixing 2 whole eggs in one litre of milk and 30 ml of castor oil. It should be fed three times a day.

Feeding of milk replacer

The most natural nutrition for calves is whole milk, but because it is expensive and competes with humans for food, it is effectively substituted by a calf milk replacer. A milk replacer is made up of high-quality, inexpensive components and has a comparable nutritional profile to milk. It is a more cost-effective option than whole milk for producing calves. It is given to calves until their rumens begin to operate properly. On the tenth day after birth, it is introduced, and its volume can be gradually increased by reducing the amount of whole milk. Calves' proper growth and development are aided, and calves' mortality is decreased. In order to sustain core body temperature in cold weather, extra energy should be consumed. The calf needs to be given care in cold and chilly weather. The calf needs to be given 25–50% more whole milk or milk replacer during colder months. Milk replacers should have 10 to 22% crude fat, 18 to

22% crude protein, and 0.52% crude fiber. Milk replacers with 15 to 20 percent fat may help to reduce scours and encourage faster growth. The ingredients used to create milk replacer should be of high biological value and good quality. Volatile Fatty Acids are the primary products of rumen fermentation and contribute to rumen epithelium development in calves. Studies suggested that infusion of sodium propionate or sodium butyrate greatly promotes the development of the rumen papillae in calves.

Composition of milk replacer

Ingredients	Quantity (kg)
Wheat	10
Fish meal	12
Linseed meal	40
Milk	13
Coconut oil	7
Linseed oil	3
Citric acid	1.5
Molasses	10
Mineral mixture	3
Butyric acid	0.3
Antibiotic mixture	0.3
Vitamin- A, B_2, D	0.015

Water: Water is the most overlooked aspect of calf-raising program. One very important role that water plays is in rumen development. When calf drinks water, the water goes into the rumen, as does the starter feed, while milk fed from bottle or bucket, by-passes the rumen via esophageal groove, and is deposited into the abomasum of the calf. Water in the rumen helps the microbes to live in and grow. Without water ruminal development is slowed. Offering water separate from

milk will increase weight gains by increasing dry feed intake at an early stage. So start offering fresh and clean water (free from *E coli* and *Salmonella* etc) to calf from first week of life.

Feeding of calf Starter

Calves are completely dependent on a diet of whole milk or milk replacers until they are 15 days old. Following that, they are fed fluids and calf starter till the establishment of the rumen. Ground grains, oil cakes, animal protein supplements, and brans supplemented with vitamins, minerals, and antibiotic feed additives make up the solid feed known as "calf starter". Calf starter ingredients can be changed based on availability and feed price. It ought to contain more protein, energy, and digestibility. Calf starter should contain between 23 and 26 percent crude protein and between 75 and 80 percent total digestible nutrients. As a low proportion of fibrous material promotes starting intake and calf growth, it should have less than 7% fibre. Because it contains easily fermentable carbohydrates, feeding high-quality, palatable calf starter is important. These carbohydrates are fermented to produce volatile fatty acids, which are necessary for the early development of the rumen. This might facilitate the calf's early weaning. Good quality fibre in hay/forage helps in saliva production and rumination activity which ultimately aid in rumen development. It also mitigate the chances of ruminal acidosis.

Composition of Calf starter

Ingredients	Quantity (Kg)
Barley	40
Groundnut cake	25
Soybean cake	25
Dried skim milk	8
Mineral mixture	1
Vitamins	1
Irradiated yeast	25 g/100 Kg

Feeding schedule of calves up to three months

Age (days)	Colostrum (kg/day)	Whole milk (kg/day)	Calf starter (kg/day)	Good quality hay (kg/day)
1-4	$1/10^{th}$ of BW	-	-	-
5-14	-	$1/10^{th}$ of BW	-	-
15-28	-	$1/20^{th}$ of BW	0.1	0.25
29-42	-	1.0	0.3	0.40
43-56	-	0.5	0.5	0.50
57-70	-	-	0.75	0.75
71-90	-	-	1.0	1.00

Micronutrient dietary supplements

Some vitamins and minerals, including Fe, Zn, and Vitamin C, are inadequate in calves given milk and milk replacers. These micronutrients can be given as supplements to calves starting on day one or included in milk replacers or calf starters. To increase the calves' resistance to diseases and antioxidant status, several vitamins and minerals, including Cu, Zn, Se, Mn, Vitamin A, C, and E, can be given as supplements. When compared to inorganic minerals, organic mineral chelates have been proven to yield more favorable outcomes. For example, organic zinc has been reported to be more bioavailable, to show better average daily gains, and to use feed energy more efficiently (Malik et al., 2017).

Post-weaning feeding of the calf

Early weaning is a possibility if the calf begins consuming dry feed earlier (about 1.0-1.5 kg/day), and it is also cost-effective for the farmer. At the end of the second week, calf starter should be offered. Feed should not contain more than 10 per centfiber. Because calves have a limited ability to use non-protein nitrogen, urea is not added to feed, however, 5-7 percent molasses can be added to make the meal more palatable. Adult animals ration or lactating animal

ration should not be fed to calves as it have less protein, high fiber content and sometime also contain urea.

Feeding of Pre and probiotics to Calves:

Probiotics are viable and beneficial microorganisms that help maintain GI microbial balance and promote rumen development.Prebiotics are the substances that aid in development of benefecial microorganism. Feeding probiotics to calves around weaning age may facilitate the development of rumen bacterial communities and help calves with a transition from liquid feed to dry feed.

Feeding of heifers and growers

The transition from starter to growth ration occurs when the calf reaches the age of three months. To feed during this stage, high-quality ad-lib fodder, 0.5 kg of wheat straw, and 1.5–2 kg of grower concentrate are required. Protein should make up 15 to 18 percent of the diet. In order to create the grower concentrate, add 55 kg of maize, 15 kg of oats, and 20 kg of soyabean. 7 kg of molasses, 2 kg of limestone powder, 2 kg of DCP, 0.5 kg of minerals, and lower doses of vitamins A, D, and E are also included. Between 80 and 90 days of age, calves are offered grower feed. Growers switch to heifer meal at six months of age.

After attaining the age of 6 months, heifers can be fed on concentrate with 20% CP and 63% TDN. Avoid high nutrition levels since they slow down the growth of the milk secretory tissue, thus reducing the capacity for lifetime milk production. They can get all the nutrients they need from about 5-7 kg of maize silage (>30% DM), 2 kg of wheat straw, and 2 kg of concentrate mixture per head each day. 30 kg maize, 15 kg mustard cake, 15 kg soyabean meal, 20 kg wheat bran, 17 kg rice bran, 2 kg mineral combination, and 1 kg salt can be combined to create the concentrate mixture. The dietary addition of vitamins, feed additives, and appropriate management will also be proven to be a significant step in enhancing heifer performance.

References:

Afzal M, Javed MH, Anjum AD. Calf mortality: Seasonal pattern, age distribution and causes of mortality. Pakistan Vet J, 3:30-33, 1983.

Bebe BO, Abdulrazak SA, Ogore PO, Ondiek JO, Fujihara T. A note on risk factors for calf mortality in large-scale dairy farms in the tropics: A case study on Rift Valley area of Kenya. Asian-Aust J AnimSci, 14:855-857, 2001.

Blom JY. The relationship between serum immunoglobulin values and incidence of respiratory diseases and enteritis in calves. Nord Vet-Med, 34:276-284, 1982.

Blood DC, Radostits OM. Veterinary Medicine, 7th Ed, ELBS, Oxford, 1989.

Jenny BF, Cramling GE, Glaze TM. Management factors associated with calf mortality in South Carolina dairy herds. J Dairy Sci, 64:2284-2289, 1981.

Malik TA, Mir SH, Pal RP: Chelated Versus Inorganic Zinc Supplementation in Ruminants. Nutri Food SciInt J, 4:555633, 2017.

Moran JB. Factors affecting high mortality rates of dairy replacement calves and heifers in the tropics and strategies for their reduction. Asian-Aust J AnimSci, *24*:1318-1328, 2011.

Umoh JU. Relative survival of calves in a university herd in Zaire, Nigeria. British Vet J, 138:507-514, 1982.

White DG, Andrews AH. Adequate concentration of circulating colostral proteins for market calves. Vet Record, 119:112-114, 1986.

Chapter 2

NUTRITIONAL MANAGEMENT OF DAIRY ANIMAL DURING TRANSITION PERIOD

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Essentially, one involved in dairy farming desires cows produces milk up to its genetic potential, calves every year and minimum health problems. Nutritional management determines the success dairy farming, especially during the transition period. The transition stage is defined as a two-month period before and after calving, with the most critical time being 14-21 days before and 14-21 days after calving. Typically, dairy animals calve and transition from the dry to milking stage as a result of physiological and hormonal changes. However, maintaining these targets at the farm level is difficult. Cow is at highest risk of health disorders during this stage as a result of failure of management including nutrition and others, affects physiological and metabolic adaptation.

The DMI of dairy cows begins to decline at 3 weeks prepartum because a foetus grows rapidly during this period and various stresses can negatively impact hormone secretion. The pre-partum transition period's average dry matter intake (DMI) has been reported to range between 1.7 and 2.0 percent of body weight (BW). The drastic decrease in voluntary DMI is primarily due to a decrease in rumen volume caused by the increased weight of the foetus and uterus. At four days postpartum, the mammary energy requirement is estimated to be three times that of the uterus. To match the rapidly increasing milk production, the demand for glucose for lactose synthesis increases immediately after calving, when feed intake has not yet reached its maximum. Milk production peaks around 5-8 weeks postpartum, while diet consumption peaks around 10-14 weeks postpartum. As a result, dairy cows typically suffer from negative energy balance (NEB) for 6-8 weeks after giving birth. The significant increase in nutrient requirements following parturition has been quantified in terms of mammary demands for milk lactose, fat, and protein synthesis versus the conceptus's lower prepartum needs for glucose, fatty acids (FAs), and amino acids (AAs). Similarly, requirement for calcium and other mineral also

increases with increase in a milk volume. Animals with superior genetic homeorhetic traits can direct the majority of their nutrients toward milk production. Elevated non esterifies fatty acids (NEFA) concentrations frequently result in significant NEFA conversion to ketones, such as BHBA, in the liver. Elevated NEFA concentrations may impair neutrophil viability and high BHBA concentrations in the blood can cause Subclinical ketosis (SCK).SCK is known to suppress the immune system and acts as a gateway to several other production diseases. The calcium requirement of the mammary gland for producing 10 kg of colostrum on the day of parturition is more than double that of foetal growth in late gestation. The onset of lactation places such a high demand on calcium homeostasis mechanisms that most cows develop hypocalcaemia at calving. In some cases, plasma calcium concentrations become insufficient to support nerve and muscle function, may result in parturient paresis or milk fever.

Reduced Dry matter intake

The drops in DMI can be up to 30-35 percent during the last three weeks of pregnancy. Low DMI can also be caused by health problems that cause inflammatory conditions (such as metritis, mastitis, lameness, and so on) by releasing pro-inflammatory cytokines, which are proteins (5-20 kDa). Cows with the highest inflammatory indices appear to be affected by a NEB and increased -hydroxy butyric acid levels (BHBA). Retained placenta, metritis, and endometritis are all diseases of immune function that begin at least 2 weeks before calving. The distribution of production diseases indicates which areas of nutrition require attention during the transition period. A high rate of lameness, for example, is more indicative of rumen acidosis, whereas a high rate of displaced abomasum could be due to subclinical ketosis (SCK) and/or hypocalcaemia. NEB, BCS, rumen health, calcium status, trace elements, and antioxidant status in blood are areas to monitor.

BCS management

The energy intake through diet cannot fill the requirement of peak lactation curve at initial phase. As a result, animals experience negative energy balance (NEB) and begin mobilising body reserves for at least 6 weeks, which a normal physiological procedure. Negative energy balance (NEB) induces catabolism of body tissue to meet energy requirements during late gestation and early lactation, resulting in decreased BCS, rumen fermentation, and milk production, as well as the possibility of triggering metabolic syndrome. The BCS during the

lactation cycle is most likely the most important aspect of dairy cow management for ensuring a healthy transition from gestation to lactation. Over conditioned dairy cows (BCS>4.0) have a much greater reduction in feed intake immediately before calving than under conditioned cows. While feeding such cows, the goal should be not to lose more than one BCS (in the scale of 1–5) after calving, which could be managed by maximizing DMI during the close-up period, which in turn depends on the dietary factors affecting hunger and satiety centres in the brain of mammals.

Nutritional Strategies for transition cows

Increasing feed intake

The first goal of gradually increasing DMI in transition animals by providing easily digestible diet is to stabilise rumen health, improve ruminations, and increase feeding frequency. Different interventions in diet formulation, such as monitoring fibre content and effective particle size, as well as soluble fibre sources, can improve DMI intake during the close-up stage and for a few days after calving. Few studies have found that including aromatic compounds improves DMI in animals. These include essential oils, herbs, spices, and extracts with flavouring properties, particularly aromatic phenolic compounds found in thyme, clove, and other herbs and spices. A phytogenic feed additive improved feed and energy intakes (1.2 percent) in cows in a recent trial in the Czech Republic. Furthermore, milk yield increased by 5.1 percent, as did daily milk solids production (fat by 6.7 percent, protein 1.7 percent, lactose by 4.8 percent). The energy corrected milk (ECM) was improved by 3.5%. More importantly, optimal nutrient composition, roughage concentrate ratio, and particle size, as well as increasing or decreasing dietary NFC or NDF, can all strongly stimulate DMI during the transition stage.

Increasing feed conversion efficiency (FCE)

The second tool for assisting the newly calved cow (i.e., immediately following parturition) is to increase FCE by increasing digestibility and efficiency of nutrient utilisation from the feed. This can be accomplished by feeding beneficial rumen microbiota functional components such as nucleotides and glucans. While a variety of yeast products contain such components, it has been demonstrated that autolysis of whole yeast cells into fragments increases microbial numbers in the rumen. The increased microbial population improves feed digestibility

and increases acetate, propionate, and butyrate production, resulting in a better energy balance and higher beef or milk production.

Increasing density of ration

The energy requirements during advanced pregnancy and early lactation are unusually high in the entire production cycle. Because DMI is the most important constraint, the alternative is to either increase energy density (by feeding more maize or adding oil seeds or protected fat) or reduce the roughage to concentrate (R: C) ratio. It has also been reported that milk production is positively related to DM and water intake. Maize, wheat, sorghum, and pearl millet are common feedstuffs used to provide energy to animals. The particle size of hard coat cereal grains is very important in terms of nutrient utilisation efficiency.The total starch content of a high yielder's diet should be between 20 and 25 percent. Such cows' diets must provide the required 10 MJ ME/kg DMI with 16 percent CP. The net energy of lactation (NEL) required in diets of cows and heifers during the transition period to meet requirements for maintenance and gestation revealed that heifers require more dietary energy due to lower feed intake and additional energy requirements to support growth. Grain must be introduced into the cow's ration at least three weeks before the due date, and heifers at least five weeks. The energy density per kilogramme of DM intake should be between 1.56 and 1.62 M cal NEL (NRC, 2001). The total starch content of a high yielder's diet should be between 20 and 25 percent. Such cows' diets must provide the required 10 MJ ME/kg DMI with 16 percent CP. Dietary supplementation of oils can also increase the density of a ration. Bypass fat is poorly soluble in the rumen and is not bio-hydrogenated in the rumen.

Manipulating dietary protein

By increasing microbial protein synthesis in the rumen, a 5% increase in DMI can reduce CP requirements by 1%. It has been reported that increasing the UDP content by up to 56% through dietary manipulation improves milk yield, quality (milk protein), weight loss, first service conception rate, and pregnancy rate. The level of UDP has a significant impact on the availability of N for rumen microbes. Low UDP will not only impair the animal's performance but will also result in ammonia loss from the rumen. *In-vitro* studies revealed that diet containing ME at 110 or 120% of NRC with UDP (24% of dietary CP) supplemented with niacin gave the best response as far as digestibility of nutrients and availability of ME was concerned.

Increasing dietary minerals

Cow's milk contains calcium, chloride, potassium, magnesium, and sodium. Milk contains the majority of the B-complex vitamins. A minor vitamin and mineral deficiency may not have a significant impact on milk quantity and quality. However, calcium is one of the critical elements in the ration that must be carefully considered in transition cows. The demand for the calcium increases drastically at the start of lactation, resulting in a sudden drop in blood calcium levels, a condition known as hypocalcaemia. This promotes the release of parathyroid hormone (PTH), which promotes bone resorption. The PTH activates vitamin D3, which increases intestine Ca absorption and mobilised bone Ca. To avoid milk fever, the best feeding management practise is to provide a low Ca (50 g/day) diet during the final 2-3 weeks of pregnancy, which should be increased to 100 g/d at least two days before parturition. Following parturition, the diet should include enough Mg, an essential activator of vitamin D3 in the liver. The anionic diet (12 meq/100 g DM) had no effect on mastitis or milk fever in cows. Metabolic acidosis caused by a negative dietary cation- anion difference (DCAD) promotes calcium mobilisation from bone, whereas high dietary potassium levels and positive DCAD inhibit this process. The subclinical hypocalcaemia is believed to be a contributing factor in disorders such as displaced abomasum and ketosis.

Manipulating dietary fiber level and particle size

For optimal animal production, the rumen should be as efficient as possible in terms of mixing, rumination, and emptying. Active bouts of rumination aid in feed breakdown and saliva secretion, which aids in rumen pH control. Complete feed for high-yielding cows should contain no less than 21% ADF or 28% NDF (NRC, 2001). The forage DMI as a percentage of BW should be between 1.4 and 2.4 percent. Individual NDF intake by high-yielding cows can reach 1.3-1.4 percent of BW. Forage NDF intake as a percentage of bodyweight should be around 0.8-0.9or it should not exceed 17-18% of total DMI. However, it can be increased to 30-34 percent by using wheat bran, orange peel, or citrus pulp, which are high in soluble fibre (pectins). Rumen pH is stabilised by a proper balance of slowly (fibrous) and rapidly fermentable NFC, as well as an optimal PEF length, which stimulates rumen mixing, rumination, and saliva production. Researchers fed small amounts of cereal straw (0.5 kg/day) as a source of structural fibre and of appropriate length (4-8 cm), and structure was fully incorporated into mixed rations, with

encouraging animal performance results. Smaller particle size (6 mm) may reduce dairy cattle's feed bunk sorting behaviour. Because of sorting activity, rations with a higher proportion of longer particles (>19 mm) are more likely to have a larger difference between the feed initially offered and that consumed throughout the day.

In conclusion, appropriate feeding during, 3 weeks before and 3 weeks has significant impact of milk yield, sustenance of production potential and reproductive efficiency of the animal. Drastic physiological and metabolic adaptations take place during transition phase. Nutritional imbalances may depress performance, reproductive status or can predispose to other metabolic disorders. With the advancement of gestation, the dry matter intake (DMI) of declines by 1.7–2.0 % body weight at parturition, while daily milk production increases to peak production at about 5-8 weeks. Typically, animal suffer from a 6–8-week period of negative energy balance, mobilize body reserves especially fat and protein. At cellular stage hepatic gluconeogenesis and beta oxidation metabolism initiates to produce β-hydroxy butyric acid and non-esterified fatty acids, which may further develop number of sever metabolic diseases. Appropriate nutritional management and precision in transition animal feeding can improve animal term of milk yield, FCE and reproductive efficiency, as well as minimum incidence of metabolic disease. Mainly, targeted to increase the DMI in animals, enrich nutrient density, improve quality of protein, concentrate and forage ratio, balanced for NDF & NFC content, optimize particle size.

Chapter 3

IMPORTANT ZOONOTIC DISEASES ENCOUNTERED IN DAIRY FARMING

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India is a land of farmers. Indian farmers have been involved in crop production as well as animal husbandry. With the largest bovine population in the world, India ranks first in production and consumption of milk. Dairy farming along with other animal husbandries contributes significantly to the economy of our nation. For a fruitful dairy business, it is important to adopt good animal husbandry and management practices, for example, cleanliness and sanitation of the sheds, regular deworming and vaccination of animals, bio security, clean milk production, guidance from veterinarians etc. Such practices not only lead to healthy animals but also help to keep ourselves healthy because there are several diseases of animals that can be transmissible to humans, and are known as zoonotic diseases/zoonoses. In this article, we will discuss about important zoonotic diseases encountered during dairy farming practices especially cattle farming.

Anthrax

Anthrax is caused by a spore forming bacterium known as *Bacillus anthracis*; spores are resistant to the heat and several disinfectants. Anthrax spores remain stable in the environment for long periods, sometimes for decades. While grazing on the contaminated soil, animals can get exposed to the spores (usually via ingestion or sometimes through inhalation) which become activated inside the animal body. Within the body, vegetative form of this bacterium produces toxins which results into severe disease and death. Carcass of dead animal or fluid oozing from the natural orifices of dead animals have heavy bacterial loads which could lead to contamination of the environment. When come in contact with air, bacteria develop into spores (Fig 1). Herbivore animals (e.g., cattle and small ruminants) are considered most susceptible as compared to omnivores and carnivores. In cattle, peracute and acute form of the diseases are found. In peracute form, staggering, trembling, convulsions, difficult breathing and death have

been observed in cattle, sheep and goats. In acute form, fever, cardiac or respiratory distress are seen; fever is high (may reach 107 C). Animals are found dead without showing any symptoms as the course of disease is very rapid. Oozing of bloody discharges from the natural orifices are also observed. Necropsy of the dead animal is not recommended, as it could lead to contamination of the environment with spores.

People get infected with *B. anthracis* via (1) handling contaminated carcasses/animal products (2) inhalation of spores and (3) ingestion of meat (Fig 1). Three forms of anthrax have been reported in humans: Cutaneous (most common), inhalational (pulmonary) and gastrointestinal.

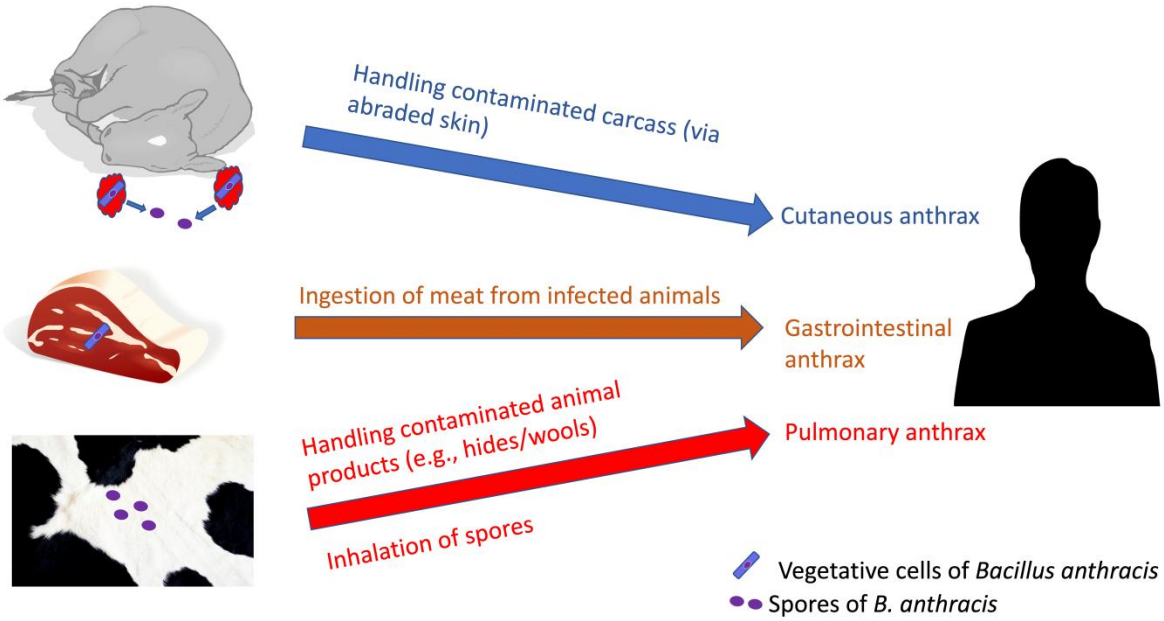

Fig 1: Routes of transmission of *Bacillus anthracis* in humans

In cutaneous form, lesions in the form of depressed black scars (eschar) are seen on the skin of infected person. In gastrointestinal form, clinical manifestations (depending on the part of gastrointestinal system (oropharynx/abdomen) affected are observed: fever, anorexia, dysphagia, cervical edema, nausea, vomiting, abdominal pain and diarrhea. In inhalational (pulmonary) form, non-specific symptoms are observed, for example, fever, chills and malaise. Sometimes, symptoms related to respiratory distress are also observed.

Anthrax is a notifiable disease in humans in India. Vaccination of animals could be helpful to prevent this problem in animals. In case of an outbreak, carcasses of infected/suspected animals should be disposed of properly (incineration is considered best disposal technique; in case, carcass is to be buried, it must be covered with lime, so that stray dogs/carnivores don't open the pit and get access to the carcass). Contaminated beddings and feed should also be disposed of. Never perform necropsy of the suspected/infected animals (as it may lead to formation of spores and thus, contamination of environment). Contaminated equipment and pens/shed should be properly cleaned and disinfected. Sick animals should be isolated from the healthy animals. Proper veterinary inspection during the slaughtering is a cardinal measure to control this issue. While handling sick animals, veterinarian and farmers should use proper personal protective equipment.

Brucellosis

Brucellosis is caused by a bacterium belonging to the genus *Brucella*; several species of *Brucella* are zoonotic in nature. In cattle, brucellosis is commonly caused by *B. abortus*; in addition, *B. melitensis* and *B. suis* have also been reported in cattle. Animals acquire infection via ingestion of bacteria from the contaminated feed or water, or by licking the aborted fetus, placental membranes, discharges (Brucellae are present in large number), genitals of infected animal. Transmission via natural insemination in cattle is not common, however artificial insemination (contaminated semen) could lead to infection if semen is deposited at the mid cervix, but not in case it is deposited in the uterus.Abortion is a major clinical manifestation; infected animals abort in their third trimester of pregnancy. Retention of placenta, still birth or weak calves have also been observed. Epididymitis has also been seen in male animals. Humans become infected when they come in contact with the dead calves, or uterine discharges while handling and caring of animals (Fig 2). As brucellae are present in large number in dead calves, or uterine discharges,

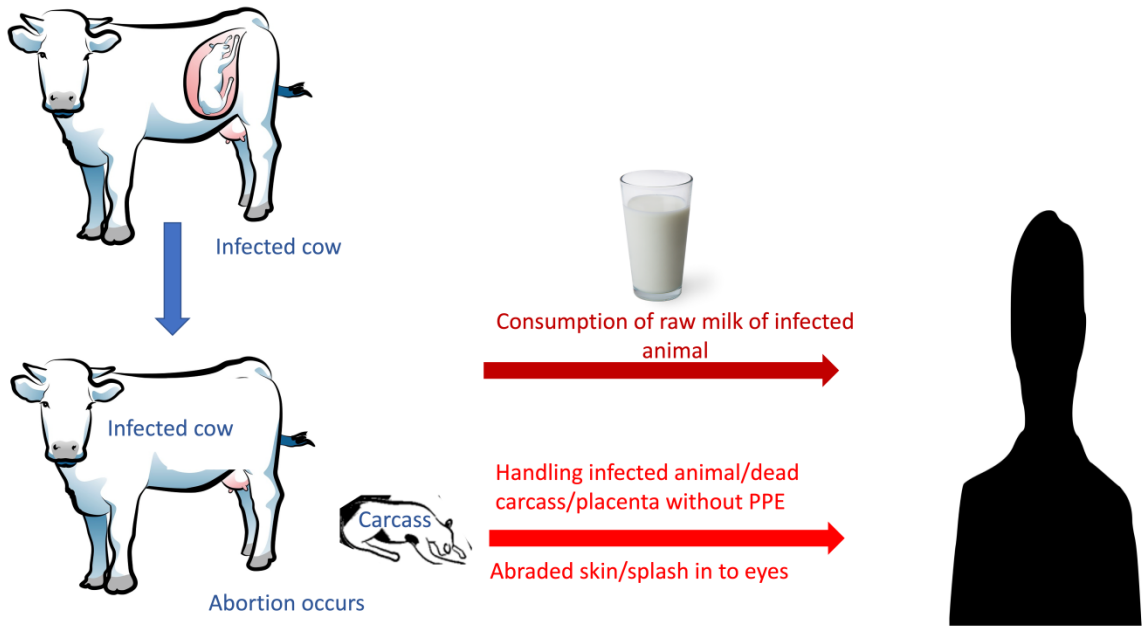

Fig 2: Routes of transmission of *Brucella* spp. in humans

they can enter human body via cuts (on the skin) or mucous membranes (splash of discharges in to the eyes). Another route of transmission in humans is via ingestion of raw/unpasteurized milk of infected animals. Brucellae are shed in the milk for a variable period of time.

Intermittent fever, profuse sweating, anorexia, swelling of joints, headache and backache are some of the clinical manifestations in people. Farm workers or animal handlers should dispose of the dead calf, placenta, contaminated beddings carefully, and must use PPE. Infected animals should be isolated from the others. The sheds/contaminated areas should be cleaned and disinfected. New animals should be quarantined and tested (for *Brucella* spp.) before their release in to the farm. Vaccination is an effective method of controlling brucellosis. *Brucella* S19 or RB51 vaccines are commonly used for the control. Vaccine (Brucella S19) is administered only in the female calves (at age of 4-8 months). Veterinarians while vaccinating animals should also use PPE, in order to avoid accidental splash (into eyes) or injection (into skin) as *Brucella* vaccine are live attenuated. Milk should be pasteurized before drinking.As this is an occupational disease, it is suggested that the livestock farmers, farm workers, lab workers and veterinary personnel get themselves tested for this infection biannually.

Bovine Tuberculosis

Tuberculosis (TB) in humans in caused mainly by *Mycobacterium tuberculosis* and *M. africanum.* There are several zoonotic species of *Mycobacterium*which could be transmitted from animals to humans: *M. bovis* (bovine tuberculosis), *M. caprae* (caprine tuberculosis), *M. orygis*, *M. pinnipedii*, *M. microti* and *M. mungi*. In cattle, *M. bovis*is a primary causative agent of bovine tuberculosis; *M. caprae* and*M. orygis*have also been reported in cattle. *Mycobacterium bovis* can be found in the respiratory secretions, milk, urine and faeces. Cattle acquire infection mainly via inhalation, especially when animals are confined with infected animals in closed spaces.

Humans usually get this infection either by inhalation of organisms from the environment or from ingestion of raw/unpasteurized milk of infected animal (Fig 3).

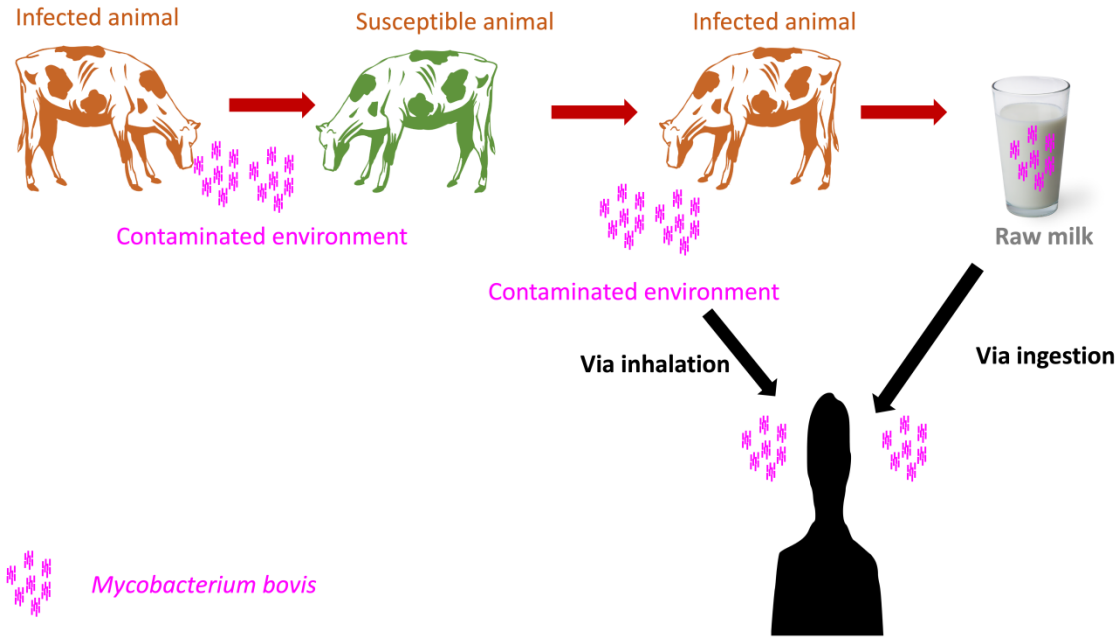

Fig 3: Routes of transmission of *Mycobacterium bovis* in humans

In cattle, clinical manifestations may include anorexia, weakness, weight loss (emaciation in terminal stage of the disease), and fever (fluctuating intermittent). Swelling of retropharyngeal lymph node may also occur. Respiratory signs for example moist intermittent cough, difficult/fast breathing can also be seen. In case, GI system is affected, intermittent diarrhea and constipation are observed. In humans, non-specific symptoms are observed; fever, malaise, weakness, anorexia as well as symptoms related to the system affected (Respiratory, skin, CNS,

gastrointestinal, urogenital systems). In humans, respiratory signs are usually observed in TB caused by *M. tuberculosis*, however in case of *M. bovis* infection (which usually occurs via ingestion in humans), symptoms are usually extrapulmonary. Clinical manifestations depend on the body system affected. For example, cutaneous lesions in case skin are affected, and meningitis, meningoencephalitis are commonly reported when CNS system is affected. Proper cleaning and sanitation and ventilation of sheds could help in control of the disease. Overcrowding in the animal shed should also be avoided. Test and segregation/slaughter programs are used for the control of this disease across the world. Milk should be pasteurized before drinking. While handling infected animals, use PPE.

Cryptosporidiosis

Cryptosporidiosis, a protozoan zoonosis is caused bythe parasite *Cryptosporidium*. There are several species of *Cryptosporidium*; *Cryptosporidium parvum* along with *C. andersoni, C. bovis and C. ryanae*are common species in cattle.*Cryptosporidium hominis and C. parvum* are the most commonly reported species in humans; C. *hominis* is anthroponotic in origin (man to man transmission), however *C. parvum*is zoonotic in nature (cattle to man transmission). There are other species of *Cryptosporidium*, which are also zoonotic, and infrequently transmitted from other animal host species to people. Transmission is via faecal-oral route through the ingestion of food or water contaminated with the oocysts. Cattle shed sporulated oocysts of *Cryptosporidium*in the faeces and usually do not show symptoms. Sporulated oocysts are immediately infectious. Clinical manifestations are usually observed in 1-3 weeks old calves, and include pale/brown diarrhea, anorexia and weight loss. Infection is self-limiting in healthy humans, however abdominal pain, mild to severe diarrhea and weight loss are observed. For prevention and control, cleaning and disinfection of the sheds is important. Avoid overcrowding of animals. Calves must be provided with the colostrum. Humans should also follow good personal hygienic practices; for example, washing hands after handling animals and working in the sheds. Also, wear proper PPE while handling sick animals. Wash vegetables and fruits properly before eating. To avoid acquiring infection from contaminated food, cook it properly; for example, drink pasteurized/boiled milk.

Leptospirosis

Leptospirosisis caused by a bacterium belonging to genus *Leptospira*. There are many serovars (>250) of *Leptospira*. Cattle act as primary reservoir host for the serovar Hardjo. Rodents are important reservoir host for*Leptospira* serovars: Icterohaemorrhagiae, Grippotyphosa and Sejroe. Serovars Icterohaemorrhagiae, Grippotyphosa and Sejroe can also cause infection in cattle. Cattle, rodents, dogs, sheep, goats and pigs act as carrier animals, and may shed leptospires in urine for years. Transmission among animals usually occurs through direct contact of skin (abraded) or mucus membrane with the infected urine, and placental fluids or via feed/water/areas/soil contaminated with urine.

Acute leptospirosis is observed in calves, and symptoms may include fever, anorexia, jaundice, haemoglobinuria, meningitis. Infection in adult cattle results in abortion, still birth or birth of weak calves. Rodents and dogs are considered to be responsible for most of the human cases. Humans usually acquire infection through contact with contaminated water/soil. Two phases of leptospirosis are observed in humans: (1) phase I (known as septicaemic phase): symptoms include fever, cough, chest pain, myalgia, (2) phase II (immunephase) canresult in to optic neuritis, peripheral neuropathy and pulmonary hemorrhage. Weil syndrome is a severe form of leptospirosis, and is also known as icteric leptospirosis. In Weil syndrome, jaundice and anemia are observed. In addition, epistaxis, hemoptysis and GI haemorrhages are also seen.

Prevention and control measures include vaccination, proper sanitation of sheds, use of personal protective equipment, biosecurity system (control of rodents), good personal hygienic practices, and use of gum boots in rice fields/while working in water with possibility of contamination with urine.

Pseudo cowpox

Pseudo cowpox is viral zoonosis and caused by parapox virus. Small red papules are formed on the teats or udder of the infected cows. Papules develop into vesicles, nodules and scabs; the small scabs appear as a circular ring or horseshoe (this is characteristic of pseudo cowpox). Transmission between cattle occurs through humans usually during milking or by milking equipment. Humans can also acquire this infection from contact with infected cows; condition is more common in milkers, and is known as milkers' nodules. Painless purplish red

nodules are formed on hands, fingers or arms. Reinfection can be observed in cows but not in humans. For prevention and control, good hygienic practices need to be followed: washing and disinfecting hands between milking of cows, post-teat dipping, and disinfection of milk unit.

Chapter 4

INNOVATIONS IN DAIRY ANIMALS FEEDING

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Increasing human population, shrinkage of agricultural land and volatility of conventional feed ingredients prices are the major constraints for sustainable dairy farming. The key to economic dairy production is meeting the nutrient requirements of dairy animals precisely in cost effective way as feeding constitutes about 60–70% of total cost of milk production. Similarly, availability of nutritious green fodder throughout the year is very essential for profitable dairy farming. However, in tropical countries like India the availability of green fodder varies from season to season and dairy farmers are facing shortage of green fodder twice a year particularly during November-December and May-June. Hence various efforts have been in dairy feedings to have green fodder throughout the year and to improve the efficiency of animals for better utilization of available nutrients. All these technologies and innovations are discussed briefly in this chapter.

A. Total mixed ration:

Total mixed ration (TMR) is most widely used method of animals feeding that combines roughage and concentrate feed ingredients (forages (green or dry), grains, protein sources, minerals, vitamins and feed additives) formulated to a specific nutrient content into a single blend of feed to supply all required nutrients to the animal, in a form that excludes selection (NRC 2001; ICAR 2013).

Types of TMR: 1) Loose form 2) Complete feed block 3) Feed pellets

Advantages of TMR

1. Eliminate selective feeding by animals and with every bite dairy animal will eat a nutrient-balanced ration. It's harder for cows to be picky while eating.
2. By TMR feeding rumen microorganisms have a uniform supply of protein and carbohydrates throughout the day. This will lower the risk of digestive upset, stabilizes

rumen pH and maximizes rumen fermentation as well as production of rumen microbial protein

3. A TMR provides more control and accuracy over the amount of feed given than separate ingredients which is needed for good production (increase milk production 1 to 2.5 kilograms per cow daily) and health.
4. Allow use of low palatable feeds/by-products or crop residues in feeding of dairy animals which will not only makes dairy farming more economical but also helpful in mitigating the adverse effect on the environment by crop residue burning.
5. TMR allows the feeding of larger groups of cows faster and more economically than feeding forages and concentrates separately
6. Nonprotein nitrogen compounds, especially urea, can be more easily and safely fed as TMR
7. TMR mixers can reduce the work of feeding cows and save labour costs.

Critical points for formulation of TMR:

1. **Moisture level:** The amount of moisture/dry matter (DM) in TMR has a significant effect on DM intake and ingredient separation. TMR having DM lower than 45% (or >55 % moisture) resulted in decrease DM and nutrient intake because of gut fill limitations and not able to meet the requirement of animals precisely. This situation can occur when ensiled forages or large amounts of wet by-products (wet distillers' grains) were used for TMR preparation. On the contrary, TMR that is too dry (>60 % DM), which can occur with large amounts of hay and concentrates, may increase ingredient separation and decrease total DMI. Therefore, optimal DM content of TMR should be greater than 45% and less than 60% DM (Schingoethe 2017).
2. **Particle size:** Particle size reduction is the prerequisite for mixing of various feed ingredients. Chaffing of forages and grinding of concentrate ingredients plays very important role in preparation of good quality TMR. Very fine size makes TMR dusty, lower its palatability and lower animals' performance as ground forages pass through the rumen faster than long or chopped material and reduce the digestibility of the fibre

fraction by as much as 20 % with 5–15 % decrease in DM digestibility. Therefore, medium fine grinding is best which contains coarse, medium and fine particle in 1:6:3 ratio

3. **Accuracy of weighing and ratio of forage:** The DM intake of dairy animals is also affected by the dietary forage to concentrate ratio. Irrespective of the forage type, as the concentrate component increases, there is generally a linear increase in DM intake and digestibility. However, if the DM intake through concentrate increases beyond 60 % of the total diet, it could lead to ruminal acidosis and thereby, severely affecting the fibre fermentation in rumen.
4. **Forage quality:** Type of forage and its quality affects the overall quality of TMR.
5. **Proper mixing:** There should be proper mixing of each and every feed ingredient so that animals get balanced nutrient with every bite. Further, under mixing of ingredients may lead to poor performance of animals.Mixing time, order of adding ingredients to the mixer wagon, and style of mixer wagon are important factors which affects the quality of TMR.
6. **Monitoring of feed intake:** Dominant animals (older and larger ones, but not necessarily the high milk yielders), tend to spend more time in eating than those with a lower social rank in competitive situations. This may affect the intake of TMR and overall performance. Grouping of animals into various categories is required to meet their nutrient requirements such as grouping first-lactation cows separately from older cows places less stress on first-lactation cows and possibly results in better health and production. Further, having separate groups for prefresh and fresh cows has several benefits like monitoring intake and health status of animals

Table 2. Example of total mixed rations for dairy animals, % DM basis

Ingredients	**TMR_1**	**TMR_2**	**TMR_3**	**TMR_4**	**TMR_5**
Maize grains	18.0	12.0	12.0	16.0	12.0
Mustard cake	7.0	6.0	16.0	12.0	16.0

Rice bran	8.0	10.0	8.0	8.0	10.5
Wheat bran	5.5	10.5	4.0	2.5	9.0
Berseem hay	45	-	-	-	-
Green berseem	-	45.0	-	-	-
Green maize/sorghum	-	-	48.5	-	-
Oat hay	-	-	-	45.0	-
Maize/bajra silage				-	25.0
Fermented wheat straw	-	-	10.0	15.0	26.0
Wheat straw	15	15	-	-	-
Mineral mixture	1.0	1.0	1.0	1.0	1.0
Common salt	0.5	0.5	0.5	0.5	0.5

Disadvantages of TMR Feeding

1. The same ration is fed to a group of animals therefore some animals may be overfed and some underfed. Further, the no. of animals in a group is not specified yet.
2. Mixer wagons that thoroughly blend ingredients are expensive, and electronic load cells are highly recommended to quantitate the blending process.
3. The use of TMR feeding allows a dairy to group cows into many categories based on nutritional needs less economically feasible in smaller herds
4. Hay that is stored in baled or long form must be chopped before it can be blended with silage or grain

B. Fodder conservation: In order to overcome the shortage of green fodder during lean period and avoidance of nutrients loss from green fodder at the time of abundant availability, fodder conservation is very helpful. Depending on the type of forage or grasses these can be conserved in its original wet form known as silage or in dry form known as hay.

A. Silage: It is a fermented, high moisture green fodder prepared by preserving fodder crops (maize), crop residues/by-products in anaerobic conditions and can be stored over log period. The process of preparing silage is called ensiling is a process which involves the conservation of green fodder crops, grasses and the storage over long period. The basic principle of silage making is to convert the sugars in the ensiled fodder into lactic acid under anaerobic condition, reduces the pH of the silage to about 4.0 or lower which inhibit the biological activities of microbes that are responsible for spoilage.

B. Guidelines for preparing silage

Crops suitable for silage:

a) All thick stem fodder crops such as maize, sorghum, oats, pearl millet, and hybrid Napier which are rich in soluble carbohydrates are most suitable for fodder ensiling.

b) Natural and cultivated grasses can be ensiled with addition of molasses at 3-3.5%.

c) Mixture of grasses/ cereal fodders and legume (berseem, lucerne) in 3:1 ratio

d) Unwilted leguminous leafy fodders and dry forage in 4:1 ratio

e) Dry roughage (like wheat and paddy straw) with fruits and vegetable wastes in 25:75 ratio (depending on quality of waste)

Stage of harvesting the crop: The fodder crop should be harvested at a stage when nutrient content is at peak stage and neither immature nor over mature. Flowering to milk stage is recommended for making silage from maize fodders containing between 25-35 % dry matter. If DM content is lower than 25 % then partial wilting by drying in sun for 12 hours should be done to reduce the water concentration to about 70%.

Chaffing of fodder crop: For proper filling and compaction as well as to remove the gases, fodder crops particularly those having thick and pithy stems, should be chaffed to 2-3 cm size using chaff cutter.

Filling of silo pits and bags: Chopped fodder should be filled layer by layer of within a day and compacted properly with tractor to remove trapped air. The entire pit should be filled in the same manner up to a height of about 1.5 to 2 meters above the ground, to ensure that after complete setting the silage mass is well above the ground level, in order to avoid water collection in the pit. Similarly, in case of polythene bags compaction can be done manually with foots or using a thick log of wood

Closing the silo pit: After properly shaping-up the mass on the top layer (dome shape), the silage pit should be covered as soon as possible with a layer of straw or waste fodder, and subsequently with a plastic sheet of 250 to 275 micron thickness to prevent oxygen from coming in.

Opening the silo: Silo pit and bags can be open after 42 days from the closing of silo pit or bags. While opening the silo, the cover should be removed properly and a plastic sheet is taken out in a section of the pit, taking care that the minimum possible surface is exposed to the atmosphere. Some mouldy material may be found on top and also on the side, which should be removed before taking the silage for feeding. Well-preserved silage will be of yellowish green colour, having a pleasant acidic smell.

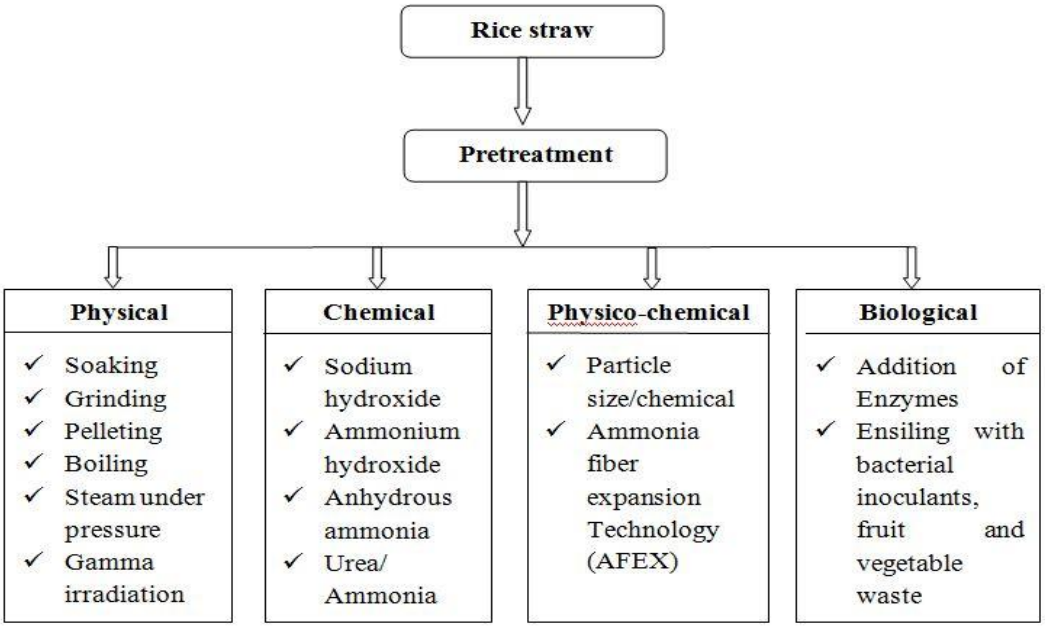
C. Hay

D. The basic principle of hay making is to reduce the moisture concentration (< 15%) in the green forages sufficiently as to permit their storage without spoilage or further nutrient losses. Hence, crops suitable for hay making are with thin stems and many leaves i.e. leguminous fodder crops as they dry very quickly

Innovations in improving quality and utilization of crop residues

Straws (wheat and paddy) are naturally available cheap roughage used as feed material for dairy cattle worldwide. These contains 30 to 40% cellulose, 20-25% hemicellulose, 15-20% lignin and minor organic compounds. Special nutritional characteristics of straws such as low protein, high silica and lignin contents result in lower feed digestibility. The several methods such as

pretreatment, enrichment and recycling are generally used to improve the quality and economic feasibility of straw (Fig 1). Moreover, it can be mixed with other ingredients to make a certain formulation with enriched nutritional characteristics.



Urea treatment of rice straw

One of the most effective and widely used method to improve nutrient utilization of straws is its with urea to maintain an appropriate carbon to nitrogen ratio. Under this process around 14 kg urea is dissolved in 200 liters of water and then sprinkled over 386 kg of straw, which is then thoroughly mixed and stacked for 9 days. More than 85% of the added urea is hydrolyzed by 9^{th} day, thereby eliminating the chances of urea toxicity to the animals fed on urea treated straw. The shelf life of naturally fermented straw is one year without any deterioration in the quality. After the stipulated time of 9 days, the stack should not be dismantled; rather the quantity required for feeding should be taken from one side of the stack. The fermented rice straw is found to have higher crude protein and lower fiber content in comparison to the untreated rice straw and it can be fed *ad lib* to the adult ruminants.

Ammonia Fiber Expansion Technology (AFEX)

AFEX an ammonia-catalysed biomass processing method is the advanced form of urea/ammonia treatment of straw in which biomass is contacted with ammonia and water at moderate temperatures (70-120˚C) and pressures (5 to 20 atmospheres) for up to 30 minutes, following which the ammonia is recycled and reused, and the biomass is optionally dried have been developed to enhance the utilization low quality roughages. Recovering the majority of ammonia following the reaction and reusing it in subsequent processing is crucial to the economic viability of AFEX process.

Bypass nutrient and feed additives

Ruminants have unique ability to form microbial protein in rumen from rumen soluble N sources which along with rumen undegradable protein reaches small intestine and form the protein or amino acid pool to meet the requirements of maintenance and low producing animals (NRC 2001).However, in case of high yielders and physiological stress periods (transition, heat stress, etc.) this becomes a limiting factor. Under above circumstances, rumen protected/bypass nutrients are promising strategy for delivering nutrients directly to the intestine in utilizable and efficient form. Supplementation of rumen protected protein, fat and vitamins has improves milk yield, reduces heat stress and boosts immunity of the animals.

Bypass fat: Cereal grains and fats are mainly used as source of energy in the ration of dairy animals. However, inclusion of cereals is limited due to ruminal acidosis and unprotected fat over 3% of dry matter (DM) intake, decreases fibre digestibility along with depressing effect on rumen cellulolytic microbial activity (NRC, 2001). Thus, supplementing high energy sources like fat in such a form that resists interaction with rumen microorganisms, but gets digested in lower digestive tract to dairy animals is very crucial for enhancing the energy density of ration and this form is known as bypass fat or rumen protected fat or inert fat.It is recommended that first 3% fat of total DM intake in animals should be provided through various oilseeds while, that in excess of 3% should be supplied as inert fat.Under Indian conditions bypass fat should be supplemented at 10 g and 20 g per kg milk production in lactating cows and buffaloes, respectively or at 2-2.5 % in the concentrate mixture.

Bypass protein: Feeding a diet containing high rumen degradable protein is not a satisfactory strategy as > 10% RDP in the diet is associated with delayed first ovulation or oestrous, decreased conception rate and increased number of days open (Tamminga, 2006). Therefore, efforts have been made to balance dietary protein in ruminant's ration on the basis of degradable and undegradable protein sources in order to improve feed efficiency, reduce feed cost and N losses to the environment. For optimum milk protein synthesis, lysine and methionine should be about 7.2 and 2.4 % of metabolizable protein respectively, in the diet of dairy cattle. Thus,for adult cattle (400 kg body weight and 10 kg milk yield) and buffaloes (600 kg body weight and 10 kg milk yield) lysine and methionine requirements will be around 43.9 and 17.9 g/d and 70.6 and 22.5 g/d, respectively(Sarkar et al. 2022).

Rumen protected vitamins:

Niacin**:** A dose upto 3.5 g/d is optimum for early lactating animals, while, during heat stress a dose of 12 g/d should begiven to mid and late lactating animals.

Choline: A dose of 15 to 60 g/day of rumen-protected choline is to be supplemented to dairy animals during transition period for enhancing productivity and reducing the chances of metabolic disorders.

B complex:A dose of 100 g/cow/day of rumen-protected B vitamins (choline, riboflavin, folic acid, and vitamin B12 from 3 weeks before parturition to2 weeks after parturition should be given for optimum milk production and health of the dairy animals. Then 4 g of rumen-protected B vitamins (folic acid, vitamin B12, biotin, pyridoxine, and pantothenic acid) for lactating cows upto 72 days in milk is required (Kaur 2019).

Role of protected probiotic in ruminants

Probiotics are live microbial feed supplements that beneficially affect the host by improving its intestinal microbial balance" (Fuller, 1992). They mainly belong to the genera *Lactobacillus* and *Bifidobacterium*, but strains of *Bacillus, Pediococcus* and some live yeasts have also shown probiotic attributes. For dairy animals'yeast and *Aspergillus oryzae* are commonly used prebiotics which have direct impact on maintaining rumenmetabolic profile.The biggest challenge of probiotic feeding in adult ruminants is to protect them from extensive rumen degradation and its postruminal delivery with original potency. To overcome this

constraint,probiotics are encapsulated to prevent dissolution in rumen pH and permitting slow release in lower gastrointestinal tract. A level of 20 g unprotected probiotic per day per animal is found beneficially effective for maintain optimum rumen environment and to augment their productivity.

References

Fuller, R. (1992). History and development of probiotics. In *Probiotics* (pp. 1-8). Springer, Dordrecht.

ICAR (2013). Nutrient Requirements of Cattle and Buffaloes. 3rd edn. Indian Council of Agricultural Research, New Delhi, India

Kaur, M., Hartling, I., Burnett, T. A., Polsky, L. B., Donnan, C. R., Leclerc, H., ... and Cerri, R. L. (2019). Rumen-protected B vitamin complex supplementation during the transition period and early lactation alters endometrium mRNA expression on day 14 of gestation in lactating dairy cows. *Journal of Dairy Science*. *102*(2): 1642-1657.

NRC. (2001). Nutrient requirements of dairy cattle: 2001. National Academies Press Washington D.C., USA.

Sarkar, S., Sharma, A., Tariq, H., & Satapathy, D. 2022. Role of Rumen Bypass Nutrients in Dairy Animal's Health and Productivity: A Review. *Indian Journal of Animal Research*, 1, 9.

Schingoethe, D.J. (2017). A 100-Year Review: Total mixed ration feeding of dairy cows. *Journal of Dairy Science*, 100(12), 10143-10150.

Chapter 5

METABOLIC DISEASES OF DAIRY ANIMALS AND THEIR ALLEVIATION

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Metabolic diseases continue to be a substantial challenge in the dairy industry everywhere around the world where modern dairy breeds are managed and fed for high milk yields. Undoubtedly, some of this continuing challenge is related to progressive improvement in dairy cow genetics and the ever-increasing average milk yields of modern dairy cattle. Improvements in production capacity challenge us to manage and feed animals to allow them to adapt to the tremendous metabolic demands of high milk production. Dairy animals metabolic disorders are the diseases related to disturbance of one or more metabolic processes in the organism. The transition period which includes three weeks before and three weeks after parturition is very critical for dairy animals. This period of time is associated with multiple changes including hormonal changes, moving from non-lactating to lactating state as well as a major drop in feed intake and switching of the diet from a roughage-based diet (hay and grass) into a diet rich in rapidly fermentable carbohydrates (high-grain diets). One in two dairy animals in a herd is affected by one or multiple metabolic disorders.

Metabolic disorders are a group of diseases that affect dairy animals immediately after parturition. There are several metabolic disorders identified and the most frequent ones are: (1) subacute and acute ruminal acidosis, (2) laminitis, (3) ketosis, (4) fatty liver, (5) left displaced abomasum (LDA), (6) milk fever, (7) downer cow, (8) retained placenta, (9) liver abscesses, (10) metritis, (11) mastitis, and (12) bloat. The reason that these diseases are called metabolic disorders is related to the fact that they are associated with the disturbance of one or more blood metabolites in sick cows. For example, ketosis is associated with enhanced ketone bodies (BHBA) in the blood; fatty liver is associated with enhanced NEFA and their accumulation in the liver; acidosis is associated with increased production of organic acids (e.g. acetic, propionic, and butyric acids) in the rumen and low rumen pH and milk fever is associated with decreased

blood calcium. The most interesting observation with regards to the occurrence of metabolic disorders is that they are highly associated with each other. For example, animals affected by milk fever are more prone to mastitis, retained placenta, metritis, LDA, distocia, udder edema and ketosis; cows affected by acidosis are more prone to laminitis, LDA, milk fever, mastitis and fatty liver.

1. Milk Fever

Milk fever is an important production disease occurring most commonly in adult dairy animals within 48-72 hours after parturition, which is characterized clinically by hypocalcemia, general muscular weakness, circulatory collapse and depression of consciousness. This disease has been known by a number of terms including parturient paresis, milk fever, parturient apoplexy, eclampsia, and paresis peurperalis. When milk fever results due to imbalance in blood Ca, P and Mg levels, it is known as "Milk fever complex". Generally the milk fever is sporadic but on individual farms the incidence may rarely reach 25-30% of susceptible cows and increases with age and yield.

Pathogenesis

The onset of lactation places such a large demand on the calcium homeostatic mechanisms of the body that most cows develop some degree of hypocalcemia at calving. Milk fever occurs when calcium leaves the blood to support milk production faster than Ca can be put back into the blood from the diet, skeletal Ca stores and renal conservation of calcium. The entire extracellular pool will have only 8 to 9g Ca. In some cases, plasma calcium concentrations become too low to support nerve and muscle function, resulting in milk fever. A cow producing 10 kg of colostrum (2.3g of Ca/kg) will loose 23g of Ca in a single milking. This is about 9 times as much Ca as that present in the entire plasma Ca pool of the cow. Normally extracellular Ca concentration is around 10,000 greater than intracellular resting Ca concentration. A 50% decline in extracellular ionosed Ca concentration, typical of the cow with milk fever. This hypocalcemia is caused by an imbalance between Ca output in the colostrum and influx of Ca to the extracellular pool from intestine and bone. In order to prevent blood calcium from decreasing, the cow must replace calcium lost to milk by withdrawing calcium from bone or by increasing the efficient absorption of dietary calcium. Plasma Ca concentration is under the control of parathyroid hormone, calcitonin, and the metabolites of vitamin D (Goff *et al.*, 1995). Bone calcium mobilization is regulated by parathyroid hormone (PTH) produced by the parathyroid glands. Whenever there is

a drop in blood calcium, blood PTH levels increase dramatically. Renal tubular reabsorption of Ca is also enhanced by PTH. However, the total amount of Ca that can be recovered by reducing urinary Ca excretion is relatively small.

Clinical signs The main clinical manifestations are divided into three stages.

First stage/ Stage of excitement	**Second stage/ Stage of sternal recumbency**	**Third stage/ Stage of lateral recumbency**
Nervousness or hypersensitivity	Depression, Sternal recumbency	Lateral recumbency
Anorexia (decreased appetite)	Characteristic "S" shaped posture	comatose condition, progressing to loss of consciousness
Mixed excitement or tetany without recumbency	Fine muscle tremors	Flaccid muscles
Weakness or weight shifting	Pupils dilated and unresponsive to light	Profound gastrointestinal atony
Stiffness of hind legs	Cold extremities	
Rapid heart rate	Rapid heart rate, decreased intensity of heart sounds	Impalpable pulse and almost inaudible heart sounds
Rectal temperature is usually normal or above normal (>39°C)	Decreased rectal temperature (35.6-37.8°C)	

Diagnosis

- History taking
- Clinical examination
- Laboratory investigation: The major change in the blood of cows with milk fever is blood calcium

a) Normal level in a dairy cow is 8-10 mg/dl.
b) The level drops to 8 mg/dl at calving.
c) In milk fever cows, blood calcium level drops to 6.5 mg/dl in stage I, 5.5 mg/dl in stage II and 4.5 mg/dl in stage III.

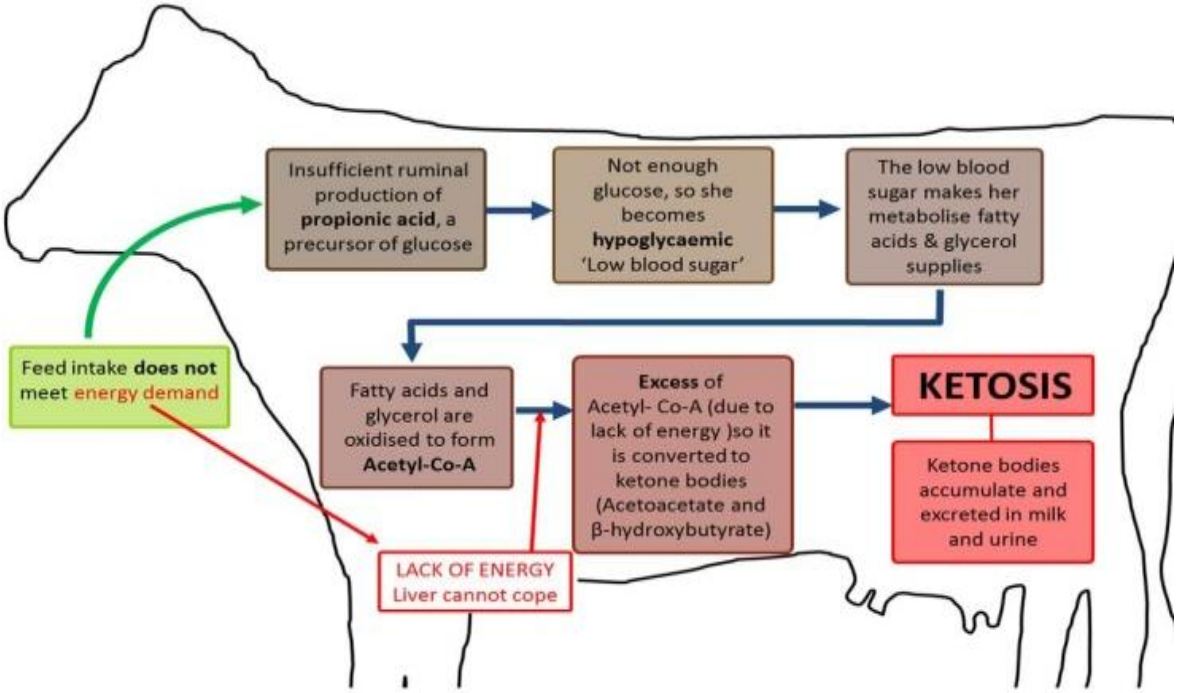
Treatment

The treatment of choice for milk fever is slow, intravenous infusion of 8-12 g of calcium as soon as possible after the onset of clinical signs. Heart rate should be closely monitored for toxic effects. Calcium borogluconate containing products with or without added magnesium and phosphorus are mostly used usually 400 ml of 40% calcium borogluconate. If a response is not evident by 5-6 hours, the diagnosis should be reassessed and if necessary, a further intravenous infusion of 8-12 g of calcium administered. Relapses of milk fever occur in 25% of cases treated. Twelve hours after treatment, all calcium administered, whether by the intravenous or subcutaneous route, has been eliminated from the body.

2. Ketosis and Fatty Liver

Ketosis is a production disease of dairy cows caused due to hypoglycaemia and is characterized by ketonaemia and ketonuria due increased level of ketone bodies in the blood. These ketone bodies are betahydroxybutyric acid, acetoacetic acid and acetone. Most commonly, ketosis is seen either in high producing cows or cows on a poor diet. Signs of the disease can be seen before calving, but they occur most commonly during the first 10 to 60 days after calving. The three-week period after calving seems to be the most critical time. Subclinical ketosis (SCK) is more important because it causes heavy economic losses due to reduced milk production. SCK is "a condition marked by increased levels of circulating ketone bodies without the presence of clinical signs of ketosis. Blood glucose concentrations in clinically affected cows fall below the level required to support nerve and brain function and cows often exhibit stumbling while walking, head pressing, and other signs of central nervous system dysfunction. Ketotic cows also are inappetant, which further exacerbates their negative energy balance. Milk production falls precipitously.

Pathogenesis



Types of Ketosis		
Type I Ketosis	**Type II Ketosis**	**Subclinical Ketosis**
Occurs around 4-6 weeks after parturition (time of peak milk production)	1–2 weeks postpartum, very early lactation	high serum ketone body concentrations without clinical signs
underfed cattle, metabolic shortage of gluconeogenic precursors than with excessive fat mobilization	usually associated with fatty liver	increased risk of clinical ketosis, metritis, and displaced abomasum

Clinical signs

- dullness, depression, a staring expression
- loss of appetite, pick at her feed, and leave some grain, may progress from leaving most of the grain and some silage to the stage of eating only small amounts of hay and preferring to eat bedding

- smell in breath/milk
- Decreased milk production
- In mild cases, the only observation may be that the cow is not "doing well."
- Nervous ketosis
    - false chewing movements
    - frothing and salivating profusely
    - pressing forward in the stanchion
    - walking in an unusual "goose-stepping" manner
    - licking themselves continuously (especially the forearms)

Diagnosis

- History taking
- Clinical examination
- Laboratory investigation

Qualitative test (Rothera's test)		**Quantitative test**	
No color change	negative	Blood glucose	normal is 50mg/dl <40mg/dl
Slight lavender	+	Blood ketone	> 10mg/dl to 100mg/dl
Deep lavender	++	Urinary ketone	>70mg/dl
Beet red or purple	+++		
Deep beet red or purple and opaque (strongly positive)	++++		

Treatment

Two types of treatment are commonly used, Replacement therapy and Hormonal therapy. However, presently a combination of both is required. The various recommendations are as follows:

- **Glucose therapy**:
    - Dextrose 40%, 500 ml IV.
    - Fructose @ 0.5 g as 50% solution, IV

    - 500 g Oral glucose following premedication with sodium bi-carbonate-100 % recovery observed.
- Sodium and Magnesium propionate- Give 80-200 g orally twice daily up to 10 days. Used as a preventive or as a "follow up" after initial intravenous injection of 500 ml of 40% dextrose
- Propylene glycol- Propylene glycol 125 ml + 12 g of Niacin daily for 5-7 days
- **Glucose and hormones combined therapy**:
    - 540 ml of Rintose + 80 units of insulin is better than Rintose alone.
    - 20 % glucose + 0.5 units / kg insulin in buffaloes- recovery in 2 days.
    - Corticosteriods (Tramcinolone acetonide)- Give 0.05 mg /kg b. wt. alone.
    - Adrenocorticotropin- Give 200 - 800 units IM.
- **Miscellaneous therapy:**
    - Vit.B12 and Cobalt to promate propionate production.
    - Nicotinic acid 12 g daily to promote gluconeogenesis.
    - Chloral hydrate. Give one ounce (28.5 g) BID for 3-5 days.

3. Ruminal Acidosis

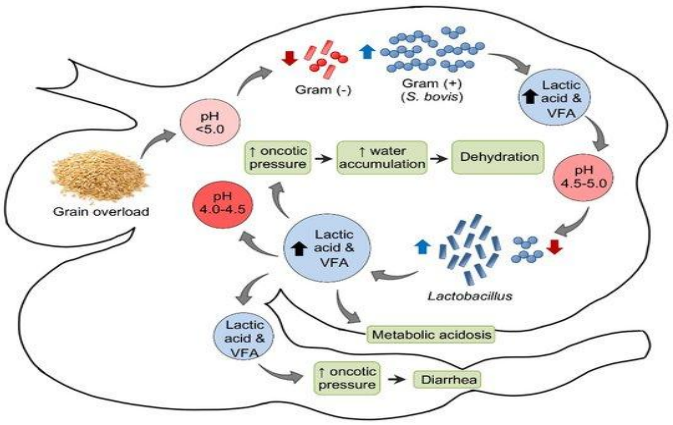
Ruminant animals are adapted to digest and metabolize predominantly forage diets; however, growth rates and milk production are increased substantially when ruminants consume high-grain diets. One consequence of feeding excessive amounts of rapidly fermentable carbohydrates in conjunction with inadequate fiber to ruminants is subacute ruminal acidosis. It is a metabolic status defined by decreased blood pH and bicarbonate, caused by overproduction of ruminal D-lactate. It will appear when animals ingest excessive amount of nonstructural carbohydrates with low neutral detergent fiber. Animals will show ruminal hypotony/atony with hydrorumen and a typical parakeratosis-rumenitis liver abscess complex, associated with a plethora of systemic manifestations such as diarrhea and dehydration, liver abscesses, infections of the lung, the heart, and/or the kidney, and laminitis, as well as neurologic symptoms due to both cerebrocortical necrosis and the direct effect of D-lactate on neurons.

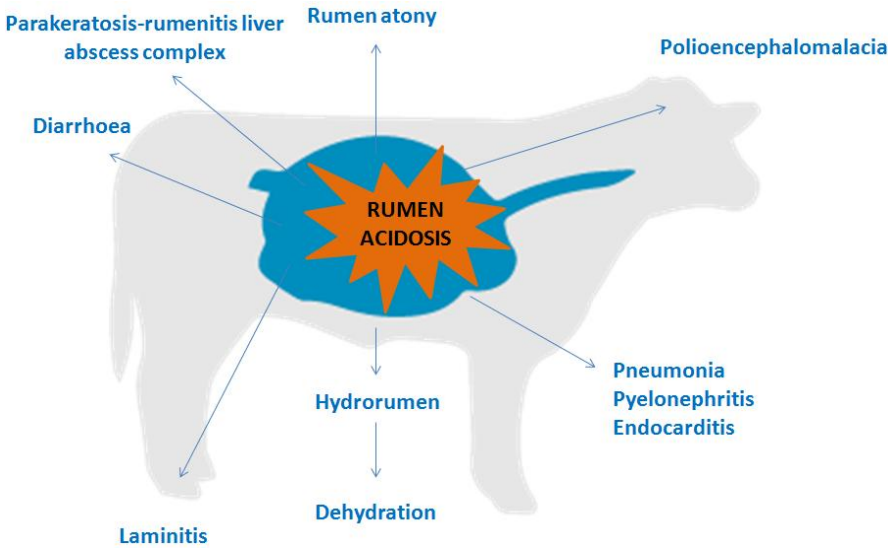
Pathogenesis

Main difference between two clinical forms of ruminal acidosis

Type	Acute	Subacute
Presence of clinical signs	Yes	Maybe
Mortality	Yes	No
Rumen pH	<5	5.0-5.4
Lactic acid	Increase	Normal
Gram negative bacteria	Decrease	Normal
Gram positive bacteria	Increase	Normal
Blood pH	Low	Borderline

Clinical signs





Treatment

- Thiamine is often a highly recommended injection to give to cattle with acute acidosis, as it is very important in treating and stopping a sudden acidosis attack as it halts the production of thiamine through digestion
- Baking soda will also work as a treatment for cattle with acidosis.
- Removal of rumen contents and replacement with ingesta taken from healthy animals is necessary.
- In animals that are still standing, rumenotomy is preferred to rumen lavage, because animals may aspirate during the lavage procedure and only rumenotomy ensures that all ingested grain has been removed.
- Fluid therapy to correct the metabolic acidosis and dehydration and to restore renal function. Initially, over a period of ~30 min, 5% sodium bicarbonate solution should be given IV. During the next 6-12 hr, a balanced electrolyte solution, or a 1.3% solution of sodium bicarbonate in saline, IV.
- Procaine penicillin G should be administered IM to all affected animals for at least 5 days to minimize the development of bacterial rumenitis and liver abscesses.

Chapter 6

MASTITIS: EARLY DIAGNOSIS AND CONTROL

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Worldwide, mastitis is considered as one of the most important diseases in dairy cattle. It is important because of its frequent occurrence and associated production losses. Moreover, mastitis affects the milk quality and has, therefore, concerns of the dairy industry. Further, the disease constitutes a major reason for the use of antibiotics and hence the threat of drug residues in dairy production systems. Because of concerns about drug residues, and bacteria of mastitis origin, it has also become a worry to the public. The paper summarizes the current status of mastitisand future directions for disease control at field level.

Socio-economics

The Meta analysis of the available published literature in Indian contest revealed that on an average mastitis affects 46.91% of cows and 33.57% of buffaloes at subclinical levels(Bansal et al. 2009). The quarter wise occurrence shows 23.04% in cows and 15.96% in buffaloes. The clinical mastitis affects almost equally the cows (6.23%) and buffaloes (6.02%). The 2009 calculations revealed the estimated annual economic losses from mastitis to the country as Rs. 7165 crores, much by subclinical mastitis. Subclinical mastitis though in apparent causes 10-25% loss in milk production, whereas in clinical mastitis, there may be total loss of milk. Mastitis results in increase in the somatic cell count (SCC) and bacterial load of milk. The European Union has set up a threshold of 400 $\times 10^3$cells/ml for the milk to be from a healthy quarter of a cow. The studies conducted on a limited number of farms (129 farms)in Punjab revealed that 47% of cow farms and 31% of buffalo farms produced bulk tank milk with SCC > 400×10^3 cells/ ml (EU Limit for quality milk). The BTM analysis of antibiotic residues revealed 7/129 (6.42%) samples positive for one or the other drug at higher than MRL levels.The high SCC in mastitis milk has a lipolytic effect on fat and there is increased tendency for rancidity of milk and milk products. Rancid milk with acid degree value > 1 could be detected at 400, 000 cells/ml. Also,

the mastitis milk with a total bacterial count of more than 100 000 cfu/ml could release hydrolytic enzymes, which spoil the milk and milk products. In mastitis milk good things such as lactose, casein, butterfat, solids not fat, calcium and phosphorus decrease while undesirable milk components such as lipase, whey proteins, immuno-globulins, sodium and chloride increase.

Etiology of disease

The disease is mainly caused by bacterial organisms withstaphylococci (*S. aureus* and coagulase-negative staphylococcal (CNS), as the most common pathogens, constituting more than 70% of infections in India. The others include streptococci (*Streptococcus agalactiae, Str. dysgalactiae, Str. Uberis*) coliforms (e.g. *E. coli, Klebsiella*) and corynebacteria. The causative agents are categorized into two groups; the contagious organisms viz., *Staphylococcus aureus* and *Streptococcus agalactiae* which are frequently present on the teat and udder skin of animals, and may be transmitted from one animal to another animal at the time of milking through milking utensils, milker' s hands and cups of milking machine. The other group comprised environmental organisms such as coliform and *Str. uberis,* which are frequently present in dung, animal bedding, manure, soil, feed stuffs, uterine discharges and urine etc., may be transmitted to animal at any time, even in-between the milkings. The relative importance of the mastitis pathogens varies between countries, regions and farms. *S. aureus* remains an important pathogen in most countries. This organism can produce numerous putative virulence factors that allow adhesion to membranes and resistance to phagocytises. Mastitis due to coliform, mainly *E. coli*, is common in high-producing cows with a low milk somatic cell count. In *E. coli* mastitis, severity of clinical signs depends mainly on the immune response of the animals where effective elimination of the bacteria by neutrophils is important for the resolution of infection. *S. uberis*, an environmental pathogen, can produce a persistent infection that often is unresponsive to treatment. *S. uberis* uses host milk proteins to establish intramammary infection. This organism may also behave as a contagious pathogen. *The S. agalactiae* has been considered more or less non-existent. But, this pathogen is re-emerging. The CNS has gained increasing interest, and ismost prevalent udder pathogens in India and someother countries. The corynebacteria have been found as quite frequent organisms in some cases, particularly in machine milked cows.

Involvement of pathogens in tissue damage

Staphylococcus aureus produces toxins that destroy cell membranes, directly damage milk-producing tissue, and induce necrosis in bovine mammary glands. Initially, the bacteria damage tissues lining the teat and gland cisterns within the quarter. If unchecked, they invade the duct system and establish deep-seated pockets of infection in the milk-secreting cells (alveoli). The secretory cells revert to non-producing state and alveoli begin to shrink. Substances released by PMN completely destroy the alveolar structure which is replaced by connective and scar tissue. Thisis followed by the walling-off of bacteria by scar tissue and the formation of abscesses. In addition, the clots formed by the aggregation of PMN and blood clotting factors may block small ducts and prevent complete milk removal.

Escherichia coli produce a number of proteinases, including collagenolytic enzymes, which contribute to the degradation of ECM components. In moderate cases of *E. coli* mastitis, there is minimal alveolar tissue damage. In very severe cases, the infection progressed via the ductile system to producea limited inflammatory reaction, but with an extensive involvementof the secretary tissue. In its most severe form with uncontrolled bacterial multiplication, alllactiferous sinus epithelia were lost, interstitial tissue became hemorrhagic, and often the animal died of toxemia within a fewhours of infection.

Diagnosis of mastitis

In its clinical form, mastitis may be diagnosed well by the classical signs of inflammation and visible alterations in milk consistency, colour and appearance etc. The changes in levels at which certain components in the mammary secretion are present are commonly utilized in diagnosing subclinical mastitis. A variety of mastitis diagnostic tests are available which differ markedly with respect to sensitivity, specificity, simplicity, rapidity and cost. Some simple test systems that can be used on farms as **cow-side** tests are described below.

CMT/ Sodium Lauryl Sulphate (SLS) test:

It is based on the principle that reagent ruptures somatic cell releasing cellular proteins (DNA) that results in gel formation, and depending upon the degree of gel formation the reaction is scored as 0, Trace, 1, 2 and 3. Thus, this test gives the indirect estimate of milk somatic cell count. To conduct the test, 2-3 ml of milk sample from each quarter is drawn separately into the cups of the specially designed plastic paddle. The reagent is added in equal quantity, and the

contents are mixed by a rotatory movement of the hand. The reaction is scored while mixing the contents and by tilting the paddle in between.

Electrical conductivity (EC) test:

The ions in milk conduct electricity, such that any change in the concentration of ions is reflected as a change in conductivity. Dissociated, inorganic salts such as sodium, chloride and potassium are the main contributors to conductivity. In mastitis, there is damage to tight junctions between epithelial cells and an increased permeability of the blood capillaries occurs. As a result Na^+ and Cl^-, which are higher in extracellular fluid, pour into the lumen of alveolus and in order to maintain osmolarity K^+ and lactose levels decrease proportionately. The alterations of the concentrations of Na^+, K^+ and Cl^- in mastitis without a concomitant change in osmotic pressure are the main reason for the higher EC of mastitis milk. Any quarter having relatively 15% or more conductivity than the quarter with the lowest value within cow is taken as evidence for mastitis. The EC could be measured by digital conductivity meters, which are easily available in the market.

Bromothymol blue card (BTB) card test:

It is based on the principle that in mastitis, the pH of milk rises due to entry of bicarbonate salts from the blood into milk. Depending upon the health status of the quarter and hence pH, the colour of the dye changes from yellow (normal) to greenish-yellow (+), green (++) and blue (+++) when a drop of quarter milk is placed on the card. This is a comparatively somewhat less sensitive test, particularly in buffaloes.

Microbiology:

The microbial isolation, identification and drug sensitivity testing is essential for undertaking the rational treatment and disease control programs. To achieve this, the correct sampling and microbial procedures are important and those recommended by the NMC and IDF are widely accepted. Currently, PCR-based tests are exploited, but generally these are more expensive than classical bacteriology. The molecular diagnostic methods are not only useful for the diagnosis of mastitis organisms, they may also help to identify particularly virulent strains of an organism or distinguish between clonal and non-clonal infection outbreaks. In a clonal outbreak, the observed predominance of a single strain could indicate contagious transmission of the organism or exposure of multiple cows to an environmental point source. For example *S. aureus* strains

possessing *sed, sej,* and *bla*Z genes, often in combination with penicillin resistance, have been typically found in connection with persistent IMI.

Treatment of mastitis

In vitro testing of milk samples revealed that the drug sensitivity pattern of mastitis organisms goes on changing. So, treatment should be given, preferably based on culture and sensitivity test particularly in mild cases. In acute or per acute cases, there is no time for these tests, so the therapy in such cases is based on the past data of herd infection and sensitivity reports. However, before starting therapy in such cases, the milk sample should be invariably taken and put to culture sensitivity so that the therapy may be changed if needed in the light of sensitivity report. Moreover, it may also be made clear that there is no surety that *in vitro* sensitivity determination will correlate with the *in vivo* treatment results. For example, enrofloxacin that shows high *in vitro* sensitivity and is pharmacologically considered to distribute well in the udder clinically proved to be less efficacious against staphylococcal mastitis because of its inability to kill intracellular organisms. On the other hand, amino-glycosides (gentamicin and neomycin) that are considered to have poor distribution in the udder, *in vivo* proved very much effective in treatment of clinical mastitis. The organism involved in mastitis also affects the efficacy of treatment. Streptococci respond well, staphylococci less and coliform are difficult to treat due to severe per acute reaction. However, enrofloxacin could be best recommended for treatment of per acute mastitis caused by coliform. For taking specific therapy, clinical mastitis is generally divided into three forms viz., per acute, acute and chronic form.

Peracute mastitis:

It is generally caused by coliforms and it occurs commonly around calving but may develop at any time during lactation. The disease is usually sudden in onset: the cow may appear normal at one milking and at the next milking shows pronounced signs, including anorexia, rise of temperature, depression, shivering and rumen stasis. Inflammatory signs in the udder may be minimal at this time and swelling may be detectable only after the udder is milked out. Later, the quarter is swollen and hard, the teat may be thickened, oedematous, hot to touch and sensitive. In the early stages, the milk may appear normal or faintly watery. Subsequently, it may be serous and contain tiny particles. In severe cases it may become blood tinged. Recommended therapy includes the following:

- Removal of bacteria, toxins and inflammatory exudates by frequent stripping of quarters at 1-2 h intervals, even oxytocin inj. (20-30 IU I/M) may be given for complete milking out.
- Appropriate antibacterial therapy to start with systemic administration that may be later (after 12-24 h/ after last milk out) supplemented with suitable intramammary infusion.
- Fluid therapy; dextrose saline solution (10-20 L in the first hour, up to 60 L in severe cases) to restore vital body fluids, dilute toxins and counteract acidosis. Even 5% sodium bicarbonate (150-250 G) with the first 3-5 L of fluid may be given.
- Systemic glucocorticoids, Dexamethasone @ 1-3 mg/kg IV or IM once or may be repeated after 8-12 hours.The disadvantage of corticosteroids is that they tend to be immunosuppressive and inhibit bacterial clearance. Use of steroids may be replaced by nonsteroidal, anti-inflammatory drugs.
- Nonsteroidal anti-inflammatory drugs such as flunixin meglumine 1 mg/Kg IV, aspirin (30 grams P.O. bid), phenylbutazone (4-8 mg/kg orally or 2-5 mg/kg IV) have also been used extensively and found to reduce pain and inflammation, and restores appetite.
- Calcium borogluconate 20% @ 500 ml IV to counteract hypocalcaemia induced by endotoxin. Administer with care to avoid damaging effects on the heart in animals in shock.Calcium therapy may be delayed until after initial volume expansion with fluids.
- Antihistaminic drugs & multivitamins. Antihistamines are of the empirical value if administered repeatedly every 3 to 4 hours for at least 2 days. Histamine appears to be the major factor responsible for the early phase of circulatory shock.
- In cases where there is DIC possibility, heparin may be given at 75 IU/kg BW (56,000 units)/day.

Acute mastitis:

In this form there is no systemic reaction. Primarily changes are observed in milk, which may contain flacks, become watery or thick, and sometimes may contain blood. The udder may become swollen and hard. The line of treatment includes use of antibacterial drugs plus calcium and multivitamin therapy. The combination therapy, i.e. intramammary plus parenteral works well than the alone parenteral or intramammary. The important recommendations in mastitis therapy are (i) Use antibacterial on need based on culture sensitivity data (ii) Use appropriate dose, dosing

interval and duration, minimum 3-5 days (iii) Stick to the recommended milk withdrawal times. Depending upon the sensitivity report following combination may be used:

Option	Parenteral	Intramammary infusion
1.	Gentamicin sulphate 3-5 mg/Kg IV or IM twice daily	Gentamicin sulphate 100-150 mg twice daily or commercially available i/mam preparations of neomycin for 3 days
2.	Ampicillin sodium 20 mg/Kg IV followed by 10mg/kg IM twice daily	Cephapirin sodium 200 mg at every milking for 3 times or cloxacillin sodium 200 mg at every milking for 6 times
3.	Sulfadiazine/ trimethoprim 25 mg/Kg IV or IM once daily	Amoxycillin sodium 62.5 mg or Hetacillin potassium 62.5 mg every milking for 6 times
4.	Erythromycin lactobionate or tylosin tartrate 10 mg/Kg IV followed by 5 mg/Kg IM bid	Erythromycin 300 mg at every milking for 6 times
5.	Ceftiofur up to 1 G IM once daily	Ceftiofur 100 mg
6.	Sulfamethazine sodium 100 mg/Kg IV followed by 50 mg/Kg IV	Option 3 or procaine penicillin 1 Lac IU at every milking for 6 times

(iii) Chronic mastitis:

A case is considered chronic when (i) there is formation of fibrotic cord inside the teat canal (ii) there is a thick pus discharge, not responding to treatment (iii) there is a frequent recurrence of mastitis in the same quarter. The treatment/surgery of chronic mastitis is not rewarding. Rather, such cases should be isolated from the milking herd or the affected quarter may be permanently dried-off by producing a chemical mastitis. Infusing 30-60 ml of 3% silver nitrate solution or 20 ml of 5% copper sulphate solution can do it. If a severe local reaction occurs, the quarter should be milked out and stripped frequently until the reaction subsides. If no reaction occurs, the quarter is stripped out 10-14 days later. Two infusions may be given.

Control of mastitis

The ten point mastitis control plan recommended by the NMC comprised of establishment of goals for udder health; maintenance of a clean, dry, comfortable environment; proper milking procedures; proper maintenance and use of milking equipment; good record keeping; appropriate

management of clinical mastitis during lactation; effective dry cow management; maintenance of biosecurity for contagious pathogens and culling of chronically infected cows; regular monitoring of udder health status; and periodic review of the mastitis control program.These points may be put into practice as comprehensive mastitis control program as under:

(i) Minimising the source of infection

(ii) Elimination of existing udder infections

(iii) Prevention of new intramammary infections (IMI)

(iv) Increasing the udder resistance to mastitis

(i) Minimising the source of infection:

It can be achieved by maintaining optimal **Environmental and Milking Hygiene**; segregation and prompt treatment of clinical mastitis cases, culling of carriers and drying off of chronically infected quarters. The adoption of hygienic measures depends upon the epidemiology of the causative organisms. For example, in case of contagious organisms, which are transmitted from one to another animal through the milking equipment and milker's hands, proper washing of udder, cleanliness of milker's hands/milking machine clusters in between each milking and post-milking teat dipping in germicidal solution will reduce the infection to a great extent. On the other hand, for the organisms that come from the environment like coliform, animal environment should be kept clean by frequent removal of dung, proper drainage, and adequate milking and feeding space should be provided.Environmental udder pathogens are often of faecal origin and cannot live on teat skin for long periods of time. If these bacteria are present in large numbers on teat skin, it is the result of recent contamination. Therefore, the number of these bacteria on teat skin is a reflection of the cow's exposure to the contaminating environment. A significant source of udder pathogens in confinement systems is the material used for bedding cows. Organic bedding materials such as straw, wood products and recycled manure commonly contain few udder pathogens prior to use as bedding. However, these organic products rapidly become contaminated, with the mastitis pathogen populations increasing 10,000-fold within 24 hours. Efforts to control pathogen populations in organic beddings by composting or the use of sanitizing and disinfecting agents have been unsuccessful. Inorganic bedding, such as sand, supports lower bacterial populations compared with organic bedding. The bacterial contamination of sand bedding is directly related to the moisture and organic contamination. Rates of new IMI caused by environmental pathogens are greater during involution and

lactogenesis than during lactation. Pathogen exposure and subsequent rates of mastitis during the dry period are increased by use of stalls and maternity pens bedded with moist organic bedding. Manure packs generally contain extremely high counts of pathogens dangerous to both dam and calf.

(ii) Elimination of existing udder infections:

It can be best achieved by **Dry Therapy**. The dry (non-lactating) period is essential for the adequate proliferation and differentiation of the mammary secretory epithelium so as to achieve optimal synthetic and secretary function in the ensuing lactation of cows. Studies have shown that the dairy cows with 10-40 days dry period produced significantly less milk in the subsequent lactation than the cows with a 40-60 day nonlactating period. The mammary glands are markedly susceptible to new IMI during the early dry period and towards the end of dry period; may be due to (i) the flushing effect of milking on bacteria colonizing the teat canal is terminated (ii) increased intramammary pressure may causethe opening of teat orifice and bacterial penetration (iii) the defence mechanisms of the mammary gland are compromised. The new IMI in the dry period are important. During the first month of lactation, a quarter newly infected in the dry period will sustain a production loss equal to that of a quarter that retains an established infection throughout the dry period. In addition, most clinical mastitis cases in early lactation are the result of dry period infections. Dry therapy, intramammary infusion of long acting antibiotic at dry off, that maintains effective drug concentration for 6-8 weeks i.e. throughout the dry period is considered not only eliminates the subclinical infections of previous lactation but also prevents new IMI and increases the milk production by about 8-10%. In addition, it improves the milk quality at calving and prevents the occurrence of clinical mastitis around calving. Studies conducted at this institute showed dry therapy very much effective in the prevention of mastitis and lowering the milk SCC. The milk production improved more than 15% in dry treated buffaloes (unpublished data).To minimize the adverse effects of antibacterial treatment, dry treatment of infected quarters only (Selective quarter therapy) or cows (treat all quarters of cows infected in one or more quarters; Selective cow therapy) may be a preferred concession than the blanket or complete therapy (treat all cows in the herd at dry off irrespective of quarter health status) for herds with low mastitis infection rate. However, studies have shown that in herds in which selective therapy was used, thehigher infection rate was reported at calving

than at drying off. From this consideration, it seems clear that selective therapy, as compared with complete one cannot be justified economically in most herds.

(iii) Prevention of new intramammary infections (IMI):

It can be achieved by (a) Post-milking teat disinfection (b) Teat Sealants at dry off

(a) Post milking teat disinfection

Today teat disinfection products are available in a variety of physical forms and application techniques. These include: dips, sprays, high viscosity, barrier / film-forming products, foam products, dry powders, winter dips, and concentrates. Disinfectant application via dipping is widely preferred because it is easier to ensure adequate teat coverage. High viscosity products require dipping, which results in an improved procedure. The teats of all the lactating cows, and dry cows during first and last 10-14 days of the dry period, are dipped regularly after every milking in a germicidal solution. Some of the products recommended for teat dipping are:

- **Iodine-based dips (iodophor):** The iodine present in these acts like a reservoir to provide a slow release of disinfectant. They have an acidic base so requires emollients to condition the teat skin and avoid dryness and cracking of the skin.So, it is used asIodine (0.5% available iodine) solution + Glycerine @ 10-15% of iodine solutionIt treats various types of teat lesions and injuries also.
- **Chlorhexidine-based dips:** These products are very effective against most bacterial species and are not as affected by organic contaminants as others. Chlorhexidine is an irritant to teat skin, so it can be used as Chlorhexidine (0.5%) solution + Glycerine @ 06% of solution.Chlorhexidine gluconate is better than the acetate salt due to cost and solubility.
- Barrier dips are compounded with iodine and used for post-milking only. They are a gel-like substance that dries create a plasticised barrier that covers the teat and requires removal immediately before the next milking. This barrier stops bacteria from colonising on the teat surface or invading the teat orifice. Some of the barrier teat dips marketed are:
    - Lactic acid based teat dip. The lactic acid has broad-spectrum,long lasting disinfectant capabilities and because lactic acid is an all-natural substance,there is no problem of drug residue in milk. E.g. Lacti Fence barrier film (De Laval), Synofilm barrier film
    - Iodine based barrier technology that protects IMI from the environment between milkings. E.g. Blockade^{TM}(DeLaval), Bio-Secure (Dairy Dynamics)

The choice of type of teat dip depends on the management and the environment. Generally, for cooler, drier climates non-barrier teat dips are suited, and for hot, humid, rainy seasons, barrier teat dips are best. Some dairies switch teat dips with the seasons. To assess teat dip practicality from management point, walk through your barns. If you need boots, your cows are likely fighting environmental mastitis pathogens, so a barrier would be a smart choice. If you can wear "church shoes" into the barns, cows are more likely to face contagious mastitis spread during milking, so a non-barrier teat dip would suffice.

(b) Teat Sealants:

Cows are naturally protected against intramammary infections during the dry period by formation of a keratin plug in the teat canal. However, time of teat canal closure varies among cows. In one study, 50% of teat canals were classified as close by 7 days after dry off, 45% closed over the following 50-60 days of the dry period, and 5% had not closed by 90 days after dry off. Teats which do not form a plug-like keratin seal remain more susceptible to infection. Although, antibiotic dry cow therapy has been considered the major management tool for the prevention of IMI, even with the use of antibiotics, 50% of new IMI occur during the dry period. Moreover, the rate of new IMI is related to the length of the dry period. Longer dry periods have been associated with an increase in the incidence of new IMI.A large percentage of new infection results from environmental organisms. The National Mastitis Council recommends the use of teat sealant on dry cows as part of mastitis control program. Teat sealants have been tried as "External teat sealant" and "Internal teat sealant". The external teat sealant is applied as a teat dip at dry-off, after antibiotic infusion, and again 7 to 10 days prior to calving. The teat sealant functions by preventing environmental organisms from entering the udder. Duration of adherence of the sealant to the teat, and protection of the teat orifice, are critical factors in the performance of the teat sealant. The average time of sealant adherence following dry off application was found 3-4 days. Studies have shown that when used in combination with antibiotic infusion, teat sealants can provide an additional reduction in dry period mastitis. They also demonstrated a 46.8% reduction in new IMI for cows that received a teat sealant. The external teat sealant are available with active ingredients Ethyl Alcohol (53% w/w) and Triclosan 0.1%.

The internal teat sealant, comprising of an inert substance containing 65% bismuth subnitrate in a paraffin base, are infused directly into the teat canal. It produces an effective physical barrier before the keratin plug in the teat canal has formed and after it has broken down, preventing the entry of pathogens into the udder. The best mastitis protection is expected from a combined use of DCT and internal teat sealant. Because of the growing concern about antibiotic overuse, for uninfected quarters, the use of internal teat sealant alone has been advocated.

(iv) Increasing the udder resistance to mastitis:

Future trends in mastitis control are aimed at increasing the immunity of the udder to mastitis pathogens. This can be achieved by use of non-specific (cytokines/ proper nutrition) and specific (vaccination) immuno-modulators.

(a) **Cytokines:**

Cytokines include interferons, interleukins, colony stimulating factors (CSF), and a variety of other proteins that modulate the activity of immune cells and thus enhance the phagocytic cell functions in the udder. It has been shown that interferon treated cells exhibit significantly more phagocytosis and intracellular killing of *Staphylococcus aureus.* Interleukins enhance the production of local antibodies and accelerate the involution process that will further promote resistance to mastitis during the dry period. Similarly, the granulocyte-macrophage colony stimulating factors significantly increase the chemo tactic and bactericidal activities of mammary gland neutrophils.

(b) **Nutrition:**

Even slightest deficiencies of certain vitamins (Vit E, C, A and ß-carotene) and micro-nutrients (Cu, Se, Zn, Co) are reported to have a detrimental impact on the efficient functioning of the immune system. Vitamin A is involved in maintaining a functional epithelium that provides a physical barrier to the entrance of pathogens. ß-carotene also referred as pro-vitamin A enhances the immune function and disease resistance. Zinc supplementation prevents the infection by strengthening the skin and stratified epithelium (keratinocytes) of the teat canal. The biological role of Cu is exerted through a number of Cu containing proteins, including ceruloplasmin and superoxide dismutase (SOD). These proteins protect the host tissues from membrane oxidation by acting as an antioxidant and scavenging oxygen free radicals produced during the inflammatory response. Similarly, vitamin E and the Se containing enzyme

glutathione peroxidase (GSH-pX) also act as integral part of the antioxidant system. Studies have shown that supplementation of cows during dry period and around calving (first 8-10 weeks) with the following nutrients per head per day proved beneficial in preventing mastitis/ lower milk SCC.

(i) Vitamin 53000 IU + Beta- carotene 300 mg

(ii) Zinc-methionine (180-360 mg Zn, 360-720 mg methionine)

(iii) Copper @ 20 ppm i.e. about 200 mg

(iv) Vitamin E 1000 IU during dry period and 500 IU for lactating cows

(v) Selenium is recommended as 3 mg during dry periods and 6 mg during lactation

(c) Vaccination:

The effective immunization against mastitis has been a goal of mastitis researchers for many years. But, the nature of disease creates a number of unique challenges for the production of successful immunity against mastitis. Commercially, few mastitis vaccines are currently available in the developed world for immunization against mastitis caused by *Staphylococcus aureus* and *E. coli.* Several studies have evaluated these; the outcomes have been inconsistent and confusing to interpret. However, it is generally accepted that *S. aureus* vaccine has limited ability to prevent new infections and clinical mastitis cases. The best use of the vaccine is the reduction of chronic infections rather than prevention of new infections. The use of vaccine against coliform mastitis has been considered efficacious even though the rate of intramammary infection is not significantly reduced in vaccinated animals but because they significantly reduced the severity of clinical disease. The farmer may expect a mastitis vaccine to eliminate existing infections, prevent new mastitis cases and reduce the severity of mastitis. While these expectations seem reasonable, it is unlikely that any one vaccine will be able to achieve all of these outcomes. So, we may expect the role of vaccination as the one component of mastitis control program, but alone its use may not be giving much encouraging results.

As an extension activity of the university, this department is supplying mastitis diagnostic kit (BTB card, CMT reagent and Paddle) and undertaking culture sensitivity and somatic cell count analysis of milk samples. You may avail these services from the department (Phone No. 0161- 241 4008).

Chapter 7

MAJOR DISEASES RELATED TO NUTRITIONAL DEFICIENCIES

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In dairy animals, major nutritional deficiencies are mineral deficiencies. Minerals play a central role in maintaining optimal health, reproduction and production of dairy animals. Baseline surveys have been conducted by researchers of department of Veterinary Medicine in the state for over two decades to identify prevalent mineral imbalances and to address corrective measures in dairy animals. Testing of blood and fodder samples from different parts of Punjab state have shown that deficiencies of calcium, phosphorus,iodine, zinc and copper are prevalent in significant proportion of lactating animals.

About 22 minerals are believed to be essential for optimal growth, biological functions, and productivity in dairy animals, have been classified into two groups; i) macromineralsincluding calcium, phosphorus, potassium, sodium, chlorine, magnesium and sulphur andmicromineralsincluding iodine, iron, zinc, copper, manganese, cobalt, selenium, molybdenum, chromium, tin, nickel, arsenic, vanadium, silicon. Deficiency of any of these minerals in the body leads to impaired production and reproduction in the dairy animals causing huge economic losses. Often mineral imbalances are subclinical and thereby go unrecognized. Under such circumstances health, production and reproduction is compromised and inflict economic loss.

Copper (Cu) deficiency is the most widely prevalent nutritional deficiency in dairy animals of Punjab. Based on blood Cu levels, 25-30% animals were deficient in central region of Punjab. In the sub-mountainous region of Punjab prevalence of Cu deficiency was higher in cross- bred cattle (25.6 %) than buffaloes (10.1 %).Non-infectious hemoglobinuria and leucoderma were common signs of Cu deficiency in buffaloes. Unthriftiness, chronic diarrhoea, reduced growth rates, lameness, weak immune function, loss of hair colour and reduced reproductive function

were commonly encountered with Cu deficiency. Deficiency in sheep caused loss of wool crimp and depigmentation of hair.

Infertility has been reported in experimental Cu deficiency in ewes and delayed/depressed estrus has been observed in cows fed on Cu deficient pastures. Reduced conception rate, calving problems, retention of placenta have been observed in Cu deficient cows. Copper deficiency has been known to be associated with decreased gonadotropin release by pituitary glands. In bulls, poor quality semen is associated with Cu deficiency.

Slow release supplements have been developed and found effective in treatment and prevention of this deficiency. Subclinical deficiency compromises immunity which can be reversed by single injection of copper glycinate.

Zinc (Zn) deficiency in animals affects growth and reproduction in dairy animals. It has been recorded in **one third** of the blood samples analyzed from the central zone of the Punjab and about 10% animals were deficient in submountainous region. Zn plays a vital role in DNA synthesis, Nucleic Acid & protein metabolism. Zn deficiency results in reduced appetite, depressed growth, skin abnormalities and impaired reproduction. In males spermatogenesis & development of primaryand secondarysex organs are adversely affected.The major abnormality in males is testicular hypo function affecting both spermatogenesis and testosterone production by leydig cells. On the other hand, in females - all the phases of reproduction starting from estrus to parturition and lactation can be adversely affected. Zinc plays a role in numerous enzymatic reactions. Severe zinc deficiency in cattle and buffalo has been found to cause reduction in growth and feed intake, loss of hair, skin lesions that were most severe on the legs, neck, head, and impaired reproduction.

Administration of 360 mg of elemental zinc daily for 30 days in form of Zn propionate and Zn methionine in Zn deficient animals was effective in reversing clinical signs. Improvement was observed in coat and body condition of the Zn supplemented buffaloes.

Iodine is an essential component of thyroid hormones and its deficiency leads to impairment of physical, mental and reproductive health. Repeat breeding, anoestrous, stillbirths, birth of hairless young one and retention of placenta are common clinical abnormalities

associated with iodine deficiency in animals. Young animals are more affected and show goiter and hypothermia and sometimes poor physical mental and sexual developments (Chhabra et al., 2008). In iodine deficient areas hypothermia was found to be cardinal sign of in buffalo calves during winter season in Punjab (Chhabra et al., 2008). Assessment of I status is done by measurement of serum I, protein-bound I, thyroxine (T_4), or the presence of goiter.

Concentrations of inorganic iodine in plasma (PII) are significantly affected by dietary I. Maternal plasma I can be more useful than T_4 for assessment in gestating ruminants, because low I intakes during pregnancy can result in goiter in newborn lambs even though serum T_4 of the dam may not be affected by the temporary low I intake.

Sub-clinical deficiency of iodine has been recognised to be widely prevalent in Punjab. Prevalence of subclinical iodine deficiency in sub-mountainous region of Punjab was 48.8 and 35.2 % in cross-bred cattle & buffaloes, respectively. In central districts of Punjab, the deficiency is affecting 22-57% animals. In south-western zone, high fluoride content adversely affected bioavailability of iodine and more than half of the blood samples of the region were iodine deficient. Reproduction is influenced through the action of iodine on thyroid gland. Inadequate thyroid function reduces conception rate and ovarian activity. Marked improvements in conception rate and reduction of irregularities of estrus after iodine supplementation (Injection of iodized oil @ 750 mg/ animal) in deficient animals have been observed. Iodized oil in injectable form has been successfully used to control iodine deficiency for 90 days of its administration.

Manganese deficiency is associated with infertility and anestrous in dairy animals. Low blood plasma manganese (Mn) levels have been detected in Ferozepur district of Punjab which was a confirmed area of Mn deficiency in soil-plant system;the data shows that 68.8% of dairy animals were Mn deficient showing signs of infertility and anestrous. Blood Mn levels were low in more than 25% of the animals of various districts of Punjab. Manganese deficiency causedanestrous to sub estrus, delayed ovulation, low conception rate and birth of deformed calves in cattle.Manganese deficient calves were weak, small and developed enlarged joints. Oral supplementation of manganese sulphate has been recommended for the deficient dairy animals.

In laying hens, decreased rate of egg production, poor shell quality, reduced hatchability have been observed due to manganese deficiency. Testicular degeneration has been observed in rats, mice and rabbits as a result of manganese deficiency. It has been reported that lack of manganese inhibits the synthesis of cholesterol and its precursors and in turn limits the synthesis of sex hormones, resulting in infertility.

Baseline surveys have shown that occult **deficiency ofcalcium** (Ca) is prevalent in dairy cattle and buffaloes of Punjab which adversely affects milk production. Low plasma Ca concentration in more than 2/3rd of the crossbred cattle of the state. In the sub-mountainous region of Punjab prevalence of Ca deficiency in cows and buffaloes was 29.23 and 22.78 per cent, respectively. The highest prevalence was among cattle of younger age group (35.7%) and minimum in mature animals (19.0%). Among buffaloes, deficiency of Ca was highest in 3-6 year age group (26.9 %) & least in young animals (16.7 %). In central zone, 22-42 per cent of dairy animals were deficient in different districts where animals of district Patiala were showing maximum deficiency. Subclinical postparturient hypocalcaemia was recorded in high yielding dairy animals and it affected appetite and milk production. Chronic secondary deficiency either due to low dietary Ca levels or low Ca:P ratio in diet resulted in osteodystrophy syndromes (Rickets and Osteomalacia). The best and cost effective way of treatment and prevention of subclinical calcium deficiency in early phase of calving is mineral mixture supplementation.

Post parturient haemoglobinuria (blood in urine) is the most common disease syndrome associated with acute **phosphorus deficiency**. Subclinical hypophosphatemia (low levels of phosphorus in blood) is affecting approximately 1/3rdof crossbred cows of Punjab. The phosphorus deficiency was affecting 27.38 and 21.70 per cent of cattle and buffaloes, respectively in sub-mountainous region of Punjab. In central zone, 33-71 per cent of dairy animals were deficient in different districts. Initially there is reduction in milk yield, slight depression followed by brownish red colour urine. In less acute cases, haemoglobinuria is the first sign. The earliest sign of chronic phosphorus deficiency is reduction in appetite, weight loss, reduction in milk yield and reduced fertility. In more severe form of deficiency, symptoms of osteomalacia develop. Affected animals manifest stiffness, shifting lameness and slab shaped chest. Rickets (enlarged painful swelling of physis and metaphysis of long bones, bowing of

forelegs) develops in young growing animals. Longer term phosphorous deficiency resulted in poor reproductive performance, bone abnormalities, and pica.

Selenium toxicity has been identified in few villages of Nawanshahhar, Hoshiarpur and Jalandhar districts, which is not only affecting animals but human health also. Major clinical signs are abnormalities of hooves and horns, emaciation and poor reproductive performance. Clinical signs of Se toxicity appear in animals consuming diet containing 5 ppm Se on DM basis in diet for several weeks/ months. Level of Se in hair could be the best indicator of in the diagnosis of Se toxicity. Chronic poisoning due to Se results in slow death, hair loss and malformation of hooves and horns of animals.

Se toxicity in buffaloes was treated successfully in India by Arora (1985), using a daily oral dose consisting of a mixture of sulphates. Sulphur (S) and Se are Group VI elements. Being closely related, S antagonises Se in soil and in the body. The mixture of sulphates consists of-

Ingredient (sulphate salt) in dose	***(kg)***
Magnesium sulphate	1.000
Ferrous sulphate	0.166
Copper sulphate	0.024
Zinc sulphate	0.075
Cobalt sulphate	0.015
Total	***1.280***

Daily dose to adult cattle should be 30 g each.

Chronicfluoride toxicity (fluorosis) has been recorded in cattle, buffaloes, sheep, goat and horses in many parts of Punjab. Cross sectional study in South-west Punjab has shown the incidence to be 7.71%. Buffaloes are more prone to chronic fluorosis than cattle. In Punjab hydrofluorosis (fluorine content 3ppm) is the most common cause of chronic fluorosis. Its major clinical signs are present in teeth and bones. Mottling, colouring of the developing teeth, was the first sign and most sensitive reaction to fluorine excess. Affected animals had abnormal shape,

size and colour of teeth. Incisors became pitted and molars had cavitations due to wear. In advanced fluorosis reluctance to move and severe lameness were noticed. Poor growth rate and weight loss occurred together with signs of reduction in fertility and milk production. Affected animals were anaemic and production decreased.

Blood levels of fluorine are 0.2 mg/dl of blood and 2-6 ppm in urine of normal non-fluorotic cattle. If confirmation is required urine sample should be submitted for fluorine level. Normal fluorine level is 5ppm and the concentration of 20 to 30ppm suggests border line toxicity and above 30ppm suggests clinical toxicity.

Potassium: Common clinical signs of severe potassium deficiency include: diminished feed intake, reduced water intake, pica, poor productivity, weakness, and recumbency. Tissue concentrations of potassium poorly correlate with dietary deficiency. Serum potassium is the best indicator of deficiency; but disease states can cause electrolyte shifts that result in lowered serum potassium when dietary deficiency has not occurred. In addition, serum that is haemolysed or left on the clot too long may have falsely increased potassium content due to loss from the red blood cells. Thus, dietary potassium concentrations are a better guide to potassium deficiency.

Sodium: Common clinical signs of severe sodium deficiency include: diminished feed intake, decreased water intake, poor productivity, and pica. Tissue concentrations of sodium poorly correlate with dietary deficiency. Serum and urine are the best for measuring sodium deficiency; but disease states can cause electrolyte shifts that result in lowered serum or urinary sodium even when dietary concentrations are adequate. Thus, dietary sodium concentrations are a better guide to diagnose deficiency.

Chapter 8

FEED RELATED TOXICITIES IN DAIRY ANIMALS

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Toxicosis, poisoning, and intoxication are synonymous terms for the disease produced by a toxic agent. Toxicity (sometimes incorrectly used instead of poisoning) refers to the amount of a toxic agent necessary to produce a detrimental effect. Acute toxicosis refers to effects during the first 24-hour period. Effects produced by prolonged exposure (≥3 months) are referred to as chronic toxicosis. Terms such as subacute and subchronic are used to cover the large gap between acute and chronic.

The main toxicities occurring in dairy animals through feed are mycotoxicosis, salt toxicosis and urea toxicosis.

Mycotoxicosis

Mycotoxin contamination of feedstuffs occurs either as a result of invasion of crops by field fungi or by the growth of storage fungi in crops held under improper storage conditions. Many cereal grains show a rapid increase in respiration during storage when the moisture content is increased. Most important species of molds normally associated feed related toxicities is Aspergillus species.

Toxins of the Aspergilli

Aflatoxins are bisfuranocoumarin metabolites produced by toxigenic strains of Aspergillus flavus, A parasiticus, and A nomius on peanuts, nuts, corn (maize), rice, cottonseed and other cereals, either in the field or during storage when moisture content and temperatures are sufficiently high for mold growth. Usually, this means consistent day and night temperatures >21.1°C (70°F). Climatic conditions of drought or prolonged hot weather, corn variety, and insect damage to crops can influence aflatoxin production. Earlier recognized disease outbreaks called moldy corn toxicosis, poultry hemorrhagic syndrome, and Aspergillus toxicosis may have

been caused by aflatoxins. The primary aflatoxins analyzed in crops include aflatoxins B1, B2, G1, and G2, with the letter indicating the fluorescence color under ultraviolet light (ie, blue for B1 and B2 and green for G1 and G2). Aflatoxin B1 is recognized as the predominant toxin found in crops, and the most toxic and carcinogenic metabolite. Under optimal conditions, high concentrations of aflatoxins can be produced quickly or within a few days in field corn.

Aflatoxin Types- The major types of aflatoxin are B1, B2, G1, G2, M1, and M2 .

Feed Limits

The U.S. Food and Drug Administration (FDA) has set an action level for aflatoxin M1 (AFM1) of 0.50 µg/kg in liquid milk, total aflatoxins of 20 µg/kg (20 ppb) in feed ingredients offered to dairy cattle, 100 µg/kg (100 ppb) for breeding cattle, 300 µg/kg (300 ppb) for finishing beef cattle and 20 µg/kg (20 ppb) in foods intended for human consumption . The European Commission set up an action level for AFM1 of 0.05 µg/kg in liquid milk, AFB1 of 20 µg/kg in all feedstuffs, 10 µg/kg in complete feeds, and 5 µg/kg in complete feeds for dairy animals .

Mechanism of action - Aflatoxins are apparently readily transported to liver cells of most species and are metabolized to an epoxide that binds to macromolecules, especially nucleic acids and nucleoproteins. Their toxic effects include mutagenesis due to alkylation of nuclear DNA, carcinogenesis, teratogenesis, reduced protein synthesis, and immune suppression. Reduced protein synthesis results in reduced production of essential metabolic enzymes and structural proteins for growth. The liver is the principal organ affected. High dosages of aflatoxins result in hepatocellular necrosis; prolonged low dosages result in reduced growth rate, immune suppression, and liver enlargement.

Risk Factors and Contamination

In general, stressors such as temperature, drought, moisture, insect infestation, diseases, hail and other factors that can physically damage plants or interfere with the growth of plants can predispose crops to aflatoxin contamination. In terms of weather, dry and hot conditions predispose growing plants with aflatoxin contamination, while warm and wet conditions favor the contamination after maturation.

Temperature Stress- Aflatoxin production is highly temperature sensitive because temperature can affect the expression of aflatoxin biosynthetic genes. Aflatoxin production is not related to

the growth rate of A. flavus; maximal growth of A. flavus at 29–35 °C and A. flavus has maximum aflatoxin production at 28–30 °C but production decreases at temperatures close to 37 °C. Below 7 °C or higher than 41 °C, there is no aflatoxin production even after 12 weeks of fungal growth. The type of aflatoxin produced and the ratio of AFB1 to AFG1 also varies with temperature. The different aflatoxin-producing rates at different temperatures may be due to modulation of the gene expression. All aflatoxin biosynthetic genes in A. flavus, are more highly expressed at 28 °C , are downregulated above 37 °C and all aflatoxin biosynthetic pathways genes of A. flavus are shut down beyond 42°C.

Drought Stress - Dry and hot weather, particularly prolonged droughts, predispose crops to aflatoxin contamination.while irrigation reduces fungal infection and AFB1 contamination. Diseases, Insects and Other Physical Damage - Wound-inoculated corn has drastic increase in aflatoxin contamination than naturally infected corn during a drought. Corn silage made from rust infected plants has very high aflatoxin contamination, whereas no aflatoxin is detected in insect damage-free corn.

Clinical Findings in Animals

The toxic response and disease in mammals and poultry varies in relation to species, sex, age, nutritional status, and the duration of intake and level of aflatoxins in the ration.

In general, symptoms of acute aflatoxicosis in mammals include: inappetance, lethargy, ataxia, rough hair coat, and pale, enlarged fatty livers. Symptoms of chronic aflatoxin exposure include reduced feed efficiency and milk production, icterus, and decreased appetite. Reduced growth rate may be the only clue for chronic aflatoxicosis and other mycotoxicoses. The mechanism by which aflatoxins reduce growth rate is probably related to disturbances in protein, carbohydrate and lipid metabolism.

Performance and Health - Dietary aflatoxin contamination can negatively altere concentrations of plasma metabolites in ruminant, which indicates compromised animal health. Consumption of aflatoxis increases aspartate transaminase, gamma–glutamyl transferase activities, prothrombin time, and concentrations of cholesterol, uric acid, and triglyceride values, indicating suppression of liver function. Decreased concentrations of glucose, albumin, urea nitrogen and urea-to-creatinine ratio in the serum of growing wethers is also noted. In dairy cows there is an

increased concentration of plasma haptoglobin and β-integrin, indicating an increased inflammatory response caused by aflatoxins. AFB1 also reduces RBC count and hemoglobin concentration , bacteriostatic activity of the serum and in vivo cellular immunity, which indicates poor immune response to infectious diseases.

Ruminal Fermentation - Aflatoxins have toxic effects on ruminal microorganisms as they inhibit synthesis of DNA and RNA and interact with enzymes to lower ruminal microbial protein synthesis and activity. Aflatoxins, inhibit in vitro DM degradation of alfalfa hay by upto 50 %, and also reduce the total volatile fatty acids concentration by 12.7 % and 9.6% from alfalfa or ryegrass hay, respectively.

Reproduction- Aflatoxins have a negative impact on reproduction in both male and female animals. AFB1 reduces sperm viability of bulls, indicated by lower integrity of the plasma membrane, and it also reduced mitochondrial membrane potential and DNA integrity of sperm. There is differential expressions of 345 genes that are involved in cellular pathways, such as, cell cycle, DNA repair and histone modification and signaling pathways. In addition, aflatoxin impairs oocyte and the preimplantation development of embryos by inducing overproduction of reactive oxygen species (ROS), through suppression of superoxide dismutase and glutathione peroxidase activity, thus reducing antioxidant capacity. In addition, ROS can be generated by many pathways including the metabolic processes by the cytochrome P450 system, which is known to metabolize AFB1 to a highly reactive metabolite, AFB1-8,9-epoxide. AFB1 can inhibit development of preimplantation bovine embryos by reducing the percentage of oocytes becoming blastocysts partially through overproduction of ROS, causing potential embryonic loss in dairy cows.

Overall, aflatoxins can impair fertility of both female and male animals by affecting reproductive organs and cells and hinder embryonic development both pre- and post-implantation. In addition, aflatoxin exposure can impair immunity, reduce performance of animals, and is a food safety hazard when present in animal products.

Diagnosis of Aflatoxicosis

- Clinical signs and signalment
- Serum biochemical analysis, CBC, and coagulation testing

- Postmortem examination
- Feed analysis

Prevention and Treatment of Aflatoxicosis

Treatment is supportive and the hydrated sodium calcium aluminosilicates (HSCAs) can act as aflatoxin binders in feed.

Prevention of aflatoxin in dairy cattle

Aflatoxicosis can only be prevented by feeding rations free of aflatoxin. Preventing aflatoxin contamination requires an on-going and thorough sampling-cum-testing program.

1. Purchase feed from reputable companies experienced in aflatoxin prevention.

2. Don't buy poor quality feed or feed ingredients. A good deal on feed prices can be the most expensive buy a dairy farmer ever makes if it proves to contain aflatoxin. To check the feed, take a representative sample of suspect feed after milling by passing a cup through a moving auger stream at frequent intervals, mixing samples thoroughly, and saving a 4.5-kg (10-lb) sample for analysis. Alternatively, use probe sampling of recently blended grain in bins or trucks at five locations in each structure for each 6 feet of depth. Freeze or dry samples, and submit for analysis in a paper bag (not plastic). Dry samples are preferable in a paper bag to prevent condensation during transport and storage. Samples should be dried at 176°–194°F (80°–90°C) for ~3 hours to reduce moisture to 12%–13%.

3. Store feed at proper moisture levels. Check feed storage bins for leaks.

4. Develop a systematic inspection and clean-up program to keep bins, delivery trucks and other equipment free of adhering or caked feed ingredients. Remove contaminated feed and clean equipment and sanitize with hypochlorite (laundry bleach).

5. Minimize dust accumulation in milling and mixing areas. Keep all feed equipment free of caked feed. Implement effective rodent and insect control program in storage areas.

6. Grains contaminated with aflatoxins have been successfully treated with ammonia, but it is expensive and dangerous to do.

Salt Toxicity in Animals

Etiology

In general, animals can tolerate high concentrations of salt or sodium in the diet if they have continuous access to fresh water. Salt toxicosis is often directly related to water consumption and can be reduced notably or abolished completely in production animals by means of appropriate management of factors such as mechanical failure of waterers, overcrowding, unpalatable medicated water, new surroundings, or frozen water sources. Increased water requirements increase the susceptibility of lactating cows to salt toxicosis, especially in response to sudden restrictions in water. High concentrations of salt in the diet (up to 13%) have been used to limit feed intake of cattle. Salt-deprived animals or those not acclimated to high-salt diets can overconsume these feeds, making the animals prone to salt toxicosis. Improperly formulated or mixed feed can be sources of excess salt. The use of whey as a feed or as a component of wet mash can add to sodium intake. Additional sources of excess sodium can include high-saline ground water, brine, or seawater.

Mechanism of Action

As serum sodium concentration increases, water moves along the osmotic gradient out of the interstitium and intracellular fluid into the extracellular fluid. Rapid development of hypernatremia results in cerebral dehydration and neuronal cell shrinkage, with the brain then pulling away from the calvarium, which disrupts the blood supply to the brain and can cause tearing of vessels and hemorrhage. To prevent excess water loss to the extracellular fluid, cells of the brain increase their intracellular osmolarity through the generation of idiogenic osmoles. Sodium diffuses passively across the blood–brain barrier and eventually redistributes into neural tissues; however, high intracellular sodium concentrations inhibit energy-dependent pathways for transporting sodium out. With changes in cellular osmolarity in chronic water deprivation, once water access is restored, due to a rapid decrease in serum sodium concentration, intracellular water influx into neurons along the osmotic gradient can lead to cerebral edema.

Clinical Findings

In cattle, signs of acute salt toxicosis involve the GI tract and CNS. Salivation, increased thirst, vomiting (regurgitation), signs of abdominal pain, and diarrhea are followed by ataxia, circling, blindness, seizures, and partial paralysis. Cattle sometimes display belligerent and aggressive

behavior. A sequela of salt toxicosis in cattle is dragging of hind feet while walking or, in more severe cases, knuckling of the fetlock joint.

Diagnosis

History, clinical signs, and excess sodium concentrations in serum, CSF, or postmortem brain tissue samples

Analysis of food, water, or suspect feed for sodium content - Serum and CSF concentrations of sodium >160 mEq/L, especially when CSF has a greater sodium concentration than serum, indicate salt toxicosis. Brain sodium concentrations >2,000 ppm (wet wt) are considered diagnostic in cattle.

Treatment

There is no specific treatment for salt toxicosis. The mortality rate may be >50% in affected animals regardless of the supportive treatment, which includes:

Monitoring serum sodium concentration is the first step in treatment. Calculate the free water deficit (FWD) in the animal, based on the following formula:

FWD = 0.6 × BW × (current [Na+]/desired [Na+] – 1) where BW is body weight (in kg), and [Na+] is serum sodium in mEq/L.

Not more than 50% of the FWD should be replaced in the first 24 hours, with the remaining deficit replaced in the following 24–48 hours, so that serum sodium concentration is lowered at a rate of 0.5–1.0 mEq/L per hour. Quickly lowering the serum sodium concentration will increase the osmotic gradient between the serum and the brain, with water following the gradient into the brain and increasing the likelihood of severe cerebral edema. Note: On a herd basis with large animals, water intake should be limited to 0.5% of body weight at hourly intervals until normal hydration is accomplished, usually taking several days. Severely affected animals can be given water via stomach tube.

- Seizures should be controlled (with diazepam or other seizure control agents); if brain edema is suspected, mannitol, dexamethasone, or dimethyl sulfoxide (DMSO) may be helpful.
- Immediate removal of offending feed, water or other suspect material is imperative.

Urea Toxicity (NPN Poisoning)

Non Protein Nitrogen (NPN) is any source of nitrogen not present in a polypeptide form. Mature ruminants are affected most commonly. After ingestion, NPN undergoes hydrolysis and releases excess ammonia (NH3) into the GI tract, which is absorbed and leads to hyperammonemia; the poisoning is usually acute, rapidly progressive and highly fatal.

Etiology

NPN poisoning occurs commonly when only natural protein was previously fed. As such, animals should be gradually acclimated to NPN so that rumen microflora can adjust. Ruminants may require days to weeks before rumen microflora can utilize gradually increasing amounts of dietary NPN; however, such adaptation is lost relatively quickly (1–3 days) once NPN is removed from the diet. The most common sources of NPN in feeds are urea and urea phosphate. Because feed-grade urea is unstable, it is pelleted to prevent degradation to NH3. Ruminants are most sensitive to toxicosis because urease is normally present in the rumen after 50 days of age. Diets low in energy and high in fibre are more commonly associated with NPN toxicosis, even in acclimated animals. Highly palatable supplements (such as large protein blocks), range cubes, or improperly maintained lick tanks may lead to consumption of lethal amounts of NPN.

A related CNS disorder in cattle fed ammoniated high-quality hay, silage, molasses, and protein blocks is caused by formation of 4-methylimidazole (4-MI) via the action of NH3 on soluble carbohydrates (reducing sugars) in these feed. Cattle fed dietary components containing 4-MI develop a syndrome known as “bovine bonkers syndrome,” named for the abnormal belligerent behavior exhibited. Clinical signs relate to CNS effects, with stampeding, ear twitching, trembling, champing, salivating, and convulsions.

Clinical Findings

The period from ingestion to onset of clinical signs is generally 20–60 minutes in cattle. Early clinical signs include muscle tremors (especially of the face and ears), abdominal pain, frothy salivation, polyuria, and bruxism. Tremors progress to incoordination and weakness. Pulmonary edema leads to marked salivation, dyspnea, and gasping.

An early sign in cattle is ruminal atony; as toxicosis progresses, ruminal tympany is usually evident on auscultation. Violent struggling bellowing, a marked jugular pulse, severe twitching,

tetanic spasms, and convulsions may be seen. The PCV and serum concentrations of NH3, glucose, lactate, potassium, phosphorus, BUN, and activities of AST and ALT are usually considerably increased.

As death nears, affected animals become cyanotic, dyspneic, anuric, and hyperthermic, with blood pH decreasing from 7.4 to 7.0. Death related to NPN toxicosis usually occurs within 2 hours in cattle. Surviving animals recover in 12–24 hours with no apparent sequelae.

Diagnosis

- Based on history and clinical signs, with testing of NPN concentration in postmortem samples. Ruminal or cecal pH > 7.5 also aids in diagnosis.

Treatment

- Removal of the suspected feed source. Supportive care.
- Ruminal infusion of 5% acetic acid and water after collection of ruminal-reticular fluid specimens.

References

US FDA. COMPLIANCE POLICY GUIDE (CPG) (1994)Sec. 683.100 Action levels for aflatoxins in animal feeds. Revised 8/28/94.

US FDA. COMPLIANCE POLICY GUIDE (CPG)(2005) Sec. 527.400 Whole Milk, Lowfat Milk, Skim Milk - Aflatoxin M1. Updated 11.29.2005.

U.S. Food and Drug Administration (2019). Compliance Policy Guide (CPG) Action Levels for Aflatoxins in Animal Food, Available online: https: // www. fda.gov/media / 121202/ download

Meerdink GL. (2004). Aflatoxins. In: Plumlee KH, ed. Clinical Veterinary Toxicology. St Louis: Mosby; 231-235.

CAST (Council for Agricultural Science and Technology). Mycotoxins: Risks in Plant, Animal and Human Systems, Task Force Report No. 139. Ames, Iowa: Council for Agricultural Science and Technology, 2003.

Mostrom MS, Jacobsen BJ.(2011). Ruminant mycotoxicosis. Vet Clin North Am Food Anim ; 27:315-344

Kerr LA, Groce AW, Kersting KW. (1987). Ammoniated forage toxicosis in calves. J Am Vet Med Assoc. 1; 191(5):551-2.

Haliburton JC, Morgan SE. (1989). Nonprotein nitrogen-induced ammonia toxicosis and ammoniated feed toxicity syndrome. Vet Clin North Am Food Anim Pract. ;5(2):237-49.

A.F. Kertz (2010). Urea Feeding to Dairy Cattle: A Historical Perspective and Review, The Professional Animal Scientist, Volume 26 (3); 257-272.

Kosicki R., Błajet-Kosicka A., Grajewski J., Twarużek M. (2016). Multiannual mycotoxin survey in feed materials and feedingstuffs. Anim. Feed. Sci. Technol. ; 215:165–180.

Chapter 9

PRACTICAL APPROACHES TO COUNTER THE COMMON REPRODUCTIVE DISORDERS IN DAIRY ANIMALS

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Reproduction is the most important aspect in dairy animal production for the future propagation of the generation. The reproductive problems are the most common complications encountered by the veterinarians in the field conditions. The major reproductive disorders in dairy animals are related to infertility viz. delayed puberty, anestrus, repeat breeding, cystic ovarian disease, uterine infections etc. as compared to peri-partum complications like dystocia, genital prolapse, metabolic disorders etc. This chapter is destined to provide the insights into the common practical approaches for the diagnosis as well as therapeutics of the various reproductive disorders encountered by the filed veterinarians.

Common reproductive problems in dairy animals

Infertility Peri-partum complications

- Anestrus
- Repeat breeding
- Cystic ovarian disease
- Uterine infections
  - Metritis
  - Clinical endometritis
  - Subclinical endometritis
  - Pyometra

- Genital prolapse

 Ante-partum genital prolapse

 Post-partum genital prolapse

- Dystocia

 Uterine torsion

- Retained fetal membranes

1. **Anestrus**

Anestrus is one of the most commonly occurring reproductive problems in cattle and buffalo in India. It is a functional disorder of the reproductive cycle which is characterized by absence of overt signs of estrus manifested either due to lack of expression of estrus or failure of its detection.

Diagnosis of anestrus

- History
- Rectal palpation of genitalia: smooth ovaries in true anestrus
- Ultrasonography

Therapeutics of anestrus

- Nutritional management with supplementation of minerals and vitamins in diet
- Regular deworming of animals
- Estrus induction protocols using various hormones

 Progesterone based protocol: Controlled internal drug release device (CIDR)is an intravaginal 'T' shaped device is impregnated with 1.38 grams of progesterone is left into the vagina for 7-12 days and PGF2α is injected at the time of removal of device or one day before the removal of implant, animal will come into heat within 24-72 hours.

 Prostaglandin based protocol: Estrus induction protocols like "Doublesynch" and "Estradoublesynch" are most effective.

2. **Repeat breeding**

A repeat breeder is generally defined as any animal that has not conceived after three or more services associated with true estrus. The incidence of repeat breeding is high in cows compared to buffaloes (approximately 19 vs. 9%, respectively). Repeat breeding syndrome is responsible for long service period and inter-calving interval thereby causing low milk and calf production resulting in to greater economic losses to dairy industry. The causes of repeat breeding could be classified into three main types i.e. male factors (poor semen quality), managemental factors (nutrition and improper timing of insemination) and female factors (genetics, anovulation, delayed ovulation, luteal insufficiency, subclinical endometritis etc.).

Therapeutics of repeat breeding

- Proper timing of insemination with accurate nutritional management of animals
- Good quality semen from disease free bulls should be used for insemination.
- To counter the ovulation related defects, injection of 1500IU human chorionic gonadotropin, 10-20µg buserelin acetate at the time of insemination can be given.
- No use of progesterone injection as the time of insemination as it will lead to failure of fertilization and further implantation.
- For subclinical endometritis treatment use of antimicrobial like cephapirin or proteolytic enzymes (see uterine infections part) via intrauterine route.
- For exact timing of insemination, fixed timed insemination protocols like ovsynch, doublesynch, estradoublesynch or progesterone based protocols can be used.

3. **Cystic ovarian disease**

It is very common in dairy cattle than other species and is closely associated with milk production in that it is seen more commonly in higher producing cattle. In cattle, the incidence is 10-20%. It is high in lactating animals and more common during 2nd to 5th lactation (4 to 6 years of age) and a higher incidence usually from 1 to 4 months after calving with a peak between 15-45 days. Self-recovery occurs in 15-30% cases within 45 days of calving. The follicular cyst and luteal cyst are two types of pathological cystic conditions. Follicular cyst is characterized by presence of follicle or follicle like structures which are usually more than 25

mm in diameter if single follicular cyst or more than 17 mm in case of multiple follicular cysts and the thickness of follicular wall is less than 3mm and the condition persist for more than 10 days. Luteal cyst is characterized by diameter of corpus luteum more than 25 mm and persisting for more than 10 days with the thickness of luteal wall more than 3 mm. Follicular and luteal cyst are anovulatory i.e. origin is from the follicle which fail to ovulate whereas cystic CL occur after ovulation. Right ovary is more affected than left reflecting the relative activity of the ovary.

Diagnosis

•History: Nymphomaniac behavior, short inter-estrus interval, longer estrus duration, copious amount of cervical mucus discharge in follicular cyst while, anestrus in luteal cyst.

•Per-rectal examination: large sized ovary with soft thin walled structure as follicular cyst while, a protruding liver like consistency structure or with thick wall as luteal cyst.

•Ultrasonography: Anechoic structure with various degrees of luteinization and thick wall as luteal cyst while, anechoic with thin wall and no lutenization as follicular cyst.

Therapeutics

•Luteal cyst: Prostaglandins (cloprostenol 500µg or dinoprost 25mg, prolonged cyst may require more than one injection).

•Follicular cyst: CIDR plus ovsynch protocol is best for follicular cyst treatment as many a times the cyst does not respond to buserelin acetate or human chorionic gonadotropin.

Uterine infections

a. **Metritis**

Metritis is most common within 10 days of parturition andcharacterized by an enlarged uterus and a watery red-brown fluid to viscous off-white purulent, uterine discharge, which often has a fetid odour. The incidence of metritis varies between breed, country, and herd. However, in some studies the incidence is as high as 40% of the herd. The associated clinical signs are used to classify the severity of disease, which varies from unapparent disease to fatal toxaemicmetritis. Grade 3 metritis also known as puerperal or toxic metritis is most commonly observed which is associated with decreased feed and water intake, and can be associated with development of the

downer cow syndrome. Puerperal metritis is highly associated with dystocia, retained placenta, twin births, and abortion, which occurs during the first three weeks after parturition.

b. **Clinical endometritis**

Clinical endometritis is defined as the presence of a purulent uterine discharge detectable in the vagina of cattle 21 days or more postpartum or a mucopurulent discharge detectable in the vagina after 26 days postpartum. The incidence of clinical endometritis is around 10% to 20%, with variation between breed, country, and herd. A simple grading system based on the character of the vaginal mucus is readily used to evaluate cows with clinical endometritis and is prognostic for the likely outcome of treatment.

c. **Subclinical endometritis**

Subclinical endometritis is characterised by inflammation of the endometrium in the absence of clinical signs of endometritis, which results in a significant reduction in reproductive performance. The inflammation is presumably associated with recovery of the tissues after metritis and clinical endometritis, trauma, or other non-microbial disease. It is usually diagnosed by cytological examination after taking samples from uterus by either cytobrush or cytotape. The smear is prepared and stained with Giemsa staining for the counting of polymorphonuclear cells (PMNs). The animals having more than 5% PMNs after 50 days postpartum are said to be suffering from subclinical endometritis.

d. **Pyometra**

Pyometra is characterised by the accumulation of purulent or mucopurulent material within the uterine lumen, causing distension of the uterus, in the presence of a closed cervix and a functional corpus luteum (CL). Postpartum pyometra is uncommon and is thought to be caused by the growth of bacteria within the uterine lumen after the formation of the first CL.

- **Therapeutics of uterine infections**

Intrauterine vs systemic therapy

It has been observed that choice of whether intrauterine or systemic therapy is still a question before the veterinarians to treat the uterine infections. The systemic antimicrobial therapy must be used to treat the cases of metritis and pyometra while intrauterine treatment is preferred

treatment of choice for endometritis as it is local inflammation and does not result in systemic illness.

Principles of antimicrobial therapy

Some clear principles underlie the choice of antimicrobial and/or antiseptic agents:

- It must be effective against the wide range of aerobic and anaerobic, Gram-positive and Gram-negative bacteria that are present.
- It must be effective within the micro aerophilic environment of the uterus.
- Whether an effective bactericidal or bacteriostatic concentration can be achieved at the site of infection by the intrauterine route of administration.
- When the intrauterine route is used, the substance must be evenly and rapidly distributed throughout the uterine lumen with good penetration into the deeper layers of the endometrium.
- It must not inhibit natural uterine defense mechanisms, particularly the cellular component.
- It must not traumatize the endometrium.
- Treatment must not reduce fertility by producing irreversible changes in the reproductive system.
- Treatment must be cost effective by enhancing fertility.
- Nitrofurazone is an irritant and has an adverse effect on fertility.
- Aminoglycosides are not effective in the predominantly anaerobic environment of the infected uterus.
- Sulphonamides are ineffective because of the presence of paraaminobenzoic acid metabolites in the lumen of the infected uterus.
- Penicillins are susceptible to degradation by the large numbers of penicillinase producing bacteria that are present in the infected uterus.

- Intra-uterine administration of tetracyclines may worsen subsequent reproductive performance due to irritation and pH damage to the endometrium.

Novel therapy with proteolytic enzymes

A combination of three proteolytic enzymes viz. chymotrypsin, trypsin and papain which act as biological scalpels and have fibrinolytic and proteolytic activity in the inflamed tissue results in breakdown of products of infection, damaged cells and tissues. The gram-positive and gram-negative bacteria, yeasts, and toxins contain proteins and lipids that are degraded directly by these enzymes leading to stasis in growth or death of bacterium. The dosage of these enzymes for endometritis is 8mg, 8mg, 4mg for Trypsin, Chyotrypsin ad papain, respectively dissolved in 50mL normal saline administered intrauterine.

5. **Genital prolapse**

The genital prolapse occurs as antepartum or postpartum complication mainly where vagina with/without cervix and uterus comes outside in the pregnant animal or complete uterus comes outside the body after parturition. It can also occur in non-pregnant animals due to feeding of estrogenic compounds in large amount. Antepartum cervico-vaginal prolapse is more common in buffaloes while, post-partum uterine prolapse in cattle.

Tips for management of genital prolapse

- Epidural anesthesia (5-10 mL 2%Lignocaine hydrochloride at sacro-coccygeal or first inter-coccygeal space).
- Proper cleaning of prolapsed mass with potassium permanganate solution or clean water.
- Three R principal of reduction (using cold water, alum etc.), replacement (use of fist and palm; no use of fingers) and retention of prolapsed mass (rope truss, Buhner's sutures in post-partum prolase).
- Progesterone injection (500mg hydroxyprogestronecaproate at weeky/fortnightly interval) can be given in the pregnant animals (avoid in the last month of gestation).
- Calcium therapy (calcium borogluconate 450mL intravenous or subcutaneous).
- Reduce feeding of feeds having estrogenic compound like soyabean, berseem etc.

6. Uterine torsion

Uterine torsion is usually defined as the rotation of uterus on its longitudinal axis. Out of domesticated ruminants, dairy buffalo is more susceptible to uterine torsion but, cattle are also affected in a significant number.

Diagnosis

History: The history is critical for deciding the line of treatment. The time lapse between occurrence of torsion and its report to the veterinarian, details of previous treatment and stage of gestation should always be considered. It is important to critically examine changes in the udder, pelvic ligament relaxation, perineal edema and other signs of calving.

Clinical signs: The frequently reported signs are anorexia, frequent straining with lying down and getting up, severe abdominal pain manifested as kicking at belly, tachycardia, tachypnea, decreased rumination, restlessness, dehydration and/or fever in delayed cases. The severity of signs is proportionate to the degree of rotation and duration of torsion. Sometimes clinical signs may not be prominent, if torsion is mild or of less than 180º. The initial manifestation is abdominal pain. All the signs of parturition may be evident if torsion occurs around parturition. There is abdominal straining but no progress to second stage of labour.

Per-vaginal / per-rectal examination: Per-vaginal examination should be done first. Care should be taken to minimize contamination during vaginal examination. The direction of rotation (right or left), location of twist (pre or post cervical) and approximate degree of rotation should be determined. Per-vaginal examination confirms post-cervical uterine torsion whereas rectal examination can establish both pre and post cervical torsion by palpating the altered orientation of broad ligaments or presence of twist on the cervix. A rectal examination is necessary to make a confirmatory diagnosis in case of pre-cervical torsion and to rule out uterine adhesions with adjacent structures.

Therapeutics

A thumb rule must be followed for the uterine detorsion using Sharma's modified Schaffer's method. The cases having uterine adhesions (usually delayed than 72h) must not be rolled for distortion and should be directly treated by cesarean section. The animal should be casted on the same side of uterine torsion with rolling on the same side after fixing the uterus using a plank

with weight of 2-3 persons. If, uterine detorsion does not occur even after 3 rolls then the animal should be treated by cesarean section.

7. Retained fetal membranes (RFM)

RFM is defined as the failure of an animal to expel the fetal membranes within 12 hours of the end of parturition. Retained placenta is an alternative name used for RFM. There is some variation in the literature about the duration of retention that defines the clinical disorder. Some prefer to define retention as being for 12 hours; however, the timing is arbitrary, and most normal cows expel the fetal membranes within a few hours of parturition. The incidence of RFM varies among herds, but is typically 5% to 10% of animals. The importance of RFM is that they are associated with reduced milk yield and an increased risk of metritis. It is a common complication of bovine parturition. The predisposition to infections of the uterus means that RFM are an important contributor to bovine infertility. Occurrence of RFM is associated with the failure of the normal processes of placental dehiscence and expulsion.

Practical approaches to treat RFM

- No manual removal of fetal membranes
- Use of ecbolics like prostaglandins and ergot alkaloids, oral herbal uterine cleansers
- Use of systemic antimicrobial like Ceftiofur
- Use of antipyretics to reduce temperature
- Use of antioxidants like vitamin E and selenium

Conclusion

Reproductive disorders are very commonly encountered at field level by the working veterinarians. The infertility related disorders are found more than peri-partum complications. The proper reproductive management of dairy animals is very essential for the better economics, goof fertility, more calf crops per animal as well as for more milk production.

Chapter 10

SEASONAL MANAGEMENT OF DAIRY ANIMALS

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Environment exercises influence over health and productivity of animals to a great extent. Domesticated animals express their true production potential and normal behavior including optimum health status under favorable environmental conditions. Both macro and micro environment exert their effect on farm animals. Air temperature has primary effect among other weather conditions, with wind, precipitation, humidity and radiation having secondary effect. Normally, effective ambient temperature is used to describe the impact of the thermal environment which is a combination of various climatic factors. Longer periods of high ambient temperature along with high relative humidity prevent dissipation of excess body heat by dairy animals in Indian conditions. On the other hand, animals under cold stress adjust to the low temperature by increasing their metabolic rate and energy expenditure. Fodder scarcity due to snow and poor quality of fodder further aggravate the stress on animals. Productivity of dairy animals is greatly affected outside the thermo-neutral zone. Maintaining the lower and upper critical temperature of thermo-neutral zone is critical for optimum production from farm animals. As such health and production of dairy animals depends on the management practices.

Temperature-Humidity Index

Environmental factors such as surrounding temperature, relative humidity, radiation, and wind velocity interact to result in “Heat stress”. Heat stress can result in lowering production and reproduction related performance. Heat stress is an important stress related factor, which expresses a combination of temperature and humidity and is commonly used to measure heat stress in dairy animals. Heat stress not only limits fodder and feed availability, it also reduces dry matter intake (DMI). Environmental temperature above 35°C activates stress mechanisms in lactating dairy cows. The thermoneutral zone (TNZ) of dairy animals is understood to exist from

16℃ to 25℃. Within this range, animals maintain a physiological body temperature of 38.4-39.1°C.

THI can be calculated as: THI = tair− (0.55−[0.55*relh/100])*(tairf-58.8) (Buffington et al., 1977) where, THI=Temperature humidity index, tair=air temperature in Fahrenheit, relh=percent relative humidity.

The exotic crosses of Holstein experience discomfort above the temperature of 25℃ depending upon temperature and humidity changes. When the Cattle Heat Stress Index is at 71 or below, cattle are in the thermal neutral zone, i.e., THI of 72 is considered as optimum. Values ranging from 72 to 80 correspond to "mild stress" and values above 80 mean "severe stress". In northern India March marks the start of summer season but humidity rises with high temperature after June, thus increasing stress level for the animals. The weather between July and October brings severe stress level. Heat stress and increased THI reduce daily milk yield of cows between 35 to 40%. In the high yielding cows once the milk production decline may cause an irreversible loss of upto 8-9 L milk every day. Low yielding cows did not show any significant change when the THI was above 72 from month June to October during stress condition in Dehradun district (India). But high yielding cows shows a significant decrease ($p<0.05$) in milk yield when THI was above 80 (severe stress zone) in the month of June to October and milk production decrease from an average of 18±1.4 to 10.9±0.92 L whereas in November-December when THI declines in the zone of comfort the milk yield did not show significant rise (Kohli *et al*, 2014).

A study based on ten years of data (2001–10), based on12673 observations on fortnightly milk yield of buffaloes reared in a dairy farm near Karnal, established the relationship between weather and production of lactating buffaloes. The critical threshold level of maximum THI for buffaloes was estimated to be 74, which is higher than that of crossbred cows. The study concluded that buffaloes start experiencing discomfort starting from mid-March up to early November. Starting from early May, milk decreases by more than 1% per unit increase in maximum THI over 82. High summer THI results in major heat stress and has a negative result on milk production as well as composition in buffaloes. Hence, extra feeding, housing and management practices might be required to counter thermal stress and maintain milk production of the animals (Kalyan *et al*,2022).

Effects of Winter Stress on Dairy Animals

The stress of winters is expressed in two ways on dairy animals:

1. Direct Effects

2. Indirect Effects

1. Direct effects: Direct effects are observable on milk production, reproduction, body condition score, feed utilization and health of animal.

a. **Milk production:** Cold exposure may directly limit the capacity of the mammary gland to synthesize milk by reducing mammary gland temperature, or indirectly through blood supply to udder.

b. **Reproduction:** Low temperature can cause failure of proper follicular development, follicular atresia, loss of libido, decreased pregnancy rates and increased calving intervals. Fertility of bulls may be reduced due to underfeeding.

c. **Body Condition Score:** Higher body condition score ensures insulation against cold and cows tend to minimize surface area exposed to difficult weather. As body reserves are exhausted due to winter stress, cows losing more than 16 % body weight may not conceive in the next season. Starvation and pregnancy toxaemia may occur due to imbalanced nutrition.

d **Health:** winter stress may increase respiratory infections & hypoxia, compromise the immune response in poorly ventilated shelters, increases basal metabolic rate, frost bite, asthma, coccidiosis and postnatal mortality.

2. **Indirect effects:** These affects are mainly seen in fodder production, water quality and quantity, and housing.

a. **Housing:** Inadequate space or ventilation can cause respiratory difficulties in animals. Overcrowding can cause moisture accumulation in the floor which can lead to foot rot.

b. Forage deficiency in quantity & quality may result due to poor production and fall in nutrients (C.P and TDN)

c. **Water:** Water supplies may run out; water quality may be poor and frequency of watering may also be affected.

Animal's response to cold stress

1. Innate defense mechanisms activate adaptive processes resulting in physiological and morphological changes enabling the organism to survive with less discomfort.

2. Vasoconstriction reduces the temperature difference between skin surface and environment, thereby decreasing heat loss.

3. Entrapped air, which occupies 95% of the volume of the hair coat provides insulation. Insulation increases with coat thickness and hair density. Piloerection also contributes to hair coat insulation.

4. Below the Lower Critical temperature, heat production is almost linear with depression in temperature. When summit metabolism approaches where heat production cannot rise any further, increased feed intake, muscular activity, heat increment and shivering can generate extra heat.

5. Brown fat generates heat in animals or neonates that do not shiver.

Management of winter stress

1. **Nutrition**

a. Increasing the energy content (77% against 70 -72% TDN) and protein (17.5% as compared to 14.5% CP) may be attempted. Rations containing about 20% as compared to 17% fiber in the animal feed can increase fat percentage in milk.

b. Animals outdoors will need about 15 to 20% more feed for the season as compared to animals kept in sheds.

2. **Winter stocks:** Apart from cultivated fodders and tree leaves, other options can be considered to counter forage shortage:

a. Concentrates in the form of Feed Blocks, UMMB Licks, Cubes, Meals and cakes.

b. Conserved Forage: Hay, Silage, Haylage, Leaf meal, etc.

c. Storage of crop residues: Stover, Straw etc.

d. Aquatic vegetation: Typhaangustataetc..

e. Fodder banks: Surplus fodder can be generated during summer and conserved for use during winters.

f. Urea treatment of straws: Application of urea (4-5%) increases the digestibility by about five units. Ensiling for 10 days, digestibility can be doubled. Cows fed urea treated wheat straw in diet are reported to have higher live weight gain than cows fed hay based diet.

3. **Shelter Management:**

a. Proper sheds should be constructed to protect the animals against wind.

b. Heating facilities like room heaters or strong curtains can be provided.

c. Improved bedding: Clean and dry bedding upto 6 inches and 2 inches can be provided to adults and young stock respectively

d. Draft free ventilation should be provided with relative humidity in the range of 40 to 80%.

e. Calf jackets and blankets are also helpful to keep calves warm.

f. Calvings can be planned for late in spring/ early summer, through hormone application or timed insemination.

g. Water Management: Water consumption is more when water temperature is 47°F or above.

4. Health Management

a. Vaccinations, nutritional supplementation and deworming protocols can negate disease occurrence due to winter stress

b. Animals must get exercise whenever possible. Exercise will help prevent obesity and overgrown hooves.

c. Use of teat dips protects the udder skin during cold winter.

d. Check bruises on soles, trim overgrown hooves and prevent laminitis and lameness.

Effect of Summer Stress/ Heat Stress on Dairy Animals

Heat Stress (HS) effects health of dairy animals through direct or indirect effects on physiology, metabolism, endocrine, and immunity system.

1. Feed intake and rumen physiology: Increase in environmental temperature directly effects the hypothalamus and decreases feed intake. Feed intake begins to decline at air temperatures of 25-26°C in lactating cows and reduces more rapidly above 30°C in temperate climatic conditions.

At 40°Cthe decline in feed intake may be as much as 40% which leads to negative energy balance. An Animal under HS has reduced acetate production whereas propionate and butyrate production increases. Consequently, rumen pH is also altered as rumen microflora is disturbed and feed intake is reduced.

2. **Acid-base balance:** Animal affected by HS has increased RR and sweating. When RR increases, expiration of CO_2 via the lungs increases resulting in respiratory alkalosis, as blood carbonic acid concentration decreases. Therefore, animal has to compensate for higher blood pH by excreting bicarbonate in the urine. Chronic hyperthermia induced in appetence further aggravates the increased supply of total carbonic acid in the rumen and decreases ruminal pH thereby, resulting into subclinical and acute rumen acidosis.

3. **Immune system:** The immune system protects the body against environmental stressors. Primary indicators of immunity response include white blood cells (WBCs), red blood cells (RBCs), hemoglobin (Hb), packed cell volume (PCV), glucose and protein concentration in blood get altered on thermal stress. High variation of Hb, PCV, plasma glucose, total protein and albumin have been reported in malpura ewes. Higher PCV value is understood to be an adaptive mechanism to provide necessary water for cooling by evaporative process.

4. **Milk production and composition:** HS has a negative impact on milk production and its composition, especially in high yielding animals. Research has established that environmental temperature above 35°C activates the stress response systems in lactating cows which directly impact feed intake leading to NEB and thus decline in milk synthesis. Also, maintenance requirements of energy shoot up by 30% under seat stress. West (2003) reported a reduction of 0.85 kg DMI for every 1°C rise in air temperature above Thermo Neutral Zone in cows. This decrease in intake is responsible for around 36% of the drop in milk.

5. **Effects on reproductive performance:** Heat stress affects reproduction of farm animals in many ways. Reduction in the length and intensity of estrus, anestrous and silent heat have been reported. Secretion of ACTH and cortisol is reduced and estradiol-induced sexual behavior is affected. Low estradiol secretion also affects expression of estrus, gonadotropin surge, ovulation, gametes transport and ultimately reduced fertilization. It has been further reported that heat stress can damage developed follicles which are particularly damaged when the body temperature

exceeds 40°C. It has also been reported that suppression of heat signs in buffalo may be due to low estradiol on the day of estrus during summer period.

Management of Heat Stress:

Sustainable livestock farming must aim for practical modifications of the environment, nutritional management and development of genetic tolerance in breeds.

1. **Environmental modification:** The most common method to relieve stress is by modifying the environment through sheds and shade, supplemented with nutritious feed and drinking water. Encouraging evaporative cooling with water as fog, mist or sprinkling with air movement, and possibly cooling ponds can also prove helpful. Dairy cattle have been found to respond positively to sprinklers in terms of milk production, reproduction and conversion of feed to milk. Various types of roofing materials can be used from metal to synthetic materials for shade structures among which a white galvanized or aluminum roof is considered best. Other researchers have compared thatch roof with metal, slate and cemented sheets and recommended thatch roof over the others.

2. Nutritional management: Nutritional interventions can help the animals to maintain homeostasis. Lower DMI due to hot weather reduces nutrients availability use efficiency. Use of bypass protein improves milk yield during hot weather. Cows fed with diets containing 14% versus 17 or 21% acid detergent fiber (ADF) showed improved DMI and milk yield. Feed containing low fiber rations during hot weather is logical since heat production is highly associated with metabolism of acetate compared with propionate Oxidative damage from heat stress can be minimized through vitamin supplementation especially vitamins C, E and A and also mineral such as zinc. Vitamin C supplementation along with electrolyte can boost cell-mediated immunity in buffaloes. Yeast has a role in decreasing the production of ruminal ammonia and increases microorganism population in rumen.

3. **Genetic selection:** Environmental modifications and nutritional management can reduce thermal stress to some extent, but long-term strategies are needed against climate change. Differences in thermal tolerance have to be identified to select thermotolerant animals. It has been established that cattle with shorter and thick hair and lighter coat color are more adapted to hot environments.

References:

Manzoor A, Maqbool I, Ganaie ZA, Afzal I, Khan HM and Zaffe B. Mitigating winter vagaries in dairy animals: A review. *International Journal of Veterinary Sciences and Animal Husbandry* 4(1): 01-05

Sivakumar T, Suraj PT, Yasotha A, Phukon J. 2017. Identification of suitable housing system for dairy cattle in North East Zone of Tamil Nadu, India, with respect to microclimate, *Veterinary World* 10(1): 1-5.

Das R, Sailo L, Verma N, Bharti P, Saikia J, Imtiwati, Kumar R. 2016) Impact of heat stress on health and performance of dairy animals: A review, *Veterinary World* 9(3): 260-268.

Kohli S, Atheya UK, Thapliyal A. 2014. Assessment of optimum thermal humidity index for crossbred dairy cows in Dehradun district, Uttarakhand, India, *Veterinary World* 7(11): 916-921.

Chapter-11

Factors Affecting Onset of Puberty in Dairy Animals

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The productivity of livestock depends upon a number of genetic and environmental factors. Among environmental factors nutrition, housing, calfhood disease etc plays major role to affect productive and reproductive efficiency of the animals.

Onset of puberty and further age at first calving determines the lifelong milk production and calf crop in dairy animals. Thus it is important to know the basic mechanism behind onset of puberty and factors affecting thereof. Activation of hypothalamic-pituitary-gonadal axis is major determinant of onset of puberty. Literature reveals that in cows LHRH pulses can be detected at as early as at two weeks of age. At four weeks of age exogenous administration of LHRH could cause release of LH. In one month old calf ovulation could be induced by exogenous administration of hormones, though these changes were age dependent. In older calves the exogenous administration of LHRH will induce more release of LH, similarly number of ovulations will increase if hormones are used in older calves. Wave like pattern of follicular growth has been reported at an age as early as two weeks. This information led to development of two theories. One theory says that more production and release of GnRH from hypothalamus and further pituitary hormones make gonads active, while the other theory says that there is endocrine block due to negative feedback effect of low concentrations of oestrogens at pituitary level. The later theory is more accepted as it has been proved by various experiments. In one month old calf, ovariectomy led to higher release of LH. Exogenous administration of oestrogens also diminish release of LH, which is higher in younger calves as compared to old calves. Near onset of puberty higher frequency of LH pulses and bigger size of follicle has been observed. This led to the conclusion that with increasing age negative feedback effect of oestrogens decrease at pituitary level, leading to higher release of LH. Under affect of this LH, follicular size increases to the extent that oestrogen produced from them is able to induce LH surge and

thus ovulation. Usually the first cycle shown by the animal is not associated with overt signs as there is not proper priming of progesterone. There are more chances of short cycle as well.

A number of factors affect onset of puberty in dairy animals, the first and foremost being genotype of the animal. Puberty is reached early in exotic cows as compared to indigenous zebu cattle. Delayed puberty has been observed more in buffaloes as compared to cows. Among the environmental factors nutrition of animals, calf hood disease, season of birth of calf etc affect onset of puberty. A study conducted at Punjab Agricultural University, Ludhiana revealed that as compared to fold plain zone of Punjab it is higher in sub mountain area, which seems to be due to less nutrition in the later area. Farmers often keep heifers on leftovers of milk producing animals, leading to deficiency of many nutrients in heifers leading to delayed onset of puberty in them. At well managed farms the onset of puberty has been noticed at an age as early as two years, thus indicating that buffaloes have potential to reach puberty early but environmental factor affect it largely. Similarly housing of animals also affects the trait as the state observes extremes of weather. During summer it is as high as 47 degree Celsius and during winter as low as zero degree Celsius, hence animals kept in comfortable environment reach puberty early as compared to animals where housing is poor. Farm size also affects productive and reproductive performance of buffaloes. At large farms, since the housing and feeding are better, so animals reached puberty early.

Nutrition affects the circulatory concentration of growth hormone, insulin, insulin like growth factors, which ultimately effect release of hormones. This has been shown by studies in ovariectomized animals. Dietary manipulations, especially glucogenic diets favour postpartum reproduction, should affect onset of puberty in buffaloes calves also.

Researchers have elucidated that administration of exogenous hormones in delayed pubertal heifers can act as a trigger and lead to onset of puberty. PMSG has given acceptable results in progesterone primed animals. Alone PMSG can induce cyclicity but conception rates remained poor. Studies have shown that improved nutrition before administration of hormones improves the conception rates and number of buffalo's heifers attaining puberty.

A critical amount of fat is required for early onset of puberty, though it is not the sole regulator. Body condition score of the animal is good indicator of body fat, which can be regularly checked

at farms. Many studies have shown that rather than total weight of the animal, daily weight gain is important for onset of puberty. Hence preferably weight of calf should be regularly checked and calves growing at low rate may be culled. Wherever the facility of weighing is not possible, body condition scoring should be carried out at regular intervals.

Buffalo being kept in Punjab is water buffalo hence wallowing is important for them, alternately they should be given bath. Duration of wallowing and number of times the animal was given bath affected the onset of puberty.

Suckling has positive effect on onset of puberty in buffaloes. Though the reason is not well known, but like in indigenous cows, calf-mother bonding is markable in buffaloes. Some other studies have shown that suckling has negative affect on onset of postpartum ovarian activity in buffaloes which indirectly indicates that calf-mother bonding has got the affect. Sucking also insures good nutrition for the calf. Keeping in mind the onset of puberty in calves and onset of post partum ovarian activity in mothers, a balance between two should be maintained. Keeping a bull in the herd hastened the onset of puberty, which could be due to pheromones.

Calves born during summer season reach puberty late, though this factor can be nullified by giving them proper nutrition and housing after birth. Quite often calves suffer from one or other disease, especially diarrhoea, which can be parasitic, viral, bacterial in origin. Simple deworming hastened onset of puberty. Duration of illness affects the onset of puberty. Education of farm manager also affects onset of puberty as better educated managers are aware of effect of better housing, feeding, and prevention of illness on onset of puberty in buffaloes.

In conclusion, a number of environmental factors affect onset of puberty in buffaloes, apart from its genotype. They have potential to reach puberty provided they are given proper nutrition, housing and saved from calf hood diseases. There is need to make farmers aware about economic losses due to delayed onset of puberty vis a vis role of environmental factors.

References:

Choudhary B and Sirohi S. 2019. Sensitivity of buffaloes (Bubalus bubalis) to heat stress. *Journal of Dairy Research* 86(4), 399-405. doi:10.1017/S0022029919000773.

Kalyan UPV, Vasantha SKI and Tej JNK. 2022. Effect of seasonal variation in Temperature Humidity Index on milk production and its composition in Murrah buffaloes. *J. Vet. Anim. Sci.* 53(1): 89-93.

Showkat AB, Bhushan B, Kumar N, Lone SA, Bharti P, Chandrasekar T, Godara AS and Godara RS. 2017. Role of Infrared lamps in cold stress alleviation during winter in Murrah calves. *Indian Journal of Animal Research* (51):827-831.

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