Natural toxicants and Antinutritional factors of feeds and detoxification processes

Several feedstuffs are being used in the formula feed of animals as carriers of nutrients. Most of them are added to provide good quality proteins, easily digestible energy and micro nutrients for productive efficiency and well being of animals. However some feeds, especially of plant origin may contain various undesirable anti-nutritive principles in addition to their desirable nutrients. The term metabolite includes all the nutrients (amino acids, fatty acids, sugars, minerals and vitamins) as well as the compounds formed during their intermediary metabolism. An antinutritional factor is a substance which interfere with the absorption or utilization of metabolites in the animals’ body. Many of the metabolites have gained the interest of nutritionists are those that have become known as anti-nutritional substances or anti-nutritional factors; materials that should be avoided or eliminated, or whose effects must be ameliorated. Antinutritional factors in livestock foods may be defined as the substances which interfere with food utilization and affect the health and production of animals. These deleterious substances, also referred to as anti-quality factors. An antinutritional factor is a substance which, when present in human or animal foods, reduces growth. Examples are phytate, protease inhibitors (notably soybean trypsin inhibitor) and excessive dietary fiber.

However, even among the substances that are related to this category, it is increasingly found that, in appropriate circumstances, they may also have beneficial effects, for example the potential anticarcinogenic effects of glucosinolates and phyto-oestrogens, the antioxidative and antiatherogenic effects of allyl thiosulphinate and disulphide and the potential coccidiostatic action of artemisin. Whilst these potentially beneficial effects have been studied mainly with human health in mind, it is quite possible that some of these substances may be of value in poultry nutrition as well.

A toxicant is a substance which under practical circumstances impair some aspect of animal metabolism and produce adverse biological or economic effects in animal production. This is a broad definition, but encompasses those aspects that are relevant in livestock production. Virtually everything is toxic, including oxygen, water, and all nutrients, if given in a large enough dose. Thus, the term “toxicant” refers only to those substances which normally be encountered at toxic levels. Other terms used synonymously with toxicant are “poison” and “toxin”. The term toxin includes the naturally occurring poisonous substances in some feeds. Of all the livestock species, birds are most susceptible to the presence of antinutritional factors in feeds as their smaller body weight and higher metabolic rate.

INFLUENCE OF TOXICANTS ON ANIMAL AGRICULTURE

Toxicants can influence animal agriculture in several ways.

- They can directly intoxicate animals, resulting in mortality or decreased production of animal products.
- Toxicants may be implicated in reducing the wholesomeness of meat, poultry products, and dairy products due to the presence of hazardous residues.
- Natural toxins may reduce the availability or usability of nutritious feedstuffs, or may necessitate the use of costly feed processing techniques to eliminate their effects.
WHY DO PLANTS CONTAIN TOXIC SUBSTANCES

Plants and animals are mutually dependent inhabitants of the planet. When looked at from a geologic time perspective, neither one can do without the other. The biosynthetic capacities of plant allow them to trap solar energy and synthesize organic compounds from carbon dioxide and release oxygen as a waste product. Animals feed on plants as their source of nutrients and utilize the oxygen, and excrete carbon dioxide which is used by plants. It is not in the best interest of either plants or animals, from an evolutionary perspective, for plants to be eaten to extinction by animals, or for plants to be totally resistant to animal herbivory. Either choice would lead to extinction of both classes of organisms. Thus plants and animals exist in a mutual relationship and plant toxins have an important role in this relationship.

Since plants are immobile and unable to resist herbivory by moving, they must have other means of defending themselves from being eaten. These defenses are primarily physical and chemical. Physical defenses include spines, thorns, leaf hairs and highly lignified tissue, while chemical defenses consist of compounds which in some way deter herbivory. These chemical compounds are often referred to as secondary compounds, to distinguish them from the primary substance of plant metabolism. Secondary substances exert their effects in several ways. They may cause the plant to be unpalatable through effects on taste or smell. Many plant toxins (e.g., alkaloids, glucosinolates, saponins) are bitter or otherwise unpleasant tasting, while terpenes and essential oils have unpleasant odors which reduce palatability to herbivores. Phenolic compounds have an astringent effect because they react with taste receptors in the mouth. These chemical defenses of plants confer resistance to mammalian, invertebrate, fungal and bacterial organisms. From the plant viewpoint they are chemical defenses, from our viewpoint they are toxins or poisons.

Those properties of secondary plant metabolites that confer adverse effects, when consumed by animals, in many instances also provide the plant with resistance to predatory insects and animals and plant pathogens. For that reason these substances are obvious substances to consider when seeking ‘natural’ means of protecting plants, thereby reducing agrochemical use by building insecticide resistance into crop plants. Wheat germ agglutinin, which is normally present at about 300 mg/kg, if incorporated at insecticidal levels (7000 mg/kg) will produce pancreatic enlargement, inefficient utilization of dietary protein, thymus atrophy and depressed growth in experimental animals.

HOW ANIMALS OVERCOME THE PLANT DEFENSES?

Plants and Animal have coevolved. As plants have evolved particular defense mechanisms, herbivores have evolved various means to overcome the plant defenses.

Animals have various strategies to overcome the effects of plant toxins. They can overcome physical defenses such as thorns and spines by developing feeding strategies that allow them to daintily browse leafy material while avoiding spines (e.g., giraffe, goat) or to develop a tough mouth which allows them to consume spines with no ill effect (e.g., thinoceros). Chemical defenses can be overcome in several ways. Some animals have developed tolerance to bitterness. Sheep are more tolerant to bitterness than cattle and may consume plants which cattle avoid.

A major defense mechanism of animals against plant toxins is a liver enzyme system called the ‘drug metabolizing enzyme system’ or the “cytochrome P450 system”. This is a series of liver enzymes which metabolizes a wide variety of foreign compounds to allow their excretion. Generally this involves an oxidation process, to put a hydroxyl group on an aromatic compound, allowing it to conjugate with a water-soluble compound like glutathione or glucuronic acid, with excretion in the urine. There are major species differences in liver enzyme activity, which can account for species differences in susceptibility to plant toxins. For example, sheep and are resistant to pyrrolizidine alkaloids found in senecio sp, (e.g., tansy ragwort) while cattle and horses are very susceptible. This is largely because in cattle and horses the
alkaloids are rapidly converted to reactive metabolites in the liver, while the conversion in much slower in sheep and goats.

Another detoxification site is the rumen. Ruminants are often more resistant to plant toxins than non-ruminants, because of the degradation or inactivation of toxins in the rumen. Glucosinolates in rapeseed and other Brassica sp, and gossypol in cottonseed are examples.

WHY ANIMALS EAT POISONOUS PLANTS?
A frequently asked question is: why do animals eat poisonous plants? The answer is no simple, but among the reasons are the following:

1. Total lack of sufficient palatable forage the animals are hungry.
2. Decrease in palatability and nutrients of mature, weathered range grasses, with the result that poisonous plants become more appealing, comparatively speaking.
3. Insufficient spring grass.
4. Rain, melting snow, and heavy dew may enhance the palatability of some poisonous plants and
5. Going without water too long, which results in a reduction in feed intake, then, after watering, they develop a ravenous appetite and eat anything in sight-including less palatable poisonous plants.

IS THERE A PROBLEM?
There are two steps in determining if there is a problem of toxicity;

2. By chemical analysis.

METABOLISM OF TOXICANTS
The principal rout of exposure of most natural toxicants to livestock is through the diet. Ingested toxins may be subjected to a number of metabolic processes in the digestive tract and in various tissues prior to excretion. Animals have been exposed to toxic constituents of plants during the long period of their coevolution and have developed numerous biochemical strategies for detoxification of poisonous compounds. Once a toxicant has been consumed, there are several barriers it must surmount before reaching its critical target. These include chemical and microbiological detoxification mechanisms in the gastrointestinal tract, a host of detoxifying enzymes in the liver, and similar enzymes in all other tissues.

Liver tissue has a very high level of toxin-metabolizing enzymes. This is of obvious significance, as absorbed substances are taken to the liver by the portal circulation ----- entering the general circulatory system of the body. Thus, the liver is a first line of defense and can detoxify many poisons before other tissues are exposed to them. A consequence of this activity is that the liver is particularly likely to the target organ of many toxicants.

Some generalization can be made concerning the overall fate of toxicants. They are absorbed in the lipid-soluble form and are excreted as water-soluble metabolites. Thus metabolism of toxicants involves enzymatic reactions to convert fat-soluble substances to water-soluble compounds. These metabolic processes, known as biotransformation, may either increase or decrease the toxicity of the ingested toxicant.

Reactions in the Gastrointestinal Tract
Most toxicants do not produce their toxic effects in the gut, with the exception of irritants such as saponins, selenium-containing amino acids, and oxalates, and toxicants which affect digestive processes, such as trypsin inhibitors and lectins.

The rate of absorption of toxicants is largely determined by their lipid solubility, which is commonly expressed as the lipid/water partition coefficient. Nonionic compounds are more readily absorbed than ionized substances, as they are more lipophilic. Thus, organic acids are more likely to be absorbed in an
acid environment (the stomach) while organic bases are absorbed in a basic environment (small intestine). The extent of dissociation of a compound will determine the proportion that is ionized at a given pH. Most toxicants are absorbed by simple diffusion.

In nonruminants, there may be metabolism of toxicants by microorganisms in the hindgut. Because of the anatomical location of these processes in the posterior region of the gut, metabolism by cecal organisms generally has limited effects in livestock.

The degree of absorption of toxicants can be influenced by several other factors. Anion exchange resins, bentonite and zeolite (clays with ion exchange capacity), and alfalfa meal in the diet reduce the absorption of mycotoxins such as zearalenone. Factors affecting gastrointestinal motility affect absorption can occur, and greater absorption.

**Ruminal Metabolism of Toxicants**
The rumen is a specialized area that may have pronounced effects on ingested toxicants in ruminant animals. It generally has a slightly acid pH and has an immense population of diverse types of microorganisms. Ingested material remains in the rumen for a much longer period than is the case for ingesta in the gut of nonruminants. It is an area where small water-soluble molecules such as the volatile fatty acids are absorbed.

Ruminal metabolism by microorganisms has diverse effects on toxicants. Some of the potential interactions of toxicants and ruminal metabolism are:

1. Toxicity may be increased as a result of ruminal metabolism. For example, nitrate is converted to the more toxic nitrite in the rumen. Cyanogenic glycosides are hydrolyzed more rapidly in ruminants than in nonruminants, and thus are more toxic because the enzymatic hydrolysis of the glycosides is favoured by the higher pH of the rumen than the highly acid nonruminants stomach. The toxic amino acid mimosine in Leucaena leucocephala is converted by rumen metabolism to dihydorxypyridone, a more toxic metabolite.

2. Detoxification can occur in the rumen. For example, Oxalates can be degraded by rumen microorganisms. Gossypol, a phenolic substance in cottonseed meal, is detoxified in the rumen, as are trypsin inhibitors in soybeans and glucosinolates in rapeseed meal.

3. Rumen microorganisms may produce toxins.

4. Dietary toxicants may inhibit rumen fermentation.

**Why study is needed:** Coniine, the toxic alkaloid in hemlock, is acutely poisonous to humans but quails are able not only to adapt to and accommodate coniine in whole seed, but are also able to accumulate the toxin in body tissue at levels that are toxic to mammals. Thus it is clear that any extrapolation to avian species of knowledge of the effects of secondary plant metabolites gained from mammals must be undertaken with caution. The tropical legume *Leucaena leucocephala*, is known as ipil ipil. Use of ipil ipil meal in poultry rations had no obvious ill effects. Yet the non-nutrient amino acid, mimosine that is present in *Leucaena* is held responsible for poor growth of cattle consuming high levels of *Leucaena* herbage and whose rumen microflora are not adapted to mimosine detoxification. However, when the animals were inoculated with rumen microflora from Hawaiian cattle the adverse effects were eliminated. Because of its effect on hair follicles, mimosine has even been used in ‘chemical shearing’ experiments.

**The nutritional effects of secondary plant metabolites** are achieved by a wide variety of mechanisms, some bind specifically to and inhibit digestive enzymes, some bind to proteins generally, some chelate trace metal nutrients, others disrupt membrane structures, whilst the phyto-oestrogens achieve their effects by mimicking the structure of a natural hormone.
There is a long list of these materials, the eradication, detoxification and making wholesome feeds have been suggested. Adulteration of foreign materials, low quality and low cost feeds with prime feed ingredients has also been discussed.

**Antinutritional Factors in Feed and Fodder used for Livestock and Poultry Feeding**
Toxic substances of natural origin can be classified based on their chemical properties and on the basis of their effect on utilization of nutrients.
According to their chemical properties

**Group I: Proteins**
1. Protease inhibitor
2. Haemagglutinins (Lectins)

**Group II: Glycosides**
1. Saponins
2. Cyanogens
3. Glucosinolates (Goitrogens) Or thioglucosides

**Group III: Phenols**
1. Gossypol
2. Tannins

Table 1: Anti-Nutritional factor in forage crops.

<table>
<thead>
<tr>
<th>S. No</th>
<th>Antinutritional/ Toxic substances</th>
<th>Fodder crops</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Nitrate</td>
<td>Sudan Grass, Pearl millet, Oats</td>
</tr>
<tr>
<td>2</td>
<td>Oxalates</td>
<td>Paddy straw, Guinea Grass, Bajra and Hybrid Napier, Setaria Grass, Kikyu &amp; Buffel grass</td>
</tr>
<tr>
<td>3</td>
<td>Saponins</td>
<td>Lucerne</td>
</tr>
<tr>
<td>4</td>
<td>Tannins</td>
<td>Fodder tree/Shrubs</td>
</tr>
<tr>
<td>5</td>
<td>Cynogens</td>
<td>Sorghum, Sudan grass, Jhonson grass</td>
</tr>
<tr>
<td>6</td>
<td>Glucosinolates (Goitrogens)</td>
<td>Cabbage, Turnips, Rapeseed and Mustard green</td>
</tr>
<tr>
<td>7</td>
<td>Mimosine</td>
<td>Subabul</td>
</tr>
<tr>
<td>8</td>
<td>β-N-oxalyl-L-α, β-diamino propionic acid (β-ODAP or BOAA)</td>
<td>Lathyrus</td>
</tr>
</tbody>
</table>

**Group IV: Miscellaneous**
1. Anti-metals
2. Anti- vitamins

**On the basis of nutrients that are affected directly or indirectly**

**Substances depressing digestion or metabolic utilization of proteins:**
1. Protease inhibitor (Trypsin and Chymotrypsin inhibitor)
2. Haemagglutinins (Lectins)
3. Saponins
4. Polyphenolic components

**Substances reducing solubility or interfering with the utilization of Minerals:**
1. Phytic acid
2. Glucosinolates (Thioglucosides)
3. Oxalic acid
4. Gossypol

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**Substances increasing the requirements of certain vitamins:**

1. Anti-vitamin A, D, E, K.
2. Anti-vitamin B1, B6, B12 and Nicotinic acid.

**Group – I: Proteins**

**Protease inhibitors:**

- Substances that have the ability to inhibit the proteolytic activity of certain digestive enzymes eg. Legume seeds such as Soyabean, Kidney bean and Mung bean.
- Protease inhibitors are concentrated in the outer part of the cotyledon mass.
- Two types of Protease enzymes inhibitors:
  1. Kunitz inhibitor (inhibits only Trypsin)
  2. Bowman–birk inhibitor (inhibits Trypsin and Chymotrypsin)

- The inhibitory substances are mostly heat labile and thus proper heat treatment inactivates the protease inhibitors.
- Trypsin inhibitor of Soyabean interferes with the availability of Methionine from the raw Soyabean.
- Young chicken fed raw Soyabean developed hypertrophy of the pancreas and this is not observed in large animals such as Pigs, Dog and Calves.
- The factors controlling Trypsin inhibitor destruction are
  1. Temperature
  2. Duration of heating
  3. Particle size
  4. Moisture level
- The trypsin inhibitor activity of solvent extracted Soyabean Meal was destroyed by exposure to steam for 60 min, or by autoclaving under the following conditions i.e. 5 psi for 45 min, 10 psi for 30 min or 15 psi for 20 minutes.

**Haemagglutinins (Lectins)**

- These compounds occur in many seeds and plants. They are protein in nature and have remarkable property of agglutinating red blood cells. These toxic substances combine with the glycoprotein components of RBC causing agglutination of the cells.
- Soybean, castor bean (ricin) and other legume seeds contain haemagglutinins. Ricin is toxic and causes severe inflammatory changes in the intestine, kidney, thyroid gland etc. Lectins are resistant to digestion by pancreatic juice.
- Ingestion of such toxicant cause inflammation of epithelial lining, destruction of epithelial cells as well as edema, hypermea and hemorrhages in the lymphatic tissues. Capillaries are seen to be extended and filled with blood clots. The liver undergoes fatty degeneration and necrosis.
- The Haemagglutinins can be inactivated by moist cooking with 2% sodium hydroxide. Simple heat-treatment (one hour steam heating or autoclaving at 15 pounds/sq. inch pressure for 20 min.) is also effective. The improvement in the feeding value of leguminous seeds which is observed upon their heating may be ascribed partly to the inactivation of Haemagglutinins present in them.

- Lectins are resistant to dry heat but are destroyed by moist heat (steam).
Group – II: Glycosides

Saponins
- These are glycosides characterized by bitter taste, foaming in aqueous solution and haemolyse RBC.
- Their toxicity is related to their activity in lowering surface tension in ruminants.
- The important forages which causes saponin poisoning of livestock are Lucerne, soybean etc.
- The saponin content of the leaves is twice as much as that of the stems and will decline as the plant becomes older.
- Poultry are more susceptible than Pigs. 0.4- 0.5% saponin in the feed decreases feed consumption, egg production and body weight loss in birds.
- Saponins are degraded by rumen microbes and hence, no growth depression takes place. However, upon excess feeding of green Lucerne, saponins lower the surface tension of ruminal content leading to accumulation of gas in the digesta. This condition is known as bloat or tympany or tympanitis.

Saponins are glycosides containing a polycyclic aglycone moiety of either C27 steroid or C30 triterpenoid attached to a carbohydrate. The structural complexity of saponins results in a number of chemical, physical, and biological properties, which include foaming, emulsifying, sweetness, bitterness, pharmacological and medicinal, haemolytic properties, as well as insecticidal and anti-microbial activities. Saponins reduce the uptake of certain nutrients including monosaccharide and cholesterol at the gut through intra-lumenal physico-chemical interaction. Hence, it has been reported to have hypocholesterolemic effects [4]. Sharma, et al. [5] Observed that 4-7 weeks of ad lib. feeding of Albizia stipulate (Siris) gave rise to toxic manifestation of sheep. A resinous shrub, broom breed (Gutierrezia sarothrae), the toxicity is due to its saponin content. Symptoms include anorexia, listlessness, gastroenteritis and weight loss. In non-ruminants (chicks and pigs), retardation of growth rate, due primarily to reduction in feed intake, is probably major concern [6]. Saponins are among several plant compounds which have beneficial effects. as antibacterial and anti-protozoal properties.

Precaution
Repeated washing with water reduces saponin content and makes the feed more palatable reducing the bitterness associated with saponins [7]. Incorporation of other legumes and roughes in ration along with siris leaf (which are toxic to animal) reduces the saponin toxicity.

Cyanogens
- Cyanide in trace amounts is present in the plant kingdom.
- It occurs mainly in the form of cyanogenic glycoside.
- These glucosides are hydrolyzed to Prussic acid or hydrocyanic acid by enzyme present in the same plant or while being digested by animals.
- There are three distinct glucosides
  - Amygdalin found in Almonds.
  - Dhurrin found in Jowar and other immature grasses.
  - Linamarin found in Pulses, Linseed and Cassava.
- Ruminants are more susceptible to HCN poisoning than horses and pigs since the enzyme required for the release of HCN is destroyed in horses and pigs by gastric HCl.
- It usually causes reduced growth, poor feed efficiency and result in death if consumed in increased amounts. Cattle and buffaloes are more susceptible than sheep.
- Feeding of immature jowar green fodder should be avoided to prevent HCN poisoning.
Table 2: Prussic acid (HCN) Concentration in forages

<table>
<thead>
<tr>
<th>HCN Concentration in (ppm)</th>
<th>Potential Effect on Livestock</th>
<th>Remarks</th>
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<tbody>
<tr>
<td>Dry Matter</td>
<td>Fresh Harvested</td>
<td></td>
</tr>
<tr>
<td>0-500</td>
<td>0-100</td>
<td>Forage is generally safe and should not cause toxicity.</td>
</tr>
<tr>
<td>500-1000</td>
<td>100-200</td>
<td>Potentially toxic and forage should be fed at restricted rate in the diet.</td>
</tr>
<tr>
<td>&gt;1000</td>
<td>&gt;200</td>
<td>Very dangerous to livestock and will usually cause death.</td>
</tr>
</tbody>
</table>

Cyanogens are glycosides of a sugar or sugars and cyanide containing aglycone. Enzymes found in the cytosol which can hydrolye cyanogens and release HCN which is toxic. Damage and toxicity to the plant occurs when the enzymes and glycoside form hydrocyanic acid (HCN). In the rumen hydrolytic reaction can take place by microbes. Hence, ruminants are more susceptible to CN toxicity than non- ruminants. In the liver enzyme Rhodanese rapidly detoxified absorbed HCN and converts CN to thiocyanate (SCN). Excess cyanide ion inhibits the Cytochrome oxidase which stops ATP formation, and further tissues suffer energy deprivation and death follows rapidly. In Cattle and Sheep, the lethal dose of HCN is 2.0-4.0 mg per kg body weight, respectively.

The lethal dose for cyanogens would be 10-20 times greater because the HCN comprises 5-10 per cent of their molecular weight of the substance. Forage containing HCN consumed within a few minutes and simultaneous HCN production would have to be rapid for toxic effect in the body. Young seedlings contain more HCN level than in matured seedlings. Normally in the first cut the forage prussic acid percentage is more than the second cut, probably due to degradation of the acid and a higher metabolic activity of the plant due to higher temperatures during growth processes which can reduce the prussic acid accumulation, these low amounts of Forage Prussic Acid Percentage are not toxic to animals.

**Precaution**

Forage grown on energy stress condition and crop not get proper irrigation, the levels of HCN is found higher in younger sorghum crop. Thus try to avoid these type of crop for feeding livestock. Post-harvest wilting and drying of Cynogenic leaves may decrease the effect of cyanide poisoning. Sorghum, Sudan and Johnson grass must be dried at least six hour before its use for feeding to livestock. More than 200 ppm in fresh green fodder and more than 1000 ppm in dry fodder HCN concentration is toxic. Before feeding, proper drying, ensiling and maturity of fodder reduces the HCN concentration.

**Glucosinolates:**

- Most plants of Crucifera family (Cabbage, Turnips, Rapeseed and Mustard green) contain these substance.
- These are responsible for the pungent flavours present in plants belonging to the genus Brassica.
- Ruminants appear to be less susceptible compared to pigs and poultry.
- Their main biological effect is to depress the synthesis of the thyroid hormone (Thyroxine, T4) and Triiodothyronine (T3) producing Goiter.
- Glucosinolates occur in the root, stem, leaf and seed and are accompanied by the enzyme Myrosinase (Thioglucosidase), which is responsible for their hydrolysis.

**Group – III: Phenols**

**Gossypol**

- Gossypol is present in pigment glands of leaves, stem, roots and cotton seed and cake.
• It is highly toxic to simple stomach animals.
• Pigs and rabbits are more sensitive than poultry.
• Horses are resistant.
• Ruminants are more resistant due to the formation of stable complexes with soluble protein in rumen, which is resistant to enzymatic breakdown.
• Gossypol form complex with metals like iron and the toxic effect can be overcome by supplementing iron as ferrous sulphate.
• Gossypol can occur either in free form or as a gossypol protein complex.
• New varieties of cottonseed with less than 0.01% total gossypol (0.002% in free form) are available.
• Physiological effects of Gossypol includes:- Reduced appetite
  1. Loss of body weight
  2. Accumulation of fluid in the body cavities
  3. Cardiac irregularity
  4. Reduced O2 carrying capacity of the blood (reduced HB content)
  5. Adverse affect on certain liver enzymes
• In poultry, it causes:
  • Decreased growth in chicks.
  • Decreased egg production
  • Decreased hatchability
  • Yolk will turn olive green color

Tannins
• It is a Polyphenolic substance with molecular weight greater than 500.
• The term tannin was coined by sequin in 1796.
• Two types of Tannins:-
  Hydrolysable tannins
  Condensed tannins

<table>
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<th>S. No.</th>
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<td>Feed/Fodder</td>
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<td>2</td>
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Hydrolysable tannins
These can be readily hydrolysed by water, acids, bases or enzymes and yield gallotannins and ellagitannins.

Condensed tannins
These are Flavonoids (Polymers of flavonol.) Both hydrolysable and condensed tannins are widely distributed in nature. Tannin content of certain feedstuffs are as follows:
  1. Tannins are astringent in nature.
  2. They cause a dry or puckery sensation in the mouth, probably by reducing the lubricant action of the glycoproteins in the saliva.
  3. They bind the proteins and are thus inhibitors of proteolytic enzymes.
  4. High tannin content also depresses cellulose activity and thus affects digestion of crude fibre.
  5. Tannins reduce the digestibility of dry matter, protein and other nutrients.
6. Sorghum contains high levels of condensed tannins.
7. Most of the tannins are located in the seed coat. Hence, decortications of seeds will reduce the tannin content.
8. Germination of legume also results in a decrease in the tannin content.

Methods of Demagnification: The methods available for removal or inactivation of tannins can be divided into two main categories:

- Physical Treatment: Soaking and cooking decrease the tannin content. However, these treatment cause a substantial loss of DM between 20 to 70%.

  Anaerobic storage of moist sorghum grains for two and nine days resulted in 40 and 92% reduction in tannins, respectively.

- Chemical Treatments: Addition of tannin complexing agents like polyethylene glycol (PEG) and polyvinyl pyroldone (PVP) prevent formation of complexes between tannin and protein as well as break the already formed complex thus liberating protein. Alkalies, formaldehyde, organic solvents like acetone, acids H2O2 reduce the tannin content.

Precaution

More than 4% concentration of tannin has been reported to be toxic for ruminants as they are more resistant to microbial attack and are harmful to a variety of microorganisms. Toxic level of tannin can be reduced by physical methods like soaking, heat treatment and drying before feeding of forage. Several studies reported that feeding tannin-rich leaves with concentrate rations reduces the adverse effect. This is due to spairying effect of consume protein in excess of their requirement from the concentrate and therefore, the anti-nutritional effects of tannins were masked.

**Group – IV**

**Antimetals:** Substances depressing the utilization of minerals.

**Phytic acid**
- Is an ester formed by combination of the 6 alcoholic groups of inositol with 6 molecules of hexa phosphoric acid. Hence, its name Inosital hexaphosphoric acid.
- Seeds of cereals, dried legumes, oilseeds and nuts are rich in phytic acid.
- Phytic acid concentration is more in the rind (Pericap + aleurone layer) and the embryo than the core (endosperm).
- Phytic acid depresses the utilization of several minerals elements such as Ca, Mg, Fe, Zn etc. by forming insoluble compounds which are eliminated in the faces.

**Oxalic acid**
- Plant foodstuffs have much oxalic acid while those of animal origin have relatively little oxalic acid.
- The leaves are rich in oxalates compared to other parts.
- Young leaves contain less amounts than mature leaves.
- Ageing as well as over ripening of vegetables is accompanied by an increase in the proportion of calcium oxalate.
- Pigs and poultry are affected.
- Animal response to oxalate poisoning varies with species of animal and species of plant
- Oxalate poisoning in cattle and sheep are characterized by rapid and labored respiration, depression, weakness, coma and death.
Oxalate is an anti-nutritional content, when it is digested; it comes into contact with the nutrients in the gastrointestinal tract. After released, oxalic acid binds with nutrients, rendering them inaccessible to the body. More quantity of oxalic acid is consumed regularly, nutritional deficiencies are likely to occur, as well as severe irritation to the gastrointestinal tract mucosal lining. Strong chemical and chelated bonds are formed between oxalic acid, and various other minerals, such as Calcium, Magnesium, Sodium, and Potassium. This chemical combination results in the formation of oxalate salts. Oxalates react with Calcium to produce insoluble Calcium Oxalate complex reducing calcium absorption in the gut. This leads to a disturbance in the absorbed Calcium: Phosphorus ratio, resulting in mobilization of bone mineral to alleviate the hypocalcaemia and calcium drain out from the body and excreted through urine and faeces. Continuous mobilization of bone minerals results in nutritional secondary hyperparathyroidism or osteodystrophy fibrosa. Young plants contain more oxalate than older plants. During early stages of growth, there is a rapid rise in oxalate content followed by a decline in oxalate levels as the plant matures [11]. Several researchers reported that oxalate content is highest in leaf tissue, followed by stem tissue [12].

Precaution

Rumen micro-organisms degrade dietary into formic acid and CO2. Adaptability reduces the toxicity of oxalate in the body. Ruminants adapted to diets with high oxalate content can tolerate oxalate levels that are lethal to non-adapted animals. Moreover, it has been shown that the transfer of rumen fluid from animals in Hawaii to Australian ruminants resulted in complete elimination of the toxic effects of mimosine and the bacteria involved in such effects have been identified.

Anti–vitamins

These are organic compounds, which either destroy certain vitamins or combine and from unabsorbable complexes or interfere with digestive and/or metabolic functions.

• **Anti–vitamin A**: Raw Soyabeen contains enzymes lipoxygenase, which can be destroyed by heating 5 minute steam at atmospheric pressure. Lipoxygenase catalyses oxidation of carotene, the precursor of vitamin A.
• **Anti–vitamin D**: Rachitogenic activity of isolated soya protein (unheated) has been found with chicks and pigs. Autoclaving eliminates this activity.
• **Anti–vitamin E**: Present in Kidney bean. Diets with raw kidney beans produced muscular dystrophy in chicks and lambs by reducing plasma vitamin E. Autoclaving destroys this factor.
• **Anti–vitamin K**: Eating Sweet clover cause fatal haemorrhagic condition in cattle known as Sweet clover disease. Dicoumarol present in sweet clover is responsible for this. Dicoumarol reduce prothrombin levels in blood and affects blood clotting.
• **Anti–Pyridoxine**: An antagonist of pyridoxine from linseed has been identified as 1 – amino –D-proline. It occurs naturally in combination with glutamic acid as a peptide and it is called linatine. Nutritive value of linseed meal for chicks can be considerably improved after water treatment and autoclaving.
• **Anti–Niacin**: An antagonist of Niacin, niacytin is found in Maize, Wheat bran etc. that causes Perosis and growth depression.

Other toxic factors

**Nitrate**

Nitrate is the form of nitrogen in the atmosphere take up by plant roots from the soil, and is transported to the leaves. In stress condition excess nitrates accumulated in the plants. Drought or hot dry winds causes water stress leads to nitrate accumulation. Damage caused by hail or frost impairs photosynthesis resulting in excess nitrates accumulation and causes toxicity in livestock.
Cool cloudy weather can also cause the nitrates accumulation problem. During initial growth, much of the nitrate taken up by the plant is used for root and shoot development. At this stage, the roots are able to take up more nitrate than is required and it accumulates in the stems and leaves of the plant. As the plant develops, the leaves of the plant are able to convert more nitrate into plant protein, therefore less "surplus" nitrate is found in the plant as it matures. Fodder crops such as Sudan grass, pearl millet and oats can accumulate Nitrate at potentially toxic levels. Most of the nitrate accumulates in stem, followed by leaves and very little in the grains [13]. In ruminant nitrate is normally converted to nitrate - nitrite - ammonia - amino acid to protein in the rumen in presence of microorganisms. When forages have an unusually high concentration of nitrate, the animal cannot complete the conversion and nitrite accumulates. Through the rumen wall, Nitrite is absorbed and transported to the blood stream directly and converts haemoglobin (the O2 carrying molecule) in the blood to met haemoglobin, which cannot carry oxygen. The blood turns to a coffee colour rather than the usual bright red. An animal dying from nitrate (nitrite) poisoning actually dies from lack of oxygen (asphyxiation). The rate and quantity of fodder consumption, type of forage, energy level or adequacy of the diet are the factors affecting the severity of nitrate poisoning.

Precaution

Annual forages are more susceptible than perennial forage for Nitrates accumulation and toxicity. Adverse climate eg. period of drought or wet, dull weather condition are more prone to Nitrate toxicity. Following steps can reduce the risk of nitrate toxicity:

- Dilute the nitrate content of the total ration by feeding a combination of low and high nitrate feeds. Animal should be fed the ration, three or four times daily rather than just one meal per day. Allow cattle to sensitize with nitrate slowly to increase the nitrate content of the ration. Ensure balanced ration feeding to livestock for the level of production that is expected. Balance concentrate diet should be given along with feed contain nitrate to cattle to reduce toxicity.

Table 3: Level of Nitrate in forage (DM Basis) and potential effects on animals

<table>
<thead>
<tr>
<th>S.No.</th>
<th>Nitrate Content (ppm)</th>
<th>Effect on Animals</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0-1000</td>
<td>This level is considered safe to feed under all conditions.</td>
</tr>
<tr>
<td>2</td>
<td>1000-1500</td>
<td>This level should be safe to feed to non-pregnant animals under all conditions. It may be best to limit its use to pregnant animals to 50% of the total ration on a dry basis.</td>
</tr>
<tr>
<td>3</td>
<td>1500-2000</td>
<td>Feeds are fed safely if limited to 50 per cent of ration’s total dry matter.</td>
</tr>
<tr>
<td>4</td>
<td>2000-3500</td>
<td>Feeds should be limited to 35-40 per cent of total dry matter in the ration. Feeds containing over 2000 ppm nitrate nitrogen should not be used for pregnant animals</td>
</tr>
<tr>
<td>5</td>
<td>3500-4000</td>
<td>Feeds should be limited to 25 per cent of total dry matter in ration. Do not use for pregnant animals.</td>
</tr>
<tr>
<td>6</td>
<td>&gt;4000</td>
<td>Feeds containing over 4000 ppm are potentially toxic. Do not feed</td>
</tr>
</tbody>
</table>

B. β-N-oxalyl-L-α, β-diaminopropionic acid (β-ODAP or BOAA)

Potent neuro-toxic activity is due to excess feeding of Lathyrus containing β-N-oxalyl-L-α, β-diaminopropionic acid, a naturally occurring amino acid, possesses and has been shown to be responsible for outbreaks of neurolathyrism. β-ODAP occurs naturally as two isomeric forms with the α-form being approximately 5% of the total and β-isomer is major concern of toxicity. Genetic factors and environmental conditions effects the level of β-ODAP in dry seed. Zinc-deficiency or rich in ferrous iron in Lathirus sativus
grown in nutrient solutions have elevated levels of β-ODAP. β-ODAP is biosynthesized during the ripening of the seed and is further increased during germination. The ingestion of ODAP causes neuro-lathyrism, a neuro-degenerative disease that damages upper motor neurons, causing irreversible paralysis of the lower limbs and sometimes death in humans and animals [15]. In Ethiopia, other studies reported ODAP content in seeds varying from 5.4 to 8.9 g/kg DM or 2.0 to 4.5 g/kg DM [16]. The green parts and the straw contain lower concentrations of ODAP: 1.9 to 3.4 and 1.3 to 2.1 g/kg DM respectively.

**Precaution**

Soft and tender part of plant and leaves in excess quantity should be avoided for feeding animals. Toxicity can be reduced by water soaking or hot water soaking for few hours. Concentrates along with dry wheat or paddy straw should be feed along with Lathyrus to the big animals to reduce toxicity.

**Mimosine**

Mimosine, a non-protein amino acid structurally similarity with tyrosine, present in genus *Leucaena leucocephala* in which the level of mimosine in the leaf is about 2-6% and varies depending on season and maturity of leaf and stem. In non-ruminants mimosine toxicity cause alopecia, eye cataracts, poor growth and reproductive problems. More than 5-10% of Leucaena meal of the diet for poultry, rabbit and swine generally result in poor growth, reproduction and performance The main clinical symptoms of toxicity in ruminants includes poor body growth, alopecia, dullness, poor wool development, swollen and raw coronets above the hooves, lameness, mouth and oesophageal lesions, depressed serum thyroxine level and goiter. Symptoms may be due to metabolite of mimosine in the rumen and other’s to 3,4 dihydroxypyridine. Decrease in calving percentage due to Leucaena feeding has also been noted by Jones RM., *et al* [16].

**Precaution**

Mimosine problem could be solved by genetic selection of strain of Leucaena species containing low mimosine contents. But it is found that, low mimosine types of fodder are found to be unproductive and low vigour. This problem can be solved by feeding leucaena mixed with other feed fodder and concentrates. Hiremat, N.B. [18], suggested that use of Leucaena fodder may be restricted to 30% of green forage in the case of cattle and buffalo, and 50% for goats show better results in terms of production and growth. Physical treatment like heat treatment and chemical treatment and supplementation with amino acids or with metal ions such as, and Fe, Al and Zn reduces the mimosine toxicity.

**Methods of reduce the deleterious effect of ANF’s**

A number of methods have been tried to overcome the deleterious effect of different anti-nutritional factor includes through making hay, silage with inoculants, using PEG; Urea or biological treatment with fungi can be applied to either take off or minimized and decrease anti-nutritional factors concentration. It is well know that alkali treatment includes polyethylene glycol (PEG), which a tannins-binding agent, was shown to be a powerful tool for isolating the effect of tannins on various digestive function. Economical point of view it is not recommended. Although the adding of polyethylene glycol (PEG), which binds with and inactivates tannins and other ANF is quite effective, success of its adoption depends on the cost: benefit ratio. Russsell and Lolle suggest feed animals with 1% urea which not only provides extra N but also deactivates the leaf tannins.

**Toxins and adulteration**

The cereals and their by-products usually form about three quarter of the poultry ration. The proceeding paragraphs have a discussion on adulteration of common cereal grains and protein sources.
Maize: Two types of maize as “White maize” and “Yellow maize” is used. Yellow maize is adulterated by white maize/corn, soil or dust, damaged corn and weed seeds. There is more danger of “Aflatoxin” in maize as compared to other grains.

Wheat: Wheat is adulterated by weed seeds, soil, damaged wheat and can be affected by aflatoxin. Newly harvested wheat has gluten, that form indigestible dough inside both crop and gut.

Rice: Rice is adulterated by rice polishing, rice husk/bran, damaged rice and there is a problem of rancidity and aflatoxin.

Sorghums: White Milo contains little of bitter principal (Tannin) formed in the darker grains. There is a problem of prussic acid. Sorghum grains are adulterated by soil, weed seeds and damaged sorghums.

Rice Polishings: The main problems are rancidity and adulteration of rice bran/husk.

PLANT / VEGETABLE PROTEIN SOURCES: The main vegetable protein feeds are the residues remaining after the removal of most of the oil from certain oily feeds and fruits. The various vegetable protein sources used in poultry ration are as under:

1. Cottonseed meal: Cotton seed meal is prepared from the dehulled seeds of the cotton plant. The fresh kernels contain two toxic principles, gossypol and gossypurpurin. These are destroyed by steam cooking the seeds before the oil is removed. When unheated meal is fed in high amounts the yolk colour is adversely affected (olive yolks). It affects (reduces) the keeping quality of the eggs.

2. Rapeseed meal: Rapeseed contains glucosinolates. During processing care must be taken to destroy the enzymes myrosinase which converts the progoitrin to a goitrin.

3. Soybean Meal: Raw soybean contains soin- a toxic protein which acts as a depressant to the appetite and a trypic inhibitor. Both of which can be destroyed by heat.

4. Sunflower Meal: The hard pericarp of the sunflower seed accounts for about 40% of the weight, hence decorticated sunflower meal is much better than undecorticated meal.

5. Sesame Meal: The protein is a good source of arginine and leucine but is low in methionione and lysine. Aflatoxin is common sesame meal.

6. Linseed Meal: This is prepared by the removal of most of the oil from the seed of the flax plant. The oilseed meal containing a toxic principle, cyanogenetic glucosides, which yields prussic acid and which is destroyed by boiling.

7. Guar Meal: It is a source of guar gums utilized in so many industries. Most of the gums remain in the meal and untoasted guar is dangerous for poultry due to the presence of HCN and gums.

8. Maize Gluten Meal: These are residues from maize starch manufacture. Aflatoxin is very common in this feed ingredient.

ANIMAL PROTEIN SOURCES

1. Blood Meal: This is a product obtained when whole blood is cooked, dried and ground to meal. There is much adulteration due to its high price. The common adulterated materials are meat meal, ingesta of animals, coal, ash, burnt protein materials and urea.

2. Meat Meal: Meat unfit for human consumption whether because of disease or parasitic infestation cooked, dried and ground is called meat. The meat meal is not sterilized properly. There is danger of E. coli, salmonella. The salt is added to dry the meat that can cause salt poisoning when fed to birds. There is adulteration of soil, ingesta, residue of tannery, hair, hooves and horns.

3. Fish Meal: Fish meal is produced by cooking, drying and grinding fish. The raw fish contains antithiamine factors. There is much adulteration of hooves, horns, unwanted see animals, salt, urea, meat meal and feather meal.
4. **Feather Meal**: The production of feather meal by a steam processing method is now in operation. There is a problem of rancidity and adulteration of meat meal and burnt protein sources.

<table>
<thead>
<tr>
<th>Feedstuff</th>
<th>Toxicant</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Grains</strong></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>Phytates, mycotoxins</td>
</tr>
<tr>
<td>Rye, triticale</td>
<td>Trypsin inhibitors, ergot</td>
</tr>
<tr>
<td>Milo</td>
<td>Tannins</td>
</tr>
<tr>
<td>Grain amaranth</td>
<td>Oxalates, Saponnins</td>
</tr>
<tr>
<td><strong>Tubers</strong></td>
<td></td>
</tr>
<tr>
<td>Potatoes</td>
<td>Solanum alkaloids</td>
</tr>
<tr>
<td>Cassava</td>
<td>cyanogenic glycosides</td>
</tr>
<tr>
<td><strong>Proteins Supplements</strong></td>
<td></td>
</tr>
<tr>
<td>Soybeans</td>
<td>Trypsin inhibitors, lectins, goitrogens, saponins, phytates, mycotoxins</td>
</tr>
<tr>
<td>Cottonseed</td>
<td>Gossypol, tannins, mycotoxins, cyclopropenoid fatty acids</td>
</tr>
<tr>
<td>Rapeseed</td>
<td>Glucosinolates, tannins, eruc acids, mycotoxins</td>
</tr>
<tr>
<td>Linseed meal</td>
<td>Linatine, linamarin</td>
</tr>
<tr>
<td>Fava beans</td>
<td>Trypsin inhibitors, vicine, lectins</td>
</tr>
<tr>
<td>Field beans</td>
<td>Trypsin inhibitors, lectins</td>
</tr>
<tr>
<td>Lupin</td>
<td>Alkaloids</td>
</tr>
<tr>
<td><strong>Forages</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Legumes</strong></td>
<td></td>
</tr>
<tr>
<td>Alfalfa</td>
<td>Saponins, phytoestrogens, bloating agents</td>
</tr>
<tr>
<td>White Clover</td>
<td>Cyanogenes, phytoestrogens, bloating agents</td>
</tr>
<tr>
<td>Red clover</td>
<td>slaframine phytoestrogens, bloating agents</td>
</tr>
<tr>
<td>Alsike clover</td>
<td>Photosensitizing agents</td>
</tr>
<tr>
<td>Sweet clover</td>
<td>Coumarin</td>
</tr>
<tr>
<td>Crown vetch</td>
<td>B-nitroproponal glycosides</td>
</tr>
<tr>
<td>Leuceana spp.</td>
<td>Indospecine</td>
</tr>
<tr>
<td><strong>Grasses</strong></td>
<td></td>
</tr>
<tr>
<td>Forages sorghums</td>
<td>Cyanogens</td>
</tr>
<tr>
<td>Tall fescue, Rye grass</td>
<td>alkaloids</td>
</tr>
<tr>
<td>Tropical grasses</td>
<td>Oxalates</td>
</tr>
<tr>
<td><strong>Others</strong></td>
<td></td>
</tr>
<tr>
<td>Forage brassicas</td>
<td>Brassica anemia factor</td>
</tr>
</tbody>
</table>

**Aflatoxin in Feeds**

Most people are familiar with mold in one form or another. Mold is the wooly-like growth that develops frequently on the surface of damp organic matter held under aerobic conditions at temperatures between 45°F and 100°F. There are many different types and strains. Molds produce metabolites, many of which are toxic to other organisms. Such toxic metabolites are referred to as mycotoxins. Certain mycotoxins are harmful to large animals, including livestock. Such mycotoxins sometimes are present in feed that has been held under mold-producing conditions.

Of the many different mycotoxins that occur sometimes in feeds, the aflatoxin group seems to pose the greatest threat to animal health and efficient livestock production. The aflatoxin group embraces several different types. Aflatoxin B1,B2,G1, and G2 are the most common. Of the different aflatoxins, aflatoxin B1 usually is the most prevalent and the most toxic. The other types rank as follows, in descending order of toxicity: G1, B2, G2. Aflatoxin is produced primarily by the mold Aspergillus flavus, although other
Aspergillus molds sometimes are present and aflatoxin-like mycotoxins are produced by other molds. The aflatoxin-producing mold Aspergillus flavus is most apt to occur at harmful levels in grains and seeds that have been stored too wet for an extended period but may, under certain conditions, develop prior to harvest. Corn has received the greatest attention as a possible aflatoxin source, but barley, grain sorghum wheat, cottonseed, peanuts, and soybeans as well as processed feeds produced from these grains and seeds have sometimes shown a high level of aflatoxin. Grains and seeds that have had insect damage in the field especially are susceptible to preharvest mold infestation. Mechanical damage to grains and seeds at the time of harvest also is conducive to mold development. A proper state of dryness probably offers the surest protection against mold damage to grains, seeds, and other feeds in storage. Moisture levels of more than 13% in grains and seeds are generally favorable for mold growth in storage, although this varies depending on factors such as the following:

- Type of grain or seed
- Temperature
- Humidity
- Amount of aeration
- Time in storage

To avoid mold formation in processed feeds, the moisture level should not exceed about 11%, depending on the circumstances. Leaky bins and bins in which sweating occurs will tend to produce pockets of grain, seed, or feed that contain mold. The treatment of high-moisture feed grains with propionic acid or some similar preservative sometimes is used to keep down mold formation. Aflatoxin-producing molds are most active at 75°F to 90°F. Cold weather, of course, tends to check mold growth.

Should grains, seeds, or other products that are to be fed to livestock or used in preparing livestock feeds show any evidence of mold, they should be checked for aflatoxin before being used and then be used in a manner that is commensurate with their aflatoxin content. Grains and seeds that show mold damage may be given a preliminary check for the presence of aflatoxin by exposing such products to black light and observing them for a bright greenish yellow (BGY) fluorescence, which would indicate aflatoxin contamination. However, basically this is a test for the presence of aflatoxin and does not provide a measure of the amount present. It is effective with whole grains only. Aflatoxin determinations ordinarily are made by either thin layer or column chromatography. The different aflatoxin types normally are quantitated separately and then totaled together. Aflatoxin levels normally are reported in parts per billion (PPB), which is the same as micrograms per kilogram (mcg/kg).

Aflatoxin is thought to have its harmful effects on livestock in several possible ways.

1. It may disrupt the normal fermentation processes of the rumen, causing inefficient digestion.
2. A disruption of the rumen fermentation might cause a breakdown in the normal biosynthesis of critical amino acids and vitamins, thereby resulting in a deficiency of certain nutrients.
3. The aflatoxin may be absorbed from the digestive tract into the body, system where it disrupts certain metabolic processes.
4. It has been suggested that aflatoxin harms animals by interfering with proper antibody production, thereby making them more susceptible to disease.
5. Aflatoxin is considered to be a potent carcinogen.

Aflatoxin B1 is acutely toxic. It is reported that diets containing as low as 0.03 ppm of aflatoxins B1 from ground nut cake may developed liver tumors if fed over a long period. Symptoms of aflatoxicosis in
chickens include loss in weight gain and feed consumption, fatty degeneration and enlargement of liver. Aflatoxins have been reported in the carcasses and eggs from birds fed diets containing the toxin. Ducks and turkeys are more susceptible to the toxin than chickens. Aflatoxin can be detected in milk from cows on a ration containing as little as 15-20 PPB of this contaminant on an air-dry basis. Milk will be free of aflatoxin after 4 days of feeding noncontaminated feed. There is some evidence that aflatoxin is less harmful to sheep than to cattle. There has been very little research done on the aflatoxin tolerance of horses; it has been assumed, however, that they would be more like the hog than the cow in this regard since they also are nonruminants.

Ammonia treatment of groundnut meal is reported to detoxify the meal safe enough for feeding. Diets containing higher protein levels, vitamin D and magnesium have variously been reported to provide protection against aflatoxins. Chances of production of the toxins in various feeds increase if they are harvested and dried during moist weather. Feeds having higher oil content are more susceptible to the production of toxin. Feeds containing more than 12 per cent moisture should not be stored in order to prevent fungal growth.

**Suggested maximum levels of aflatoxin for different classes of livestock**

<table>
<thead>
<tr>
<th>Class of Livestock</th>
<th>Suggested Maximum Level of aflatoxin in Air-Dry Ration PPB (mcg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactating dairy cows</td>
<td>20</td>
</tr>
<tr>
<td>Young pigs</td>
<td>20 (in starter or creep ration)</td>
</tr>
<tr>
<td>Young colts</td>
<td>20 (in creep ration)</td>
</tr>
<tr>
<td>Cows, Ewes and mares nursing young</td>
<td>100</td>
</tr>
<tr>
<td>Sows nursing young</td>
<td>100</td>
</tr>
<tr>
<td>Young calves and lambs</td>
<td>100 (in creep ration)</td>
</tr>
<tr>
<td>Young lambs</td>
<td>200</td>
</tr>
<tr>
<td>Dry sows and boars</td>
<td>200</td>
</tr>
<tr>
<td>Dry mares, geldings, and stallions</td>
<td>200</td>
</tr>
<tr>
<td>Growing-finishing pigs</td>
<td>400</td>
</tr>
<tr>
<td>Dry cows and bulls</td>
<td>400</td>
</tr>
<tr>
<td>Dry ewes and rams</td>
<td>500</td>
</tr>
<tr>
<td>Feedlot cattle</td>
<td>500</td>
</tr>
<tr>
<td>Feedlot lambs</td>
<td>More than 500</td>
</tr>
</tbody>
</table>