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Review Article

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Literature Review on major Antinutritional factors (Toxins) in Animal feed and their effects

Mesele Kebede Mareto
Gurage Zone Livestock and Fishery Resource Office, Gurage, Ethiopia

Abstract

Plants naturally co-exist with their predators and have therefore developed certain defense mechanisms against them. Anti-nutritional factors(toxin) are a chemical compounds synthesized in natural feedstuffs by the normal metabolism of species and by different mechanisms. Their wide distribution in plants is determined by age, cultivar, geographic distribution, and storage condition after harvesting. These antinutritional factors include the of cyanogenic glycosides, protease inhibitors, lectins, tannins, alkaloids, and saponnins in feeds may induce undesirable effects in animals if their consumption exceeds an upper limit. Anti-nutritonal factors(plant toxin) are substances which either by themselves or through their metabolic products, interfere with feed utilization and affect the health and production of animal or which act to reduce nutrient intake, digestion, absorption and utilization and may produce other adverse effects. Some of these metabolites, known as anti-nutritional factors, affect the nutritive value of forages and feedstuffs, and hence animals (including humans) that feed on them. Certain harmful effects might also be due to the breakdown products of these compounds such as depressive effect on protein digestion and utilization of protein (such as protease inhibitors, tannins and saponins), affect mineral utilization (phytates), stimulate the immune system and may cause a damaging hypersensitivity reaction (such as alopecia or physiological such as disruption of metabolic reactions and synthesis of important biochemical components of tissues, antigenic proteins) and negative effect on the digestion of carbohydrates (such as amylase inhibitors, phenolic compound). The negative effects of antinutritional factors in animal should be minimized by feeding feed with containing low (free) antinutrtional factors and reduce (remove) antinutrional factors using different methods.

Keywords: Antinutrition, Toxin, Factors, Animals, Feed

1. Introduction

Plants feeds have co-evolved with predator populations of bacteria, insects, fungi and grazing animals, and have developed defense mechanisms for their survival. Many plants also produce chemicals which are not directly involved in the process of plant growth (secondary compounds), but act as deterrents to insects and fungal attack. These compounds also affect animals (including humans) and the nutritive value of forages. The antinutrit ional factors may be regarded as a class of compounds, which are generally not lethal but reduce animal productivity and may cause toxicity during periods of scarcity or confinement when the feed rich in these substances, is consumed by animals in large quantities (Cheeke and Shull, 1985).

Anti-nutritional factors are a chemical compounds synthesized in natural feedstuffs by the normal metabolism of species and by different mechanisms(for example inactivation of some nutrients, diminution ofthe digestive process or metabolic utilization of feed which exerts effect contrary to optimum nutrition (Soetanand Oyewol, 2009). Anti-nutritonal factors are substances which either by themselves or through their metabolic products, interfere with feed utilization and affect the health and production of animal or which act to reduce nutrient intake, digestion, absorption and utilization and may produce other adverse effects(Akande *et al*., 2010).

These anti-nutritional factors are also known as ‘secondary metabolites’ in plants and they have been shown to be highly biologically active. Some of these plant chemicals have been shown to be deleterious to health or evidently advantageous to human and animal health if consumed at appropriate amounts (Ugwuand Oranye, 2006).One major factor limiting the wider feed utilization of many tropical plants is the ubiquitous occurrence in the diverse range of natural compounds capable of precipitating deleterious effects in man, and animals compound which act to reduce nutrient utilization and feed intakeare often referred to as anti-nutritional factors (Shanthakumari, 2008).

Anti-nutritional factors may be divided into two major categories. They are proteins (such as lectins and protease inhibitors) which are sensitive to normal processing temperatures.Other substances which are stable or resistant to these temperatures and which include, among many others, polyphenolic compounds (mainly condensed tannins), non-protein amino acids and galactomannan gums. The major ones includes: toxic amino acids, saponins, cyanogenic glycosidestannins, phyticacid, gossypol, oxalates, goitrogens, lectins (phytohaemagglutinins), protease inhibitors, chlorogenic acid andamylase inhibitors(Akande *et al.*, 2010).

These anti-nutritional factors need to be removed or inactivated by various procedures before the use of the ingredients in the diet. Many seeds, which were once used in traditional human and animal diets, have now fallen into disuse as agricultural andnutritional needs are re-assessed. Seeds often contain factors such as lectins, which are deleterious or toxic to animal. Seed lectins present major problems as they are resistant to heat treatment and some seeds such as kidney bean, haveto be heated for several hours at temperatures above 80ºC or boiled for 10-20 minutes to ensure the elimination of their lectin activity. Great caution should therefore be taken in the use of these seeds as dietary materials. This is particularly important since recent studies suggest that long-term exposure to relatively low levels of some anti-nutritional or toxic factors may have deleterious effects on body metabolism (Adeniyi, 2009). This review is aimed to provide comprehensive information on the major types, the potential health benefits, and adverse effects, ways of reducing of major antinutritional factors in animal feeds

2. Literature Review

2.1 Antinutritional Factors in Plant feed

2.1.1Tannins

Tannins are secondary compoundsof various chemical structures widely occurring in plant kingdom and are generally divided into hydrolysable and condensed tannins. Their anti-nutritional effects include interference with the digestive processes either bybinding with the enzymes or by binding to feed components like proteins orminerals (Liener, 1989). Hydrolysable tannins are easily degraded in biologicalsystems, forming smaller compounds that can enter the blood stream and over a period of time cause toxicity to the vital organs such as liver and kidney. Tannins are heat stable and they decreased protein digestibility in animals and humans, probably by either making protein partially unavailable or inhibiting digestive enzymes and increasing fecal nitrogen. Tannins are known to be present in feed products and to inhibit the activities of trypsin, chemotrypsin, amylase and lipase, decrease the protein quality of foods and interfere with dietary iron absorption (Felix, 2000).Tannins are known to be responsible for decreased feed intake, growth rate, feed efficiency and protein digestibility in the animals. If tannin concentration in the diet becomes too high, microbial enzyme activities including cellulose and intestinal digestion may be depressed (Aletor, 2005). Tannins also form insoluble complexes with proteins and the tannin-protein complexes may be responsible for the antinutritional effects of tannin containing feeds (Kyriazakis and Whittenmore, 2006).

2.1.2 Phytate

Phytate, which is also known as inositol hexakisphosphate, is a phosphorus containing compound that binds withminerals and inhibits mineral absorption. The cause of mineral deficiency is commonly due to its low bioavailability in the diet. The presence of phytate in feeds has been associated with reduced mineral absorption due to the structure of phyate which has high density of negatively charged phosphate groups which form verystable complexes with mineral ions causing non-availability for intestinal absorption. Phytates are generally found in feed high in fibre especially in wheat bran, whole grains and legumes (Walter*et al*., 2002).

Phytate is the salt form of phytic acid, are found in plants, animals and soil. It is primarily present as a salt of the mono- and divalent cations K+, Mg2+, and Ca2+ and accumulates in the seeds during the ripening period. Phytate is regarded as the primary storage form of both phosphate and inositol in plant seeds and grains. In addition,phytate has been suggested to serve as a store of cations, of high energy phosphoryl groups, and, by chelating free iron,as a potent natural anti-oxidant (Mueller, 2001).Phytate is ubiquitous among plant seeds and grains,comprising 0.5 to 5 percent (w/w) (Loewus, 2002). The phosphorus bound to phytate is not typically bio-available to any animal that is non-ruminant. Ruminant animals, such ascows and sheep, chew, swallow, and then regurgitate their food. This regurgitated food is known as cud and is cheweda second time. Due to an enzyme located in their first stomach chamber, the rumen, these animals are able to separate, and process the phosphorus in phytates. Humans and other non-ruminant animals are unable to do so (Harold, 2004).

Phytate works in a broad pH-region as a highly negatively charged ion, and therefore its presence in the diet has a negative impact on the bioavailability of divalent,and trivalent mineral ions such as Zn2+, Fe2+/3+, Ca2+, Mg2+,Mn2+, and Cu2+. Whether or not high levels of consumption of phytate-containing foods will result in mineral deficiency will depend on what else is being consumed. Inareas of the world where cereal proteins are a major and predominant dietary factor, the associated phytate intake isa cause for concern (Mueller, 2001).

2.1.3 Oxalate

A salt formed from oxalic acid is known as an Oxalate: for example, Calcium oxalate, which has been found to bewidely distributed in plants. Strong bonds are formedbetween oxalic acid, and various other minerals, such as Calcium, Magnesium, Sodium, and Potassium. This chemical combination results in the formation of oxalatesalts. Some oxalate salts, such as sodium and potassium, are soluble, whereas calcium oxalate salts are basically insoluble. The insoluble calcium oxalate has the tendency to precipitate (or solidify) in the Kidneys or in the Urinary tract, thus forming sharp-edged calcium oxalate crystals when the levels are high enough. These crystals play a role to the formation of kidney stones formation in the urinarytract when the acid is excreted in the urine (Nachbar *et al*., 2000).

Oxalate is an anti-nutrient which under normal conditions is confined to separate compartments. However, when it is processed and/or digested, it comes into contact with the nutrients in the gastrointestinal tract (Noonan, 1999).When released, oxalic acid binds with nutrients, rendering them in accessible to the body.If food with excessive amounts of oxalic acid is consumed regularly; nutritional deficiencies are likely to occur, as well as severe irritation to the lining of the gut. In ruminants oxalic acid is of only minor significance as an anti-nutritive factor since ruminal microflora can readily metabolize soluble oxalates, and to alesser extent even insoluble Ca oxalate. While the importance of the anti-nutritive activity of oxalic acid hasbeen recognized for over fifty years it may be a subject of interest to nutritionists in the future (Liebman and Wahsh, 2011).

Oxalic acid forms water soluble salts with Na^+, K^+, andNH_4^+ ions, it also binds with Ca^{2+}, Fe^{2+}, and Mg^{2+}rendering these minerals unavailable to animals. HoweverZn^{2+} appears to be relatively unaffected. In plants with a cell sap of approximately pH 2, such as some species of Oxalis and rumex oxalate exists as the acid oxalate(HC_2O_4), primarily as acid potassium oxalate. In plantswith a cell sap of approximately pH 6, such as some plants of goose foot family it exists as oxalate $(C_2O_4)^{2-}$ ionusually as soluble sodium oxalate and insoluble calcium and magnesium oxalates. Calcium oxalate is insoluble at a neutral or alkaline pH, but freely dissolves in acid (Liener, 2005).

2.1.4 Saponins

Saponins are secondary compounds that are generallyknown as non-volatile, surface active compounds which arewidely distributed in nature, occurring primarily in theplant kingdom. The name 'saponin' is derived from the Latin word sapo which means 'soap', because saponinmolecules form soap-like foams when shaken with water.They are structurally diverse molecules that are chemically referred to as triterpene and steroid glycosides. They consist of nonpolar aglycones coupled with one or more monosaccharide moieties. This combination of polar and non-polar structural elements in their molecules explainstheir soap-like behaviour in aqueous solutions.The structural complexity of saponins results in anumber of physical, chemical, and biological properties, which includes weetness and bitterness, foaming and emulsifying properties, pharmacological and medicinalproperties, haemolytic properties, as well as antimicrobial, insecticidal, and molluscicidal activities. Saponins havefound wide applications in beverages and confectionery, aswell as in cosmetics and pharmaceutical products. Due tothe presence of a lipid-soluble aglycone and water solublesugar chain(s) in their structure (amphiphilic nature),saponins are surface active compounds with detergent, wetting, emulsifying, and foaming properties (Shanthakumari, 2008).

Saponins were treated as toxic because they seemed tobe extremely toxic to fish and cold-blooded animals' andmany of them possessed strong hemolytic activity. Saponins, in high concentrations, impart a bitter taste and astringency in dietary plants. The bitter taste of saponin is the major factor that limits its use. In the past, saponins were recognized as antinutrient constituents, due to theiradverse effects such as for growth impairment and reduce their food intake due to the bitterness and throat-irritating activity of saponins. In addition, saponins were found toreduce the bioavailability of nutrients and decrease enzyme activity and it affects protein digestibility by inhibit various digestive enzymes such as trypsin and chymotrypsin (Liener, 2003).

2.1.5 Lectins

Lectin comes from the Latin word "legere", which means "to select". Lectins have the ability to bind carbohydrates. Nowadays, proteins that can agglutinate red blood cells with known sugar specificity are referred to as"lectins" The name "hemagglutinins"is used when the sugar specificity is unknown. Lectins and hemagglutinins are proteins/glycoproteins, which have at least one non-catalytic domain that exhibits reversible binding to specific monosaccharides or oligosaccharides.They can bind to the carbohydrate moieties on the surfaceof erythrocytes and agglutinate the erythrocytes, without altering the properties of the carbohydrates(Fereidon, 2014).

Lectins are glycoproteins widely distributed in legumes and some certain oil seeds (including soybean) which possess an affinity for specific sugar molecules and arecharacterized by their ability to combine with carbohydrate membrane receptors. Lectins have the capability to directly bind to the intestinal muscosa, interacting with the enterocytes and interfering with the absorption and transportation of 0.01% free gossypol within some low gossypol cotton nutrients (particularly carbohydrates) during digestion and causing epithelial lesions within theintestine. Although lectins are usually reported as being labile, their stability varies between plant species, manylectins being resistant to inactivation by dry heat and requiring the presence of moisture for more complete destruction (Mueller, 2001).

Lectins are carbohydrate binding proteins present inmost plants, especially seeds like cereals, beans, etc., intubers like potatoes and also in animals. Lectins selectively bind carbohydrates and importantly, the carbohydrate moieties of the glycoproteins that decorate the surface of most animal cells. Dietary lectins act as protein antigens which bind to surface glycoproteins (or glycolipids) on erythrocytes or lymphocyte. They function as both allergens and hemagglutinins and are present in small amounts in 30% of foods, more so in awhole-grain diet. Lectins have potent *in vivo* effects. When consumed in excess by sensitive individuals, they can cause3 primary physiological reactions: they can cause severe intestinal damage disrupting digestion and causing nutrient deficiencies; they can provoke IgG and IgM antibodies causing food allergies and other immune responses (Felixand Mello, 2000) and they can bind to erythrocytes, simultaneously with immune factors, causing hemagglutination and anemia. Of the 119 known dietarylectins, about half are panhemagglutinins, clumping allblood types. The remainder are blood-type specific. Ingeneral, lectins alter host resistance to infection, cause failure to thrive and can even lead to death in experimental animals (Fereidon, 2014).

2.1.6 Alkaloids

Alkaloids are one of the largest groups of chemicalcompounds synthesised by plants and generally found as salts of plant acids such as oxalic, malic, tartaric or citricacid. Alkaloids are small organic molecules, common toabout 15 to 20 per cent of all vascular plants, usually comprising several carbon rings with side chains, one ormore of the carbon atoms being replaced by nitrogen.They are synthesized by plants from amino acids. Decarboxylation of amino acids produces amines which react with amine oxides to form aldehydes. The characteristic heterocyclic ring in alkaloids is formed from Mannich-type condensation from aldehyde and aminegroups (Felixand Mello, 2000).

The chemical type of their nitrogen ring offers the meansby which alkaloids are subclassified: for example, glycoalkaloids (the aglycone portion) glycosylated with acarbohydrate moiety. They are formed as metabolic byproducts. Insects and hervibores are usual1y repulsed bythe potential toxicity and bitter taste of alkaloids (Fereidoon, 2014).Alkaloids are considered to be anti-nutrients because of their action on the nervous system, disrupting or inappropriately augmenting electrochemical transmission.For instance, consumption of high tropane alkaloids will cause rapid heart beat, paralysis and in fatal case, lead todeath. Uptake of high dose of tryptamine alkaloids willlead to staggering gate and death. Indeed, the physiologicaleffects of alkaloids have on humans are very evident.Cholinesterase is greatly inhibited by glycoalkaloids, which also cause symptoms of neurological disorder. Other toxicaction includes disruption of the cell membrane in thegastrointestinal tract (Fernando*et al*.,2012).

2.1.7 Protease Inhibitors

Protease inhibitors are widely distributed within the plant kingdom, including the seeds of most cultivated legumesand cereals. Protease inhibitors are the most commonly encountered class of antinutritional factors of plant origin. Protease inhibitors has the ability to inhibit the activity of proteolytic enzymes within the gastrointestinal tract ofanimals. Due to their particular protein nature, protease inhibitors may be easily denatured by heat processingalthough some residual activity may still remain in thecommercially produced products. The antinutrient activityof protease inhibitors are associated with growth inhibitionand pancreatic hypertrophy. Potential beneficial effects of protease inhibitors remain unclear, although lower incidences of pancreatic cancer have been observed inpopulations where the intake of soybean and its products is high (Giri and Kachole, 2004).

While protease inhibitors have been linked with pancreatic cancer in animal, they may also act asanticarcinogenic agents. The Bowman-Birk inhibitors derived from soybean have been shown to inhibit orprevent the development of chemically-induced cancer ofthe liver, lung, colon, oral and esophagusTryps (Finottiin*et al*., 2006). Inhibitor and chymotrypsin inhibitor are protease in hibitors occurring in raw legume seeds. Trypsin inhibitors that inhibit the activity of the enzymes trypsin and chymotrypsin in the gut, thus preventing protein digestion, are found in many plant species mainly in different grain legumes. Trypsin inhibitors are a unique class of proteins found in raw soybeans that inhibit protease enzymes in the digestive tract by forming indigestible complexes with dietary protein. These complexes are indigestible even inthe presence of high amounts of digestive enzymes. Protease inhibitors reduce trypsin activity and to a lesser extent chymotrypsin; therefore impairing protein digestion by monogastric animals and some young ruminant animals (Friedman*et al*.,2003).

2.1.8 Cyanogens

Cyanogens are glucosides of a sugar and a cyanide containing aglycone.Intact cyanogens are harmless. Cyanogens can be hydrolyzed by enzyme torelease hydrocyanic acid (HCN) which is toxic. The hydrolytic reaction takesplace in the rumen by microbial activity. In pig and horse, enzyme concernedin the release of HCN is destroyed by the gastric HCl. Hence, ruminants are more susceptible to cyanide than monogastric animals. The HCN is rapidlyabsorbed and some is eliminated through the lungs, but the greater part israpidly detoxified in the liver by conversion to thiocyanate. Excess cyanideion can quickly produce anoxia of the central nervous system through inactivating the cytochrome oxidase system and death can result within fewseconds. Based on the intensity, animals show nervousness, abnormal breathing, trembling or jerking muscles, blue colouration of the lining of themouth, spasms or convulsions and respiratory failure (Harris and Shearer, 2003).

High levels of cyanogens can be found in certain grasses such as jowar(sorghum) and Sudan grass, linseed and cassava root. In plants the glucosideis non-toxic in the intact issues and as stated earlier, when the plants aredamaged or begin to decay, hydrolytic enzyme from the same plant is releasedand liberates HCN. This reaction can take place in the rumen by microbial activity. Forage sorghum, sorghum-Sudan hybrids, Sudan grass, and Johnson grass can accumulate cyanide under drought conditions if rain fall is scantyor irrigation is not plenty(Golden,2009). When the plant wilts, its cells rupture and the HCNis freed from the sugar molecule. If consumed by grazing livestock, the free HCN is readily absorbed into the blood stream, where it prohibits the animal'sability to take oxygen from the blood. Even in unwilted plants, chewing and digestion in the rumen can release toxic levels of HCN.Many other conditions adversely affecting the normal growing functionsof a plant can cause accumulation of HCN, such as prolonged cloudiness, soilacidity, abnormally high or low temperature (especially sudden changes intemperature), herbicide treatments, and low soil phosphorous. Proper curingor ensiling of forages containing high levels of HCN greatly reduces oreliminates the danger because free HCN volatilizes fairly quickly after it isreleased. Animals which have shown much evidence of toxicity may be injected intravenously with 3 g of sodium nitrate and 15 g sodium thiosulphate in 200ml H_2O for cattle, for sheep, 1g sodium nitrate and 2.5 g sodium thiosulphatein 50 ml H2O (Harris and Shearer 2003).

2.1.9 Nitrate

Nitrate itself is not toxic to animals, but at elevated levels will cause adisease called nitrate poisoning. Nitrates are normally found in forages are converted by the digestion process to nitrite, and in turn the nitrite is convertedto ammonia. Nitrate accumulation can occur in virtually any plant with the ability to grow rapidly and use soil nitrogen efficiently. Under normal conditions, nitrate in the soil is absorbed by plant roots, transported through the stems, and converted in leaves to proteins and other substances that are useable by the animal. Nitrate typically is used by the plant about as fast as itis absorbed from the soil. Nitrate poisoning occurs when this excessive nitrate is consumed and converted to nitrite faster than the animal can use it. Free nitrite in the rumen is readily absorbed into the blood stream, where it destroys the blood’s ability to absorb and carry oxygen(Harris and Shearer, 2003).

The ammonia is then converted to protein by bacteria in the rumen. If cattle rapidly ingest large quantities of plants that contain high levels of nitrate, nitrite will accumulate in the rumen. Nitrite is ten times as toxic to cattle asnitrate. It cause an acute toxicosis in cattle resulting from formation of methemoglobin (a true oxidation product of haemoglobin), which is unableto transport oxygen because the iron is in the ferric (Fe+++) rather than the usual ferrous (Fe++) state. Symptoms seen in acute toxicity include labouredbreathing (dyspnoea), grinding of the teeth, uneasiness and excessive salivation. Drying or ensiling forages tends to have a small effect on nitrate levels. Hays and silage from high-risk plants may remain toxic. Nitrate poisoning is usually treated by intravenous injection of methylene blue. Ahigh dose of concentrates in the daily ration and adequate feeding of VitaminA have a protective effect (Harris and Shearer.

Table 1.Plant toxins sources and concentrations

Toxins	Principal sources	Typical concentration
Lectins	Jackbean	73 units/mg protein
Wingedbean		40-320 units/mg
	Lima beans	59 units/mg protein
Trypsin	Inhibitors soybean	88 units/mg
Cyanogens	Cassava root	186mg HCN/kg
Condensed tannins	Acacia spp	65g/kg
Quinolizidine	Alkaloid lupin	10-20 g/kg
Glusosinolates	Rapeseed	100mmol/kg
Gossypol	cotton	0.6-12g/kg

Source: D’Mello, (1995)

2.1.10 Moulds and mycotoxins

Mycotoxins are the secondary metabolites of fungi that have the capacity to impair animal health and productivity (D’Mello and Macdonald, 1998).Principal mycotoxins occurring in feeds and forages are presented in Table 4.Mycotoxin contamination of forages and cereals frequently occurs in the field following infection of plants with particular pathogenic fungi or withsymbiotic endophytes. Contamination may also occur during processing and storage of harvested products and feed whenever environmental conditions are appropriate for spoilage fungi. Moisture content and ambient temperatureare key determinants of fungal colonization and mycotoxin production. Mycotoxigenic species may be further distinguished on the basis of geographicalprevalence, reflecting specific environmental requirements for growth and secondary metabolism. Thus, *Aspergillus flavus*, *A. parasiticus* and*A. ochraceus* readily proliferate under warm, humid conditions, while *Penicillium expansum* and *P. verrucosum* are essentially temperate fungi.Consequently, the *Aspergillus* mycotoxins predominate in plant products emanating from the tropics and other warm regions, while the *Penicillium* mycotoxins occur widely in temperate foods, particularly cerealgrains. *Fusarium* fungi are more ubiquitous, but even this genus contains toxigenic species that are almost exclusively associated with cereals from warmcountries.A new source of neutraceuticals has some potential antifungal and cytotoxic properties have been identified (Mokhlesi *et al.*, 2012) from seacucumber (*Holothuria leucospilota)*.The common organisms are susceptible to*Aspergillus niger, Candida albicans, Pseudomonas aeruginosa, Staphylococcusaureus and Escherichia coli etc*.

2.1.11 Aflatoxins and Gossypol

This group includes aflatoxin B1, B2, G1 and G2 (AFB1, AFB2, AFG1 and AFG2, respectively). In addition, aflatoxin M1 (AFM1) has been identified in the milk of dairy cows consuming AFB1-contaminated feeds. The aflatoxigeni*c* Aspergilliare generally regarded as storage fungi, proliferating under conditions of relatively highmoisture/humidity and temperature. Aflatoxin contamination is, therefore, almost exclusively confined to tropical feeds such as oilseed by-products derived from groundnuts, cottonseed and palm kernel. Aflatoxin contamination of maize is also an important problem in warm humid regions where *A. flavus* may infect the cropprior to harvest and remain viable during storage. Surveillance of animal feeds for aflatoxins is an ongoing issue, owing to their diverse forms of toxicity and also because of legislation in developed countries. Gossypol pigment in cotton seed occurs free and bound forms. In whole seeds, gossypol exists essentially in the free form, but variable amounts may bind with protein during processing to yield inactive forms. Free gossypol is the toxic entity and causes organ damage, cardiac failure and death. Cottonseed meal fed to bulls can induce increased sperm abnormalities and decreased sperm production (D'Mello andMacdonald, 1998).

Table 2: Different mycotoxin producing fungi and their principal toxin

Fungal Species	Mycotoxins
Aspergillus flavus; *A. flavus* Cyclopiazonic acid *A. ochraceus;* *P. citrinum;* *P. expansum* Patulin *P. citreo-viride* Citreoviridin *Fusarium culmorum;* *F. sporotrichioides;* *F. sporotrichioides; F. graminearum;* *F. culmorum; F. graminearum* Zearalenone *F. moniliforme*	*A. parasiticus* Aflatoxins *Penicillium viridicatum; P.cyclopium* Ochratoxin A *P. expansum* Citrinin *F. graminearum*Deoxynivalenol *F. poae* T-2 toxin *F. poae* Diacetoxyscirpenol*;* *F. sporotrichioides* *Alternaria alternata Neotyphodium* *coenophialum lolii* *laviceps purpurea* *ithomyces chartarum* *homopsis leptostromiformis*

(D'Mello and MacDonald, 1998)

3. Mechanism of toxicity

Tannins may form a less digestible complex with dietary proteins and may bind and inhibit the endogenousprotein such as digestive enzymes. The tannin-protein complexes are astringent and adversely affect feed intakeand all plants contains phenolic compounds but their type and concentration may cause negative animal responses (*Smitha et al., 2013)*. The concentration ofcondensed tannins above 4 per cent has been reported to be toxic for ruminants as they are more resistant tomicrobial attack and are harmful to a variety of microorganisms (Waghorn , 2008). It has been reported thatsaponins can affect animal performance and metabolism in a number of ways as follows: erythrocyte haemolysis, reduction of blood and liver cholesterol, depression of growth rate, bloat (ruminants), inhibition of smooth muscle activity, enzyme inhibition and reduction in nutrient absorption (Akande *et al.,* 2010).

Phytic acid acts as a strong chelator, forming protein and mineral-phytic acid complexes; the net result being reduced protein and mineral bioavailability. Phytic acid is reported to chelate metal ions such ascalcium, magnesium, zinc, copper, iron and molybdenum to form insoluble complexes that are not readily absorbed from gastrointestinal tract. Phytic acid also inhibits the action of gastrointestinal tyrosinase, trypsin, pepsin, lipase and amylase. Similarly Oxalic acid binds calcium and forms calcium oxalate whichis insoluble. Calcium oxalate adversely affects the absorption and utilization of calcium in the animal body(Akande *et al*., 2010). Trypsin inhibitors have been implicated in reducing protein digestibility and in pancreatic hypertrophy.

4. Beneficial effects of antinutrients

The positive of tannin in animal feeing includes; increased efficiency of protein utilization, reductionof parasite burden, and reduction of proteolysis during ensilage, bloat prevention, and increase quality of animal products, reduction of n emission into the environment and defaunate rumen. Condensed tannins (CT) have improved live weight gain, wool production and reproductive efficiency in sheep fed temperate forages and reduced the impact of gastro-intestinal parasitism. However, their value is also linked to environmental issues, such as reducing nitrogen pollution from animals grazing lush pastures with a high nitrogen content and lessening methane emissions from rumen fermentation (Waghorn, 2008).

Saponins have shown a variety activities such antitumor, cholesterol lowering, immune potentiating, anticancer, antioxidants (Blumert and Liu, 2003) and to presser lower risk of implicated in coronary heart diseases (Ferri, 2009), and saponins potential as ointment hydrocarbon to shape of first collagen, there is proteinhave a role in recovery process of wound healing. Potential beneficial effects of protease inhibitors remain unclear, although lower incidences of pancreatic cancer have been observed in populations where the intake of soybean and its products is high. While protease inhibitors have been linked with pancreatic cancer in animal studies; they may also act as anticarcinogenic agents (Chunmei et al., 2010).

5. Feeding mechanisms of feeds with anti-nutritional factors

A number of methods have been tried to overcome the deleterious effect of such anti-nutritional factors and tannins is came at the head. These are through making hay, silage with inoculants, using polyethylene glyco (Ben Salem*et al*., 2006),urea (Russel and Lolley, 1989) or biological treatment with fungi (Hassan, 2006)can be applied to either take off or minimized and decrease anti-nutritional factors concentration. It is will know that alkali treatment includes polyethylene glycol (PEG), which a tannins-binding agent (Jones and Mangan, 1977)was shown to be a powerful tool for isolating the effect of tannins on various digestive function (Barry *et al*., 1986).

Heat processing is a common procedure in feed manufacture, conferring improved properties as regards the safety and nutritive value of animal feeds.For example, heat treatment of dried poultry litter appears to be an effective method for controlling, or even eliminating, contamination with *Salmonella*, *E.coli* and *Campylobacter* (Jeffrey *et al*., 1998). Thermal processing is also effectivefor denaturing proteinase inhibitors, lectins and cyanogens. However, forantigenic proteins, more complex procedures involving the uses of hot aqueousethanol extraction are required (D'Mello, 1991).

Polyethylene glycol was supplied to animals on tannin-rich diets in different ways (in concentrate or feed blocks, dissolved in water, or sprayed on feed). Feed block, a solidified blend of agro-industrial by-products, was foundto be an efficient supplement for increasing intake, rumen fermentation, digestibility and daily weight gain insheep or goats fed on shrub foliage high in tannins. The advantage of these supplements lies in the synchronized, fractionated and balanced supply of main nutrients to rumen microflora and the host animal on tannin-rich diets, results attributable to the slow release of polyethylene glycol on licking of the block by the animal. This slow release characteristic provides an economic use of this relatively costly tannin-inactivating compound, andmaximizes its positive effect on the fodder potential of tanniniferous browse or tree species (*Smitha et al., 2013).*

6. Conclusions

Antinutritional factors in feeds are responsible for the deleterious effects that are related to the absorption of nutrients and micronutrients which may interfere with the function of certain organs. Most of these antinutritional factors are present in feeds of plant origin. These antinutritional factors include the of cyanogenic glycosides, protease inhibitors, lectins, tannins, alkaloids, and saponnins in feeds mayinduce undesirable effects in animals if their consumption exceeds an upper limit. Certain harmful effects might also be due to the breakdown products of these compounds such as depressive effect on protein digestion and utilization of protein (such as protease inhibitors, tannins and saponins), affect mineral utilization (phytates), stimulate the immune system and may cause a damaging hypersensitivity reaction (such as antigenic proteins) and negative effect on the digestion of carbohydrates (such as amylase inhibitors, phenolic compound). However, some antinutritional factors as well as their breakdown products may possess beneficial health effectsif present in small amounts.

7. Recommendations

The negative effects of antinutritional factors in animal should be minimized by feeding feed with containing low (free) antinutrtional factors and reduce (remove) antinutrional factors using different methods (avoid high level of this factor) and by creating awareness about the effect antinutritional factors.

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