The Pharma Innovation Journal 2022; SP-11(5): 1863-1875

www.ThePharmaJournal.com

The Pharma Innovation

ISSN (E): 2277-7695
ISSN (P): 2349-8242
NAAS Rating: 5.23
TPI 2022; SP-11(5): 1863-1875

www.thepharmajournal.com
Received: 20-03-2022
Accepted: 25-04-2022

V Kumar
Department of Agronomy
Lovely Professional University,
Phagwara, Punjab, India

SP Bainade
Department of Agronomy
Lovely Professional University,
Phagwara, Punjab, India

C Kashyap
Department of Agronomy
Lovely Professional University,
Phagwara, Punjab, India

Anuraj Singh
Department of Agronomy
Lovely Professional University,
Phagwara, Punjab, India

Corresponding Author
V Kumar
Department of Agronomy
Lovely Professional University,
Phagwara, Punjab, India

Anti-quality factors in field crops

V Kumar, SP Bainade, C Kashyap and Anuraj Singh

Abstract
Certain chemical ingredients and physical properties of forages can significantly change animal responses. Certain terpenoids, flavonoids, phenols, alkaloids, and unique exterior and internal anatomical aspects that can affect animal intake, dry matter digestion, and gains are taken into account in forages.

Keywords: Anti-quality factors, field crops, alkaloids

Introduction
The quality of forage may be determined by converting it into animal products such as milk, meat, wool, or labour. The potential for a feed to create a desirable animal reaction is measured by its forage quality (Ball, *et al.*, 2001) [8]. However, in practice, these animals are unable to reach their full potential due to number of variables that contribute to a drop in feed quality. Physical plant characteristics, nutritional or mineral-related issues, naturally occurring plant metabolites and poisons, and others are among these considerations. Toxicity caused by the eating of different forages has been observed often among farm animals. The common anti-quality variables found in different fodder crops have been examined in this lecture with reference to their toxic levels, symptoms, and preventative actions to be taken for their safe feeding to dairy cows. Variations in forage genotype, maturity, season, management, and anti-nutritional components all affect fodder quality. Certain chemicals can build up in plants to the point that when swallowed, they have a negative impact on animal reactions. Anti-quality chemicals are molecules that are regarded secondary in relation to the active players in plant-cell metabolism (Barnes, RF, and DL. Gustine, 1973) [9]. Animal responses (intake, digestibility, physiological condition, and so on) may be influenced in a subtle way by the ingestion of such chemicals. They may induce an un-favorable state in the animal without generating obvious symptoms, or they may have a more dramatic impact, resulting in death. The buildup of cyanogenic glucosides in plants is an example of the latter. Animal consumption has no negative effects on animal health or behavior until enough hydrogen cyanide (HCN) is released from the glycoside to surpass the animal's tolerance threshold. After that, death comes rapidly.

Factors impacting the quality of forages
Animal performance, whether in terms of growth or milk production, is determined by the animal's production capacity, as well as the amount of dry matter (DM) consumed, and the nutritional content of the DM consumed. As a result, (1) forage intake and (2) forage nutritive value are the two forage-related parameters that influence animal performance. These components work together to influence the forage's quality.

Factors Influencing Forage Consumption
The amount of fodder available and the properties of the forage ingested, as well as the animal's intake capacity, performance level, health, genotype, and social rank, all influence forage intake. Temperature and humidity, as well as other environmental conditions, influence fodder intake. Forage consumption is further influenced by management parameters such as stocking rate, kind, and quantity of supplements, feeding frequency, and water and feed availability.
Furthermore, forage quantity and numerous features of forages, such as particle size of stored forages and levels of fiber, protein, and minerals in the IDM, influence forage intake. The rate at which undigested DM moves through an animal influences its forage intake. Mold infection, inadequate fermentation, and any other factors that make the fodder unpalatable to animals all have an impact on livestock consumption of stored forages.

The type of pasture fodder consumed is influenced by the sward's characteristics. On pasture, accumulations of dead fodder or manure may reduce intake, but a thick, leafy canopy would boost forage intake.

When enough amounts of forage are available, no supplements of protein or energy are supplied to the animal, and adequate minerals are accessible either in the forage or as supplements the term "voluntary forage intake" is used to indicate how much forage DM an animal will ingest. Depending on the content of the forage and the type and amount of supplement administered to the cattle, energy and protein supplements may enhance or reduce forage consumption.

Factors Affecting the Nutritive Value of Forages

The nutritional value of pasture is largely measured by crude protein (CP) and "available" energy contents. TDN (total digestible nutrients) has been used as an overall measure of accessible energy in forages for many years. However, during the last 20 years, assessments of digestible forage, metabolizable forage, and net energy of forage have become more common. TDN, on the other hand, is still an appropriate and simple nutritive value metric, especially for beef cattle. Variations in forage genotype, maturity, season, and management have the greatest impact on forage quality.

Genotype

Legumes are often superior to grasses in terms of quality. Because legumes contain a larger percentage of fast digested leaves, they have higher CP concentrations and a higher consumption by animals. Because legumes have more poorly digested lignin than grasses, TDN concentrations in legumes and cool-season grasses are comparable. Generalizations concerning grass quality are dangerous, although temperate or cool-season grasses like rye and ryegrass are frequently better than tropical or warm-season grasses like bermudagrass and Bahia grass. However, fodder quality varies greatly between and across grass genera.

Maturity

Fodder quality is heavily influenced by the stage of regrowth at the time of usage, whether as hay, haylage, or grazed forage. The number of days between hay or haylage harvests, as well as the rest time in rotational grazing, define the forage-regrowth stage. Due to the buildup of stems and the depletion of poorly digested lignin in both leaves and stems, forage quality begins to deteriorate during the regrowth phase. As a result, fodder quality tends to deteriorate as the time between harvests of stored forages or the rest intervals in rotational grazing get longer.

The reproductive stage of growth can be used to determine the maturity of legumes and cool-season grasses. Weeks of regrowth, on the other hand, are a stronger sign of maturity in warm-season grasses since blooming can commence soon after regrowth begins. Harvesting Coastal bermudagrass at intervals longer than five weeks degrades the forage's quality. The most significant variation in maturity effects on perennial grasses is the decrease in voluntary intake between six and eight weeks. These and other studies suggest that feed quality will be lower than required for animal maintenance after eight weeks of regrowth Digit grass and limpo grass, on the other hand, have a somewhat greater TDN when mature than the other grasses. As a result, limpo-grass and digit grass make great fall stockpile forages. However, mature limpo grass and digit grass are frequently low in CP, necessitating protein supplementation for optimal use.

Different fodder crops include anti-nutritional substances such as nitrates, oxalates, HCN, and tannins, which harm animal health by disrupting animal metabolism. The negative impact of anti-quality components is determined by the pace at which they are degraded by microorganisms, and this has an impact on the animal's development and performance. The key anti-quality components that have a negative impact on the nutritional content of forages are discussed below.

Nitrate

Corn-stalk poisoning caused nitrate toxicity in cattle as early as 1895. During that time, however, nitrate was not recognized as the primary toxin. After an outbreak of oat-hay poisoning in the high plain's region in the late 1930s, nitrate was eventually blamed (Launchbaugh *et al.,* 2001) [59]. Sudan grass, pearl millet (Andrews and Kumar, 1992) [5], and oats (Singh *et al.*, 2000) are examples of fodder crops that can collect nitrate to potentially lethal amounts. Consuming forages is a better way to explain nitrate toxicity. When forages include an extremely high amount of nitrate, the animal is unable to complete the conversion, and nitrite builds up. Nitrite is taken straight into the circulation via the rumen wall, where it transforms hemoglobin (the oxygen-carrying molecule) to methemoglobin (which cannot transport oxygen). Instead of the customary brilliant red, the blood develops a chocolate brown tone. The death of a nitrate (nitrite) poisoned animal is caused by asphyxiation, or a lack of oxygen (Benjamin, 2006) [11].

Nitrate toxicity

Nitrate accumulation in plants is generated by an overabundance of nitrogenous fertilizers applied to crops, a lack of sunlight, bad weather, herbicides (2, 4-D), and diseases. Nitrate toxicity may be seen in a variety of fodder crops, including sorghum, Sudan grass, maize, oats, and barley.

Nitrate concentrations are also affected by the stage of growth and the parts of the plant. Young plants have the highest concentration, which gradually declines as the plant ages. The lowest part of the plant stalk or stem has the highest quantity of nitrate. Floral components > Stem > Leaves (order of nitrate accumulation)

Farmers and ranchers rely on a successful livestock-crop mix. Forage crops, in particular, are crucial to the farmer, but they must be closely managed owing to potential plant toxicants. Nitrate (N03) toxicity is one toxicosis to be concerned about. Corn-stalk poisoning caused nitrate toxicity in cattle as early as 1895. However, nitrate was not identified as the primary toxicant at the time. After an outbreak of oat-hay poisoning in the high plains region in the late 1930s, nitrate was eventually blamed. "Nitrate Toxicity" should be used instead of "Nitrate Toxicity." When nitrate is consumed by ruminant animals, it is chemically reduced to nitrite. Rumen microorganisms are responsible for the decline. Nitrite is easily taken into the circulation, where it converts the ferrous iron in haemoglobin to ferric iron, resulting in methemoglobin, a modified red blood colour. Because methemoglobin is incapable of carrying oxygen to various bodily tissues, the animal's blood turns a distinctive chocolate brown hue before and during death from asphyxiation. Methemoglobinemia is a condition in which ruminant animals are harmed in this way by excessive nitrate diets. Simple stomached species, such as

pigs and poultry, lack the bacteria necessary for this fast conversion and are therefore less sensitive to nitrate poisoning.

Management

The propensity of different plant species to collect nitrate varies. Even simple weeds in the barnyard might be problematic. Management: If nitrate buildup is an issue in your location, use feed sources with a lower tendency for accumulation. Consider the year's climatic circumstances and adjust inputs to account for the decreased accumulation potential. The nitrate level of stalks is greatest, followed by leaves and grain in decreasing order. Management: The lowest six inches of the stem of piper Sudan grass, sorghum Sudan grass, and pearl millet have three times more nitrate than the top section of the plant, according to study. Raising the cutter bar above the six-inch mark may help to reduce nitrate levels. Plants that are immature or young have a higher nitrate accumulation potential than older plants (such as those with seed in the hard dough stage). Management: Turning cattle in on a field that is still young in development should be done with caution. Allow hungry cattle to feed before releasing them into a field, since they are less likely to selectively graze leaves over stems. Prior to releasing cattle on a field of concern, it is always a good idea to test it. Any meteorological situation that inhibits plant development might lead to an increase in nitrate buildup. Drought and chilly, overcast weather are examples of this. Management: Obviously, you have no influence over this situation. However, you should be aware of the meteorological conditions for that year and how they impacted your produce. The amount of nitrate in a plant that is toxic to ruminants is just enough for 2 to 4 days of vigorous plant development. As a result, weather-related issues normally go away after a few days of sunshine, enough moisture, and appropriate temperatures (70°F for small grains and 85°-90°F for sorghums). Late-planted material or regrowth that has been affected by frost and shortly after a drought-ending rain would be an exception. After a drought-ending rain, nitrate levels in millets take 7 to 14 days to recover to acceptable levels, according to research.

Prevention of nitrate toxicity in animal

Make silage from fodder that has been destroyed by the drought. The nitrate level is reduced by 40% to 60% during the ensiling process. When a crop is cultivated in conditions that promote nitrate buildup, postpone harvesting until the conditions improve and the nitrate content falls to a safe level. Before feeding to cattle, have any suspicious fodder evaluated.

Mix poisonous forage with nontoxic forages and/or energy feeds like molasses or maize to dilute the toxicity.

Oxalates

Oxalate is a kind of dicarboxylic acid present in a wide range of plants. Oxalate is engaged in a number of functions, including dealing with metal toxicity, calcium control, quenching oxidative burst during pathogenic onslaught, and programmed cell death. Excessive oxalate concentrations in plants are adequate to induce animal poisoning, despite its diverse physiological importance. Oxalate can be found in a variety of forms, including soluble oxalate, insoluble calcium oxalate, and a mixture of the two. Insoluble oxalate is formed by the ions calcium (Ca2+), magnesium (Mg2+), and iron (Fe2+), whereas soluble oxalate is formed by the ions sodium (Na+), potassium (K+), and ammonium (NH4 +). Soluble oxalate is one of a handful of anti-nutrients present in forage plants. As a result, there are disruptions in the environment. Causes and effects of calcium and phosphorus metabolism excessive bone mineral mobilisation. The dehydrated bones of animals become fibrotic and misshapen, resulting in lameness and a 'bighead. 'Oxalate can be metabolised in four different ways in the ruminant system. First, rumen bacteria may breakdown soluble oxalate (Allison *et al.*, 1977) [3].

Second, when Ca2+ or Mg2+ ions interact with oxalate ions in the rumen or stomach to make insoluble oxalate, Ca2+ or Mg2+ ions form insoluble oxalate. These crystals are excreted in the faeces and are incapable of being absorbed by the body. Finally, when Ca2+ levels in the food are low, soluble oxalate remains soluble in the liquid portion of the gut contents and is easily absorbed into the circulation. If the oxalate ion concentration in the kidnapped's blood rises to dangerously high levels. It can mix with Ca2+ or Mg2+ to produce insoluble oxalate crystals, which can obstruct urine flow and lead to renal failure (Lincoln and Black, 1980; Blaney *et al.*, 1982) [62, 18]. Fourth, insoluble oxalate from plants can travel through the digestive tract without causing damage to the body's metabolism (Ward *et al.*, 1979) [105].

Various tropical grasses contain significant amounts of soluble oxalates to cause calcium shortage in grazing animals. Buffel grass (*Cenchrus ciliaris*), pangola grass (*Digitaria decumbens*), Setaria (*Setaria sphacelata*), and kikuyu grass (*Setaria sphacelata*) are among them (*Pennisetum clandestinum*). Although the oxalate level of Guinea grass, bajra, and Napier bajra hybrid is below acceptable limits, they may be harmful if fed over a prolonged length of time. Oxalates combine with calcium to form calcium oxalate, which is insoluble and reduces calcium absorption. This causes a change in the absorbed calcium: phosphorus ratio, causing bone mineral mobilization to compensate for the hypocalcemia. Nutritional secondary hyperparathyroidism or osteodystrophy fibrosa is caused by prolonged bone mineral mobilization (Rahman and Kawamura, 2011) [80].

Because oxalate is degraded in the rumen, cattle and sheep are less harmed. On Setaria pastures, however, cattle have died from oxalate poisoning due to severe hypocalcemia, and sheep have been poisoned when grazing buffel grass. In horses, soluble oxalate levels of 0.5 percent or greater in pasture grasses can cause nutritional hyperparathyroidism. In ruminants, soluble oxalate levels of 2% or more can cause acute toxicosis. In situations of fast development, grasses have the greatest oxalate content, with concentrations as high as 6% or more of dry weight (Cheeke, 1995) [23].

Ones that are young have more oxalate than plants that are older. The amount of oxalate in the plant increases rapidly during the early stages of development, then decreases as the plant grows (Davis, 1981) [27]. It's also been discovered that changing the harvesting period might affect the oxalate concentration of Napier grass. As the harvest interval increased, the oxalate content decreased. Bamboo has three times the amount of oxalate in younger regions of the shoot compared to older parts, indicating that oxalate is distributed evenly in plants. When Napier grass was grown, however, the amounts of oxalate in the leaf and stem tissues were virtually equal. Due to peak growth in July and wet conditions, was harvested in early summer (Rahman *et al.*, 2006) [83]. Seasons, ruminants, especially little ones, prefer leaves over stems, but because leaves often contain more soluble oxalate than other

portions of the plant, grazing animals' plant sources should be carefully monitored.

Plant parts

Plant parts Leaves normally contain higher oxalate content than stem so cultivars with high proportion of leaves may also have a higher oxalate level than that of varieties with low proportion of leaves. Compared to a non-dwarf variety, a dwarf variety of napiergrass exhibited higher oxalate content, though the difference was not significant (Rahman *et al.*, 2006) [83]. In a study on NBH, total oxalate content was found to be positively correlated to leaf/stem ratio in autumn season (Kaur *et al.*, 2016) [49]. Leaf tissue had much higher oxalate than stem tissue in earlier research on kikuyugrass (Marais, 1990) and setaria (John and Ford, 1972) [67, 46]. In early summer, oxalate concentration was greater in stem (3.44%) than leaf tissues (3.01%), while in late summer, leaf tissue showed the highest values (Rahman *et al.*, 2006) [83].

Seasonal variation

Seasonal fluctuation has a significant impact on the amount of oxalate in NBH. In NBH, soluble oxalate concentration was maximum in the summer (2.08 percent) i.e. the month of June, and dropped in the succeeding harvest seasons (monsoon, autumn, pre-winter) (Kaur *et al.*, 2016) [49]. Higher oxalate levels in June and July might be attributable to a surge in growth during the summer and wet seasons (Singh 2002) [94]. In setaria, a similar trend in soluble oxalate was found over harvest seasons (Rahman *et al.*, 2014) [85]. In another pot experiment, napiergrass samples with the greatest oxalate content (3.77 percent) and the lowest oxalate content (1.76 percent) had the highest oxalate content (3.77 percent) in early summer samples and the lowest oxalate content (1.76 percent) in late autumn samples (Rahman *et al.*, 2006) [83]. In the spring and fall seasons, Atriplex species had oxylate levels of 8.29 and 4.92 percent of dry weight, respectively (Abu-Zanat *et al.*, 2003) [1]. In NBH, greater oxalate content was reported in harsh winter and summer months than in the usual season, according to Sidhu *et al.* (2014) [92].

Nitrogen fertilization

The oxalate content of rhodegrass, guineagrass, and sudangrass did not rise appreciably as the rate of N fertilisation was increased (Rahman *et al.*, 2008a) [86]. The findings indicate that these grasses do not require additional N fertiliser (Standard 2 or Standard 4) to produce large levels of oxalate salts. Furthermore, in terms of animal toxicity, the amounts of oxalate found in these grasses are fairly low. However, these findings contradict Jones and Ford (1972) [46], who found that when N fertiliser (as urea) levels climbed from 0 to 200 kg/ha, setaria oxalate concentration increased ranging from 3.3 to 5.6 percent. The concentration of oxalate in a bajra x Napier hybrid (cv. Phule jaywant) grew with N fertili. The concentration of oxalate in a bajra x Napier hybrid (cv. Phule jaywant) grew with N fertilisation and reached at 125 percent of RDN in a recent research (Sonane *et al.*, 2017). In another research on napiergrass, N treatment had an effect on the initial cut, but not on successive cuts (Rahman *et al.*, 2010a) [82]. Unlike total oxalate, soluble oxalate level in NBH exhibited a positive relationship with nitrogen fertilisation dosages (Kaur *et al.*, 2016; Table 2) [49]. ratio. The concentration of oxalate in a bajra x Napier hybrid (cv. Phule jaywant) grew with N fertilisation and reached at 125 percent of RDN in a recent research (Sonane *et al.*, 2017). In another research on napiergrass, N treatment had an effect on the initial cut, but not on successive cuts (Rahman *et al.*, 2010a) [82]. Unlike total oxalate, soluble oxalate level in NBH exhibited a positive relationship with nitrogen fertilisation dosages (Kaur *et al.*, 2016) [49]. and reached at 125 percent of RDN in a recent research (Sonane *et al.*, 2017). In another research on napiergrass, N treatment had an effect on the initial cut, but not on successive cuts (Rahman *et al.*, 2010a) [82]. Unlike total oxalate, soluble oxalate level in NBH exhibited a positive relationship with nitrogen fertilization dosages (Kaur *et al.*, 2016.) [49].

Clipping interval

The oxalate concentration of various plants is substantially influenced by the clipping interval. In setaria, the young plant has more oxalate than the mature plant (Jones and Ford, 1972) [46]. The oxalate content in napiergrass, NBH, and pearl millet crops decreased as the harvest interval increased (Kaur *et al.* 2012) [48] According to Rahman and colleagues (Rahman *et al.*, 2009b) [81] oxalate concentrations in napiergrass decreased as the plant material aged.

It's possible that this is due to a faster rate of oxalate synthesis in the early stages of development in these species (Singh, 2002) [94]. The decrease in oxalate concentration with crop maturity might be attributed to oxalate breakdown to carbonates or bicarbonates, or oxalate dilution. Plants like rhubarb (Rheum rhabarbarum), halogeton, and water hyacinth (Eichhornia crassipes), on the other hand, gathered greater oxalate content as they matured. The toxicity of Halogeton increased as it grew, reaching a peak at maturity (Torell *et al.*, 2005) [100]. Abu-Zanat and his colleagues (2003) [1].

Animal Type /Toxic level

Ruminants (such as goats) are more resistant to oxalate than non-ruminants (e.g. horses).Oxalate is degraded by rumen bacteria into formic acid and carbon dioxide, making it non-Cattle and sheep that graze tropical grasses for an extended period of time might develop severe hypocalcemia (Seawright *et al.*, 1970) [87]. sheep given 0.12 g oxalic acid/ kg live weight/d, a modest degree of hypocalcemia was seen (Kyriazakis *et al.*, 1997) [58]. Panda and Sahu (2002) [76] found that when total oxalate consumption was 0.58 percent of DM diet, bulls were unaffected, but when it was increased to 1.19 percent, a negative calcium balance was produced. Because of the high oxalate concentration in buffel grass (Cenchrus ciliaris) pastures, sheep poisoning has been observed (McKenzie *et al.*, 1988) [69]. Higher mortality has been documented in cow and buffalo calves after eating napiergrass (Pennisetum purpureum cv.

Prussic acid / cyanogens (HCN)

Cyanogens are cyanide-containing aglycones that are glycosides of a sugar or sugars. It can be hydrolyzed by enzymes in the cytosol to release HCN. When enzymes and glycosides combine to generate HCN, the plant is harmed. Ruminants are more sensitive to CN poisoning than non-ruminants because the hydrolytic process might occur in the rumen due to microbial activity. The enzyme rhodanese, which converts CN to thiocyanate, absorbs the HCN and quickly detoxifies it in the liver (SCN). The cytochrome oxidase is inhibited by too much cyanide ion. As a result, ATP production is halted, tissues are deprived of energy, and death occurs quickly.

When animals are pastured on sorghum-type plants, such as grain sorghum, forage sorghum, sudangrass, sorghum-sudangrass crosses, Johnson grass, sweet sorghums, and other sorghum-type plants, they might become poisoned by prussic acid. Prussic acid induces asphyxiation by preventing the enzyme that connects oxygen to red blood cells from working (Allison, 2002) [2]. Ruminants are more vulnerable to the disease than non-ruminants. Young seedlings will have a higher HCN content than adult seedlings (Sultan, 2003) [4].

Factors that influence the amount of prussic acid in plants

Sudangrass has around 40% less prussic acid than most other sorghums (Sorghum > Sudangrass > Sudangrass). Prussic acid levels in leaf blades are usually greater than in leaf sheaths or stems. Prussic acid is found in higher concentrations in upper leaves than in lower leaves. Before the boot stage, the highest amounts of prussic acid are attained. Drought- Prussic acid poisoning is most likely caused by severe drought. Fertilizer- When high nitrogen rates are given to soils that are lacking in phosphate and potassium, the levels of prussic acid normally rise.

Prussic Acid Poisoning and How to Avoid It

Green Chop-Because it is not selectively grazed, green chop feed is typically safer than pasture forage. Silage- During the fermentation process, much of the toxin escapes as a gas.

Hay- The amount of prussic acid in sorghum hay drops by up to 75%. Allowing hungry cattle to graze in areas where prussic acid is an issue is not a good idea. Stock should never graze sorghum that is less than 50 cm tall.

Tannins

These are phenolic compounds that are water soluble and have a molecular weight larger than 500 and may precipitate proteins from aqueous solution. They can be found in practically all vascular plants. These chemicals are divided into two groups: hydrolysable tannins and condensed tannins. Tannins of both sorts can be found in the leaves of trees and shrubs (Reddy, 2001). The nutritional and harmful impacts of the two varieties differ. Condensed tannins reduce digestibility more effectively than hydrolysable tannins, which may generate a variety of toxic manifestations in the rumen owing to hydrolysis. Tannins may bind to and inhibit endogenous proteins such as digestive enzymes, forming a less digestible combination with ingested proteins (Cheeke, 1995) [23]. All plants have phenolic compounds, but their kind and concentration may cause negative animal reactions. Tannin-protein complexes are astringent and adversely impact feed intake, and all plants contain phenolic compounds, but their type and concentration may cause negative animal responses.

Ruminants have been shown to be poisonous when condensed tannin concentrations above 4% because they are more resistant to microbial assault and are detrimental to a few bacteria. Many methods have been tried to overcome the negative effects of tannins, including alkali treatments with ferrous sulphate, Polyethylene glycol-4000, which prevents the formation of tannic acid-protein complexes and aids in the breakdown of already formed complexes, thus liberating protein (Reddy, 2001), and three months of feeding Prosopis cineraria leaves and *Cenchrus spp*. with per cent urea to adult sheep. Sheep, cattle, and goats are domestic ruminants of considerable economic importance in the Mediterranean region, where extensive livestock production systems are frequently based on the consumption of tannin-rich feed, such as shrub and tree foliage, or even agro-industrial by-products, which provide energy and nutrients during times of pasture scarcity. Aside from their potential as nutritional components, these plants are gaining popularity due to their tannin content, which may have a significant impact on animal performance as well as product quality.

Tannins are thought to have both negative and positive effects, depending on their chemical structure and concentration in meals, the composition of the baseline diet, and other characteristics unique to the animals, such as species and physiological stage (Makkar *et al.*, 2007 [66]; Waghorn, 2008 [103]; Piluzza *et al.*, 2014) [78]. Reduced feed intake, fibre and nitrogen digestibility, and animal performance are all negative impacts (Min *et al.*, 2003 [72]; Makkar *et al.*, 2007 [66]; Waghorn, 2008) [103]. Tannins, on the other hand, may reduce bloating, promote protein consumption during digestion, manage internal parasites, and boost growth performance, wool development, and milk production (Min *et al.*, 2003 [72]; Waghorn, 2008 [103]; Piluzza *et al.*, 2014) [78] Furthermore, tannins are known to have antioxidant properties, and some research suggests that dietary tannins may help animals' antioxidant status (Gladine *et al.*, 2007; López-Andrés *et al.*, 2013) [63].

In recent years, there has been a surge in interest in tannin-rich plants and plant extracts for use in nutritional strategies to improve the quality of ruminant products, notably fatty acid composition and oxidative stability. According to several research, using tannin-rich plants or plant extracts in ruminant diets increases the amounts of health-promoting FA in meat and milk while also improving the oxidative stability of meat. However, the potential negative effects of tannins on animal performance and product organoleptic qualities are a substantial impediment to the practical implementation of this dietary approach.

Tannins adverse effects

Because tannins reduce the intake, digestion, and absorption of proteins, polysaccharides, and minerals, and because they can cause ulcers, irritation, and desquamation of the intestinal mucosa, lesions in the liver and kidneys, and, in extreme cases, death (Kumar and Singh, 1984 [56]; Kumar and Vaithiyanathan, 1990 [57]; The ingestion of HT, which is enzymatically depolymerized in the rumen by cleaving the ester linkages between glucose and phenolic subunits, yielding gallic acid, which is metabolized to pyrogallol and resorcinol, which are absorbed and responsible for cellular damage, is normally associated with the severe damage caused by tannins in ruminants (renal and hepatic lesions and death) (Murdiati *et al.*, 1992 [75]; Spier *et al.*, 1987) [97]. Several incidences of severe poisoning in cattle, sheep, and llamas have been reported following ingestion of high quantities of HT from oak (Quercus spp.) Leaves of yellow-wood (Terminalia oblongata) (Chamorro *et al.*, 2013 [22]; Garg *et al.*, 1992.) [31]. (Filippich and colleagues, 1991) [30]. CT scans, on the other hand, are not degraded or absorbed into the bloodstream, making them unlikely to injure organs (McSweeney *et al.*, 1988 [71]; Terrill *et al.*, 1994 [98]; Makkar, 2003 [65]; McSweeney *et al.*, 1988 [71]; Terrill *et al.*, 1994 [98]; According to Makkar, CT causes organ harm only when there is intestinal irritation and tannins reach the blood (2003). CT's antinutritional effects in animal diets are mostly due to reduced protein and carbohydrate intake and digestibility, as well as digestive enzyme suppression and animal performance

(Yisehak *et al.*, 2014 [109]; Silanikove *et al.*, 1996a [93]; Barry and McNabb, 1999) [10]. Tannins may form complexes with a variety of substances, such as proteins, polysaccharides, and minerals (McSweeney *et al.*., 2001; Min and Hart, 2003 [72]; Le Bourvellec and Renarda, 2012) [60]. Tannins' phenolic hydroxyl groups form complexes predominantly with proteins, but also with polysaccharides, nucleic acids, and metal ions to a lesser extent (Makkar, 2003) [65]. The capacity of tannins to form protein complexes is the most essential feature of their nutritional and toxicological impacts (Hagerman and Butler, 1981).

Depending on the kind of connection generated, such as covalent or non-covalent bonds, the complexes formed (tannins/proteins) might be reversible or irreversible. The complexes are reversible if the connections are non-covalent (hydrogen, hydrophobic, and van der Waals forces). The complexes produced by covalent bonds are generally irreversible (Hagerman, 1989) [38]. The type of interactions between proteins and tannins is influenced by the relative concentrations of both tannins and proteins, tannin structure (molecular weight, conformation, flexibility of tannins, and water solubility), protein structure (size, conformation [secondary or tertiary structure], and amino acid composition), and the nature of the medium in which the interactions occur (solvent, temperature, pH, ionic strength) (Le Bourvellec and Renard).

Although the results of studies on the effect of including tannins in ruminant diets on animal performance, FA profile, oxidative stability, and organoleptic properties of meat and milk are still mixed, many have shown that this nutritional strategy is a promising tool for improving ruminant fat FA profile and oxidative stability. The impact of dietary tannin sources on the FA profile of ruminant products has been studied in both meat and milk; however, to our knowledge, prior research has focused mostly on meat in terms of oxidative stability and organoleptic qualities. As a result, more research into milk and dairy products is required. There is a broad variety of tannins sources and inclusion levels in the research evaluated here, both in the context of supplementation and diet composition, which contributes to the great variability in the outcomes obtained. However, it appears that tannin effects are influenced by a number of parameters, including tannin source and concentration, as well as dietary makeup. As a result, more research is needed to determine the optimal conditions of application for each tannin source, such as supplementation level and basal diet composition, in order to develop nutritional strategies based on the use of tannins to improve product quality without compromising animal health and performance. Furthermore, more research into the underlying processes of tannins' capacity to change the FA profile and increase product oxidative stability is needed.

Mycotoxins

D' Mello and Macdonald (1998) define mycotoxins as secondary metabolites of fungus that have the potential to harm animal health and production. The many symptoms caused by these substances are commonly referred to as "mycotoxicosis," which encompasses both syndrome and non-specific disorders. A list of the most common mycotoxins found in feeds and forages, together with the fungus species responsible for their synthesis. Mycotoxin contamination of forages and cereals occurs commonly in the field as a result of plant infection with certain pathogenic fungi or symbiotic endophytes. When the environmental circumstances are right for rotting fungus, contamination can happen during the processing and storage of harvested goods and feed.

Mycotoxins are molds secondary metabolites that have harmful effects on humans, animals, and crops, resulting in sickness and financial losses. Food and feed have been contaminated by mycotoxins all across the world, which is a severe problem. The most important agro-economic mycotoxins include aflatoxins, ochratoxins, trichothecenes, zearalenone, fumonisins, tremorgenic toxins, and ergot alkaloids. More than one mycotoxin can be generated by some mold, and some mycotoxins are produced by many fungal species. On a contaminated substrate, more than one mycotoxin is frequently discovered. Mycotoxins are more common in locations with a hot, humid environment, which is conducive to mold spores, although they may also be found in temperate zones. Mycotoxins are mostly ingested, although they can also be ingested through the skin or inhaled. In a few words, mycotoxin is tough to characterise. Mycotoxins are low-molecular-weight natural products (small molecules) generated by filamentous fungus as secondary metabolites. These metabolites form a toxicologically and chemically diverse ensemble that has only been grouped together because the members can cause sickness and death in humans and animals. Many mycotoxins have overlapping toxicities to invertebrates, plants, and microbes, which is not surprising (Bennett, 1987) [14]. In the context of an unprecedented veterinary crisis outside London, England, in which about 100,000 turkey poults died, the word mycotoxin was created in 1962. When scientists discovered that the strange turkey X sickness was connected to a peanut (groundnut) meal contaminated with Aspergillus flavus secondary metabolites (aflatoxins), it alerted them to the possibility that additional concealed molds metabolites may be lethal (Bennett and Klich, 2003) [12]. Mycotoxins are harmful substances generated by fungus, although not all hazardous compounds produced by fungi are named mycotoxins. Both the goal and the metabolite concentration are crucial. Antibiotics are fungal compounds that are mostly harmful to bacteria (such as penicillin). Plant pathologists refer to fungal compounds that are hazardous to plants as phytotoxins. Fungi produce mycotoxins, which are hazardous to vertebrates and other animal species at low doses. Other low-molecular-weight fungal metabolites, such as ethanol, are not called mycotoxins since they are dangerous only at large doses (Bennett, 1987) [14].

Mycotoxins are a structurally diverse group of mostly small molecular weight compounds produced primarily by the secondary metabolism of some filamentous fungi, or molds, which can develop on a variety of foods and feeds under the right temperature and humidity conditions, posing serious health risks to humans and animals. Molds are secondary metabolites with no biochemical relevance in fungal growth and development. They range in complexity from basic C4 compounds like moniliformin to complex molecules like the phomopsins (Dinis *et al.*, 2007). More than 300 mycotoxins are known at this time, however scholarly attention is mostly focused on those that have been demonstrated to be carcinogenic and/or poisonous. Human exposure to mycotoxins can occur through the eating of contaminated plant-derived foods, the presence of mycotoxins and their metabolites in animal products such as meat and eggs (CAST, 2003), or exposure to polluted air and dust, (Jarvis, 2002) [42].

The most significant genera of mycotoxigenic fungus include

Aspergillus, Alternaria, Claviceps, Fusarium, Penicillium, and Stachybotrys, and mycotoxins can be found in human food at various levels in the food chain (Bennett and Klich, 2003) [12]. A metabolite of Aspergillus flavus and Aspergillus parasiticus, aflatoxin B1 (AFB1), is the most powerful hepatocarcinogenic chemical known, and it has recently been discovered to be genotoxic. Another issue in dairy cattle is the conversion of AFB1 and AFB2 into hydroxylated metabolites, aflatoxin M1 and M2 (AFM1 and AFM2), which are present in milk and milk products from animals that have eaten contaminated feed (Boudra *et al.,* 2007) [20]. The WHO-International Agency for Research on Cancer (WHO-IARC) evaluated the carcinogenic potential of AF, OT, trichothecenes, ZEN, and F in 1993 (World Health Organization International Agency for Research on Cancer (WHO-IARC), 1993a, World Health Organization International Agency for Research on Cancer (WHO-IARC), 1993b). Naturally occurring AF has been categorised as a human carcinogen (Group 1), whereas OT and F have been classed as potential carcinogens (Group 2B). Trichothecenes and ZEN, on the other hand, were not identified as carcinogenic in humans (Group 3). Mycotoxins' health risks to people and animals have been intensively studied in recent years (Yaling *et al.,* 2008 [108], Averkieva, 2009) [7].

Mycotoxins have harmful impacts on people.

Mycotoxicoses can be classified as acute or chronic, much like any other toxicological disease. Acute toxicity is defined by a quick start and a clear harmful reaction, whereas chronic toxicity is defined by low-dose exposure over a long period of time, culminating in malignancies and other often irreversible consequences (James, 2005) [41]. Fusarium species have been linked to multiple human mycotoxicosis epidemics prior to the discovery and application of current milling procedures. From 1932 to 1947, cereal grains infected with F. sporitrichoides and F. poae were linked to alimentary toxic aleukia in Russia. Mucous membrane hyperaemia, oesophageal discomfort, laryngitis, asphyxiation, gastroenteritis, and vertigo were among the symptoms (Lewis *et al.,* 2005) [61]. Aflatoxicosis is a kind of toxic hepatitis that causes jaundice and, in the most serious instances, death. Kenya (in 1981, 2001, 2004, and 2005), India, and Malaysia have all seen similar instances (Shephard, 2004, Lewis *et al.*, 2005) [89, 61]. The International Agency for Research on Cancer (IARC) designated AFB1 as a human carcinogen (Group 1 carcinogen) after significant research linking it to human primary liver cancer in which it operates synergistically with HBV infection (IARC, 1993). In poorer areas, this combination constitutes a significant cancer burden. A recent comparison of Kenya's anticipated population danger to France's emphasised the increased weight that underdeveloped countries may bear (Shephard, 2006) [90].

Individuals' chronic food consumption is usually the cause of the biggest risk of AF. Human hepatocellular carcinomas have been linked to dietary AF exposures, which may be exacerbated by the hepatitis B virus. Hepatocellular carcinomas are responsible for over 250,000 fatalities each year in China and Sub-Saharan Africa, and are linked to risk factors such as a high daily consumption of AF (1.4 g) and a high prevalence of hepatitis B. (Wild *et al.,* 1992) [105]. Aflatoxins have been found in the tissues of children with Kwashiorkor and Reye's syndrome, and they are thought to have a role in both conditions. Reye's syndrome causes liver and kidney enlargement, as well as cerebral edoema, and is characterised by encephalopathy and visceral degeneration (Blunden *et al.,* 1991) [19]. Aflatoxin has long been related to Kwashiorkor, a condition that is commonly thought to be a form of protein energy deficiency, yet several of the disease's symptoms are recognised to be among the pathological consequences of aflatoxins in animals. In Gambian children, aflatoxin exposure was linked to lower levels of secretory immunoglobulin A (IgA) (Turner *et al.,* 2003) [101]. Aflatoxin exposure has been linked to changes in differential subset distributions and functional abnormalities in particular lymphocyte subsets in Ghanaian adults, suggesting that aflatoxins may impair human cellular immunity, lowering resistance to infections (Jiang *et al.,* 2005) [44].

Infectious infections, such as HIV/AIDS, infectious diarrhoea, and lower respiratory tract infection, are responsible for the morbidity and mortality associated with unsafe sex, unclean water, and indoor smoking, respectively. All of these outcomes might be harmed by the immune suppression caused by aflatoxin and perhaps DON. The modifying impact of aflatoxins on human health in situations of zinc, iron, and vitamin A deficiencies is less known, although animal nutrition research suggests it might be important (Williams *et al.,* 2004) [106]. Fumonisins were linked to borborygmy, abdominal pain, and diarrhoea in one case of acute food poisoning in India, where the ingestion of fumonisin-contaminated maize and sorghum was linked to the incidence of borborygmy, abdominal pain, and diarrhoea. The IARC categorised fumonisin B1, the most prevalent of the several fumonisin analogues, as a Group 2B carcinogen (probably harmful in humans) (IARC, 2002). Fumonisins, which block folic acid absorption through the folate receptor, have also been linked to a high rate of neural tube abnormalities in rural regions where contaminated maize was consumed, such as the former Transkei region of South Africa and parts of Northern China (Marasas *et al.,* 2004) [68].

Mycotoxins have negative impacts on non-ruminants.

Early research on the effects of acute aflatoxicosis revealed a wide range of toxicities in numerous life forms. Variable reactions to all mycotoxins have been seen in monogastrics. Pigs have been demonstrated to be extremely susceptible to T-2 toxin, DON, and ZEN, for example. Both T-2 and DON have negative effects on poultry, although ZEN has a very low oestrogenic impact on them (Cheeke, 1998a). Because of the vast range of feed components utilised and the variances between and within groups, various degrees of mycotoxicoses from natural sources exist in different animals. The following sections detail experiments and case studies on mycotoxicoses in non-ruminant animals (Hussein and Brasel, 2001) [40].

1. Poultry

Numerous studies have shown that mycotoxins have a deleterious impact on chicken performance. For example, giving broilers a high dose (3.5 mg/kg of feed) of an AF combination (i.e. 79 percent AFB1, 16 percent AFG1, 4 percent AFB2, and 1 percent AFG2) lowered their body weight while increasing the weights of their liver and kidneys (Smith *et al.,* 1992) [95]. Aflatoxins also boosted blood urea-N levels while lowering total protein, albumin, triglycerides, and phosphorus levels in the blood. When broilers were fed OTA (0.3–1 mg/kg of feed), glycogenolysis was inhibited, and glycogen buildup in the liver was dose-dependent. The suppression of cyclic adenosine 3′,5′-monophosphate-dependent protein kinase was blamed for the poor metabolic

responses, which included lower feed consumption efficiency and teratogenic abnormalities (Bitay *et al.*, 1979). Other enzymes (such as alkaline phosphatase, acid phosphatase, lactate dehydrogenase, and succinate dehydrogenase) in many organs (such as the heart, liver, spleen, and pancreas) of 1-week-old chicks were similarly affected by swallowing Fusarium roseum-contaminated feed. Metabolic and cellular respiration issues, decreased body weight growth, and tissue necrosis occurred from this reduction in enzyme activity (Beri *et al.*, 1991) [15]. Mycotoxins produced by Fusarium have been demonstrated to harm chickens. When 7-day-old chicks were administered T-2 toxin (4 or 16 mg/kg of feed) or DAS (4 or 16 mg/kg of feed), buccal-oral ulceration and plaque development were seen, in addition to lower feed intake and body added weight. T-2 toxin at 6 mg/kg of feed was shown to have similar effects in 1-day–3-week-old chicks, as was DAS at 20 mg/kg of feed in 24–25-week-old chickens. When DAS was provided at 5 mg/kg of feed to hens (67–69 weeks old) and 10 mg/kg of Fertility was increased in hens (67–69 weeks old) but decreased in roosters (25–27 weeks old) when roosters (25–27 weeks old) were fed (Brake *et al.*, 2000) [21].

2. Pigs

Swine are one of the most vulnerable animals to mycotoxins. The immunological response of pigs to AF has been inconclusive. Feeding mixed AF at doses ranging from 0.4 to 0.8 mg/kg of feed to acutely lethal levels as high as 500 mg/kg of feed had no effect on the swine humoral immune response. The inhibition of DNA synthesis in pig lymphocytes when AFB1 was added to the medium at various doses (0.1–10 000 ng/ml of medium) and the immunosuppression produced by AF (140 or 280 g/kg of feed) occurred solely at the cellular level and not at the humoral level (Pang and Pan, 1994). The mycotoxin ZEN has been shown to have negative effects on pig reproductive function (Diekman and Green, 1992) [29]. The poisonous forms of ZEN have been found to be drawn back from the circulating glucuronide conjugate in pigs. As a result, ZEN's oestrogenic effects in pigs have been strong and long-lasting. In sexually mature gilts fed ZEN-contaminated feed, enlargement of the vulva and mammary glands,On a big study in Hungarian farms, as well as occasional vaginal and rectal prolapses, were observed (Glavitis and Vanyi, 1995) [37]. Edematous uterus, ovarian cysts, increased follicular maturation and frequency of stillborns, and lower other oestrogenic effects of ZEN on gilts or sows included fertility rate. In the same study, ZEN produced germinal epithelial degeneration and reduced sperm production in boars. Reproductive problems (such as ovarian and uterine shrinkage, ovarian degeneration, and endometrial glandular malfunction) have also been recorded in sows fed T-2 toxin-contaminated diet. Suckling piglets showed signs of prenatal T-2 toxicosis (endometrial glandular malfunction, gastrointestinal edoema, and hematopoesis leading to mortality) (Hussein and Brasel, 2001) [40].

3. Horses

Asquith has studied the history of horse myctoxicosis and poisoning (1991). Mature horses fed AFB1-contaminated feed (58.4 g/kg) were jaundiced and anorexic before dying in a case study. Examinations after death indicated enlarged livers, renal damage, and bile-duct hyperplasia lesions. Equine aflatoxicosis has been associated with sadness, lameness, and mortality in certain cases. Subcutaneous and enteric bleeding, enlarged kidneys, enlarged necrotic livers, and hepatic, nephritic, and cardiac abnormalities were discovered during post-mortem exams. When acute fatal dosages of AFB1 were given to ponies, damage to the skeletal muscles and heart, as well as liver failure, was seen. Horses feeding maize contaminated with a combination of AF (AFB1, AFB2, and AFM1 at 114, 10, and 6 g/kg, respectively) had significant liver lesions, according to post-mortem testing (Vesonder *et al.*, 1991) [102].

Toxins generated by Fusarium moniliforme, which have been linked to equine leukoencephalomalacia and acute neurotoxicity, have been recognised as the largest mycotoxin danger to horses thus far. The eating of corn infected with FB1 and moniliformin toxins was blamed for the disorders. Ataxia, paresis, apathy hypersensitivity, reduced locomotor performance, necrosis of cerebral white matter, and abnormalities in the cerebral cortex are all symptoms of horse leukoencephalomalacia. Bean-hull poisoning is another mycotoxin-related ailment that has been recognised in Hokkaido (Japan) for seven decades due to the cheapness of bean-hulls as a feed and bedding for horses (Asquith, 1991) [6]. Central nervous system dysfunction, fast heartbeat, reduced ocular reflexes, and mortality are some of the clinical signs (Placinta *et al.*, 1999) [79].

4. Dogs and cats

Mycotoxins have serious consequences for companion animals and can even result in death. A instance of canine hepatitis was connected to the ingestion of mouldy food as early as 1952. The agent responsible for the 1952 case was identified as AFB1 after the discovery of AF, and the symptoms of aflatoxicoses in dogs were clarified. Following the intake of commercial dog food contaminated with AF, three dogs on a farm in Queensland grew unwell (severe depression, anorexia, and frailty) and died at separate periods within a months. High quantities of AF were found in the vomitus of one dog (100 g/g of AFB1 and 40 g/g of AFG1) (Devegowda and Castaldo, 2000) [28]. Deoxynivalenol is a severe health risk for companion animals, and even after processing, it contaminates petfood via maize. It was proposed that DON levels in petfood should not exceed 0.5 g/kg due to the varied toxicity reactions to DON in dogs and cats. T-2 toxin, administered intravenously at 2 mg/kg to cats, caused hypovolemia and mortality in a case study. White blood cell counts in cats have been demonstrated to be reduced by sub-lethal T-2 poisoning (Devegowda and Castaldo, 2000) [28].

Plant toxins

Many plant components have the potential to have negative impacts on farm cattle production (D' Mello, 2000). Almost every plant utilized in practical feeding has these chemicals in its leaf and/or seeds. Toxin concentrations in their most common forms. Heat-labile plant toxins, such as lectins, proteinase inhibitors, and Cyanogens, are sensitive to standard processing temperatures; heat-stable plant toxins, on the other hand, include antigenic proteins, condensed tannins, quinolizidine alkaloids, glucosinolates, gossypol, saponins, the non-protein amino acids S-methyl cysteine sulphoxide and mimosine, and phytoestrogens. D'Mello (2000) goes into detail about the role of these drugs as antinutritional factors, but the key facts are worth repeating. Toxins from hazardous range plants have devastating effects on the embryo, fetus, and newborn, and the consequences are costly to the livestock

sector. The pervasive nature of poisonous plants, as well as cattle exposure on rangelands and pastures, are major factors in the significance of toxicoses from poisonous plants. Thousands of plant species are known to be poisonous around the world (Kingsbury 1964, Everist 1974) [55] many plants have been implicated in the cause of fetal mortality, abortion, or teratogenesis in livestock over the last 40 years, according to research. Because of the detrimental impact on the sale of breeding stock or because the mortality of children was not associated with any abnormality such as cleft palate or cardiac defects, these malformations were often undetected and/or unreported in the past. Poisonous plants, on the other hand, have been shown to cause many cattle losses owing to embryonic mortality, abortion, and teratogenesis, according to experimental research. Grazing management solutions based on 40 years of research and including certain basic toxicological and teratology concepts have decreased livestock producer losses. Wilson (1977) [107] first introduced these basic notions, which Keeler then expanded on (1978) (1) Plant toxicity and teratogenicity are affected by changes in livestock species and breeds; (2) the deformities generated are dependent on the amount of plant maternally consumed and the following dose of putative compound delivered to the embryo; (3) The concentration of teratogenic chemicals is affected by plant growth stages; (4) the embryo or foetus must be exposed to the teratogen during a vulnerable period of gestation. Each of these elements, as well as others, have a part in grazing management measures that aim to prevent losses due to noxious plant-related birth abnormalities. Plants that affect embryonic, foetal, and neonatal development, susceptible livestock species, high-risk plant growth stages, and gestational times of sensitivity will all be examined in this review. Veratrum species are included. The genera Lupinus, Conium, Nicotiana, Astragalus, and Oxytropis are among the most common. The fetotoxic effects of these plants are summarized in Table 1

Veratrum

False hellebore (Veratrum californicum Durand) is a common mountain range plant that grows in damp locations and is responsible for various congenital birth abnormalities in sheep (Keeler 1984a) [53]. The teratogenic substance is cyclopamine, a steroidal alkaloid. Congenital cyclopia and similar craniofacial deformities, also known as "monkey faced lamb illness," are among the most well-known anomalies. The facial deformities are caused by neural tube defects that are caused when pregnant ewes consume the plant on day 14 of pregnancy (Binns *et al.* 1965) [17] Embryonic mortality is common during this early stage of pregnancy (14th day), as well as the 19th and 21st days (Keeler 1990) [54]. The cyclopic deficiency has been experimentally produced in calves and goats when their dams were exposed on the 14th day of gestation (Binns *et al.* 1972) [16].

Lupinus, Conium, and Nicotiana

Lupinus species are plants that belong to the *Lupinus* genus. Conium and Nicotiana both cause acute neurotoxic and fetotoxic effects, as well as teratogenesis. Conium. Piperidine alkaloids are found in Nicotiana and some *Lupinus* species, but quinolizidine alkaloids are found in the majority of *Lupinus* species. *Lupinus formosus* Greene (lunara lupine) (Keeler and Panter 1989) [51], *Lupinus caudarus* Kelb (tail cup lupine) (Shupe *et al.* 1967) [91], *Lupinus sericeus* Pursh. (Silky lupine) (Shupe and al. 1967) [91], and *Conium maculaturn* L. (poison-hemlock) (Shupe *et al.* 1967 (Keeler 1974) [91], Burley tobacco *Nicotiana rabacum* L. (Crowe 1969) and Nicotianaglauca Graham (wild tree tobacco) (Keeler 1979) [52] have all caused birth abnormalities in cattle species. Multiple congenital contractures (MCC), such as over- or under-extension or flexure of the joints, joint rigidity, and immobility, are examples of birth abnormalities. Laterally rotated, bent, or severely flexed front limbs are possible (arthrogryposis). Curvature of the spine (scoliosis), neck twisting (torticollis), rib cage anomalies, spinal depression (kyphosis), cranial dissymmetry, and cleft palate are frequently seen.

Table 1: Poisonous plants; the livestock species affected; the stage of gestation when fetus is most susceptible; and the type of birth defects associated with their ingestion

Plant	Animal species	Stage of gestation	Birth defect
Veratrum californicum Durand (False hellebore)	Sheep	14th day	Embryonic death, craniofacial defects [i.e., congenital cyclopic (monkey-faced) lamb].
		19-21 days	Embryonic death
		27-32 days	Limb defects [i.e., shortening of metacarpus, metatarsus and tibia].
		31-33 days	Tracheal defects; tracheal collapse, suffocation and death.
Conium maculatum L. (Poison-hemlock)	Cow	55-75 days	Limb, spine and neck malformations [i.e., arthrogryposis, scoliosis and torticollis].
	Sow	30-45 days	Cleft palate.
		43-62 days	Skeletal defects similar to the cow.
	Sheep	30-60 days	Skeletal defects similar to the cow.
	Goat	30-60 days	Skeletal defects similar to the cow and cleft palate.
Lupinus caudatus Kell. And sericeus Pursh. (Tail cup lupine and silky lupine)	Cow	55-75 days	Skeletal malformations [i.e., arthrogryposis, scoliosis, torticollis, multiple contractures].
L. formosus Greene (Lunara lupine)	Cow	40-70 days	Cleft palate and skeletal defects similar to L. caudatus-induced.
Nicotiana glauca Graham (tree tobacco)	Cow	40-75 days	Skeletal defects similar to Conium and Lupinus-induced.
	Sow	18-68 days	Cleft palate and skeletal defects similar to those induced in the cow by Conium and Lupinus.
	Sheep	30-60 days	Cleft palate and skeletal defects similar to the sow.
	Goat	30-60 days	Cleft palate and skeletal defects similar to the sow.
Astragalus and *Oxytropsis* spp.	Cow	All stages	Fetal death, absortion.
	Sheep	All stages	Fetal death, abortion and contraction limb defects.

Saponins

Saponin is derived from the Latin letters'sapo,' which meaning soap, and saponin-containing plants have traditionally been used for washing. Saponins are surface-active glycosides that exist naturally. Plants (Alfalfa, Soyabean, Lucerne, Berseem, Yucca, Mahua, Guar, and others) but also lower marine creatures (sea cucumbers, starfish, and others) and some rhizo bacteria generate them (Yoshiki *et al.*, 1998) [110]. Saponins are a broad group of amphiphilic steroid and triterpene glycosides found in plants and certain marine creatures. Saponins display a wide range of biological and pharmacological activities by expressing a huge diversity of structures on both sugar chains and aglycones, and serve as significant active ingredients in folk remedies, particularly in traditional Chinese medicine. Due to the microheterogeneity of saponins in nature, isolating saponins from natural sources is typically a difficult operation. Chemical synthesis can yield huge quantities of natural saponins and congeners, allowing researchers to better understand their structure–activity connections and mechanisms of action. Saponins, bitter glycosides with a peculiar foamy feature found in oats and spinach that boost and expedite the body's ability to absorb calcium and silicon, are bitter and diminish the palatability of cattle feeds. Saponins aren't a concern in tropical pasture legumes in general. They are, nonetheless, prevalent in several temperate forage legumes.

Biological role in animal systems

Consequences on cell membranes: Saponins' action on membranes has been attributed to a broad number of biological effects. Their propensity to generate pores in membranes has actually contributed to their widespread usage in physiological studies. Saponins have been known for a long time to have a lytic effect on erythrocyte membranes, and this property has been utilized to identify them. Saponins' hemolytic activity is thought to be due to the aglycone moiety's affinity for membrane sterols, particularly cholesterol, with which they form insoluble complexes**.** Saponin-induced lesions are hypothesized to be caused by a micelle-like aggregation of saponins and cholesterol in the membrane's plane, potentially with saponin molecules structured in a ring with their hydrophobic moieties coupled with cholesterol around the outer perimeter. Saponins created membrane pores or flaws that lasted for a long time, allowing big molecules like ferritin to pass through (Seeman, 1974) [88]. In addition, soyasaponins I and II, as well as dehydrosoyasaponin I, have been demonstrated to activate large Ca-dependent K conductance channels, resulting in membrane hyperpolarization, electrical activity suppression, and smooth muscle relaxation (McManus *et al.*, 1993) [70].

Biological effects of dietary saponins on nutrient uptake through the intestinal membrane: Because dietary saponins are inadequately absorbed, their biological effects occur in the digestive system (Cheeke, 1996) [24]. Some saponins increase the permeability of intestinal mucosal cells *in vitro*, impede active mucosal transport, and enable the uptake of drugs that are ordinarily not absorbed, according to Johnson *et al.* (1986) [45]. Saponins (from Gypsophila, Quillaja, clover, guar, and lucerne) lower the transmural potential difference (TPD, an electrochemical gradient that functions as a driving force for active nutrient transport across the brush border membrane of the gut) in the rat's small intestine (Gee *et al.*, 1989) [32]. An increase in the apparent permeability of the brush boundary reported at sublethal levels of saponins could have substantial ramifications for the uptake of macromolecules like allergens, which are generally restricted in their transit through the epithelium (Gee *et al.*, 1996) [34]. In brown Norway rats, the presence of Gypsophila saponins increased the absorption of B lactoglobulin, a milk allergy, in the jejunal loops' tips (Gee *et al.*, 1997) [33]. According to certain sources, the absorption is obstructed. Dietary saponins play a role in the absorption of micronutrients Gypsophila, for example. Dietary saponins reduced mean liver iron levels (Fe) by impairing Fe concentrations and total liver Feabsorbent (Southon *et al.*, 1988) [96]. Saponin is most likely to blame. compounds with dietary Fe, preventing it from being absorbed for the purpose of absorption Saponins from lucerne have been demonstrated to when there is an increase in Fe and magnesium (Mg) excretion present in rat diets, and lower calcium levels in the blood (Ca)Zinc (Zn) and copper (Cu) in pigs (Southon *et al.*, 1988) [96]. These *In vitro*, saponins could form complexes with Fe and Zn, which could have hampered their effectiveness. Absorption. Gypsophila saponins are triterpenoid saponins. Quillaja, which was included in the diet, seems to cause problems. Vitamin A and vitamin E absorption in chicks Jenkins and Atwal (Jenkins and Atwal, 1994) [43].

References

1. Abu-Zanat MM, Al-Hassanat FM, Alawi M, Ruyle GB. Mineral assessment in *Atriplex halimus* L. and *Atriplex nummularia* L. in the arid region of Jordan. African Journal of Range and Forage Science. 2003;20(3):247-251.
2. Allison CD, Baker RD. Prussic acid poisoning in livestock [Guide B-808]. Las Cruces: New Mexico State University Cooperative Extension Service. 2002.
3. Allison MJ, Littledike ET, James LF. Changes in ruminal oxalate degradation rates associated with adaptation to oxalate ingestion. Journal of Animal Science. 1977;45(5):1173-1179.
4. Al-Sultan SI. Sorghum halepenses and its cyanide content. Pak. J. Nutr. 2003;2(3):123-124.
5. Andrews DJ, Kumar KA. Pearl millet for food, feed, and forage. Advances in agronomy. 1992;48:89-139.
6. Asquith RL. Mycotoxicoses in horses. Mycotoxins and Animal Foods. CRC Press Inc., Boca Raton, USA, 1991, 679-688.
7. Averkieva O. Mycotoxins in grains harvested, 2009.
8. Ball DM, Collins M, Lacefield GD, Martin NP, Mertens DA, Olson KE, *et al.* Understanding forage quality. American Farm Bureau Federation Publication, 2001, 1(01).
9. Barnes RF, Gustine DL. Allelochemistry and forage crops. Anü-Quality Components of Forages antiqualitycomp, 1973, 1-13.
10. Barry TN, McNabb WC. The implications of condensed tannins on the nutritive value of temperate forages fed to ruminants. British Journal of Nutrition. 1999;81(4):263-272.
11. Benjamin DN. Effects of fertilizer application and cutting interval on nitrate accumulation in bermudagrass. M. Sc. (Agri.) (Doctoral dissertation, Thesis, University Tennessee, Martin, TN), 2006.
12. Bennett JW, Klich M. Mycotoxins. Clinical microbiology reviews. 2003;16(3):497-516.
13. Bennett JW, Klich M. Mycotoxins. Clinical microbiology reviews. 2003;16(3):497-516.
14. Bennett JW. Mycotoxins, mycotoxicoses,

mycotoxicology and Mycopathologia. Mycopathologia. 1987;100(1):3-5.
15. Beri HK, Vadehra DV, Gupta JK. Proportionate incidence of mycotoxic fungi: Fusarium and its effect on ingestion by poultry. Journal of food science and technology (Mysore). 1991;28(5):329-331.
16. Binns W, Keeler RF, Balls LD. Congenital Deformities in Lambs, Calves, and Goats Resulting from Maternal Ingestion of *Veratrum californicum*; Hare Lip, Cleft Palate, Ataxia, and Hypoplasia of Metacarpal and Metatarsal Bones. Clinical toxicology. 1972;5(2):245-261.
17. Binns W, Shupe JL, Keeler R, James LF. Chronologic evaluation of teratogenicity in sheep fed *Veratrum californicum*. Journal of the American Veterinary Medical Association. 1965;147(8):839-842.
18. Blaney BJ, Gartner RJW, Head TA. The effects of oxalate in tropical grasses on calcium, phosphorus and magnesium availability to cattle. The Journal of Agricultural Science. 1982;99(3):533-539.
19. Blunden G, Roch OG, Rogers DJ, Coker RD, Bradburn N, John AE. Mycotoxins in food. Medical Laboratory Sciences. 1991;48(4):271-282.
20. Boudra H, Barnouin J, Dragacci S, Morgavi DP. Aflatoxin M1 and ochratoxin an in raw bulk milk from French dairy herds. Journal of dairy science. 2007;90(7):3197-3201.
21. Brake J, Hamilton PB, Kittrell RS. Effects of the trichothecene mycotoxin diacetoxyscirpenol on feed consumption, body weight, and oral lesions of broiler breeders. Poultry science. 2000;79(6):856-863.
22. Chamorro MF, Passler T, Joiner K, Poppenga RH, Bayne J, Walz PH. Acute renal failure in 2 adult llamas after exposure to Oak trees (*Quercus* spp.). The Canadian Veterinary Journal. 2013;54(1):61.
23. Cheeke PR. Endogenous toxins and mycotoxins in forage grasses and their effects on livestock. Journal of animal science. 1995;73(3):909-918.
24. Cheeke PR. Biological effects of feed and forage saponins and their impacts on animal production. Saponins used in Food and Agriculture, 1996, 377-385.
25. Chemistry and Significance of Condensed Tannins.
26. Hemingway RW, Crowe MW. Skeletal anomalies in pigs associated with tobacco. Modern veterinary practice. 1969.
27. Davis AM. The oxalate, tannin, crude fiber, and crude protein composition of young plants of some *Atriplex* species. Rangeland Ecology & Management/Journal of Range Management Archives. 1981;34(4):329-331.
28. Devegowda G, Castaldo D. Mycotoxins: Hidden killers in pet foods. Is there a solution. In Technical Symposium on Mycotoxins. Alltech, Inc Nicholasville, KY, 2000.
29. Diekman MA, Green ML. Mycotoxins and reproduction in domestic livestock. Journal of animal science. 1992;70(5):1615-1627.
30. Filippich LJ, Zhu J, Oelrichs P, Alsalami MT, Doig AJ, Cao GR, *et al*. Hepatotoxic and nephrotoxic principles in *Terminalia oblongata*. Research in Veterinary Science, 1991, 50(2),
31. Garg SK, Makkar HP, Nagal KB, Sharma SK, Wadhwa DR, Singh B. Oak (*Quercus incana*) leaf poisoning in cattle. Veterinary and human toxicology. 1992;34(2):161-164.
32. Gee JM, Price KR, Ridout CL, Johnson IT, Fenwick GR. Effects of some purified saponins on transmural potential difference in mammalian small intestine. Toxicology *in vitro*. 1989;3(2):85-90.
33. Gee JM, Wal JM, Miller K, Atkinson H, Grigoriadou F, Wijnands MVW, *et al*. Effect of saponin on the transmucosal passage of β-lactoglobulin across the proximal small intestine of normal and β-lactoglobulin-sensitised rats. Toxicology. 1997;117(2-3):219-228.
34. Gee JM, Wortley GM, Johnson IT, Price KR, Rutten AAJJL, Houben GF, *et al*. Effects of saponins and glycoalkaloids on the permeability and viability of mammalian intestinal cells and on the integrity of tissue preparations *in vitro*. Toxicology *in vitro*. 1996;10(2):117-128.
35. Gladine C, Morand C, Rock E, Gruffat D, Bauchart D, Durand D. The antioxidative effect of plant extracts rich in polyphenols differs between liver and muscle tissues in rats fed n-3 PUFA rich diets. Animal Feed Science and Technology. 2007;139(3-4):257-272.
36. Gladine C, Rock E, Morand C, Bauchart D, Durand D. Bioavailability and antioxidant capacity of plant extracts rich in polyphenols, given as a single acute dose, in sheep made highly susceptible to lipoperoxidation. British Journal of Nutrition. 2007;98(4):691-701.
37. Glávits R, Vanyi A. More important mycotoxicoses in pigs. Comprehensive clinico-pathological communication. Magyar Állatorvosok Lapja. 1995;50(7):407-407.
38. Hagerman AE. Chemistry of tannin-protein complexation, In. 1989.
39. Hagerman AE, Butler LG. Tannins and lignins, In: A., R. G., R., B., 1991.
40. Hussein HS, Brasel JM. Toxicity, metabolism, and impact of mycotoxins on humans and animals. Toxicology. 2001;167(2):101-134.
41. James B. Public Awareness of Aflatoxin and Food Quality Control in Benin. International Institute of Tropical Agriculture, 2005, 1-26.
42. Jarvis BB. Chemistry and toxicology of molds isolated from water-damaged buildings. Mycotoxins and Food Safety, 2002, 43-52.
43. Jenkins KJ, Atwal AS. Effects of dietary saponins on fecal bile acids and neutral sterols, and availability of vitamins A and E in the chick. The Journal of Nutritional Biochemistry. 1994;5(3):134-137.
44. Jiang YI, Jolly PE, Ellis WO, Wang JS, Phillips TD, Williams JH. Aflatoxin B1 albumin adduct levels and cellular immune status in Ghanaians. International immunology. 2005;17(6):807-814.
45. Johnson IT, Gee JM, Price K, Curl C, Fenwick GR. Influence of saponins on gut permeability and active nutrient transport *in vitro*. The Journal of nutrition. 1986;116(11):2270-2277.
46. Jones RJ, Ford CW. Some factors affecting the oxalate content of the tropical grass *Setaria sphacelata*. Australian Journal of Experimental Agriculture. 1972;12(57):400-406.
47. Karchesy JJ. (Eds.). Springer US, New York, 323-333.
48. Kaur G, Mandhania S, Chaudhary DP. Calcium oxalate in Napier Bajra Hybrid: trends. Indian Journal of Animal Nutrition. 2012;29(1):58-62.
49. Kaur R, Goyal M, Tiwana US. Influence of seasonal variation on oxalate accumulation in Napier Bajra hybrid under different nitrogen nutrition. Range Management

and Agroforestry. 2016;37(1):62-68.
50. Keeler RF, Balls LD. Teratogenic effects in cattle of *Conium maculatum* and conium alkaloids and analogs. Clinical toxicology. 1978;12(1):49-64.
51. Keeler RF, Panter KE. Piperidine alkaloid composition and relation to crooked calf disease-inducing potential of *Lupinus formosus*. Teratology. 1989;40(5):423-432.
52. Keeler RF. Congenital defects in calves from maternal ingestion of *Nicotiana glauca* of high anabasine content. Clinical toxicology. 1979;15(4):417-426.
53. Keeler RF. Mammalian teratogenicity of steroidal alkaloids. Biochemistry and Function of Isopentenoids in Plants (WD Ness, G. Fuller, and L. S. Tsai, eds). New York: Dekker Inc, 1984, 531-562pp.
54. Keeler RF. Early embryonic death in lambs induced by Veratrum californicum. The Cornell Veterinarian. 1990;80(2):203-207.
55. Kingsbury JM. Poisonous plants of the United States and Canada. Soil Science. 1964;98(5):349.
56. Kumar R, Singh M. Tannins: their adverse role in ruminant nutrition. Journal of agricultural and food chemistry. 1984;32(3):447-453.
57. Kumar R, Vaithiyanathan S. Occurrence, nutritional significance and effect on animal productivity of tannins in tree leaves. Animal feed science and technology. 1990;30(1-2):21-38.
58. Kyriazakis I, Papachristou TG, Duncan AJ, Gordon IJ. Mild conditioned food aversions developed by sheep towards flavors associated with plant secondary compounds. Journal of Chemical Ecology. 1997;23(3):727-746.
59. Launchbaugh KL, Provenza FD, Pfister JA. Herbivore response to anti-quality factors in forages. Rangeland Ecology & Management/Journal of Range Management Archives. 2001;54(4):431-440.
60. Le Bourvellec C, Renard CMGC. Interactions between polyphenols and macromolecules: quantification methods and mechanisms. Critical reviews in food science and nutrition. 2012;52(3):213-248.
61. Lewis L, Onsongo M, Njapau H, Schurz-Rogers H, Luber G, Kieszak S, *et al.* Aflatoxin contamination of commercial maize products during an outbreak of acute aflatoxicosis in eastern and central Kenya. Environmental health perspectives. 2005;113(12):1763-1767.
62. Lincoln SD, Black B. Halogeton poisoning in range cattle. Journal of the American Veterinary Medical Association. 1980;176(8):717-718.
63. López-Andrés P, Luciano G, Vasta V, Gibson TM, Biondi L, Priolo A, Mueller-Harvey I. Dietary quebracho tannins are not absorbed, but increase the antioxidant capacity of liver and plasma in sheep. British Journal of Nutrition. 2013;110(4):632-639.
64. M. (Eds.), Herbivores: their interactions with secondary plant metabolites.
65. Makkar HPS. Effects and fate of tannins in ruminant animals, adaptation to tannins, and strategies to overcome detrimental effects of feeding tannin-rich feeds. Small ruminant research. 2003;49(3):241-256.
66. Makkar HPS, Francis G, Becker K. Bioactivity of phytochemicals in some lesser-known plants and their effects and potential applications in livestock and aquaculture production systems. Animal. 2007;1(9):1371-1391.
67. Marais JP. Effect of nitrogen on the oxalate and calcium content of kikuyu grass (*Pennisetum clandestinum* Hochst). Journal of the Grassland Society of Southern Africa. 1990;7(2):106-110.
68. Marasas WF, Riley RT, Hendricks KA, Stevens VL, Sadler TW, Gelineau-van Waes J, *et al.* Fumonisins disrupt sphingolipid metabolism, folate transport, and neural tube development in embryo culture and *in vivo*: A potential risk factor for human neural tube defects among populations consuming fumonisin-contaminated maize. The Journal of nutrition. 2004;134(4):711-716.
69. McKenzie RA, Bell AM, Storie GJ, Keenan FJ, Cornack KM, Grant SG. Acute oxalate poisoning of sheep by buffel grass (*Cenchrus ciliaris*). Australian Veterinary Journal. 1988;65(26):1751-0813.
70. McManus OB, Harris GH, Giangiacomo KM, Feigenbaum P, Reuben JP, Addy ME, *et al.* An activator of calcium-dependent potassium channels isolated from a medicinal herb. Biochemistry. 1993;32(24):6128-6133.
71. McSweeney CS, Kennedy PM, John A. Effect of ingestion of hydrolysable tannins in Terminalia oblongata on digestion in sheep fed Stylosanthes hamata. Australian Journal of Agricultural Research. 1988;39(2):235-244.170-177.
72. Min BR, Hart SP. Tannins for suppression of internal parasites. Journal of Animal Science. 2003;81(14-2):E102-E109.
73. Min BR, Hart SP. Tannins for suppression of internal parasites. Journal of Animal Science. 2003;81(14-2):E102-E109.
74. Min BR, Barry TN, Attwood GT, McNabb WC. The effect of condensed tannins on the nutrition and health of ruminants fed fresh temperate forages: A review. Animal feed science and technology. 2003;106(1-4):3-19.
75. Murdiati TB, McSweeney CS, Lowry JB. Metabolism in sheep of gallic acid, tannic acid and hydrolysable tannin from Terminalia oblongata. Australian Journal of Agricultural Research. 1992;43(6):1307-1319.
76. Panda N, Sahu BK. Effect of dietary levels of oxalic acid on calcium and phosphorus assimilation in crossbred bulls. Indian Journal of Animal Nutrition. 2002;19(3):215-220.
77. Panter KE, Keeler RF, James LF, Bunch TD. Impact of plant toxins on fetal and neonatal development: a review. Rangeland Ecology & Management/Journal of Range Management Archives. 1992;45(1):52-57.
78. Piluzza G, Sulas L, Bullitta S. Tannins in forage plants and their role in animal husbandry and environmental sustainability: A review. Grass and Forage Science. 2014;69(1):32-48.
79. Placinta CM, D'Mello JF, Macdonald AMC. A review of worldwide contamination of cereal grains and animal feed with Fusarium mycotoxins. Animal feed science and technology. 1999;78(1-2):21-37.
80. Rahman MM, Kawamura O. Oxalate accumulation in forage plants: some agronomic, climatic and genetic aspects. Asian-Australasian Journal of Animal Sciences. 2011;24(3):439-448.
81. Rahman MM, Ishii Y, Niimi M, Kawamura O. Effect of clipping interval and nitrogen fertilisation on oxalate content in pot-grown napier grass (*Pennisetum purpureum*). TG: Tropical Grasslands. 2009;43(2):73.
82. Rahman MM, Ishii Y, Niimi M, Kawamura O. Interactive effects of nitrogen and potassium fertilization on oxalate content in napiergrass (*Pennisetum*

purpureum). Asian-Australasian Journal of Animal Sciences. 2010;23(6):719-723.
83. Rahman MM, Niimi M, Ishii Y, Kawamura O. Effects of season, variety and botanical fractions on oxalate content of napiergrass (*Pennisetum purpureum* Schumach). Grassland Science. 2006;52(4):161-166.
84. Rahman MM, Niimi M, Ishii Y, Kawamura O. Effects of season, variety and botanical fractions on oxalate content of napiergrass (*Pennisetum purpureum* Schumach). Grassland Science. 2006;52(4):161-166.
85. Rahman MM, Tateyama M, Niimi M, Abdullah RB, Khadijah WE, Kawamura O. Changes in oxalate and some mineral concentrations of *Setaria sphacelata* under cutting and uncutting conditions. Pakistan journal of biological sciences: PJBS. 2014;17(4):586-589.
86. Rahman MM, Yamamoto M, Niimi M, Kawamura O. Effect of nitrogen fertilization on oxalate content in Rhodesgrass, Guineagrass and Sudangrass. Asian-Australasian Journal of Animal Sciences. 2008;21(2):214-219.
87. Seawright AA, Groenendyk S, Silva King. An outbreak of oxalate poisoning in cattle grazing *Setaria sphacelata*. Australian Veterinary Journal. 1970;46:293-296.
88. Seeman P. Ultrastructure of membrane lesions in immune lysis, osmotic lysis and drug-induced lysis, 1974.
89. Shephard GS. January. Mycotoxins worldwide: current issues in Africa. In Meeting the mycotoxin menace. Netherlands: Wageningen Academic. 2004;56(4):88-97.
90. Shephard GS. Mycotoxins in the context of food risks and nutrition issues. The mycotoxin factbook: Food and feed topics, 2006, 21-36.
91. Shupe JL, Binns W, James LF, Keeler RF. Lupine, a cause of crooked calf disease. Journal of the American Veterinary Medical Association. 1967;151:198-203.
92. Sidhu PK, Lamba JS, Kumbhar G, Sekhon GS, Verma S, Gupta MP. Role of epidemiological factors in accumulation of oxalates in forage crops. Am. Int. J. Res. Formal. Appl. Nat. Sci. 2014;7(1):48-52.
93. Silanikove N, Gilboa N, Nir I, Perevolotsky A, Nitsan Z. Effect of a daily supplementation of polyethylene glycol on intake and digestion of tannin-containing leaves (*Quercus calliprinos*, *Pistacia lentiscus*, and *Ceratonia siliqua*) by goats. Journal of Agricultural and Food Chemistry. 1996;44(1):199-205.
94. Singh A. A note on seasonal variations in oxalate content of napier bajra hybrid. Indian Journal of Animal Nutrition. 2002;19(3):282-284.
95. Smith EE, Kubena LF, Braithwaite CE, Harvey RB, Phillips TD, Reine AH. Toxicological evaluation of aflatoxin and cyclopiazonic acid in broiler chickens. Poultry Science. 1992;71(7):1136-1144.
96. Southon S, Johnson IT, Gee JM, Price KR. The effect of Gypsophila saponins in the diet on mineral status and plasma cholesterol concentration in the rat. British Journal of Nutrition. 1988;59(1):49-55.
97. Spier SJ, Smith BP, Seawright AA, Norman BB, Ostrowski S, Oliver MN. Oak toxicosis in cattle in northern California: clinical and pathologic findings. Journal of the American Veterinary Medical Association. 1987;191(8):958-964.
98. Terrill TH, Waghorn GC, Woolley DJ, McNabb WC, Barry TN. Assay and digestion of 14C-labelled condensed tannins in the gastrointestinal tract of sheep. British journal of nutrition. 1994;72(3):467-477.
99. The chemical participants. Academic Press, New York, US, 355-388pp.
100. Torell R, Young JA, Kvasnicka B. Halogeton poisoning. Cooperative Extension. University of Nevada. Fact sheet-00-20. 2005.
101. Turner PC, Moore SE, Hall AJ, Prentice AM, Wild CP. Modification of immune function through exposure to dietary aflatoxin in Gambian children. Environmental health perspectives. 2003;111(2):217-220.
102. Vesonder R, Haliburton J, Stubblefield R, Gilmore W, Peterson S. Aspergillus flavus and aflatoxins B 1, B 2, and M 1 in corn associated with equine death. Archives of Environmental Contamination and Toxicology. 1991;20(1):151-153.
103. Waghorn G. Beneficial and detrimental effects of dietary condensed tannins for sustainable sheep and goat production-Progress and challenges. Animal Feed Science and Technology. 2008;147(1-3):116-139.
104. Ward G, Harbers LH, Blaha JJ. Calcium-containing crystals in alfalfa: their fate in cattle. Journal of Dairy Science. 1979;62(5):715-722.
105. Wild CP, Hudson GJ, Sabbioni G, Chapot B, Hall AJ, Wogan GN, *et al.* Dietary intake of aflatoxins and the level of albumin-bound aflatoxin in peripheral blood in The Gambia, West Africa. Cancer Epidemiology and Prevention Biomarkers. 1992;1(3):229-234.
106. Williams JH, Phillips TD, Jolly PE, Stiles JK, Jolly CM, Aggarwal D. Human aflatoxicosis in developing countries: a review of toxicology, exposure, potential health consequences, and interventions. The American journal of clinical nutrition. 2004;80(5):1106-1122.
107. Wilson JG. Current status of teratology. In General principles and etiology. Springer, Boston, MA, 1977, 47-74pp.
108. Yaling W, Tongjie C, Guozhong L, Chunsan Q, Huiyong D, Meiling Y, *et al.* Simultaneous detection of airborne aflatoxin, ochratoxin and zearalenone in poultry house by immunoaffinity column and high performance liquid chromatography. Environ. Res. 2008;107:139-144.
109. Yisehak K, De Boever JL, Janssens GPJ. The effect of supplementing leaves of four tannin-rich plant species with polyethylene glycol on digestibility and zootechnical performance of zebu bulls (B os indicus). Journal of animal physiology and animal nutrition. 2014;98(3):417-423.
110. Yoshiki Y, Kudou S, Okubo K. Relationship between chemical structures and biological activities of triterpenoid saponins from soybean. Bioscience, biotechnology, and biochemistry. 1998;62(12):2291-2299.
111. Zain ME. Impact of mycotoxins on humans and animals. Journal of Saudi chemical society. 2011;15(2):129-144.