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REVIEW ARTICLE

Ruminal Microflora, Mycotoxin Inactivation by Ruminal Microflora and Conditions Favouring Mycotoxicosis in Ruminants: A Review

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ABSTRACT

Unlike poultry, pigs and other animals, ruminants are quite resistant to adverse effects of mycotoxins. This is because the ruminal microflora and microfauna act as first line of defence by detoxifying and degrading the mycotoxins originated from feed. Ruminal fluid has a diverse ecosystem containing 50 genera of bacteria, 25 genera of cilliate protozoae, 5 genera of anaerobic fungi and 10^8-10^9/ml of bacteriophages. This number might be much larger as many of them are non-culturable. Ruminal ecosystem converts ochratoxin, aflatoxin, diacetoxyscirpenol (DAS), T-2 toxins and deoxynivalenol (DON) to less toxic ochratoxin-α, aflatoxicol, de-epoxy DAS, HT-2 toxin and de-epoxy DON respectively. While fumonisins are tolerant to ruminal degradation and zearalenone is converted to more toxic α-zearalenone. However, their oral bioavailability is very low due to which they do not cause intoxication in ruminants. This detoxifying ability of ruminal microflora is saturable. Different factors like metabolic disorders (rumen acidosis, milk fever etc), abrupt change in diet, high protein diet, negative energy balance, antimicrobial activity of some mycotoxins etc can decrease the detoxifying ability of ruminal microflora. Under field conditions, animals are under the exposure of different mycotoxins present in concentrates and roughages as a result of which detoxifying ability of microflora becomes exhausted producing a high internal challenge in animals.

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INTRODUCTION

Mycotoxins are the distinct group of secondary metabolites produced by *fungi imperfecti*. These are several hundred chemically different toxic compounds (Sweeney and Dobson, 1998). About 300 different mycotoxins have been identified which are found harmful to animals and humans. Among this diverse group, common mycotoxins are aflatoxins, ochratoxins, trichothecenes, zearalenone and fumonisins (Huwig *et al*., 2001).

Ruminants are quite tolerant to adverse effects of mycotoxins as ruminal microflora and microfauna act as first line of defense against mycotoxins. However, some reports of mycotoxicosis in large animals are available like ergotism in Old Testament (Schoental, 1984), decline of Etruscan civilization by T-2 toxin and zearalenone (Schoental, 1991), Athenian crisis in fifth century B.C. (Schoental, 1994), contamination of Egyptian tombs with ochratoxin A causing mysterious deaths of several archaeologists. Mycotoxins can affect animals by changing nutrient content absorption and metabolism, modifying endocrine, neuroendocrine and enzyme functions and immunosuppression (Cast, 1989).

Mycotoxins are produced only under aerobic conditions (Ratcliff, 2002) and they affect approximately 25% crops and cereals of the world each year (Lawlor and Lynch, 2005). There are different reasons for mycotoxin production in feed ingredients like improper rotation of the crops in the field (Blaney, 2001), harvesting of crops with moisture levels more than 14-16% (Magan and Aldred, 2007), fungal spores in the soil residing from crops to crops and year to year (Bricknell *et al*., 2006). Harvested crops with high moisture levels when stored are the excellent environment for the growth of storage fungi.

Different ingredients of the concentrates are contaminated with different mycotoxins like cereal grains, corn gluten; soyabean products are contaminated mostly with aflatoxins while maize mostly with fumonisins and zearalenone; and cereal grains are generally contaminated with ochratoxins, ergot alkaloids and deoxynivalenol (Scudamore *et al*., 1998; Placinta *et al*., 1999). In dairy cattle, up to 70% of the diet is comprised of concentrates to meet the energy requirements of animals, resulting in possible exposure to more than one type of mycotoxins (Fink-Gremmels, 2008). Mycotoxins can also be present in hay, straw and silage after a prolonged period of storage (Mansfield and Kuldau, 2007). Mycotoxins already present in the plants originating from pre-harvest contamination also remain unaffected by ensiling process.

Ruminal microflora

In ruminal fluid, a diverse ecosystem of microflora and microfauna is present. It consists of more than 50 genera of bacteria (10^{10}-10^{11} cells/mL), 25 genera of ciliate protozoa (10^{4}-10^{6}/mL), five genera of anaerobic fungi (10^{3}-10^{5} zoospores/mL) and 10^{8}-10^{9}/mL of bacteriophages (Kamra, 2005). The quantity may even be higher as many of the microorganisms are non-culturable. The ecosystem is so diverse that with the change in feed ingredients, the microbial population also changes. The condition in the rumen is purely anaerobic. The growth of invading microorganisms is limited by high buffering capacity and osmotic pressure (Odenyo *et al*., 1994). Most of the bacteria in the ruminal fluid are Gram negative (G-ve). When high energy diet is given, number of Gram positive (G +ve) bacteria increases. The optimum pH and temperature for the growth of bacteria are 6.0 to 6.9 and 39 ^{o}C respectively. Cellulose degrading bacteria in ruminal fluid are *Fibrobacter succinogenes, Ruminococcus albus* and *Ruminococcus flavefaciens* (Sahu *et al*., 2004).

Cilliate protozoa of rumen are divided into two groups i.e. entodiniomorphid protozoa and holotrich protozoa (Hungate, 1986). Holotrich protozoa are sub-divided into 15 different genera like *Dasytricha, Buetschlia* and *Isotricha* (Eloff and Van Hoven, 1980). Holotrich protozoa contain different enzymes like invertase, polygalacturonase in large quantities. However these also contain enzymes for degradation of hemicelluloses and cellulose but the levels are quite low as compared to that in entodiniomorphid protozoa (Williams and Coleman, 1985).

Some of the examples of ciliate protozoa in cattle and buffalo are *Diplodinium, Elytroplastron, Ostracodinium, Isotricha*, *Dasytricha, Metadinium* etc (Shimizu *et al*., 1983). The rumen of Brazilian water buffalo showed 49 different species of ciliate protozoa (Dehority, 1979). However, the number of ciliate protozoa in both cattle and buffalo are same (Kurar *et al*., 1988). The major function of ciliate protozoa in the rumen is stabilization of pH as the pH of the rumen is always low (Santra *et al*., 1995). Elimination of cilliate protozoa from rumen results in decrease in ammonia nitrogen (Ushida *et al*., 1986) and reduction of methanogenesis (Santra *et al*., 1994).

Fungi are also present in rumen and these fungi are obligate and anaerobic in nature. These fungi with the help of their various enzymes play an important role in degradation of fibrous diet (Paul *et al*., 2003). The fungal growth is stimulated in rumen of buffalo when fibre containing diet is fed to animal as compared to carbohydrate enriched diet (Kamra *et al*., 2003). Fungi gets easily penetrated into the lignocellulosic diet as compared to cellulose-degrading bacteria and this is due to different enzymes of fungi like cellulases, hemi-cellulases, proteases and esterases (Fonty and Joblin, 1990).

Bacteriophages are also present in rumen in large numbers. They are specific for different bacteria and are responsible for the lysis of bacteria which are not useful at different feeding regimes (Klieve and Swan, 1993).

Inter-relationship between ruminal microorganisms exists. Bacteria like *Ruminococcus flavefaciens* and *Ruminococcus albus* produce a soluble protein that is responsible for inhibiting the degradation of cellulose by anaerobic fungi (Stewart *et al*., 1992).

Rumen ecosystem changes with change in the composition of diet. Starch which is an easily fermentable carbohydrate causes a significant increase in number of ruminal protozoa. However, excess of starch not fermented by protozoa is converted to lactic acid by different bacteria which lowers the pH and ultimately lowers the number of protozoa in rumen (Ozpinar *et al*., 1999).

This information about ruminal ecosystem is still incomplete as a very large proportion of ruminal microorganisms are non-culturable. There are no single media available which can support the growth of all the microorganisms of the rumen.

Inactivation of mycotoxins by ruminal microflora

Ruminal microbes cause metabolism of ingested material which is considered to be a first line of defense against toxic material present in the diet. However, ruminal degradation might be a disadvantage as toxic substances may be converted into much toxic compounds (Kiessling *et al*., 1984). Protozoa are found to be more active than bacteria when mycotoxins are present in the rumen.

Degrading ability of rumen ecosystem depends upon the composition of diet fed to animals. Diets containing 40-60% of concentrates stimulate highest density of protozoa (Dehority and Orpin, 1988). Table 1 shows the degrading and inactivating effects of ruminal microflora on different mycotoxins.

Ochratoxin A

Ochratoxin A (OTA) is produced mainly by *Aspergillus alutaceus, A. ostianus, A. quercins, A. sulphureum* and *Penicillium verrucosum* (Federico *et al*., 2009). OTA causes severe pathological alterations in different non-ruminants like poultry (Dwivedi and Burns, 1986; Abidin *et al*., 2011), or pigs (Jelinek and Poland, 1989) whereas few data are available regarding intoxication of OTA in ruminants. Young ruminants when exposed to OTA show some serious signs of ochratoxicosis like depression, polyuria, and degeneration of kidney leading to death (Sreemannarayana *et al*., 1988).

OTA consists of ochratoxin-α linked through an amide bond with L-β-phenylalanine. Ruminal microflora causes enzymatic hydrolysis of this bond producing less

Table 1: Effect of ruminal microflora on different mycotoxins

Mycotoxin	Microflora involved	Findings	References
Ochratoxin A	Ruminal microflora	Enzymatic hydrolysis of amide bond producing OTα and L-β-phenyalanine which are almost non-toxic	Sreemannarayana *et al*., (1988); Xiao *et al*., (1991); Ozpinar *et al*., (1999)
Ochratoxin A	Ruminal content	Degradation of OTA was higher in diet containing 40% roughages and 60% concentrates as compared to 100% concentrates (in vitro experiment)	Ozpinar *et al*., (1999)
Ochratoxin A	*Acinetobacter calcoaceticus, Lactobacillus acidophilus*	Considerable degradation of OTA	Fuchs *et al*., (2008)
	Bifidobacterium animalis	95% reduction of OTA in vitro	
Ochratoxin A	Ruminal protozoae	Degradation up to 12 mg/kg OTA	Pettersson *et al*., (1982)
Deoxynivalenol (DON)	*Eubacterium* strain BBSH797	Converted DON in to less toxic DOM-1	Binder *et al*., (1998); Schatzmayr *et al*., (2006)
DAS, DON, OTA	*Butyrivibrio fibrisolvens*	Degraded derivatives of toxins in vitro	Kiessling *et al*., (1984); Westlake *et al*., (1987)
Zearalenone (ZEN)	*Butyrivibrio fibrisolvens*	Caused metabolism of ZEN	Kennedy *et al*., (1998); Yiannikouris and Jouany, (2002)
Patulin	*Bifidobacterium animalis*	Reduction of 80% patulin in vitro	Fuchs *et al*., (2008)

or non-toxic OTα and phenylalanine (Sreemannarayana *et al*., 1988; Xiao *et al*., 1991; Ozpinar *et al*., 1999). This degradation occurs in all compartments of the rumen except abomasums. After OTA administration, 50% of OTA is degraded during first 15 minutes while 95% of total OTA is degraded in following 4 hours. The left over OTA is then degraded during its passage through reticulum and omasum. The ruminal degradation rate of OTA ranges from 2 to 12.5 hours (Ozpinar *et al*., 1999). Non-toxic OTα does not further metabolize into other products however it remains as such in the rumen like environment (Muller, 1995). OTα is almost non-harmful to cells (Creppy *et al*., 1983). Organs and muscles of ruminants are not considered to be contaminated with OTA (Garies and Scheuer, 2000). Ochratoxin C (OTC) has toxicity similar to OTA. OTC when enters the rumen through feed, it is first converted into OTA and then finally degraded to OTα.

In rumen, Gram +ve bacteria are mostly involved in OTA degradation at a pH lower than 7. Certain bacteria of rumen cause considerable OTA degradation like *Acinetobacter calcoaceticus* (Hwang and Draughon, 1994), *Bifidobacterium animalis* and *Lactobacillus acidophilus* (Fuchs *et al*., 2008). Yoghurt producing bacteria like *Streptococcus salivarius, Lactobacillus delbruecki* (Skrinjar *et al*., 1996) and beer producing bacteria (Baxter *et al*., 2001) cause OTA degradation only to some extent.

Non-ruminating calves are more prone to adverse effects of OTA than ruminating calves having a functional population of microbes in ruminal fluid (Sreemannarayana *et al*., 1988). In rumen acidosis, pH as well as protozoa concentration of the rumen decreases, leading to greater adsorption and lower degradation of OTA (Kiessling *et al*., 1984). Also high starch concentration in the diet of ruminants decreases OTA degradation (Ozpinar *et al*., 1999).

90-100% of the total ingested OTA is excreted via urine as OTα (Xiao *et al*., 1991). Although transfer of OTA to milk has very less significance (Fink-Gremmels, 2008), some studies showed that ruminant milk contained OTα when OTA contaminated feed was given to them. It is noted that OTA ranging from 33-72 mg per day (repeated doses) to cattle is degraded by rumen (Muller, 1995). There is some reports available suggesting that OTA up to 12 mg/kg of feed can be degraded by ruminal protozoae showing high tolerance of ruminants to OTA (Pettersson *et al*., 1982).

Aflatoxins

Aflatoxins belong to difuranocoumarins group and are sub-divided into B_1, B_2, G_1 and G_2 which are produced by three species of *Aspergillus* i.e. *Aspergillus flavus, A. parasiticus* and *A. nomius* and various other species of *Rhizopus* and *Penicillium* (Smith, 2002). *A. flavus* only generates aflatoxins B_1 and B_2 while *A. parasiticus* produces all four types (David and Diener, 1983). Regarding dairy cattle, mature animals are less susceptible to aflatoxins than pregnant, growing and young animals. Aflatoxins are poorly degraded (only 10%) in the rumen (Westlake *et al*., 1989). Aflatoxin B1 (AFB_1) is converted to aflatoxicol in rumen (Auerbach *et al*., 1998) but the percentage of its formation is still unknown as it is readily converted back to parent AFB_1 (Nakazato *et al*., 1990). Aflatoxin present in feed binds with ruminal contents and only 2-5% of the total ingested aflatoxin reaches intestine. High levels of AFB_1 (up to 100µg/kg of feed) are considered to be toxic for cattle (Radostits *et al*., 2000). However some reports showed that AFB_1 up to concentrations of 60-300µg/kg feed had no effects in ruminants (Helferich *et al*., 1986). Doses between 200 and 500µg/kg feed for 14 days cause severe pathological effects in calves (Pier *et al*., 1976). Ray *et al*., (1986) reported a third trimester abortion in dairy cows due to feed containing 77µg/g AFB_1.

AFB_1, which escapes from the ruminal degradation, goes to liver and is converted to AFM_1 by hepatic metabolism. AFM_1 is 4-hydoxymetabolite of AFB_1 (Kuilman *et al*., 2000) and is released in milk 2 days after ingestion of AFB_1. Almost 1-2% of total ingested AFB_1 is excreted in milk as AFM_1. This percentage increases up to 6.2% in case of animals receiving high concentrates diet. However, AFM_1 disappears from milk 4 days after removal of AFB_1 from diet (Whitlow *et al*., 2000). The

average transfer of AFB_1 in cattle from diet to milk as AFM1 is 1.7% (Jouany and Diaz, 2005). The maximum acceptable level of AFM_1 in milk is 0.05µg in Asia (Van Egmond *et al*., 2007). Some of the aflatoxicol also comes in milk (Van Herwaarden *et al*., 2006). To avoid aflatoxin residues in milk, the dietary levels of AFB_1 must be lower than 25ppb (Jones *et al*., 1994).

Fumonisins

Fumonisins are produced by *Fusarium moniliformis* and *Fusarium proliferatum*. There are six different types of fumonisins i.e. FA_1, FA_2, FB_1, FB_2, FB_3 and FB_4 but Fumonisin B_1 (FB_1) is considered to be the most toxic among all the fumonisin mycotoxins (Gelderblom *et al*., 1992). Fumonisins are tolerant to microbial degradation in the rumen. However, its oral bioavailability remains very low due to which acute or severe intoxications do not occur at farm levels (Fink-Gremmels, 2008) although high levels may cause microscopic lesions in liver and kidney (Osweiler *et al*., 1993).

Fumonisins pass the rumen unaffected (Caloni *et al*., 2000). Prelusky *et al*., (1996) found that feeding of FB_1 at the rate of 400 µg/g of feed resulted in 80% excretion of un-metabolized FB_1 in feces and traces were also found in urine. No hydrolyzed metabolites were detected. Neither FB_1 nor its metabolites were present in the blood.

60-90% of orally fed FB_1 is excreted as metabolites in feces. This FB_1 is extensively hydrolyzed in cattle into metabolites (aminopolyols and aminopentol) which are easily absorbed in body and are much toxic than FB_1 (Gelderblom *et al*., 1992). However, low toxicity of FB_1 in ruminants is due to low bioavailability of FB_1 after oral administration (Prelusky *et al*., 1995). Fumonisin at the rate of 3 mg/kg body weight per day in Jersey cows for a period of 14 days resulted in decreased feed intake and milk production (Sahu *et al*., 2004).

Trichothecenes

Trichothecenes is a diverse group of mycotoxins, the important ones of which are diacetoxyscirpenol (DAS), T-2 toxins and deoxynivalenol (DON) also known as vomitoxin. Trichothecenes are produced by a variety of fungal species but the most important producers are *Fusarium* species. DON is produced particularly by *Fusarium roseum* (Federico *et al*., 2009) while T-2 toxins *by F. sporotrichioides* and *F. poae* (Whitlow and Hagler, 2005).

Micro-organisms in the rumen of sheep have deacetylation function. In rumen, DAS is de-acetylated to monoacetoxyscirpenol (MAS) and scirpenetriol. Further it is converted to de-epoxy MAS and deepoxyscirpenetriol while T-2 toxin is metabolized to HT-2 and neosolaniol (Cote *et al*., 1986b) under anaerobic conditions and they are less toxic products than parent compounds (Kiessling *et al*., 1984). However, DON is converted into de-epoxy DON also known as DOM-1 which is non-toxic (Binder *et al*., 1998). DON occurs in a large quantity in concentrates because of its high grain quantity. It is readily degraded in rumen. However, in animals already suffering from rumen acidosis, the degradation of DON is usually incomplete and measurable levels can be detected in blood samples (Speijers and Speijers, 2004). T-2, HT-2, DON and DAS are neutralized by ruminal contents when toxins are given at the rate of 10µg/ml (Prelusky *et al*., 1986). *Eubacterium* strain BBSH797 isolated from rumen of bovines convert DON into DOM-1 (Binder *et al*., 1998; Schatzmayr *et al*., 2006).

Bacteria such as *Butyrivibrio fibrisolvens, Selenomonas ruminantium* and *Anaerovibrio lipolytica* use T-2 toxin as an energy source through two enzyme systems (Westlake *et al*., 1987). Other strains of *B. fibrisolvens* are able to degrade the derivatives of toxins like DAS, DON and OTA *in vitro* (Kiessling *et al*., 1984; Westlake *et al*., 1987).

Ruminants can tolerate up to 8.5 mg/g DON in the diet for several weeks without showing major effects on the health of animals. DON is completely degraded within 24 hours (King *et al*., 1984) or even within 6 hours (Prelusky *et al*., 1996) in the rumen. However, if animals suffer from rumen acidosis, there occurs microorganism imbalance in the rumen leading to incomplete ruminal detoxification of DON (Swanson *et al*., 1984).

Zearalenone

Zearalenone (ZEN) is a myco-estrogen mainly produced by *Fusarium graminearum, F. culmorum, F. crookwellense, F. semitectum* and *F. moniliforme* (Marasas *et al*., 1984) but the most important producer is *F. graminearum* (Zinedine *et al*., 2007). In rumen, it is metabolized to its hydroxyl-metabolite (α-zearalenol) by ruminal protozoa (Malekinejad *et al*., 2006), which is ten times more toxic than ZEN (Danicke *et al*., 2005) and further metabolized by bacterium *Butyrivibrio fibrisolvens* present in the ruminal fluid (Kennedy *et al*., 1998; Yiannikouris and Jouany, 2002). To less extent, it is also converted to β-zearalenol having lower toxicity. Alpha-zearalenol and ZEN together can produce zeranol which is an oestrogenic hormone enhancing growth of animal (Kennedy *et al*., 1998). ZEN and its metabolites (α-zearalenol) bind to oestrogen receptors and induce a state of hyperoestrogenism in ruminants (Minervini *et al*., 2001). Although it binds with oestrogen receptors, the cases of hyperoestrogenism are very rare in ruminants (Coppock *et al*., 1990). This is because of the reason that α-zearalenol is more polar and has poor rate of absorption. This secondary metabolite has good oral bioavailability and is often used as a growth-promoting agent in fattening cattle (Launay *et al*., 2004; Blokland *et al*., 2006). Due to this reason, ZEN concentration should not be more than 250ppb in total diet of the ruminants (Jones *et al*., 1994). ZEN and its metabolites are also secreted in milk but levels are very low often below measurable levels.

Patulin

Patulin is produced by *Penicillium expansum, P. urticae* and *Aspergillus clavatus* (Yiannikouris and Jouany, 2002). It is an electrophilic molecule present in very high concentration in silage due to its high maize contents, however, only seldom produces its specific neurotoxic signs but adversely affects ruminal ecosystem due to its antimicrobial activity (Schneweis *et al*., 2000).

Conditions favouring mycotoxicosis in ruminants

Different mycotoxins when present together in feed pose greater negative effects as compared to those produced individually (Smith and Seddon, 1998). Despite

degrading ability of ruminal microflora, there are different conditions which favor the mycotoxicosis in ruminants.

High yielding cows encounter many metabolic disorders like ketosis, milk fever, rumen acidosis etc. These disorders occur in high frequency during the transition period starting from dry period in late pregnancy up to onset and increase in milk production. Onset of lactation demands high digestible energy and calcium requirements in animals but the feed intake by animals reduce up to 20% few days before calving and it remains low during first days of parturition leading the animals in a state of negative energy balance (NEB). It results in depletion of calcium stores and body fat leading to lower body condition scoring (Geelen and Wensing, 2006). During NEB, easily fermentable carbohydrates are given in the form of high grain diets. The disadvantage of such feeding is that the organic acid load increases and there is reduced salivary secretions which can buffer the acids of rumen probably due to reduced fibre intake resulting in acute ruminal acidosis (Enemark *et al*., 2002). Storage fungi produce microbial volatile organic compounds (MVOCs) which produce mouldy odour in the feed. Ruminants also dislike this odour and reduce feed intake. In NEB, there are high energy demands and supplementation of diet with mouldy odour further contributes to NEB (Fink-Gremmels, 2008).

Various mycotoxins have antimicrobial, antifungal and antiprotozoal activity hence modifying the ruminal ecosystem and their degrading activity too. Patulin has a vast antimicrobial activity upon both G+ve and G-ve bacteria and different protozoa (Morgavi *et al*., 2003). It has adverse effects on the protein synthesis as well as upon the production of volatile fatty acids in ruminal fluid (Tapia *et al*., 2002). Fusarium toxins inhibit the multiplication of *Ruminoicoccus albus* and *Methanobrevibacter ruminatum*. Deoxynivalenol which is often present in animal feed does not have any effect on the growth of these bacteria (May *et al*., 2000). DON is almost completely degraded in the rumen; however, it reduces the ability of rumen microflora of using dietary proteins (Danicke et el., 2005).

Patulin is an electrophilic molecule that binds with thiol containing proteins such as glutathione and those bind with covalent bonds (Wooters and Speijers, 2003). This binding causes depletion of glutathione resulting in oxidative stress (Morgavi *et al*., 2003).

Another important factor for mycotoxicosis in ruminants is the abrupt change in diet such as high protein diet which modifies the ruminal ecosystem leading to decreased detoxifying and degrading ability of ruminal microflora (Xiao *et al*., 1991a; Muller *et al*., 2001).

Under field conditions, ruminants are under the exposure of different mycotoxins present in concentrates and roughages as a result of which the detoxifying capacity of ruminal fluid becomes exhausted and mycotoxins remain as such which are absorbed through duodenum producing a high internal challenge (Fink-Gremmels, 2008).

Conclusions

Nature has provided the ruminants a distinct ability of detoxifying and degrading mycotoxins present in feed in the form of ruminal microflora and microfauna; however, this ability is saturable. From the above mentioned discussion, it can be concluded that there should be proper monitoring of levels of different mycotoxins in feed to improve the health of ruminants. Management should be improved to avoid mycotoxicosis in ruminants. There should be improved conditions for harvesting and storage of crops. Harvesting should be done when moisture level of crops is below 14%. If not so, the moisture level should be brought below 14% soon after harvesting to avoid the growth of storage fungi. Addition of organic acids should be done during silage preparation. Mycotoxicosis in ruminants can be controlled by feeding optimum ratio of concentrates and roughages to animals. Use of probiotics can also prove beneficial in improving the health of ruminants.

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