Mycotoxins and Their Biotransformation in the Rumen: A Review*

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ABSTRACT : Mycotoxins are secondary metabolites produced by fungi. These toxins pose serious health concerns to animals as well as human beings. Biodegradation of these mycotoxins has been considered as one of the best strategies to decontaminate food and feedstuffs. Biodegradation employs the application of microbes or enzymes to contaminated food and feedstuffs. Ruminants are considered to be resistant to the adverse effects of mycotoxins presumably due to the biodegrading ability of rumen microbes compared to mono-gastric animals. Therefore, rumen microbial source or microbial enzyme could be a great asset in biological detoxification of mycotoxins. Isolation and characterization of pure culture of rumen microorganisms or isolation and cloning of genes encoding mycotoxin-degrading potential would prove to have overall beneficial impact in the food and feed industry. (**Key Words :** Mycotoxins, Biodegradation, Ruminants, Microbes, Enzymes)

INTRODUCTION

Mycotoxins are toxic metabolites produced by fungi. According to WHO, about 25% of world's food is contaminated by mycotoxins. Fungi belonging to Aspergillus, Penicillium and Fusarium species are responsible for causing mycotoxins of agro- economic importance. Aflatoxin, Ochratoxins, Trichothecenes, Zearalenone and Fumonisins are mycotoxins of greatest agricultural concerns (Vasanthi and Bhat, 1998).

Food and feedstuffs are prone to mycotoxin infection at field condition or during storage because intrinsic as well as extrinsic factors such as environment, climatic condition and fungal species variation contribute to mycotoxin infection (Hussein and Brasel, 2001).

The consumption of these mycotoxin-contaminated feedstuffs by animals leads to adverse effect on animal health and the effects are more serious in monogastric animals depending on the species and the susceptibility to toxins within the species. Ruminants, however, are considered generally more resistant to adverse effects of mycotoxins (Fink-Gremmels, 2008). This assumption is based on the findings that rumen microbiota has the biotransformation ability of mycotoxins to less toxic or non toxic metabolites. However, this is not applicable to all mycotoxins and the impact of mycotoxins in ruminant animals also depends upon age, breed, sex, dose level and immune status of individual animal (Dien Heidler and Schatzmayr, 2003). The carryover of toxins from animal food may have severe consequences on human health.

Mycotoxicosis is difficult to diagnose, because few signs of poisoning is produced.

The impact of mycotoxins upon animals extends beyond their obvious effect in producing death in a wide variety of animals. The economic impact of lowered productivity, reduced weight gain, reduced feed efficiency, damage to body organs, interference in reproduction is many times greater than that of immediate mortality and morbidity (Wu et al., 2004). Potential threats of cancer induced by mycotoxins in feeds and human foods along with the unknown effects of these mycotoxins are coupled to the universal concern about health risk (Marquardt, 1996). Consumption of some mycotoxins at levels does not cause overt clinical mycotoxicosis but may suppress immune function and lower resistance to diseases.

Thus, effective measures for detoxification of mycotoxins are essential for the improved production and productivity of livestock.

This paper focuses on different mycotoxins of agricultural importance, their effects on animal health and

explores the potential role of ruminants in mycotoxin biotransformation.

EFFECTS OF DIFFERENT MYCOTOXINS ON ANIMAL HEALTH

Mycotoxins in food and feedstuffs affect both the organoleptic characteristics and the nutritive value of feed, leading to risk of toxicosis. However, the biological effects of mycotoxins depend on the amount ingested by the host, varieties of occurring toxins, time of exposure to mycotoxins and animal sensitivity. Also, mycotoxins can induce health problems that are specific to each toxin or suppress the immunity power of animals, favoring infections. This is the major reason for the difficulty of diagnosing mycotoxicoses. Major mycotoxins of animal health concerns are as follows:

Aflatoxin

Aflatoxin is the most prevalent and significant mycotoxin. It is produced by *Aspergillus flavus* and *A. parasiticus* (Deiner et al., 1987; Kurtzman et al., 1987). It is stable once formed in grains and is not degraded during normal milling and storage (Brown, 1996). Aflatoxin contamination is more likely in grains grown in tropics or subtropics because the toxigenic fungi produce toxin in warm and high humid conditions. Commodities affected by aflatoxin include corn, peanuts, cottonseed, millet, sorghum and other feed grains (Phillips, 1999).

Due to the fact that aflatoxins are unavoidable contaminants in a variety of food and feeds, it has attracted the attention of many researchers (Verma, 2007). Like many microbial secondary metabolites, aflatoxins are a family of closely related compounds (Moss, 1996) that includes aflatoxin B1, B2, G1 and G2, but usually aflatoxin B1 (AFB1) is in the highest concentration. Following ingestion of aflatoxin-contaminated feeds, a part of the ingested aflatoxin B1 is degraded in the rumen, resulting in the formation of aflatoxicol. The remaining fraction is absorbed in the digestive tract by passive diffusion and is hydroxylated in the liver to aflatoxin M1 (Kuilman et al., 2000).

Aflatoxin lowers resistance to diseases and interferes with vaccine-induced immunity in livestock (Diekman and Green, 1992). Suppression of immunity by aflatoxin B1 has been demonstrated in turkeys, chickens, pigs, mice, guinea pigs, and rabbits (Sharma, 1993).

All animal species are susceptible to aflatoxicosis, and the sensitivity varies between species (Coppock and Christian, 2007). For example, monogastric animals such as fish, birds, cats, dogs, and swine are more susceptible than mature ruminants. Engel and Hagemeister (1978) reported

that 42% of aflatoxin was degraded when incubated *in vitro* with rumen fluid. Upadhaya et al. (2009) reported that aflatoxin B1 degradation in rumen fluid was influenced by the species of animal and types of forage fed to the animals.

Symptoms of acute aflatoxicosis in mammals include: inappetance, lethargy, ataxia, rough hair coat, and pale, enlarged fatty livers. Symptoms of chronic aflatoxin exposure include reduced feed efficiency and milk production, icterus, and decreased appetite (Nibbelink, 1986).

AFB1 inhibits both DNA and RNA synthesis (Butler and Neal, 1977). Lillehoj (1991) indicated that the activated AFB1 metabolite, AFB1-8,9 epoxide forms a covalent bond with the N7 guanine and forms AFB1-N7guanine adducts in target cells leading to G-T transversion, DNA repair, lesions, mutation and tumor formation (Foster et al., 1983).

AFB1 is also known as a potent hepatotoxin and hepato-carcinogen. The liver is considered to be the primary target organ for aflatoxin (Towner et al., 2000). It was reported that AFB1 could induce lipid peroxidation in rat livers causing oxidative damage to hepatocytes (Shen et al., 1994). Bonsi et al. (1999) demonstrated that cyclic nucleotide phospho diesterase activity in the brain, liver, heart and kidney tissues can be inhibited by AFB1.

Ochratoxin A (OTA)

Ochratoxin A is a complex compound consisting of OTA α linked through a 7-carboxy group to L-B phenylalanine by an amide bond (Mobashar, 2010).

It is produced by Asperigillus and Penicillium species that contaminate cereals, coffee beans, grape and other fruits, beer and wine (Halasz et al., 2009). Ochratoxicoses has rarely been found in ruminants because the microorganisms of the rumen are able to hydrolyze the amide bond of OTA to produce OTA α. which has a lower toxicity. The fact that the young animals with developed rumen are reported to be affected much less by OTA than the pre-ruminant calves indicates the significance of ruminal degradation of OTA (Sreemannarayana et al., 1988). However, the detoxification capacity of the rumen may be exceeded in cases of severe poisoning (Ribelin et al., 1978). Acute ochratoxicoses chiefly affects poultry, rats and pigs and leads to kidney damage, anorexia and weight loss, vomiting, high rectal temperature, conjunctivitis, dehydration, general weakening and animal death within two weeks after toxin administration (Chu et al., 1972; Chu, 1974). Chronic poisoning induces a decrease in ingestion, polydipsea and kidney lesions. Pigs are particularly sensitive to OTA (Elling et al., 1973). Such poisoning has a significant effect for toxin concentrations exceeding 1,400 µg/kg of feed. OTA has genotoxic properties due to DNA adduct formation (Pfohl et al., 2000). It also has immuno

toxic and carcinogenic properties by decreasing the number of natural killer cells responsible for the destruction of tumor cells.

Zearalenone (ZEN)

Zearalenone is a phyto esterogenic compound (Diekman and Green, 1992). It is caused by several Fusarium species. *F. graminaerium* is the species most responsible for estrogenic effects in farm animals (Marasas, 1991).

The main effects of ZEN are reproductive problems and physical changes in genital organs similar to those induced by oestradiol: oedemas and hypertrophy of the genital organs of pre-pubertal females, decrease in the rate of survival of embryos in gestating females, decrease in the amounts of luteinizing hormone (LH) and progesterone produced affecting the morphology of uterine tissues, decrease in milk production, feminization of young males due to decreased testosterone production, infertility and perinatal morbidity. Pigs are highly susceptible to ZEN poisoning whereas chickens and cattle show lower sensitivities (Coloumbe, 1993). ZEN is produced in very small amounts in natural conditions, and probably in insufficient quantities to cause trouble in ruminants (Guerre et al., 2000). ZEN has, however, been shown to cause infertility in grazing sheep in New Zealand (Towers et al., 1993).

Fumonisin (B1 and B2)

Fumonisins are the metabolties produced by *Fusarium proliferatum*, and F. *verticillioides.* The metabolite from FB1 is reported to be the most toxic promoting tumor in rats (Gelderblom et al., 1988). The aminopentol isomers formed by the base hydrolyis of ester linked tricaballylic acid of FB1 has been suggested to cause toxic effects because of the structural similarity to sphingoid bases (Humpf et al., 1998).

Fumonisins mostly affect horses, pigs and poultry, with ruminants seeming to be much less sensitive to this type of contamination (Yiannikouris and Jouany, 2002). However, fusarium-contaminated wheat when fed to dairy cows led to increased crude protein degradation and a lower molar percentage of propionate in the rumen (Tiemann and Danicke, 2007).

Fumonisins cause deep lesions in the liver, gastrointestinal tract, nervous system and lungs. Acute doses of fumonisins in pigs may inhibit the activity of pulmonary macrophages responsible for the elimination of pathogens, leading to pulmonary oedema (Harrison et al., 1990). In horses, contamination is manifested as severe neurological lesions leading to locomotive problems and ataxia (Yiannikouris and Jouany, 2002).

Fumonisins inhibit the synthesis of ceramides from sphinganin, blocking the biosynthesis of sphingolipid complexes. The quantity of sphinganins therefore increases and the recycling of sphingosins is blocked, resulting in cell dysfunction followed by cell death (Riley et al., 1998).

Trichothecenes

Trichothecens are produced by *Fusarium species* (e.g. *F. sporotrichioides*, *F. graminearum*, *F. poae*, and *F. culmorum*). It can also be produced by members of other genera viz *Trichothecium* (Jones and Lowe, 1960). Trichothecenes include T-2 toxin, diacetoxyscirpenol (DAS), deoxynivalenol (DON or vomitoxin), and nivalenol. Both T-2 toxin and DAS are the most toxic. Pigs and poultry have been shown to be very sensitive to T-2 toxin, DON (Friend et al., 1992). However, ruminants are less susceptible to these mycotoxins.

Tricothecenes have been reported as potential biological warfare agents. For instance, in an investigation of biological warfare agents in Cambodia from 1978 to 1981, T-2 toxin, DON, ZEN, nivalenol, and DAS were isolated from water and leaf samples collected from the affected areas (Watson et al., 1984).

These toxins cause weight loss, vomiting, severe skin problems and bleeding and may, in some cases, be responsible for the death of animals (Yiannikouris and Jouany, 2002). Like aflatoxins, they have immuno-suppressive properties acting both on the cell immune system and on the number of macrophages, lymphocytes and erythrocytes. T-2 and deoxynivalenol (DON) are known to inhibit protein synthesis and cause cell death in various parts of the body.

The findings based on the studies on toxicity of mycotoxins of agricultural importance in different animal species at different dose levels are summarized in the table below:

MYCOTOXIN PREVENTION, CONTROL AND DETOXIFICATION

Mycotoxins are toxic metabolites that can occur naturally in many agricultural products. The approaches for the prevention and control of mycotoxin formation may be taken at pre-harvest, immediately after harvest or during storage. The main approaches for pre-harvest prevention of mycotoxin formation include good agricultural practices, such as crop rotation, time of irrigation, planting and harvesting, plant breeding for resistance to toxigenic fungi, genetically modified crops resistant to insect penetration, and competitive exclusion by using of non-toxigenic strains in the field (Duncan et al., 1994). Prevention through pre-harvest and harvest management is the best method for controlling mycotoxin contamination. However, if contamination still occurs, post-harvest decontamination/ detoxification procedures can be used in order to remove or

Table 1. Aflatoxin (AF) toxicity in different animal species

Animal species	Dose	Symptoms of toxicity	Reference
Pig	0.02, 0,0385, 0.75 and 1.48 mg/kg in diet	Linear weight reduction and reduced feed efficiency	Southern and Clawson, 1979
	1-4 mg/kg	Linear weight reduction	Harvey et al., 1988
	2 mg/kg combined with 2 mg/kg OTA (barrows)	56% weight reduction	Huff et al., 1988
	140-280 µg/kg feed (*in vitro* experiment)	Immunosuppresion at cellular level	Van Heugten et al., 1994
	300 ppb (gestating and lactating sows)	Indirect toxication of piglets	Cabassi et al., 2004
Poultry	3.5 mg/kg in diet (broiler)	Reduced body weight and increased liver and kidney weight	Ortatali et al., 2005
	0.6-1.2 mg/kg feed (hen)	Reduced apparent digestible (10%) and metabolizable energy (4%)	Applegate et al., 2009
Ruminants	600 µg/kg (steer)	Reduced feed efficiency and rate of gain.	Helferich et al., 1986
	200-800 µg/kg	Reduced rumen motility	Cook et al., 1986
	2.5 mg/kg feed (lambs) for 21 days	Hepatic and nephritic lesions, increased weight and size of liver and kidney	Fernandez et al., 1997
	10- 108.5 µg/kg feed (cattle)	Significant feed intake reduction in dose dependant manner	Choudary et al., 1998
	2.5 mg/kg of feed (lamb 5 months)	Significant body weight reduction and reduced cellular immunity	Fernandez et al., 2000

Table 2. Ochratoxin (OTA) toxicity in different animal species

Animal species	Dose	Symptoms of toxicity	Reference
Pig	0.2-4 µg/kg feed for 3-4 months	Nephrotoxicity	Krogh et al., 1974
	0.06 mg/kg	10% immune response inhibition	Hult et al., 1980
	2.5 mg/kg feed	Reduced phagocytic activity of macrophage and IL2 production	Harvey et al., 1992
	5-50 µg/kg body weight for 4 weeks	Increased severity of pneumonia	Muller et al., 1999
	1-3 ppm OTA	Susceptible to salmonella infection	Stoev et al., 2000
	80-100 µg/kg in diet	Porcine nephropathy	Stoev et al., 2001
Poultry	4 mg/kg feed	Reduced IgG, IgA and IgM concentrations	Dwivedi et al., 1984
	4 ppm in 3 week old broiler chicks	Dramatic increase in mortality	Gibson et al., 1990
	0.5-2.0 ppm	Significant reduction in cell mediated immunity	Singh et al., 1990
	0.5 ppm in feed (chicks)	Depression of functional properties of macrophages and heterophils	Politis et al., 2005
	400-800 ppb (broilers)	Significant body weight, thymus weight, feed consumption reduction	Elaroussi et al., 2006
Ruminants	390-540 µg/kg barley (12 week calves)	No significant clinical symptoms	Patterson et al., 1981
	2-5 ppm (sheep)	No clinical effect seen and no OTA detection after I h incubation	Keisling et al.,1984
	100-150 ppb in goat rumen fluid (*in vitro*)	No OTA detection after 6 h incubation	Liu et al., 2010

Table 3. Zearalenone (ZEN) toxicity in different animal species

Animal species	Dose	Symptoms of toxicity	Reference
Pig	25-100 mg/kg diet	Estrus, Pseudo pregnancy and infertility	Chang et al., 1979
	3.6-20 µg/kg	Odeamatous uterus, ovarian cysts and increased still births	Cantley et al., 1982; Flower et al., 1987
	200 µg/kg bwt (immature gilt)	Follicular development and maturation	Zwierchowski et al., 2005
Poultry	0.1-2 mg/kg post orally (hen)	No symptoms (ZEN metabolized to non toxic form)	Pompa et al., 1986
Ruminants	385-1,925 ppb in feed (lactating)	No effect on milk production and no ZEN residue observed after Post mortem	Shreeve et al., 1979
	12 mg/kg feed (sheep)	Reproduction affected negatively	Dicostanzo et al., 1996
	(cattle)	No symptoms	Seeling et al., 2006

Table 4. Deoxynivenelol (DON) and T2 toxicity in different animal species

Animal species	Dose	Symptoms of toxicity	Reference
Pig	10 ppm	Neutrophils affected	Takayama et al., 2005
	5.7 mg/kg	Elevated liver RNA concentration and increased protein synthesis	Danicke et al., 2006
	0.21 ppm DON+0.004 ppm ZON or 9.75 ppm DON +0.358 ppm ZON in diet (sow)	Lesions in spleens and liver of sows No pathological lesions in piglet	Tiemann et al., 2008
Poultry	4 or 16 mg/kg diet (7 day old chicks)	Reduced body weight and feed intake, plaque formation and buccal ulceration	Hoerr et al., 1982
Ruminants	15.6 mg/kg DON (sheep)	No effects for 28 days Weight loss (extended feeding)	Harvey et al., 1986
	10-20 mg/kg T2 toxin in feed (calves)	Sloughing of papilla and omasum ulceration	Cheeke et al., 1998a
	3-5 ppm (dairy cow)	Reduced IgA , serum albumin and globulin	Korosteleva et al., 2007

reduce toxin amounts in agricultural products contaminated with unacceptable levels of mycotoxins.

Several strategies have been reported for the decontamination/detoxification of mycotoxins-contaminated grains but with limited success. This includes mechanical separation of infested grain, irradiation, solvent extraction and microbial inactivation (Karlovsky, 1999).

Different chemicals have been tested for their ability to structurally degrade or inactivate mycotoxins. This includes acids, bases, aldehydes, bisulfates and various gases as well as adsorbents (Huff et al., 1992; Raju and Devegowda, 2000; Santin et al., 2002). Ammoniation has resulted in a significant reduction in the contaminated peanuts and cotton seed meals (Marquardt, 1996). Likewise, when Neal et al. (2001) subjected a sample of peanut meal, highly contaminated with aflatoxin, for detoxification by using ammonia- diets based process, aflatoxin level was reduced to acceptable levels but different effects *in vivo* were noticeable when incorporated into animal.

Various other dietary treatments have been employed to reduce the toxicity of mycotoxins. This includes the use of chemisorbents like aluminosilicates, bentonites with the capacity to tightly bind and immobilize mycotoxins in the intestinal tracts of animals thereby reducing the bioavailability of toxins. Efficacy of adsorbents like montmorillonite nanocomposite (Shi et al., 2005), hydrated sodium aluminum silicates (Girish and Devegowda, 2006; Wang et al., 2006) were widely investigated and reported to be effective in elimination of mycotoxins. Likewise the use of freeze dried citrus peel to reduce aflatoxin contaminated feed was reported to be effective (Nam et al., 2009).

Many of these techniques proposed to decontaminate mycotoxins are perceived to be ineffective and potentially unsafe for large scale utilization (Marquardt, 1996) because toxicological safety of final product is not always guaranteed since there may be presence of chemical residues in the final product. Furthermore, these treatments may not be cost effective and there is the possibility of adsorbent to bind other nutrients leading to loss in feed nutritive value and palatability of feed.

Alternative strategy to control the problem of mycotoxicoses in animals is the application of enzymes or

microorganisms capable of biotransforming mycotoxins into nontoxic metabolites.

MYCOTOXIN BIOTRANSFORMATION IN THE RUMEN

Recently study on biodegradation of mycotoxins has been gaining grounds. A number of microbes from different niche have been reported to have biotransformation ability (Schatzmayr et al., 2006). Research studies show that biotransformation or cleaving and detoxifying mycotoxin molecules by microbes or enzyme is effective and safer method of mycotoxin control strategy (Schatzmayr et al., 2006).

Regulatory limits for mycotoxins imposed by FDA to overcome the adverse effects reflect that ruminants are less susceptible to mycotoxins. The maximum tolerable limits of mycotoxins as indicated by FDA are higher in ruminants compared to pig and poultry. For instance, allowable limits of aflatoxin for finishing swine are 200 ppb whereas it is 300 ppb for finishing beef cattle. However, for immature livestock, poultry and dairy cattle, the acceptable limits of aflatoxin is only 20 ppb.

Similarly the maximum allowable limits for fumonosins are highest for ruminants (60 ppm) and 10 ppm for swine. Likewise, maximum DON allowable limit for cattle is 10 ppm with 5 ppm for other livestock (Michael, 2006). There has been no advisory or regulatory level for ochratoxin issued by the FDA, but several research findings indicate that OTA is well tolerated by ruminants because of biodegradation by rumen microbes/enzymes.

Microbial degradation

Kurmanov (1977) reported that ruminants are more resistant to mycotoxin poisoning than monogastrics. Some microbes from the rumen have been identified for their ability to degrade mycotoxins or plant toxins. Among the first mycotoxins shown to be detoxified by ruminants were ochratoxin A (Hult et al., 1976) and aflatoxin B1 (Alcroft et al., 1968). Jones et al. (1996) reported on the disappearance of aflatoxin B1 within several weeks of incubation with broiler and turkey faeces. Engel and Hagemeister (1997) reported that 42% of aflatoxin was degraded when incubated *in vitro* with rumen fluid. Upadhaya et al. (2009) reported that aflatoxin B1 degradation in rumen fluid was influenced by the species of animal and types of forage fed to the animals. The changes in the feed composition from roughage to concentrates and a high percentage of protein-rich concentrates in the daily diet modify the cleavage capacity of rumen microorganisms (Xiao et al., 1991; Muller et al., 2001; Liu Yang, 2010).

The metabolism of different mycotoxins potentially encountered by ruminants has also been investigated (Kiessling et al., 1984) and found that the mycotoxins zearalenone, T-2 toxin, diacetoxyscirpenol and deoxynivalenol were well metabolized by whole rumen fluid; whereas aflatoxin B1 and ochratoxin A were not. Westlake et al. (1989) investigated the effects of these mycotoxins in addition to verrucarin A on the growth rate of *Butyrivibrio fibrisolvens* specifically. They found that this organism was able to degrade all but aflatoxin B1 and that none of the toxins tested inhibited the growth of *B. fibrisolvens.* Similarly, Kennedy et al. (1998) reported that 90% of ZEN was hydrolyzed to alpha ZEN by rumen microbes. Although the alpha form of ZEN is more estrogenic than its parent form, due to low rate of absorption, ruminants are less susceptible to ZEN toxicity (Seeling et al., 2006).

Kiessling et al. (1984) demonstrated that 90-100% of the metabolism of OTA, ZEN, T-2 toxin and DAS were achieved by the rumen protozoa and, therefore, they were considered as the most important ruminal microbial population in mycotoxin biodegradations. However, some studies indicated that the bacterial fraction of rumen fluid had significant capacity of OTA degradation (Schatzmayr et al., 2002; Liu Yang, 2010).

The study on effect of feed types by Korean native goat (Liu Yang, 2010) on OTA degradation indicates that the high OTA degradation in 100% roughage diet was due to shift in *Bacillus licheniformis* population in the rumen of goat. *B.licheniformis* isolated from Thai fermented soybean mean has also been reported to degrade OTA (Petchkongkaew et al., 2008).

A continuous anaerobic culture capable of deoxynivenol deepoxidation was established on the basis of a cattle ruminal fluid inoculum (Binder et al., 1997b). Binder et al. (2000) isolated a new species of bacterium of the genus *Eubacterium* (*Eubacterium* strain BBSH 797) from bovine rumen fluid which showed the potentiality of biotransforming the epoxide group of trichothecenes into a diene (Schatzmayr et al., 2006).

In another study, dietary DON concentrations ranging between 3.1 and 3.5 mg/g feed (88% DM) did not cause any significant adverse health effects; however, it increased ammonia concentrations (Seeling et al., 2006).

Taken together, these examples demonstrate the capacity of the rumen to inactivate mycotoxins. However, there exists the likelihood of adverse health effects in cattle.

For instance, some mycotoxins e.g aflatoxins are converted into metabolites that retain biological activity. The assessment of undesirable effects exerted in ruminants should include the antimicrobial activity of various mycotoxins that results in an impairment of the function of the rumen flora, followed by a poor feed utilization and reduced weight gain and productivity.

Nevertheless, there still lies the possibility of isolation,

screening, selection and characterization of potential rumen microbes or gene for mycotoxin biotransformation.

Microbial enzymatic degradation

In the past, enzymes were isolated primarily from plant and animal sources, and thus a relatively limited number of enzymes were available to the food processor at a high cost. Today, bacteria and fungi are exploited and used for the commercial production of a diversity of enzymes. Several strains of microorganisms have been selected or genetically modified to increase the efficiency with which they produce enzymes. In most cases, the modified genes are of microbial origin, although they may also come from different kingdoms. For example, the DNA coding for chymosin, an enzyme found in the stomach of calves, that causes milk to curdle during the production of cheese, has been successfully cloned into yeasts (*Kluyveromyces lactis*), bacteria (*Escherichia coli*) and moulds (*Aspergillus niger* var. *awamori*). Chymosin produced by these recombinant microorganisms is currently commercially produced and is widely used in cheese manufacture (FAO, 2004).

In view of the extensive contamination of the feedstuffs by mycotoxins originating as a secondary metabolite of different fungi, it is imperative to develop cost effective and efficient methods for their decontamination. Biodegradation is a popular and attractive technology that utilizes the metabolic potential of microorganisms or enzymes to decontaminate food or feedstuffs. Recently, the capability of different microbial enzymes for biodegradation of environmental pollutants or mycotoxins has generated a considerable research interest in this area of food, industrial or environmental microbiology (Ashger et al., 2008).

The detoxification by specific enzymes helps to avoid the drawbacks of using the certain microorganisms which have negative effect such as impairment of the nutritive value of food and feedstuffs, food safety, refusal of food or feedstuffs due to change in colour or flavour (Shapira, 2004). For instance, the ability of *Flavobacterium aurantiacum* B-184 to remove aflatoxins from foods was demonstrated in milk, vegetable oil, corn, peanut, peanut butter and peanut milk. However, the bright orange pigmentation associated with this bacterium limit its applicability for food and feed fermentations (Line et al., 1995).

The industrial production of enzymes from microorganisms involves culturing the microorganisms in huge tanks where enzymes are secreted into the fermentation medium as metabolites of microbial activity. Enzymes thus produced are extracted, purified and used as processing aids in the food industry and for other applications. Purified enzymes are cell free entities and do not contain any other macromolecules such as DNA.

Several studies demonstrated the capacity of microbial enzymes in mycotoxin biodegradation. Liu et al. (2001) reported the extraction, purification and characterization of aflatoxin degrading enzyme, aflatoxin detoxifizyme (AFDF) from *Armillariella tabascens*. In a recent study by Albert et al. (2009), laccase enzyme obtained from fungus *Peniophora* and *Pleurotus ostreatus* was found to have aflatoxin degradability by 35-40%. The enzyme responsible for OTA degradation was reported to be carboxypeptidase A (Pitout, 1969). An enzymatic extract possessing a high hydrolytic activity of ochratoxin A was isolated from *A. niger* MUM 03.55. This enzyme extract exhibited carboxypeptidase A-like hydrolytic activity on OTA (Abrunhosa et al., 2007). Carboxypeptidase A present in *Phaffia rhodozyma* is also reported to degrade OTA up to 90% (Peteri et al., 2007). De-epoxidase was reported to be responsible to detoxify DON (Binder et al., 2000). Stefan et al. (2010) reported two genes, fumD, encoding a carboxylesterase and fumI encoding an amino-transferase which is responsible for Fumonisin B1 degradation by Sphingopyxis sp. MTA144. These evidences prove the effective use of microbial enzymes for biodegradtion of mycotoxins.

In addition to the use of intact microbes or cell-free enzymatic preparations as feed additives (Erber, 1996), the expression of the respective genes in genetically manipulated organisms opens new avenues for the protection of health of farm animals. Examples of such procedures include the genetic engineering of ruminal microorganisms (Duvick et al., 1998b) and feeding transgenic mycotoxin-degrading maize to pigs (Duvick and Rood, 1998).

CONCLUSION

Biodegradation of mycotoxins with microorganisms or enzymes is considered as the best strategy for detoxification of feedstuffs. This approach is considered as environmental friendly approach in contrast to physicochemical techniques of detoxification. Since ruminants are potential source of microbes or enzymes for mycotoxins biotransformation, isolation of pure culture using enriched media or screening of candidate genes from the metagenomic library of rumen micro-organism seems to be a good strategy for overcoming the problem of some mycotoxins.

Furthermore genetic engineering technologies will not only improve the efficiency with which enzymes can be produced from these organisms or producing the engineered organism having the target genes, but they also increase their availability, bioavailability, and improve their quality. Thus, the use of enzymes or engineered micro-organisms as processing aids in the food industry would prove to have overall beneficial impact.

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