Review on biological degradation of mycotoxins

Cheng Ji, Yu Fan, Lihong Zhao

State Key Laboratory of Animal Nutrition, College of Animal Science and Technology, China Agricultural University, Beijing 100193, China

1. Introduction

Mycotoxins are a large group of secondary metabolic products from fungi, or molds, which pose serious risks for human and animal health. Fungal growth and mycotoxin production may occur in the field and/or during storage, under suitable temperature and humidity conditions (Bryden, 2012). Mycotoxin contamination occurs widely in feedstuffs of plant origin, especially in cereals, fruits, hazelnuts, almonds, seeds, fodder, and other agricultural feed or food intended for animal or human consumption (Guan et al., 2011; Wu et al., 2013, 2014, 2015a,b). It is also worth noting that human exposure to mycotoxins may be caused by not only consumption of plant-derived foods contaminated with toxins, but also the carry-over of mycotoxins and their metabolites in animal products, such as animal tissues, milk and eggs (CAST, 2002). Moreover, mycotoxins lead to huge economic losses annually, including loss of human and animal life, loss of livestock production, loss of forage crops and feeds, and so on (Mohamed, 2011).

It is well known that not all molds are toxigenic and not all secondary metabolites from molds are toxic (Mohamed, 2011). Currently, more than 300 mycotoxins have been identified, and scientific attention is focused mainly on the mycotoxins that have proven carcinogenic and/or toxic. Thus, aflatoxins (AF), zearalenone (ZEA) and deoxynivalenol (DON) elicit great public health concerns due to their high prevalence, and their teratogenic, carcinogenic, mutagenic and immunosuppressive effects (Miazzo et al., 2000; Oueslati et al., 2012).

Aflatoxins, produced mainly by Aspergillus flavus and Aspergillus parasiticus (Wogan and Pong, 1970), are recognized as the most hazardous mycotoxins. The liver is the primary target organ for AF. Long-term intake of feeds contaminated with AF results in negative effects on the liver, such as hepatic cell and tissue injury, as well as gross and microscopic abnormalities (Williams et al., 2011; Gholami-Ahangaran et al., 2016). In classical epidemiology, researchers revealed the relationship between AF exposure and high prevalence of human liver cancer in various areas of Asia and Africa (Pitt, 2000). Zearalenone, produced by several Fusarium species, in particular Fusarium graminearum and Fusarium culmorum,
competitively binds to estrogen receptors causing reproductive disorders and estrogenic dysfunction in humans and animals (Borutova et al., 2012; Schoevers et al., 2012). Swine have been shown to be very sensitive to ZEA. Zearalenone may lead to swelling of the vulva and mammary glands, vulvovaginitis, vaginal and/or rectal prolapse, disrupted conception, abortion and infertility in swine (Stoev, 2015). Deoxynivalenol, also produced mainly by Fusarium species, is one of the most frequently detected trichothecene contaminants in grains (Mishra et al., 2013). The ingestion of low or moderate dose of DON causes nausea, diarrhea, gastrointestinal tract lesions, decreased nutritional efficiency, and weight loss in animals. Higher dose of DON induces vomiting and feed refusal with severe reduction in weight, severe damage in the hematopoietic systems and immune dysregulation (Maresca, 2013; Wu et al., 2015a,b). Swine is the animal species most severely affected by DON.

In an eight-year study on occurrence of mycotoxins in feed and feed raw materials worldwide, a total of 56,672 analysis were conducted on 17,316 samples for the determination of AF, ZEA, DON, fumonisins and ochratoxin A (Streit et al., 2013). The results showed that 72% of the samples tested positive for at least one mycotoxin, and 38% were found to be co-contaminated with multi-mycotoxins. In a recent paper, Li et al. (2014) reported that DON, aflatoxin B1 (AFB1) and ZEA were detected at percentages of 97%, 70% and 100%, respectively, in feeding ingredients and complete feeds obtained from swine farms in the Beijing region of China. The co-occurrence of several mycotoxins occurs frequently because of the ability of a given mold species to produce several kinds of mycotoxins in one type of food ingredient. Besides, the complete feeds, made from different contaminated ingredients or raw materials, may also have several types of mycotoxins. Co-contaminated feeds probably lead to adverse effects even though the concentration of the individual mycotoxin does not surpass legal maximum limit (Streit et al., 2013).

Numerous traditional physical and chemical strategies for the elimination or inactivation of mycotoxins have been reported in the literature (Stoev, 2013). Nevertheless, these methods have some limitations concerning safety issues, losses in the nutritional value and the palatability of feeds, coupled with limited efficacy and cost implication. In recent years, using mycotoxin-adsorbing agents, to bind mycotoxins in gastrointestinal tract of animals and then decrease their bioavailability and toxicities, shows a promising potential in feed industrial applications. However, there are various kinds of adsorption agents and their efficacy in preventing mycotoxicosis varies (Mohamed, 2011). Adsorption agents are very useful to prevent aflatoxiosis, but are not very effective for other mycotoxins (Stoev, 2013). Therefore, seeking for an effective, specific, feasible and environmentally sound decontamination technology is in a great demand. The biological detoxification of mycotoxins, using microorganisms and/or enzymes to degrade mycotoxins to non- or less toxic compounds, can be a choice of such technology (Taylor and Draughon, 2001). This review is mainly focused on the biological detoxification of AF, ZEA and DON.

2. Biological degradation of aflatoxins

2.1. Fungi

A number of fungal species have shown an ability to degrade AF (Mishra and Das, 2003; Wu et al., 2009). Fungi Aspergillus species, including A. parasiticus, Aspergillus flavus, A. flavus and Aspergillus niger, were reported early to be able to degrade AF. Doyle and Martín (1978) found that the mycelial blend of A. parasiticus NRRL 2999 could degrade AFB1 and AFG1 in the reaction mixture, and the degradation rates of AFB1 and AFG1 proportionally increased with the increase of the initial aflatoxin concentration or the size of mycelia inoculum. Then, Huynh et al. (1984) extracted a crude mycelial protein, which could detoxify AFB1, from a 16-day-old culture of A. parasiticus using (NH4)2SO4 at 80% to 100% precipitation. In their study, the main breakdown product of AFB1 was isolated, and was confirmed to be non-fluorescent, non-mutagenic, and non-toxic for ducklings. The result showed that the dialyzed mycelial protein destructed AF by degradation of the cyclopentene moiety, principally the lactone ring, via infrared spectral analysis of the main breakdown product. Mann and Rehm (1976) reported another species of Aspergillus, A. niger, was able to convert AFB1 into aflatoxicol (AFL). Zhang et al. (2014) screened a strain of A. niger (ND-1) that could degrade 58.2% of AFB1 after 48 h of fermentation. The degradation activity of A. niger was significantly stronger in culture supernatant than cells and cell extracts, and affected by heat treatment, temperature, pH, and metal ions, indicating that the degradation reaction is enzymatic and this process mainly occurs in the extracellular environment. Mishra and Das (2003) have reviewed other Aspergillus species possessing capability to biodegrade AF.

Nakazato et al. (1985) investigated the effect of incubation period on the conversion of AFB1 into AFL by Eurotium rubrum. The accumulation of AFL in the cultures began on the 2nd day, rapidly increased between the 6th day and the 8th day, and then gradually decreased toward the 15th day, the end of the incubation period. Phoma sp. not only prevented AF formation but also degraded preformed AF (Shantha, 1999). When the reaction mixture containing cell-free extracts of Phoma sp. and AFB1 was incubated for 5 days without and with heating in boiling water for 10 min, significant degradation was observed in both the cases. This suggested a possible role for a heat stable enzyme in the degradation. Reports on other fungi detoxifying AF have been reviewed by Wu et al. (2009). Briefly, Penicillium raistrickii NRRL 2038 could convert AFB1 to a compound that is similar to AFB2 (Ciegler et al., 1966). Dactylium dendroides (NRRL 2575), Mucor griseocyanus (NRRL 3359), Mucor alternans (NRRL 3358), Absidia repens (NRRL 1336), Helminthosporium sativum (NRRL 3356), Muco rambigous (J.M.M. 115), and Trichoderma viride (ATCC 13223) could transform AFB1 to a new fluorescent-blue compound AFRe, namely AFL, through a carbonyl reduction in the cyclopentane ring (Detroy and Hesseltine, 1969; Mann and Rehm, 1976). The conversion of AFG1 to AFB1 (parasiticol) by Rhizopus stolonifer (NRRL 1477), Rhizopus arrhizus (NRRL 2585) and Rhizopus oryzae (NRRL 395) was studied by Cole and Kirksey (1971). Cole et al. (1972) revealed that two fluorescent metabolites of AFB1 accumulated during degradation by R. stolonifer, R. arrhizus and R. oryzae, were identified as hydroxylated stereo isomers derived from reduction of the ketone function on the cyclopentane ring of AFB1. Rhizopus oligosporus [F0216] was able to inhibit synthesis of AFB1 or to degrade AFB1 (Kusumaningtyas et al., 2006). Some white rot fungi exhibiting high laccase activity, e.g., Peniophora and Pleurotus ostreatus, also had the potential to degrade AFB1 (Alberts et al., 2009).

2.2. Bacteria

Aflatoxins can be metabolized by certain species of Actinomy- cetales, such as Nocardia corynebacterioides (formerly classified as Flavobacterium aurantiacum), Nocardia asteroides, Corynebacterium rubrum, Rhodococcus erythropolis, Mycobacterium fluo- ranthenivorans, and Mycobacterium smegmatis. C. rubrum eliminated more than 99% of the added AFB1 (1.48 mg/mL) in a liquid culture after 4 days (Mann and Rehm, 1976). Kong et al. (2012) adopted Plackett–Burman design and central composite design to screen the key factors and identify the optimal conditions for degradation of AFB1 by R. erythropolis, and eventually the AFB1...
degradation efficiency was increased from 28.7% to 95.8%. Taylor et al. (2010) identified nine *M. smegmatis* enzymes that utilized the deazaflavin cofactor F420H2 to catalyze the reduction of the α,β-unsaturatededester moiety of AF, activating the molecules for spontaneous hydrolysis and detoxification. These enzymes belonged to two F420H2 dependent reductase (FDR-A and -B) families. In another work of this team (Lapalikar et al., 2012), they indicated that the FDR-A enzymes had up to 100 times more activity than the FDR-B enzymes, meanwhile, they tested another ten FDR-A enzymes from other *Actinomycetales* and found that nine of them can also reduce AFB1, and AFG1. This suggested that AF reducing activity might be widespread in this family of enzymes and the order of *Actinomycetales*, as the family of enzymes was itself widespread in the *Actinomycetales*. More studies on other *Actinomycetales* for elimination of AF have been discussed in the reviews by Bata and Lasztity (1999), Mishra and Das (2003), and Wu et al. (2009).

It was reported that *Pseudomonas aeruginosa* N17-1 could degrade AFB1, AFB2 and AFG1 by 82.8%, 46.8% and 31.9% after incubation in Nutrient Broth medium at 37°C for 72 h, respectively (Sangare et al., 2014). Some *Bacillus* sp. also displayed aflatoxin reduction activity, such as *Bacillus subtilis* (Fan et al., 2013, 2015). *B. subtilis* 168 showed a capability of detoxifying AF with microorganisms, our research team has intensively studied bacterial isolates capable of biodegrading AF. Guan et al. (2008) used cumarin as the sole carbon source to screen the bacterial isolates with AFB1 reduction activity. And they found that *Stenotrophomonas maltophilia* 35-3, obtained from tapir feces, could reduce AFB1 by 82.5% after incubation in the liquid medium at 37°C for 72 h. Its culture supernatant was able to degrade AFB1 effectively, whereas the viable cells and cell extracts were far less effective. And treatments with proteinase K, proteinase K plus sodium dodecyl sulfate (SDS) and heating significantly reduced or eradicated the degradation activity of the culture supernatant, indicating that the degradation of AFB1 by *S. maltophilia* 35-3 was enzymatic. *Myxococcus fulvus* ANSM068, isolated from deer feces, was also able to transform AFB1 (Guan et al., 2010). Liquid chromatography mass spectrometry and infrared analysis indicated that AFB1 was transformed to a structurally different compound, and the lactone ring on the AFB1 molecule was modified by the culture supernatant. In addition, Gao et al. (2011) isolated a strain of *B. subtilis* ANSB060 from fish gut showing a strong ability to detoxify AF, and percentages of AFB1, AFG1, and AFB2 degradation were 81.5%, 60%, and 80.7%, respectively. Also, ANSB060 showed antimicrobial activities against Escherichia coli, Salmonella typhimurium, *Staphylococcus aureus* and resistance to the simulated gut environments. Moreover, the protective effects of supplementation of *B. subtilis* ANSB060 in the aflatoxin-contaminated diets on layers and broilers have been verified well in the studies in vivo (Ma et al., 2012; Fan et al., 2013, 2015).

### 2.3. Enzyme

A kind of pure laccase enzyme from *Trametes versicolor* (1 U/mL) could degrade AFB1 by 87.34% after incubation for 72 h (Alberts et al., 2009). Zeinwand-Lorestani et al. (2013) found that the optimal enzymatic reaction occurred in 0.1 mol/L of citrate buffer containing 20% dimethyl sulfoxide (DMSO) at 35°C, a pH of 4.5, and a laccase activity of 30 U/mL, and after two days, 67% of AFB1 was removed. Manganese peroxidase (MnP) from the white-rot fungus *Phanerochaete chrysosporium* (YK-624) could first oxidize AFB1 to AFB1-8, 9-epoxide and then hydrolyze to AFB1-8, 9-dihydrodiol (Wang et al., 2011). Similarly, Yehia (2014) purified MnP from white rot edible mushroom *P. ostreatorius*, with the final enzyme activity achieving 81 U/mL and specific activity achieving 78 U/mg. The highest detoxification power (90%) was observed after incubation for 48 h at 1.5 U/mL enzyme activities. As well, The MnP-cDNA encoding gene was sequenced and determined (GenBank accession No. AB698450.1). Wu et al. (2009) also reviewed an AF degradation enzyme from *P. ostreatorius* in the earlier time, and an enzyme named aflatoxin-detoxifyzyme (ADTZ) from *Armillariella tabescens* (E-20). Zhao et al. (2011) prepared and purified an extracellular enzyme, designated as myxobacteria aflatoxin degradation enzyme (MADE), from bacterium *M. fulvus* ANSM068 with a final specific activity of 569.44 × 10⁻³ U/mg. The pure enzyme (100 U/mL) demonstrated high degradation ability against AFG1 (96.96%) and AFB1 (95.80%) after 48 h of incubation. Enzyme MADE demonstrated high activity in a broad range of pH values between 5.0 and 7.0, and temperatures between 30 and 45°C.

Recombinant laccase produced by A. niger D15-Lcc2#3 (118 U/L) decreased AFB1 by 55% in 72 h (Alberts et al., 2009). Aflatoxin-detoxifyzyme ADTZ from *A. tabescens* was confirmed to be an oxidase and renamed aflatoxin-oxidase (AFO). Wen et al. (2011) cloned the cDNA of AFO, and expressed this enzyme as a fusion protein in *Pichia pastoris* by using pPIC9-AO as vector. The recombinant AF oxidase converted AFB1, with the Km value of 3.93 ± 0.20 × 10⁻¹ mol/L under its optimal conditions of pH 6.0 and 30°C.

### 3. Biological degradation of zearalenone

#### 3.1. Fungi

Some fungi that exhibit degradation ability against ZEA have been reviewed by Zinedine et al. (2007). Indeed, *Thamnidium elegans* (NRRL 1613) and *Mucor bainieri* (NRRL 2988) could transform ZEA into a non-estrogenic substance ZEA-4-O-glucoside. Zearalenone was converted to 8'-hydroxy-zearalenone and 2, 4-dimethoxyzearalenone by *Streptomyces rimosus* (NRRL 2234) and *Cunninghamella bainieri* (ATCC 9244B), respectively. Gliocladium roseum (synonym: *Clonostachys rosea*) could cleave the lactone ring of ZEA, and result in subsequent spontaneous decarboxylation, rendering the reaction irreversible. Utermark and Karlovsky (2007) also reported that *G. roseum* produced a zearalenone-specific lactonase which protected *G. roseum* from the inhibitory and toxic effects of ZEA. The lactonase could catalyze the hydrolysis of ZEA, followed by a spontaneous decarboxylation. *Trichosporon mycotoxinivorans* had a very high capability to degrade ZEA. Furthermore, the metabolite of ZEA by *T. mycotoxinivorans* has been investigated by Vekiru et al. (2010). The authors reported that the novel ZEA metabolite, named ZOM-1, was characterized by an opening of the macrocyclic ring of ZEA at the ketone group at C6, and identified as (5S)-5-{2,4-dihydroxy-6-[(1E)-5-hydroxypent-1-en-1-yl]benzoyl}oxo hexanoic acid. In addition, the ZOM-1 metabolite was not estrogenic in vitro and did not interact in vitro with the estrogen receptor protein, showing the loss of ZOM-1 estrogenicity. Zearalenone was found to be completely degraded by several Rhizopus isolates including *R. stolonifer*, *R. oryzae* and *Rhizopus microsporus* strains. El-Sharkawy et al. (1991) demonstrated that *R. arrhizus* (IFO-6155) could catalyze sulfation of ZEA at the C-4 hydroxyl group, forming a new metabolite determined as ZEA-4-O-sulfate conjugate. As well, A. niger was able to detoxify ZEA by sulfonation, leading to a less toxic compound (Jard et al., 2009).

#### 3.2. Bacteria

A few studies on the bio-transformation of ZEA by bacteria have been reported, and the strains in these studies include the bacterial gut flora of pigs (Kollarzick et al., 1994), mixed culture (*Alcaligenes*, *Bacillus*, *Achromobacter*, *Flavobacterium*, and *Pseudomonas*)
(Megharaj et al., 1997), Acinetobacter sp (Yu et al., 2011a), and so on. Recently, Cserháti et al. (2013) have reported that three Rhodococcus species R. erythropolis, Rhodococcus ruber and Rhodococcus pyridinivorans had >50% ZEA degrading capacity, thereinto, R. pyridinivorans strains were the most effective degraders with 70% efficiency. Pseudomonas bacteria are also capable of ZEA biodegradation. Tan et al. (2014) isolated two ZEA degrading bacteria from gyttja soil and moldy soil, named as Pseudomonas alcaliphila TH-C1 and Pseudomonas plecoglossicida TH-L1, and their degradation rate for ZEA (2 μg/mL) was about 68% and 57%, respectively, when incubated for 72 h. Altalhi (2007) described a Pseudomonas strain, Pseudomonas putida ZEA-1 isolated from rhizosphere of corn plant, is able to utilize ZEA as energy source and transform ZEA along with its derivatives, including α- and β-zearalenol, without generating harmful metabolites. Later, Altalhi and El-Deeb (2009) localized and cloned the fragment containing genes encoding for ZEA detoxification in the plasmid of P putida ZEA-1, and the cloned genes were actively expressed in E. coli. The results showed that ZEA degradation by recombinant E. coli was relatively rapid and effective, leaving no detectable ZEA after 24 h. There were more than one target proteins induced through growth of recombinant E. coli, however, the authors were unable to separate these genes, indicating that the genes are organized in a complex structure and probably the degradative genes are clustered in one or more large operons in bacteria. The efficient abilities of B. licheniformis CK1 (Yi et al., 2011) and B. subtilis (Cho et al., 2010) to degrade ZEA in the culture media were also reported. Our research team screened a strain of bacteria B. subtilis ANSB01G from normal broiler intestinal chyme, which could reduce ZEA by 88.65% (Lei et al., 2014). Under simulated intestinal tract conditions, the B. subtilis ANSB01G degraded 84.58%, 68.34% and 83.04% of ZEA in naturally contaminated corn, distiller's dried grains with solubles (DDGS) and swine complete feed, respectively. Moreover, the addition of ANSB01G to diet naturally contaminated with ZEA obviously alleviated adverse symptoms induced by ZEA in pre-pubertal female gilts (Zhao et al., 2015).

3.3. Enzyme
Takahashi-Ando et al. (2002) purified a novel lactonohydrolase in fungus C. rosea that is responsible for the detoxification of ZEA, and then cloned the encoding gene, designated as zhd101. Maximal activity of recombinant ZHD101 protein expressed in E. coli toward ZEA was observed at pH 10.5 with an extremely low molar activity (kcat = 0.51/s at 30°C) (Takahashi-Ando et al., 2004). Yu et al. (2011b) isolated enzymes from bacterium Acinetobacter sp. SM04 extracellular extracts of liquid cultures, and obtained an active fraction capable of efficiently degrading ZEA. The active fraction could degrade ZEA into smaller estrogenic products, and two intermediate products, ZEN-1 and ZEN-2, were found, which indicated that the benzene ring of zearalenone may be cleaved and oxidized into the products containing carboxyl groups. Further, enzymes in the active fraction were analyzed, and three proteins were found. They were identified as peroxiredoxin, a possible cytochrome and apputative fimbrial protein precursor.

4. Biological degradation of deoxynivalenol
4.1. Fungi
It was reported that the growing Fusarium nivale acetylated DON to afford a small amount of 3-acetyldeloxynivalenol (Yoshizawa and Morooka, 1975). The fungus Aspergillus tubingensis NJA-1, isolated from soil, demonstrated an ability to degrade DON. And the molecular weight of the biocconversion metabolite was 18.1 D (H2O) larger than that of DON, showing that DON could be hydrolyzed by NJA-1 (He et al., 2008).

4.2. Bacteria
To date, several microorganisms from various sources such as soils, animal guts, and plants have been reported to have the ability to degrade DON. Zhou et al. (2008), He et al. (2010), Awad et al. (2010) and Karlovsky (2011) have reviewed the studies on DON degradation. Shima et al. (1997) inferred that 3-OH group in DON is likely to be involved in exerting its immunosuppressive toxicity. Agrobacterium–Rhizobium strain E3-39 was obtained from soil samples by an enrichment culture procedure (Shima et al., 1997). The strain E3-39 could oxidize the 3-OH group of DON to generate 3-keto-4-deoxynivalenol (3-keto-DON), which exhibited a remarkably decreased (to less than one tenth) immunosuppressive toxicity relative to DON. Among 1,285 microbial cultures obtained from farmland soils, cereal grains, insects and other sources, Volkl et al. (2004) screened one mixed culture from spontaneously infected minimal medium with protein and glucose (MMGP), able to transform DON into 3-keto-DON. The strain could also transform trichothecenes 15-acetyl-DON, 3-acetyl-DON and fusarenon-X. However, the microorganisms responsible for the biotransformation have not been identified. The bacterium Nocardoides WSN05-2, isolated from soil samples collected in wheat fields, degraded DON to produce a novel intermediate, 3-epi-DON (Ikunaga et al., 2011). 3-epi-DON is an epimer of DON, which is different from DON in the stereochemistry at the 3-OH group. Recently, He et al. (2015) screened a bacterium Devosia mutants 17-2-E-8 from an agricultural soil. The bacterium was capable of transforming DON to 3-epi-DON (major product) and 3-keto-DON (minor product). And the metabolite 3-epi-DON was proved to be less toxic than DON through in vitro and in vivo studies. A total of thirteen aerobic DON-degrading bacteria were isolated from a variety of environmental samples, including field soils and wheat leaves (Sato et al., 2012). Of these thirteen strains, nine belonged to the Gram-positive genus Nocardoides and other four to the Gram-negative genus Devosia, indicating that aerobic DON-degrading bacteria were distributed within at least two phylogenetically restricted genera.

Microorganisms in rumen fluid from different ruminants could transform DON. The C-12, 13-epoxy group is essential for the toxicity of DON. Yoshizawa et al. (1983) were the first to characterize the deepoxidated metabolite of DON, namely DON-1, in rat urine and feces. The rumen fluid from a cow transformed all DON (up to 10 ppm) to DOM-1 within a 24-h period (King et al., 1984). The deepoxidation of DON by rumen fluid was also described by Coté et al. (1986) and Swanson et al. (1987). So far, Eubacterium strain BBSH 797 is the most intensively studied bacterial isolate able to transform DON. The strain was isolated from cow rumen fluid (He et al., 1992) and its capability of transforming DON into DOM-1 was proven in vitro and in vivo (Schatzmayr et al., 2006; Zhou et al., 2008), Fuchs et al. (2002) found that Eubacterium BBSH 797 could transform the epoxide group of trichothecenes into a diene. A BBSH 797-based commercial mycotoxin-deactivating feed additive product is available on the market. Awad et al. (2004, 2006) demonstrated that the Eubacterium sp. DSM 11798 could counteract the adverse effect of DON on poultry.

In the past three decades, transformation of DON by microorganisms from animal intestines has also been studied. Lun et al. (1988) observed a decrease in DON, when it is incubated with the fluids obtained from the main parts of hen gastrointestinal tract. Microbiota from chicken large intestines (He et al., 1992) and pure cultures of microbial isolates (LS100 and SS3) from chicken intestine (Young et al., 2007) showed the capacity for degrading DON by deepoxidation. Yu et al. (2010) utilized conventional
microbiological selection strategies guided by PCR-DGGE (denaturing gradient gel electrophoresis) bacterial profiles to isolate DON-transforming bacteria. And 16S rRNA gene sequence analysis indicated that the ten isolates obtained belong to four different bacterial groups, Clostridiales, Anaeroflobium, Collinsella, and Bacillus. Moreover, Bacillus sp. LS100 could detoxify DON in contaminated feed before feeding pigs and thus completely eliminate the adverse effects of DON on pigs (Li et al., 2011). Studies have also shown that DON can be transformed to DOM-1 by intestinal microorganisms of other animal species including rat (Worrell et al., 1989) and pig (Kollarzczik et al., 1994). In addition, Guan et al. (2009) screened one microbial community from catfish (Amiurus nebulosus) digesta, namely microbial culture C133, completely transforming DON to de-epoxy-DON after 96 h incubation.

4.3. Enzyme

3-O-acetylation of the trichotheccene ring in DON leads to its inactivation. Gene tri101 encoding trichothecene-3-O-acetyltransferase from F. graminearum was characterized (Kimura et al., 1998). Khatibi et al. (2011) cloned trichothecene 3-O-acetyltransferases genes from seven Fusarium species and compared the properties of them to identify an optimal source of the enzyme for biotechnological applications. A UDP-glucosyltransferase from Arabidopsis thaliana catalyzed transfer of glucose from UDP-glucose to the hydroxyl group at carbon 3 of DON (Poppenberger et al., 2003). However, whether acetylation of C3-OH or conjugation by glycosylation can be considered as detoxification is controversial, because acetylated and conjugated mycotoxins may be hydrolyzed and regenerated the toxins in the digestive system of animals and humans.

5. Perspective

Microbial detoxification may become a promising choice, since it can be a specific, effective, irreversible and environmental friendly strategy of detoxification that leaves no toxic residues. Despite the many publications on biological transformation of mycotoxins by microorganisms, their application in practice in detoxification of food and/or feed has been limited. Practical applications of fungi may be limited by factors, such as complicated procedures needed for obtaining the active extracts, long incubation time required for the detoxification, and incomplete detoxification process. The high degradation rate and wide reaction conditions for degradation by bacteria imply a potential and promising application for mycotoxin degradation. Innovative techniques and strategies, such as enrichments, highly selective media, PCR-DGGE bacterial profiles and effective molecular techniques, may increase the opportunities to select target microorganisms from a complex microflora. It is also of great interest to develop studies focusing on probiotic bacteria, which can be directly applied in the feedstuffs and feeds. In addition, the use of active enzyme(s) seems to be a promising opportunity for mycotoxins degradation. Many enzymes with certain function, such as phytase, have been purified from fermentation broth, and successfully applied in food and feed industries. In this respect, enzyme products for the effective detoxification of mycotoxins can be developed through preparation and purification of enzyme, identification of the genes encoding the enzyme, and overexpression of the recombinant enzyme. Meanwhile, the studies on mechanisms of biodegradation, structure of degradation products, and safety of the microorganisms and degradation products towards animals are still warranted to support the application of microorganisms in food and feed industry.

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