

MANAGING MYCOTOXINS IN NORTHEAST SILAGES

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INTRODUCTION

Dairy producers and those in the agricultural support industries have noted the presence of mycotoxins in silages as a significant concern for dairy production. The perception that mycotoxins are present in silages and that they have negative impacts on production and animal health is widespread. Research to document this is limited but that information which is available certainly supports these observations. Silage mycotoxins can result in losses due to reduced milk production, missed breeding cycles, abortions, veterinary fees, testing costs, the use of feed additives and in some cases cost of feedstuff replacement. Loss estimates for the Vermont dairy industry are between \$4.5 and 9 million per year (Thomas et al., 1998). This paper will provide an overview of silage mycotoxins, what is known about their effects on dairy cattle and suggest strategies for preventing their accumulation.

FUSARIUM MYCOTOXINS

Mycotoxins are chemicals produced by fungi that are toxic to humans and animals. The three main genera of mycotoxin-producing fungi are *Aspergillus*, *Fusarium* and *Penicillium* (CAST, 1989). Over 400 different mycotoxins have been identified however only a small fraction of these appear to have significant impact on agricultural activities. The most commonly encountered mycotoxin in silages is deoxynivalenol, also known as DON or vomitoxin (Whitlow and Hagler, 1997). DON is produced by several species of *Fusarium* including *F. graminearum* (= *Gibberella zeae*), and *F. cerealis* (formerly known as *F. crookwellense*). *F. graminearum* is the most common producer of DON in the Northeastern United States. This fungus causes head scab of wheat and barley as well as ear and stalk rot of corn. Its name means "Fusarium of the grasses" and it will infect nearly any grass including those used to make hay and haylages. Thus, DON is a potential contaminant of any silage, haylage or big bale. Infection of wheat, barley, and grasses occurs at the time of flowering and is favored by moderately cool, wet conditions. Spores of *F. graminearum* are introduced to the flowers by wind and rain splash. Infection of corn can occur through the silks, insect damage to ears and stalks, and bird damage of ears. Since *F. graminearum* overwinters on plant material left in the field no-till and minimum till operations tend to be at greater risk for these diseases unless effective crop rotation is practiced. Useful crop rotations would be those that include non-graminaceous crops such as alfalfa, soybeans or potatoes.

Deoxynivalenol contamination of dairy cattle feeds does not appear to have significant effects on milk production, milk quality, feed intake or animal health. Feeding studies utilizing DON contaminated feeds with early lactation (Ingalls, 1996), mid-lactation (Charmley et al, 1993) and

non-lactating cows (Trenholm et al, 1985) all came to similar conclusions. Ingalls reported that diets containing as much as 14.6 mg/kg DON in concentrates had no effect on feed intake or milk production. Trenholm et al. (1985) stated that mature non-lactating dairy cows were relatively insensitive to DON and noted that their observations were consistent with those of two earlier studies (Noller et al., 1979; Stojanovic, 1974). Charmley et al. (1993) observed no effect on feed intake or milk production when cows between week 13 and 22 lactation were fed diets containing a total of 6.4 mg/kg DON. None the less, many producers have observed a correlation between DON in rations and problems with reduced milk production, feed intake and herd health. It is likely that when DON is present other mycotoxins are present as well. Thus DON appears to be a marker for the presence of other toxic factors in feeds.

There are several possible explanations for the discrepancy between controlled research studies and on-farm observations regarding the effects on DON-contaminated feeds on dairy cattle. It is known that rumen microorganisms can degrade DON to a form that may be less toxic (Swainson et al., 1987). However, if the transit time of feed in the rumen is short, the resident microbes may have insufficient time to act on the DON present. Animals used in research studies may not be producing at as high a level as those on farms where problems are observed. Research cows may receive superior care that is not warranted for farm animals and this may bias results. Finally, as mentioned above it is very likely that if DON is present other mycotoxins are present as well since conditions for the growth of fungi on the feed occurred at some point either in the field, in storage or possibly at both times. Most mycotoxigenic fungi produce multiple mycotoxins. For example, *F. graminearum* the fungus that produces DON also produces the estrogenic mycotoxin zearalenone. This mycotoxin is not acutely toxic but is known to cause reproductive problems in many animal species. Some co-occurring mycotoxins may also have synergistic effects. Fusaric acid is a weak mycotoxin produced by most species of *Fusarium* and thus may be present in feeds containing fumonisin, deoxynivalenol, and or zearalenone. Studies with rats, mice and swine have shown that fusaric acid can act synergistically with deoxynivalenol and zearalenone (Smith et al., 1997; Porter et al., 1998). The effects of these combinations of mycotoxins on dairy cattle are currently unknowns but warrant study. There may be other mycotoxins that are not routinely tested for or that we are simply unaware of that could also have additive or synergistic effects with DON.

Other *Fusarium* mycotoxins that have been found in silage are fumonisin B1 and T-2 toxin. T-2 toxin producers include *F. sporotrichioides* and *F. poae*. T-2 toxin is very toxic and causes a hemorrhagic syndrome in animals including dairy cattle. Since T-2 toxin induced hemorrhagic syndrome sometimes results in death of the effected animal it is of serious concern when it occurs. Anecdotal evidence indicates however that T-2 toxin is not encountered frequently. A related compound diacetoxyscirpenol (DAS) is also quite toxic and has similar effects. The fungi that produce these toxins are most commonly associated with grasses. As such haylages and big bales are probably more likely to contain this toxin than corn silage. Fumonisin B1 is not generally considered to be a health threat to dairy cattle at the concentrations most often encountered in the Northeast. It is possible however that fumonisins may act in concert with other mycotoxins resulting in fumonisin toxicities at much lower levels.

Attention to agronomic practices in silage crops is the first step in producing quality forages. The majority of the mycotoxigenic *Fusarium* species encountered in Northeast agricultural settings are weak to virulent plant pathogens. Some general guidelines about agronomic practices are applicable to most if not all of the *Fusarium* toxins discussed in this section. Strategies to

maintain plant health and reduce plant stress such as maintaining adequate soil moisture and fertility should be employed. It should be noted that in drought years some mycotoxins levels tend to be higher. This is probably related to plant stress and possibly to fungal physiology. If possible choose disease-resistant varieties. Foliar disease may also stress plants making them more susceptible to invasion by other pathogens. If a no-till or minimum-till program is being used it is important to incorporate non-grass species into the rotation. Many mycotoxigenic *Fusarium* species over-winter in crop residue. If host plants such as corn, wheat, and hay are planted continually then the fungal inoculum levels in the field may build up to levels that simply cannot be managed by other means. Corn ear rot infections often start at the site of insect or bird damage to the ear. Control of insect pests such as the European corn borer will likely reduce mycotoxin levels in the crop.

PENICILLIUM MYCOTOXINS

Penicillium roqueforti is the most commonly found fungus in silage. Unlike the *Fusarium* species discussed in the previous section this fungus is a saprophyte and is not a plant pathogen. *P. roqueforti* thrives in acidic, low oxygen tension environments and for this reason is well-adapted to growth in silage. *P. roqueforti* produces at least four mycotoxins all of which have been documented in silage. The toxins are PR toxin, roquefortine C, patulin and mycophenolic acid. The effects of these mycotoxins on dairy cattle are not currently well understood. Rapid immunoassays such as ELISA are not commercially available for these toxins and as a result the possibility that they are the source of problems with spoiled silage is rarely considered.

Among the *P. roqueforti* toxins PR toxin is considered the most toxic based on studies with laboratory animals. It has been found in silages in the United States Midwest and in Europe (Yu et al., 1997). Although its impact on dairy cattle is not documented it has been implicated in bovine abortion and placental retention. Research is needed to determine if PR toxin does in fact cause these problems and if so and what levels. Roquefortine C and the related compounds roquefortine A, B and D have been found in silage in Europe and Japan (Auerbach et al., 1998; Ohmomo et al., 1994). Roquefortine C is a neurotoxin and it has been suggested it may have paralytic effects on cattle. This claim needs to be addressed through controlled research studies. The mycotoxin patulin is made by *P. roqueforti* as well as by other fungi found in silage such as *Paecilomyces varioti* and *Byssochlamys spp* (e.g. see Muller and Amend, 1997). Its presence has been linked to hemorrhagic syndromes in cattle but as with the other *Penicillium* toxins this idea needs to be substantiated by controlled research. Finally, mycophenolic acid has been found in corn and grass silage in Germany (Schneweis et al., 2000). This compound is known to be highly immune-suppressive and is used for immune system repression in organ transplant patients. It is not known if the levels of mycophenolic acid found in silages are high enough to be of concern with respect to dairy cattle health however. Mycotoxins produced by *P. roqueforti* appear to at least have the potential for considerable negative impact on dairy cow health. Hopefully future research will clarify the impacts of the toxins on dairy production.

Management of *P. roqueforti* growth and mycotoxin accumulation is achieved through the use of sound ensiling practices. Silos should be cleaned prior to filling and checked regularly for evidence of mold growth. Moldy, spoiled, silage should not be fed. Forages should be ensiled at the proper maturity, moisture content and chop length to ensure good packing and thus air exclusion from the silo. Silo coverings should be maintained to prevent air leaks and rainwater

entry. In some situations, the use of bacterial silage inoculants may be helpful in achieving a good fermentation. Good ensiling practices will exclude oxygen from the silo and inhibit fungal growth of all types.

AFLATOXIN AND OTHER MYCOTOXINS

Aflatoxin is arguably the most important mycotoxin worldwide. It is the only mycotoxin declared a known human carcinogen by the International Agency for Cancer Research (IARC) and the only mycotoxin subject to enforceable regulations of the United States government. Aflatoxin is of concern to dairy producers in particular because the US FDA also regulates aflatoxin residues in milk and the limits set are quite low (0.5 ppb). *Aspergillus flavus* and *A. parasiticus* produce aflatoxin. These fungi prefer to colonize oil rich seeds and are found most frequently on corn, cotton and peanuts. Colonization and subsequent aflatoxin production is favored by hot and dry conditions. Although this mycotoxin has been found in silages it is unusual to find it on corn crops produced in the Northeast particularly in the more northern or higher elevation areas because the climatic conditions that favor it do not occur frequently. Aflatoxin in the diet is still of concern however since it can be introduced on concentrates produced in other warmer locations. The most likely sources are grain corn, peanut meal and cotton seed. The agronomic practices described for prevention of *Fusarium* toxins apply to prevention of aflatoxin as well. Plant stress, poor soil fertility and moisture, and insect and bird damage will all favor *Aspergillus* growth and aflatoxin production.

Ochratoxin A is a nephrotoxin produced by several species of *Penicillium* and *Aspergillus* (CAST, 1989). It has been found in US corn silage. Production probably occurs during fermentation and storage. Although this is a fairly toxic compound, concern for dairy cattle is somewhat moderated by the knowledge that rumen microorganisms are capable of metabolizing ochratoxin A. In addition, anecdotal evidence suggests that ochratoxin A is not encountered very often in Northeast silages.

AAL toxin is produced by *Alternaria alternata*, a common plant pathogenic fungus and occurs primarily on the aerial parts of plants. AAL toxin has been found in silage in the United States. One study conducted in the Midwest on hay and silage indicated that AAL toxin is found almost as frequently as DON (Yu et al., 1997). The toxicity of AAL toxin to dairy cattle is not known but should be explored given the apparent abundance of this toxin.

Ergot alkaloids are a complex group of mycotoxins produced by *Claviceps purpurea* and other related fungi (Kuldau and Bacon, 2000). *C. purpurea* infects nearly all grasses including barley, rye, wheat and grasses used for hay and haylage. Rye is known to be especially susceptible. This fungus infects through the flower and infection is favored by cool damp conditions at the time of flowering. After the infection is established *C. purpurea* produces a structure called a sclerotia, also known as an ergot, in the location where the seed would have formed. The ergot is similar in shape to the grain but it is usually slightly to significantly larger, and is hard and dark purple or black in color. At harvest time the ergot is collected along with the grain, hay or haylage. When consumed ergot alkaloids cause reproductive problems in cattle. Difficulties resulting from insufficient blood flow to the extremities can also occur. In severe cases this can result in the loss of tails, ears and feet or hooves. Reduced milk production and hyperthermia are also symptoms of ergotism. Ergotism from barley was reported in 1997 in Iowa and

southwestern Wisconsin that effected over 1000 dairy cattle (Munkvold et al., 1997). The incidence of ergot in Northeast haylages has not been formally documented but producers, crop consultants, nutritionists and others in the dairy industry should be aware of it and be prepared to look for sclerotia if cow symptoms suggest its presence. Selection of resistant varieties, mowing of field borders and deep plowing to bury sclerotia are all part of an effective management strategy for *C. purpurea*.

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