Molds and Mycotoxins in Feeds Harvested in 2009

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Due to the wet conditions in the Midwest and Northeast U.S. in 2009, mold and mycotoxin concentrations in many feeds and forages are higher than normal. Many feeds contain some mold. Problems come when these molds grow in grain or forage. Mycotoxins are poisons that are made by mold fungi. Mycotoxin poisoning is called mycotoxicosis. It is possible to see mold in a feed but have no mycotoxin problem. It is also possible to have a mycotoxin issue with no visible mold.

Factors Affecting Disease Development

A majority of the mold and mycotoxin problems associated with corn in the northeast and Midwest U.S. arise from two distinct diseases on ears of corn caused by Fusarium species. These are Fusarium ear rot (pink ear rot) and Gibberella ear rot (red ear rot), both which can cause mycotoxin contamination of grain, including vomitoxin (a.k.a. deoxynivalenol or **DON**), T2 toxin, zearealenone, and fumonisin. Fusarium ear rot is associated with *Fusarium verticillioides*, *F. subglutinans*, and *F. proliferatum*. Giberella ear rot is caused by *F. graminearum* and less importantly by *F. culmorum*. Gibberella ear rot predominates in cooler areas or where there is higher precipitation during the growing season. Fusarium ear rot is the most common disease found in corn ears and can be found at low levels of severity in nearly all corn fields in late season. Concentrations of mycotoxins in samples of forages, concentrate feeds, and TMR submitted to Cumberland Valley Analytical Services during fall 2009 are provided in Tables 1 and 2.

Corn crop residue is the primary source of inoculum for infection, and Fusarium species survive very well on this residue. As well, Fusarium species can colonize residues of other crop and weed species that are generally not considered hosts. The *F. verticillioides*, *F. subglutinans*, and *F. verticillioides* produce large numbers of microconidia and macroconidia on crop residues. These asexual spores comprise the primary inoculums for Fusarium ear rot and for infections that may be symptomless.

Seed is a possible but minor contributor to Fusarium infections compared to airborne spores infecting through the silks. Kernels can be infected from the soil, creating a systemic infection of the plants.

The *F. graminearum* grow ascospores and macroconidia that when escaping the crop canopy can be effective at inoculation after traveling long distances by air. It is estimated that viable spores of *F. verticillioides* have traveled as much as 300 to 400 km (180 to 240 miles) (Munkvold, 2003).

Insects play a key role in the dispersal of mold spores. A variety of insect hosts have been identified, including European corn borers, sap beetles, corn rootworm beetles, and western flower thrips. Rootworm beetles feed on corn silks where

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windblown spores have settled and then cause kernel infection. Sap beetles are attracted to corn ears that have been damaged by other insects. They appear to be attracted as well to volatile compounds produced by *F. verticillioides*. These sap beetles potentially acquire Fusarium spores from infected plant material and carry them to damaged kernels that are prone to infection (Munkvold, 2003).

The primary pathway of infection is by way of the silks which are highly susceptible during the first 6 days after emergence. Spores reach the silks by way of wind, insects, and splashing. In the central United States, the degree of Fusarium ear rot is closely correlated with insect injury. Field experiments in Iowa from 1996 to 2001 showed correlations between insect injury and Fusarium ear rot severity from 0.66 to 0.92. The relationship between insect injury and fumonisin presence was 0.50 to 0.77 (Munkvold, 2003).

Fusarium ear rot and Gibberella ear rot are supported by different environmental conditions. Fusarium ear rot is more common under warmer and drier conditions. Drought stress is associated with higher levels of *F. verticillioides* and fumonisin. Gibberella is seen where there are high levels of moisture at the time of silking with moderate temperatures and higher rainfall during ear maturation.

Hot, humid conditions, drought, insect damage, and other crop stress enhance the production of mycotoxins called aflatoxins (from *Aspergillus flavus* and *Aspergillus parasiticus*) in grains prior to harvest. Aflatoxin is associated with drought primarily because the bacteria and fungi that would normally compete against it do not grow as well in a dry climate. But, these *Aspergillus* fungi do like moisture and will grow and contaminate an entire bin of grain that has not been properly dried down. Lightweight grain with kernel damage (black tips) often has higher concentrations of aflatoxin. Since aflatoxin is a carcinogen, the legal limit set by the U.S. Food and Drug Association for aflatoxin is 20 ppb in dairy feeds and 0.5 ppb in milk. Aflatoxin is the only mycotoxin regulated by the FDA (Whitlow and Hagler, 2005).

Table 3 provides a list of common corn ear molds, their associated fungi, and their visual characteristics. The presence of various molds in samples of forages, concentrate feeds, and TMR submitted to Cumberland Valley Analytical Services are provided in Tables 4 and 5. Some nutritionists have feed and forage samples analyzed for the presence of particular fungi. If a mycotoxin-producing species is present, then there is cause for concern.

Molds and mycotoxins have many effects on the cow. They can reduce palatability of feeds and decrease intake. Molds reduce the nutritive value of feed because they use feed nutrients for their own growth. Mold counts in forages, concentrate feeds, and TMR samples sumbitted to Cumberland Valley Analytical Services are provided in Figures 1 through 5. Molds may also inhibit the rumen microbes and reduce the digestibility of the ration. Mycotoxins can cause hormonal and immunity problems in dairy cows, especially in dairy cows that are stressed. Mycotoxins can result in intermittent diarrhea, general unthriftiness, rough hair coats, and early embryonic death that shows up as irregular heat cycles in the cow.

Unfortunately, much of the time mycotoxins lead to chronic problems like 2 to 3 lb/day of lost milk per cow, some extra disease in the herd, or poorer reproductive performance. These issues often are not recognized or attributed to mycotoxins, but obviously, they can be very costly over time.

Much of the research on mycotoxins has been with adding a certain amount of a pure mycotoxin to a ration and then observing the effect. In the real world, however, most feeds are contaminated with more than one mycotoxin at a

time, and laboratories don't analyze for many of those mycotoxins. Some experts think the presence of vomitoxin may indicate the presence of other unknown mycotoxins (Seglar, 2003). Mycotoxin interactions can cause problems in the cow even when concentrations of individual mycotoxins are not above what is considered to be a concern. Mycotoxin concern concentrations by different experts are provided in Table 6.

Dealing with Mycotoxins

If there are any signs or symptoms of mold or mycotoxins in the feed or the cows, always try to rule out any other nutritional or management problems first. After that, if mycotoxins are still suspected, consider testing for mycotoxins, taking out the suspected problem feed from the diet or at least diluting it in the ration, and adding a mycotoxin binder to the diet.

Testing for mycotoxins

There are hundreds of different types of mycotoxins. Unfortunately, labs do not test for every single one. So, if the lab does not find high mycotoxin concentrations in a feed, it doesn't necessarily rule out a problem. Sometimes though, especially with suspected problems with purchased commodities, it is helpful to send feed samples to a lab to try to confirm a high concentration of mycotoxin. It is best to blend forage in a TMR mixer prior to sampling for mycotoxin analysis in order to obtain a truly representative sample rather than trying to take handfuls from the face of the bunk (Mahanna, 2007). It is also recommended that mycotoxin testing be conducted via a chromatography approach (high pressure liquid chromagraphy, **HPLC**; gas chromagraphy, **GC**, or thin layer chromagraphy, **TLC**) rather than using a quick, less accurate, enzyme-linked immunosorbant assay (Mahanna, 2007).

The mycotoxin concentration in the total diet is what is important to the cow. If a feed is contaminated with mycotoxins but that feed only makes up a small percentage of the diet, it may not actually cause a problem because it is diluted. So, it is helpful to calculate the concentrations of individual mycotoxins in the total diet.

Dilution

If a significant mold or mycotoxin problem is suspected, the best solution is to take the problem feed out of the diet. The second best solution is to reduce the amount of it in the ration. This reduces the total mycotoxin load on the cow and may allow you to still feed a moderately infested feed and get some value from it. Feed fines often contain the highest concentration of contamination, so it helps to find a way to avoid feeding them.

Mycotoxin binders

Mycotoxin binders are added to diets in order to attach to toxins so that they cannot be absorbed from the cow's gastrointestinal tract. In this way, they protect the cow from the toxins and prevent them from contaminating milk. Although much research has been conducted with these products, currently there are no additives approved in the U.S. for removing toxins from feeds. Unfortunately, research methods for evaluating mycotoxin binders in the lab and in the cow have not been standardized, making it difficult to compare products.

Silicates (clay, bentonite, montmorrillonite, zeolite, and phyllosilicates) are typically sold as anticaking agents or pellet binders. Even though they are not guaranteed to prevent mycotoxin problems, many nutritionists recommend them for binding toxins. Sodium bentonite, in the form of a fine powder (200 mesh) is typically fed at a rate of 8 oz/cow/day. The U.S. has a regulated upper limit of 2% sodium bentonite in a ration. Other sodium

aluminosilicate binders are fed at 4 oz/cow/day. One specific hydrated sodium calcium aluminosilicate has been shown to bind aflatoxins in a number of livestock species. Unfortunately, it does not seem to consistently bind other mycotoxins besides aflatoxin. Chemical modification of silicates shows some promise in improving their binding to other mycotoxins, such as zearalenone. Charcoal (activated carbon) has been used with some success as a binder of zearalenone and DON, but it does not seem to work as well as silicates for binding aflatoxins (Whitlow, 2006).

Esterified glucomannans are fragments of yeast cell wall that can effectively bind mycotoxins when fed at much lower levels than silicates. At 10 g/cow/day, esterified glucomannans significantly reduced milk aflatoxins in dairy cows as much as silicates fed at 225 g (8 oz)/cow/day (Diaz et al., 2004). This indigestible carbohydrate has been shown to bind aflatoxin, ochratoxin, zearalenone and T-2 toxin in the laboratory. It improved growth of broilers when fed at a rate of 0.5% in a diet known to contain aflatoxin, ochratoxin, zearalenone, and T-2 toxin (Aravind et al., 2003).

Some nutritionists recommend that binders be fed continuously as a preventative against subclinical mycotoxin problems. Unfortunately, some binders may also bind dietary minerals, especially trace minerals, making them unavailable to the cow. For this reason, constant dietary inclusion of binders may not be desirable, especially if organic mineral sources are not included in the diet. Future research looking at the cost of subclinical mycotoxicosis and the effectiveness of binders with low dietary mycotoxin concentrations in a ration would be helpful.

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Table 1. Incidence of mycotoxins in samples analyzed at Cumberland Valley Analytical Services, Fall 2009.

Mycotoxin at respective concentrations	Hay or Haylage (% of Samples; n=89)	Corn silage (% of Samples; n=241)	TMR (% of Samples; n=75)
DON[1] = 1 to 5 ppm[2]	3.36	44.44	25.35
DON = 5 to 25 ppm	5.6	8.27	5.34
15 Acetyl DON = 0.5 to 1 ppm	0.0	3.31	2.67
15 Acetyl DON = 1 to 5 ppm	0.0	6.19	1.33
Zearalenone = 0.5 to 1 ppm	0.0	0.43	0.0
Zearalenone = 1 to 5 ppm	0.0	1.28	0.0
T2 - % Positive	0.0	0.0	0.0
Aflatoxin, AB1 = 5 to 50 ppb[3]	—	0.8	—
Aflatoxin, AB2 = 5 to 50 ppb	—	0.4	—
Aflatoxin, AG1 = 5 to 50 ppb	—	0.4	—
Aflatoxin, AG2 = 5 to 50 ppb	—	0.4	—

[1]DON = Deoxynivalenol
[2]ppm = Parts per million
[3]ppb = Parts per billion

Table 2. Incidence of mycotoxins in samples analyzed at Cumberland Valley Analytical Services, Fall 2009.

Percent of samples containing particular mycotoxin levels	Corn grain (% of Samples; n=285)	Distillers (% of Samples, n=142)	Solubles (% of Samples n=20)	Wheat midds (% of Samples n=15)
DON[1] = 1 to 5 ppm[2]	25.61	67.62	90.0	100.0
DON = 5 to 25 ppm	16.85	15.50	0.0	0.0
15 Acetyl DON = 0.5 to 1 ppm	9.12	8.46	0.0	33.0
15 Acetyl DON = 1 to 5 ppm	12.62	17.64	5.0	0.0
3 Acetyl DON = 0.5 to 1 ppm	1.05	0.0	0.0	0.0
3 Acetyl DON = 1 to 5 ppm	0.35	0.0	0.0	0.0
Zearalenone = 0.5 to 1 ppm	2.47	7.78	0.0	0.0
Zearalenone = 1 to 5 ppm	3.18	0.71	0.0	0.0
T2 - % Positive	0.0	0.0	0.0	0.0
Aflatoxin, AB1 = 5 to 50 ppb[3]	2.49	—	—	—
Aflatoxin, AB1 > 50 ppb	0.71	—	—	—
Aflatoxin, AB2 < 5 ppb	1.05	—	—	—
Aflatoxin, AB2 = 5 to50 ppb	0.35	—	—	—
Aflatoxin, AG1 = 5 to 50 ppb	0.0	—	—	—
Aflatoxin, AG2 = 5 to 50 ppb	0.0	—	—	—

[1]DON = Deoxynivalenol
[2]ppm = Parts per million
[3]ppb = Parts per billion

Table 3. Common corn ear molds (Esker, 2010).

Corn ear mold	Fungi	Identification
Fusarium ear rot	*F. verticillioides, F. proliferatum, F.subglutinans*	Whitish pink, lavendar growth
Cladosporium ear rot	*C. herbarum, C. cladosporoides*	Dark, greenish black
Gibberella ear rot	*F. graminearum (G. zeae)*	Reddish mold beginning at tip
Diplodia ear rot	*Stenocarpella maydis*	White mold at base of ear
Trichoderma ear rot	*T. viride*	Green to bluish green
Penicillium ear rot	*P. oxalicium*	Green or blue, especially at tip

Table 4. Samples (%) with identified mold type (Cumberland Valley Analytical Services, Fall 2009 to Winter 2010).

	No. Samples	Fusarium	Aspergillus	Penicillium	Mucor	Rhizopus
TMR	70	51.4	15.7	34.3	40.0	1.4
Haylage	89	13.5	6.7	14.6	13.5	0.0
Distillers grains	22	27.3	0.0	22.7	36.4	4.5
Corn silage	352	17.3	9.7	23.9	19.9	0.6
Corn grain	247	36.8	8.9	25.1	42.5	0.8

Table 5. Samples (%) with identified mold type (Cumberland Valley Analytical Services, Fall 2009 to Winter 2010).

	No. Samples	Cladosporium	Absidia	Moniliella	Alternaria	Wallemia	Ulocladium
TMR	70	7.1	7.1	1.4	4.3	0.0	0.0
Haylage	89	11.2	2.2	0.0	1.1	1.1	0.0
Distillers grains	22	18.2	0.0	0.0	0.0	0.0	0.0
Corn silage	352	9.7	1.1	0.0	0.0	0.0	0.3
Corn grain	247	18.2	6.5	1.6	1.2	0.0	0.0

Table 6. Mycotoxin concern concentrations according to a few different U.S. experts.

	DON[1] (ppm)	Zearalenone (ppb)	T2 Toxin (ppb)	Aflatoxin (ppb)
Hutjens, 2007	> 0.5	> 300 to 500	> 100 to 200	> 20
Seglar, 2001	> 5	> 25,000	> 500	> 20
Dairyland Labs, 2010	> 6	> 300	> 500	> 20
Whitlow and Hagler (ADM)	> 0.3 to 0.5	> 200 to 300	> 100	> 20

[1]DON = Deoxynivalenol.

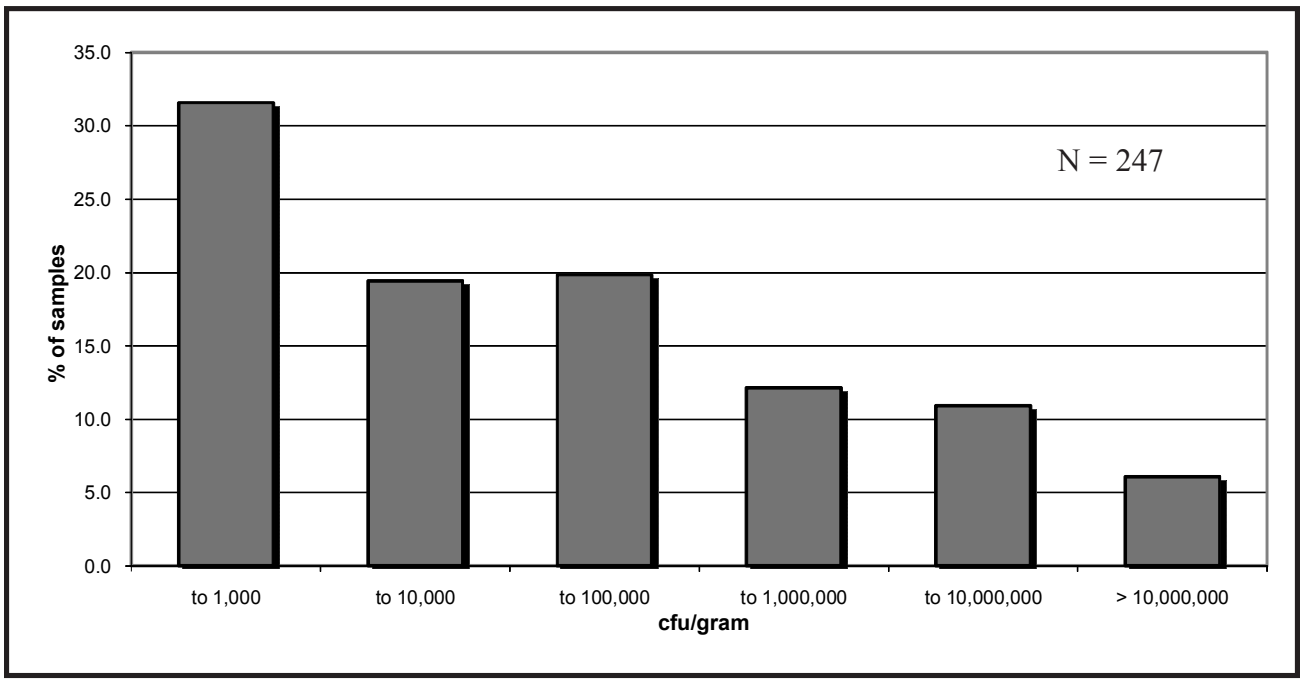

Figure 1. Distribution of mold counts in corn (cfu = colony forming units).

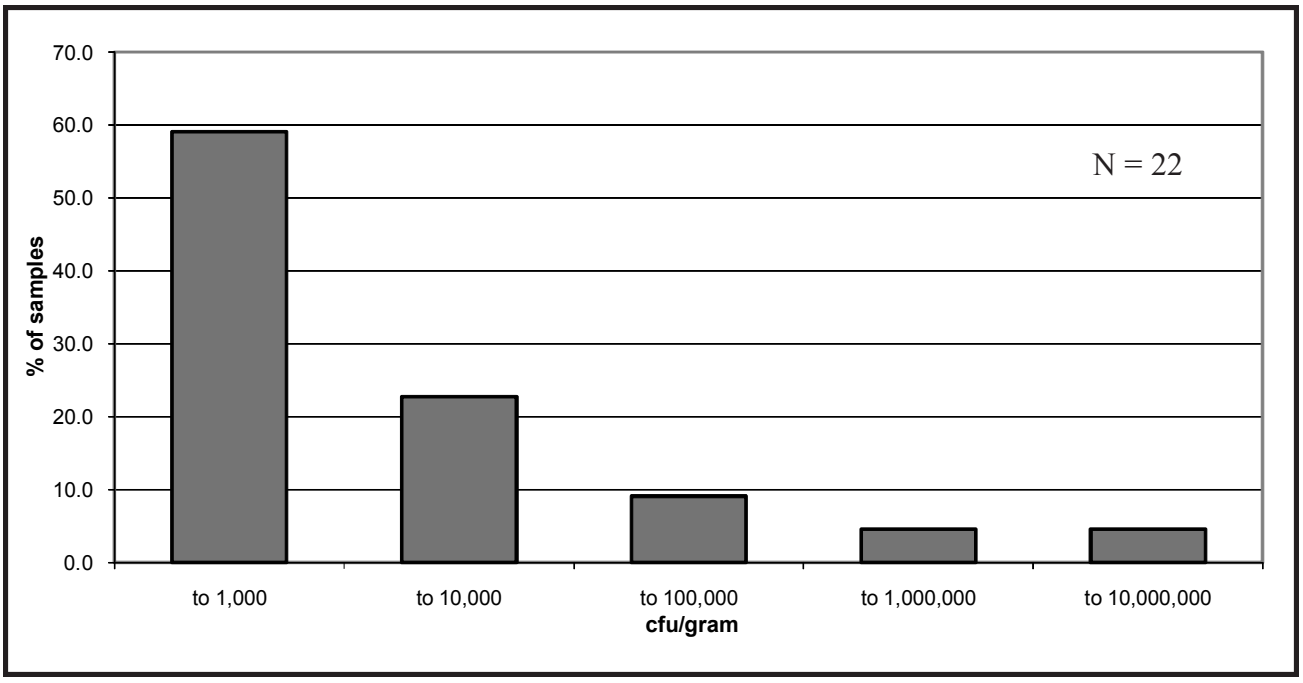

Figure 2. Distribution of mold counts in distillers grains (cfu = colony forming unit).

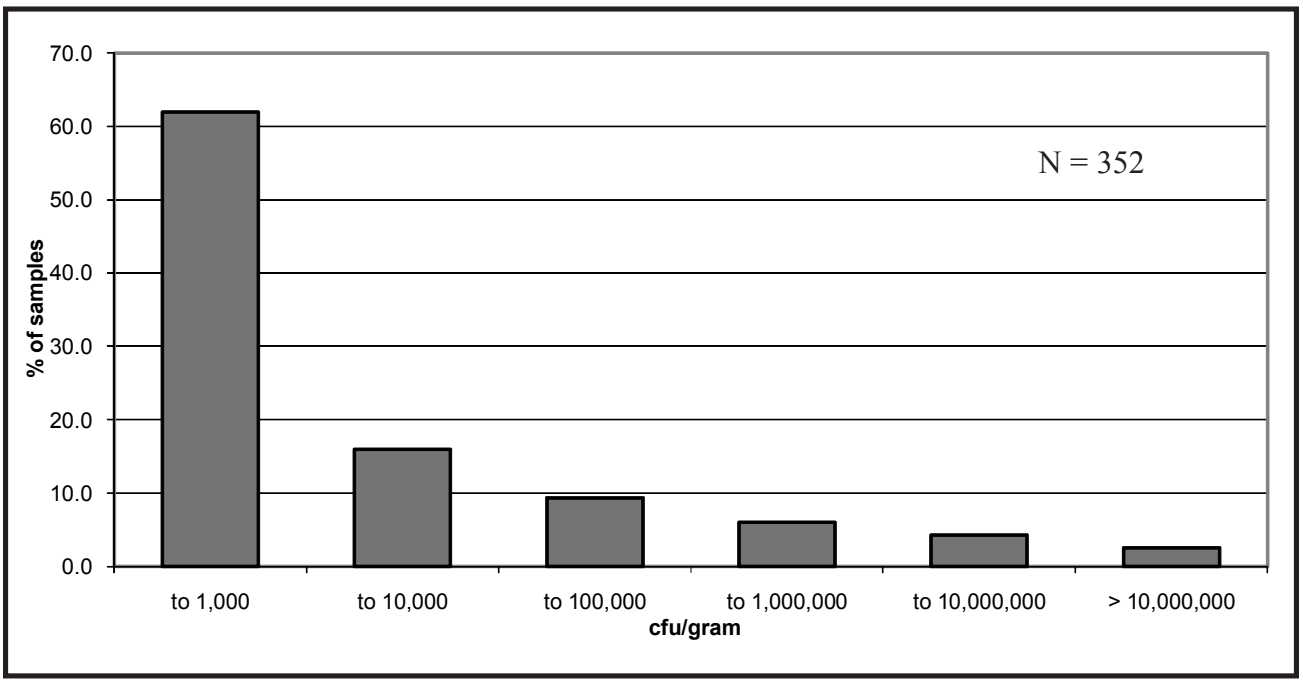

Figure 3. Distribution of mold counts in corn silage (cfu = colony forming unit).

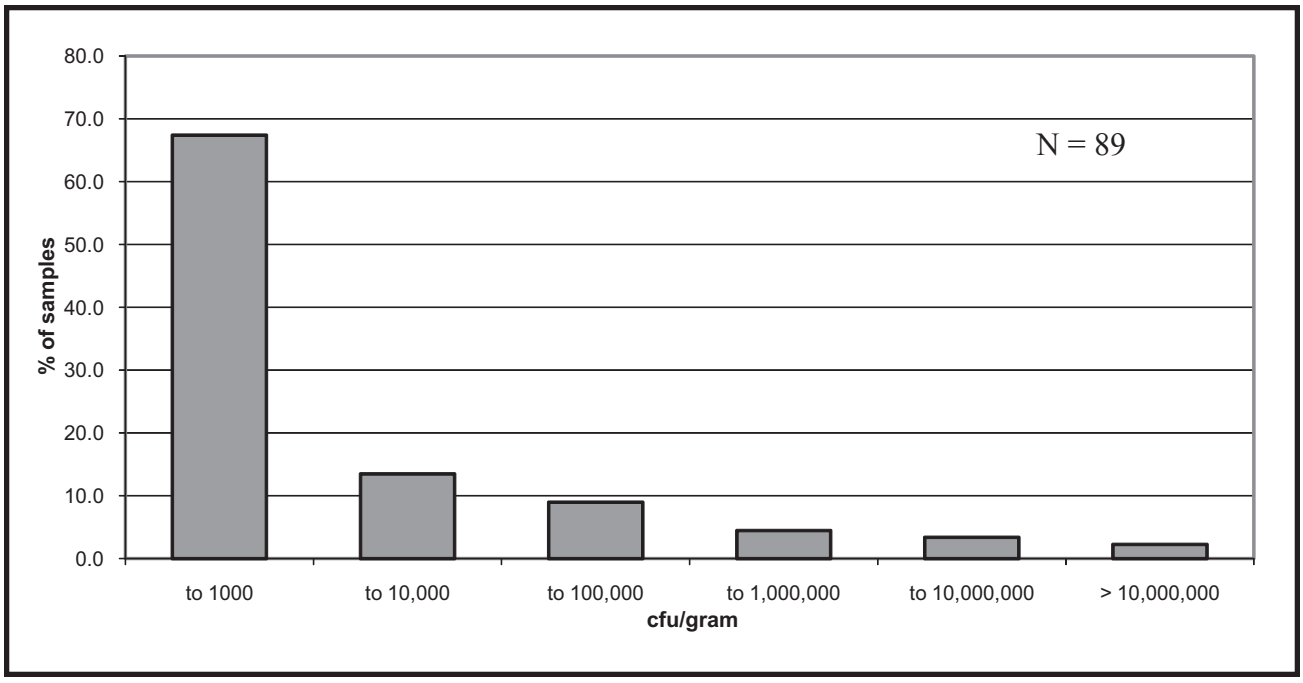

Figure 4. Distribution of mold counts in haylage (cfu = colony forming units).

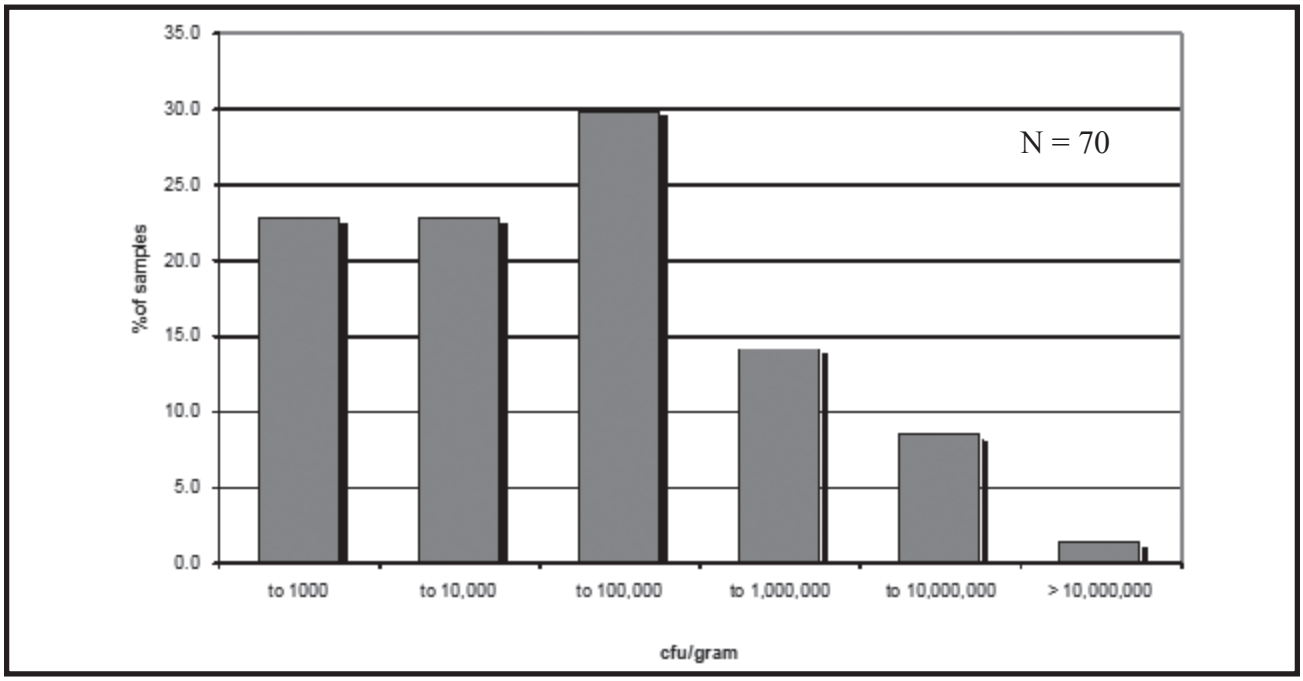

Figure 5. Distribution of mold counts in total mixed rations (cfu = colony forming units).