Mycotoxins - A Stress Factor for Dairy Cattle

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Summary

Mycotoxins occur frequently in a variety of feedstuffs and are routinely fed to dairy cattle. Sometimes mycotoxins occur at concentrations high enough to cause major losses in health and performance, however mycotoxins are more usually at lower levels that result in subclinical losses in milk production, increases in disease incidence and reduced reproductive performance. These subclinical losses are of greatest economic importance. Although a mycotoxicosis is difficult to diagnose, mycotoxins should be considered as a causative factor when existing problems can not be attributed to other typical causes. Laboratory detection methods have improved in accuracy and cost. Prevention practices need more emphasis. The potential for effective treatments has improved. Certain feed additives can reduce mycotoxin exposure of animals and thus minimize their negative effects.

Review of Problem and Literature Review

Mold Growth and Mycotoxin Formation

Mycotoxins are toxic compounds produced by actively growing molds. Molds are fungi which grow in multicellular colonies, as compared with yeasts which are single cellular fungi. Mold growth and mycotoxin production are related to weather extremes (causing plant stress or excess hydration of stored feedstuffs), to inadequate storage practices, to low feedstuff quality, and to faulty feeding conditions. Molds can grow and mycotoxins can be produced pre-harvest or during storage, processing, or feeding. Because crops can be contaminated prior to harvest, excellent storage conditions are essential to prevent further mold growth and mycotoxin formation. Molds grow over a temperature range of 10-40° C (50-104° F), a pH range of 4 to 8, and above 0.7 a_w (equilibrium relative humidity expressed as a decimal instead of a percentage). While yeast require a moist surface or water layer, molds can grow on a dry surface (Lacey, 1991). Mold can grow on feeds containing more than 12% to 13% moisture. Higher moisture levels support mold growth up to the point where water excludes adequate oxygen. Almost all molds are aerobic.

The *Aspergillus* species grow at lower water activities and at higher temperatures than do the *Fusarium* species. *Aspergillus flavus* and aflatoxin in corn are favored by the heat and drought stress associated with warmer climates. Aflatoxin is enhanced by insect damage before and after harvest. *Penicillium* species grow at relatively low water activities and low temperatures and are fairly widespread in occurrence. Since both *Aspergillus* and *Penicillium* grow at low water activities, they are considered to be storage fungi. *Aspergillus* species are more likely in warm climates while *Fusarium* and *Penicillium* species are more

likely in cooler climates.

The *Fusarium* species are generally considered to be field fungi and may be more likely to proliferate prior to storage. *Fusarium* commonly affects corn and small grains. In corn, *Fusarium* molds are associated with ear rot and stalk rot. In small grains, *Fusarium* molds are associated with diseases such as head blight (scab). These field diseases result in yield loss, quality loss and mycotoxin contamination. In wheat, excess moisture at flowering and afterward is associated with increased incidence of mycotoxin formation. In corn, *Fusarium* diseases are more commonly associated with insect damage, warm conditions at silking, and wet conditions late in the growing season (Trenholm et al., 1988).

The conditions most suitable for mold growth may not be the optimum conditions for mycotoxin formation. For example, the *Fusarium* molds associated with alimentary toxic aleukia have been reported to grow prolifically at temperatures of 25 to 30° C without producing much mycotoxin, but at near freezing temperatures, large quantities of mycotoxins are produced without much mold growth (Joffe, 1986). Field applications of fungicides may reduce mold growth reducing production of mycotoxins, however, the stress or shock of the fungicide to the mold organism may cause increased mycotoxin production (Boyacioglu et al., 1992 and Gareis and Ceynowa, 1994).

Mycotoxin Occurrence

Mycotoxins occur in most all types of feedstuffs and they occur worldwide. Table 1 shows the occurrence of five mycotoxins in corn silage, corn grain and in all feeds analyzed (Whitlow, et al., 1998). These results over a nine-year period from feed samples submitted by North Carolina farmers may contain a bias because samples are not random. Results were variable by year.

Aflatoxin >10 ppb n % Pos mean ∀ s.d.	Deoxynivalenol >50ppb n % Pos mean ∀ s.d.	Zearalenone >70ppb n % Pos mean ∀ s.d.	T-2 Toxin >50ppb n % Pos mean ∀ s.d.	Fumonisin >1ppm n % Pos
CORN SILAGE				
461 8 28 ∀19	778 66 1991 ∀ 2878	487 30 525 ∀ 799	717 7 569 ∀ 830	63 37
CORN GRAIN				
231 9 170 ∀ 606	362 70 1504 ∀ 2550	219 11 206 ∀ 175	353 6 569 ∀ 690	37 60
ALL FEEDS				
1617 7 91 ∀ 320	2472 58 1739 ∀10880	1769 18 445 ∀ 669	2243 7 482 ∀ 898	283 28

Mycotoxin Effects

Mycotoxins exert their effects through three primary mechanisms: (1) alteration in nutrient content of feed, in nutrient absorption and nutrient metabolism, (2) effects on the endocrine system, and (3) suppression of the immune system. Mycotoxins can increase incidence of disease and reduce production efficiency. The resulting cascade of symptoms may be perplexing and make diagnosis difficult (Hesseltine, 1986b and Schilfer, 1990). Diagnosis is complicated by a lack of research (especially with dairy cattle), by nonspecific symptoms, by interactions with other stress factors, and by the lack of feed analyses.

Dairy herds experiencing a mycotoxicosis that is severe enough to reduce milk production will usually display other symptoms. Deoxynivalenol, T-2 toxin, and fumonisin may result in digestive disorders while zearalenone is more likely to be associated with reproductive problems. Because of possible interactions with opportunistic diseases, symptoms may be general and variable. Symptoms may include some of the following: intermittent diarrhea, reduced feed intake, feed refusal, unthriftiness, rough hair coat, undernourished appearance, subnormal production, increased abortions or embryonic mortalities, silent heats, irregular estrus cycles, expression of estrus in pregnant cows, and decreased conception rates. Fresh cows under the stress of calving may show the most pronounced symptoms. There may be a higher incidence of displaced abomasum, ketosis, retained placenta, metritis, mastitis, and fatty livers. Cows may not respond well to typical veterinary therapy. A definitive diagnosis of a mycotoxicosis cannot be made directly from symptoms, specific tissue damage, or even feed analyses, however, experience with mycotoxin affected herds may increase the probability of recognizing a problem. Regardless of the difficulty of diagnosis, mycotoxins should be considered as a possible cause of production and health problems in the dairy herd. A process of elimination of other factors, coupled with feed analyses and responses to treatments can help identify the problem.

Safe Levels of Mycotoxins

Several factors contribute to the difficulty of establishing levels of safety and include lack of research, ruminal degradation, species differences in sensitivity, imprecision in sampling and analysis, the large number of potential mycotoxins, and interactions with other stress factors (Hamilton, 1984, and Schaeffer and Hamilton, 1991).

Because of partial degradation in the rumen, mycotoxins are less toxic to cattle than to most other animals, however mycotoxins are not completely degraded and some of the degradation products remain toxic (Kiessling et al., 1984). Extent of ruminal degradation appears to be variable and may be less for high producing cows with a faster rate of ruminal feed passage.

In controlled research, naturally contaminated feeds have been shown to be more toxic than feeds with the same level of an pure mycotoxin supplemented into the diet. This suggests the presence of other unidentified mycotoxins in naturally contaminated feeds. Therefore the presence of mycotoxins such as deoxynivalenol may serve as a marker for moldy, mycotoxin-contaminated feed.

Interactions with other stress factors make recommendations difficult. Lillehoj and Ceigler (1975) give an example where penicillic acid and citrinin were innocuous when administered alone but were 100% lethal when given in combination. Fumonisin at 100 ppm has been shown to reduce milk production in dairy cattle (Whitlow, unpublished), but to not affect average daily gain in beef cattle fed 148 ppm (Osweiler et al, 1993). Aflatoxin produced from culture was more toxic to dairy cattle than pure aflatoxin added to diets (Applebaum et al., 1982). In swine, Foster et al. (1986) demonstrated that pure deoxynivalenol added to diets was less toxic than diets with similar concentrations of deoxynivalenol, which was supplied from naturally contaminated feeds. Smith and MacDonald (1991) have suggested that fusaric acid may occur along with deoxynivalenol to produce more severe symptoms. Many such interactions are possible since *Fusarium* molds produce many mycotoxins, and it is well documented that several mycotoxins may be found in the same feed (Hagler et al., 1984). Abbas et al. (1989) demonstrated that *Fusarium* species isolated from Minnesota corn produces an array of mycotoxins.

Wannemacher et al. (1991) presents data to show that even with laboratory animals such as the mouse and rat, T-2 elicits very real species differences. Mycotoxin effects are also moderated by factors such as sex, age, and stresses of the environment and production. Certainly duration of exposure is important. The known dietary factors, which interact with mycotoxins, include dietary nutrients including, fat, protein, fiber, vitamins and minerals. Dietary pellet binders (clay) adsorb some mycotoxins reducing exposure of the animal. Thus, many factors and interactions make it difficult to relate field observations to those from controlled research.

Mycotoxin Testing

Mold spore counts may not be very useful and are only a gross indication of the potential for toxicity. Mold identification can be useful to suggest which mycotoxins that may be present. Scott (1990) states that screening methods are needed for the *Fusarium* produced mycotoxins and that one approach is to test for deoxynivalenol, diacetoxyscirpenol, T-2 toxin and nivalenol, because other *Fusarium* mycotoxins seldom occur without one of these four also present. Feeds could then be further tested for other mycotoxins. Generally laboratories provide analysis for only a limited number of mycotoxins perhaps including aflatoxin, ochratoxin, deoxynivalenol, zearalenone, fumonisin, and T-2 toxin. Analytical techniques for mycotoxins are improving (Chu, 1992). The costs are decreasing and several commercial laboratories are available which provide screens for an array of mycotoxins. Proper collection and handling of representative feed samples is essential. Since molds grow in spots, mycotoxins are not

uniformly distributed within a feed, making it difficult to obtain a representative sample especially from whole seed, coarse feeds or feeds not adequately mixed. Once collected, samples should be handled properly to prevent further mold growth. Wet samples may be frozen or dried prior to shipment. Transit time should be minimized.

Aspergillus Molds

Aflatoxin (AF) is produced primarily by *Aspergillus flavus* and is commonly found in the southern U.S., but also in other regions in some years when weather conditions are conducive. For example, 8% of samples of Midwestern U.S. corn grain from the 1988 drought season contained aflatoxin (Russel, et al., 1991). It appears that *Aspergillus flavus* does not grow well in hay or silage, however, concentrations of aflatoxin as high as 5 ppm have been reported (Kalac and Woolford, 1982). We have detected low levels of aflatoxin (<100 ppb) in corn silage and alfalfa. The FDA limits AF in corn grain according to its intended use, which for lactating dairy cattle is 20 ppb. AF is excreted into milk in the form of AFM_1 with residues approximately equal to 1.7% of the dietary level (Van Egmond, 1989). The FDA limits aflatoxin M_1 in milk to no more than 0.5 ppb. Levels of 300 to 700 ppb are considered toxic for beef cattle depending on criteria for toxicity, and other factors affecting toxicity. Garrett et al., (1968) showed that with beef cattle, gain and intake were affected at 700 ppb AF, but not at 300 ppb; however, a no effect level can not be determined from such data with few animals. Trends in the data, especially for increased liver weights, would indicate potential toxicity at levels as low as 100 ppb. Guthrie (1979) showed a decline in reproductive efficiency when lactating dairy cattle in a field situation were consuming 120 ppb AF. When cows were changed to an AF free diet, milk production increased over 25%. Patterson and Anderson (1982) and Marsi et al. (1969) also suggest that 100 ppb may reduce milk production. Applebaum et al. (1982) showed that impure AF produced by culture reduced production while equal amounts of pure AF did not.

Aspergillus fumigatus has been found in both hay (Shadmi, et al., 1974) and silage (Cole, et al., 1977). The silage was found to contain fumigaclavine A and C and several fumitremorgens. Symptoms included generalized deterioration typical of protein deficiency, malnutrition, diarrhea, irritability, abnormal behavior and occasional death. The hay was fed to goats and rats and resulted in retarded growth and histopathological changes in the livers and kidneys. Sterigmatocystin is primarily produced by *Aspergillus versicolor* and has been observed as a primary mycotoxin produced by *Aspergillus* on cereal grains in western Canada (Mills and Abramson, 1986). While it is thought to be infrequent at toxic levels in the U.S., it was detected in a grain mixture and associated with bloody diarrhea and deaths of cows in a field case in Tennessee (Vesonder and Horn, 1985). *Aspergillus ochraceus* was implicated as producing ochratoxin A associated with abortions in cattle consuming moldy alfalfa hay (Still, et al, 1971). Lacey (1991) has reviewed other cases of potential toxicities associated with a*spergillus* molds.

Fusarium molds

Fumonisin B_1 (FB_1) was isolated by Gelderblom et al. (1988) and shown to be a cancer promoter. FB_1 has been shown to cause leukoencephalomalacia in horses (Marasas, et al., 1988), pulmonary edema in swine (Harrison et al., 1990) and hepatoxicity in rats (Gelderblom et al., 1991). A USDA, APHIS (1995) survey found an average of 6.9% of 1995 corn samples from Missouri, Iowa and Illinois to contain more than 5 ppm FB_1. While FB_1 is thought to be much less potent in ruminants than monogastrics, work by Kriek et al. (1981) suggested that fumonisin was toxic to sheep. Osweiler et al., (1993) demonstrated that FB_1 in large amounts (148 ppm) can cause mild liver damage in cattle even when fed for a short term (31 days), but without an effect on feed intake or weight gain. FB_1 fed to dairy cattle at 100 ppm for approximately 7 days prior to freshening and for 70 days thereafter, significantly and dramatically reduced milk production (6 kg/cow/day) and increased serum enzymes levels indicative of liver disease Diaz et al., 2000. These results strongly suggest that FB_1 is toxic to dairy cattle at levels that are less toxic to beef cattle, or perhaps FB_1 interacts with other factors to produce different effects in beef and dairy cattle under different conditions. FB_1 carryover from feed to milk is thought to be negligible (Richard et al., 1996 and Scott et al., 1994).

Deoxynivalenol (DON) is the proper name for a commonly detected *Fusarium* produced mycotoxin often referred to as vomitoxin. Two independent Midwestern studies (Vesonder et al., 1978 and Côté et al., 1984) showed DON to be the primary mycotoxin associated with swine problems including feed refusals, diarrhea, emesis, reproductive failure, and deaths. Smith and McDonald (1991) have indicated that fusaric acid interacts with DON to produce the symptoms previously attributed to just to DON. In cattle, DON has been associated with reduced feed intake (Trenholm et al., 1985). Clinical data from 300 herds representing about 40,000 cow records showed that DON was associated with a loss in milk production, but this study did not establish a cause and effect (Whitlow et al., 1991). DON may simply be a marker for problem feeds. Field observations by others help substantiate that DON is associated with losses in milk production (Gotlieb, 1997 and Seglar, 1997). Charmley et al. (1993), demonstrated a 13% (2.85 kg) numerical decrease in 4% fat corrected milk production (statistics not available), utilizing 18 midlactation dairy cows (average 19.5 kg milk) consuming diets shown to contain no common mycotoxins other than DON which was at levels of 2.7 to 6.4 ppm in treatment diets. While the decrease in actual milk production (1.35 kg) was not statistically significant ($p<.16$), the decrease in fat test (3.92% *vs.* 3.04%) was significant. Noller et al., (1979) utilized 54 lactating dairy cows in a 3 X 3 Latin Square experiment with 21 day feeding periods. Experimental diets contained corn grain contaminated with *Gibberella zeae* and resulted in DON levels in the TMR of approximately 0, 1650 and 3300 ppb and zearalenone levels of 0, 65 and 130 ppb. DON and zearalenone were not analyzed directly but was found in corn harvested earlier from the same field. Neither dry matter intake nor milk production (average 22.9 kg) was affected by additions of contaminated grain to the diet. However, compared with controls, cows that received incremental levels of contaminated grain, gained significantly less weight. Compared with the control, daily weight gain was 0.60 lb less for cows on the diet containing 1650 ppb DON and 0.85 lb less for the diet containing 3200 ppb of DON. DiCostanzo et al, (1995a) cites results by Ingalls (1994) where lactating dairy cows were

fed 0, 3.6 10.9 or 14.6 ppm of DON for 21 days, without an apparent effect on feed intake or milk production. Milk production averaged about 30 kg daily. Beef cattle and sheep appear to tolerate relatively large amounts of DON without obvious deleterious effects (DeHaan et al., 1984, Nelson et al., 1984, DiCostanzo et al., 1995b, Boland et al., 1994, and Windels et al., 1995).

Zearalenone (ZEN) is a *Fusarium* produced mycotoxin, which elicits an estrogenic response in monogastrics (Sundlof and Strickland, 1986). However, ZEN is rapidly converted to ∀- and ∃-zearalenol in rumen cultures (Kiessling et al., 1984) and has been of less toxicity to ruminants. Ruminal degradation of ZEN was found to be about 30% in 48 hours (Kellela and Vasenius, 1982). A controlled study with cows fed up to 22 ppm ZEN showed no obvious effects except that corpora lutea were smaller in treated cows (Weaver et al., 1986b). In a similar study with heifers receiving about 13 ppm ZEN, conception rate was depressed about 25%; otherwise, no obvious effects were noted (Weaver et al., 1986a). Several case reports have related ZEN to an estrogenic response in ruminants and sometimes included abortions as a symptom (Kellela and Ettala, 1984, Khamis et al., 1986; Mirocha et al., 1968; Mirocha et al., 1974; and Roine et al., 1971). Other cattle responses may include vaginitis, vaginal secretions, poor reproductive performance and mammary gland enlargement of virgin heifers. In a field study, (Coppock et al., 1990) diets with about 750 ppb ZEN and 500 ppb DON resulted in poor consumption, depressed milk production, diarrhea, and total reproductive failure. New Zealand workers (Towers, et al., 1995a, Towers, et al., 1995b, Sprosen and Towers, 1995, and Smith et al., 1995) have related urinary zearalenone and zearalenone metabolites (zearalenone, zearalanone, ∀- and ∃-zearalenol and ∀- and ∃-zearalanol) which they refer to as "zearalenone" to intake of "zearalenone" and to reproductive disorders in sheep and dairy cattle. In sheep, "zearalenone" was related to lower conception, reduced ovulation, and increased twinning rates. With dairy cattle, herds with low fertility were found to have higher levels of blood and urinary "zearalenone" and consumed pastures containing higher levels of "zearalenone". In addition, individual cows within herds were examined by palpation and those that were determined to be cycling had lower blood "zearalenone" levels than did cows that were not cycling. Differences in "zearalenone" levels were attributed to selective grazing behavior. The reproductive problems in dairy cattle were noted with "zearalenone" concentrations of about 400 ppb in the pasture samples.

T-2 toxin (T-2) is a very potent *Fusarium* produced mycotoxin, and has been associated with gastroenteritis, intestinal hemorrhages (Petrie et al., 1977 and Mirocha et al., 1976) and death (Hsu et al., 1972 and Kosuri et al., 1970). Weaver et al. (1980) showed that T-2 was associated with feed refusal and gastrointestinal lesions in a cow, but did not show a hemorrhagic syndrome. Kegl and Vanyi, (1991) observed bloody diarrhea, low feed consumption, decreased milk production and absence of estrus cycles in cows exposed to T-2. Serum immunoglobulins and certain complement proteins were lowered in calves receiving T-2 toxin (Mann et al, 1983). Gentry et al. (1984) demonstrated a reduction in white blood cell and neutrophil counts in calves. A calf intubated with T-2 developed severe depression, hindquarter ataxia, knuckling of the rear feet, listlessness and anorexia (Weaver et al, 1980). Data with cattle are limited, but the toxicity of T-2 toxin in laboratory animals

is well documented (Wannemacher et al, 1991). In a field observation with Jersey cows, we observed a 7 lb decrease in milk production coinciding with diarrhea and apparently associated with 350 ppb of T-2 toxin in the dietary dry matter. Cows responded to a clay type feed additive. At similar levels in other herds, we have associated T-2 toxin with an increased incidence of disease in early lactation, poor adjustment of fresh cows to the lactation ration, excessive weight loss, increased death loss and a loss in milk production.

Diacetoxyscirpenol is a *Fusarium* produced mycotoxin. It may occur along with T-2 toxin and is thought to produce similar symptoms of toxicity.

Penicillium molds

Ochratoxin, produced primarily by a *Penicillium* mold but also by certain *Aspergillus* molds, is rapidly degraded in the rumen and thus thought to be of little consequence unless consumed by young pre-ruminant calves (Sreemannarayana et al., 1988). However, depending on the animal and type of diet, ochratoxin degradation may be limited. Vough and Glick (1993) reported cattle deaths diagnosed as ochratoxin toxicity. *Aspergillus ochraceus* was implicated as producing ochratoxin A associated with abortions in cattle consuming moldy alfalfa hay (Still, et al, 1971). Ochratoxin has also been detected in milk at levels suggesting that a substantial amount of ochratoxin escapes ruminal degradation (Nip and Chu, 1979). Patulin is produced by *Penicillium*, *Aspergillus*, and *Byssochlamys* molds and may be found in silage (Dutton, et al., 1984 and Hacking and Rosser, 1981). Patulin has been incriminated as a possible toxin in Europe and New Zealand (Lacey, 1991). PR toxin, produced by *Penicillium roquefortii*, has been found in silage (Hacking and Rosser, 1981) and was the suspected vector in a case study with symptoms of abortion and retained placenta (Still et al., 1972). *Penicillium* or Aspergillus molds growing on sweet clover or sweet vernal grass can cause a conversion of natural compounds in the plant to dicoumarol. Dicoumarol interferes with the function of vitamin K, resulting in a hemorrhagic syndrome. Moldy sweet clover poisoning is discussed by Radostits, et al., (1980).

Others

Stachybotrys toxicosis has been observed when the mold occurs on hay and straw but it is thought to be rarely associated with dairy cattle problems in the U.S. This mold was associated with deaths of thousands of horses in Russia during the 1930's. The mold produces a large number of spores, resulting in sooty black spots on the forage. There have been several mycotoxins isolated and identified (Eppley, 1977). There are other mycotoxins that affect ruminants. Some are thought to occur less frequently or to be less potent, but in many cases, there is a lack of information.

Treatment

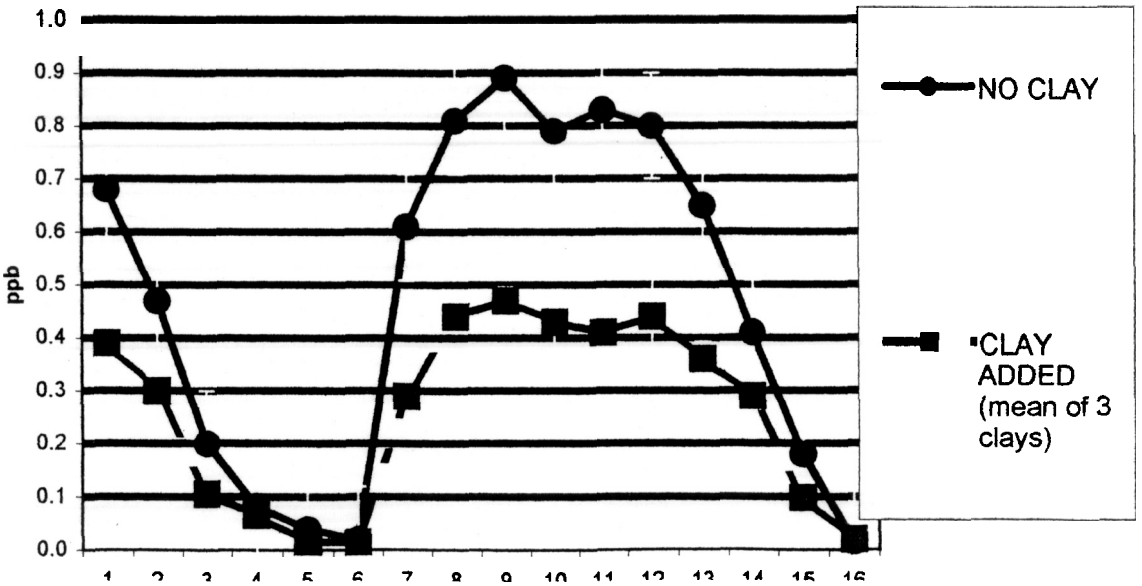
Some additives may be beneficial in reducing mycotoxins because they are effective in reducing mold growth. Ammonia, propionic acid, microbial, and enzymatic silage additives have all shown some effectiveness as mold inhibitors. Additives to enhance

fermentation may be added at ensiling. Mold growth inhibitors such as propionic acid may be helpful as a surface treatment when capping off the silo or daily after silage feed-out to reduce molding of the exposed silage feeding surface. If unacceptably high levels of mycotoxins occur, dilution or removal of the contaminated feed is preferable; however, it is usually impossible to completely replace major forage ingredients. While dilution is sometimes a viable practice to reduce exposure, reduced feeding of silage could result in such a slow feedout that mycotoxin problems within the silage increase. Ammoniation of grains can destroy some mycotoxins, but there is no practical method to detoxify affected forages already in storage. Increasing nutrients such as protein, energy and antioxidant nutrients may be advisable (Brucato et al., 1986, Chandler, 1992). Adsorbent materials such as clays (bentonites) added to contaminated diets fed to rats, poultry, swine and cattle have helped reduce the effects of mycotoxins (Diaz et al., 1997; Galey et al., 1987; Harvey, 1988; Lindemann et al., 1991; Scheideler, 1990; Hayes, 1990 and Smith, 1980 and 1984). In most cases, clay has been added to the diet at about 1%. Activated carbon at 1% of the diet effectively reduced aflatoxin in milk (Galvano et al., 1996). Aactivated carbon fed at 0.1% of the diet did not reduce aflatoxin levels in milk (Diaz et al, 1999). A glucomannan fed at 0.05% of diet dry matter or bentonites at 1% of diet dry matter were effective (Diaz et al, 1999).

Areas of needed Information

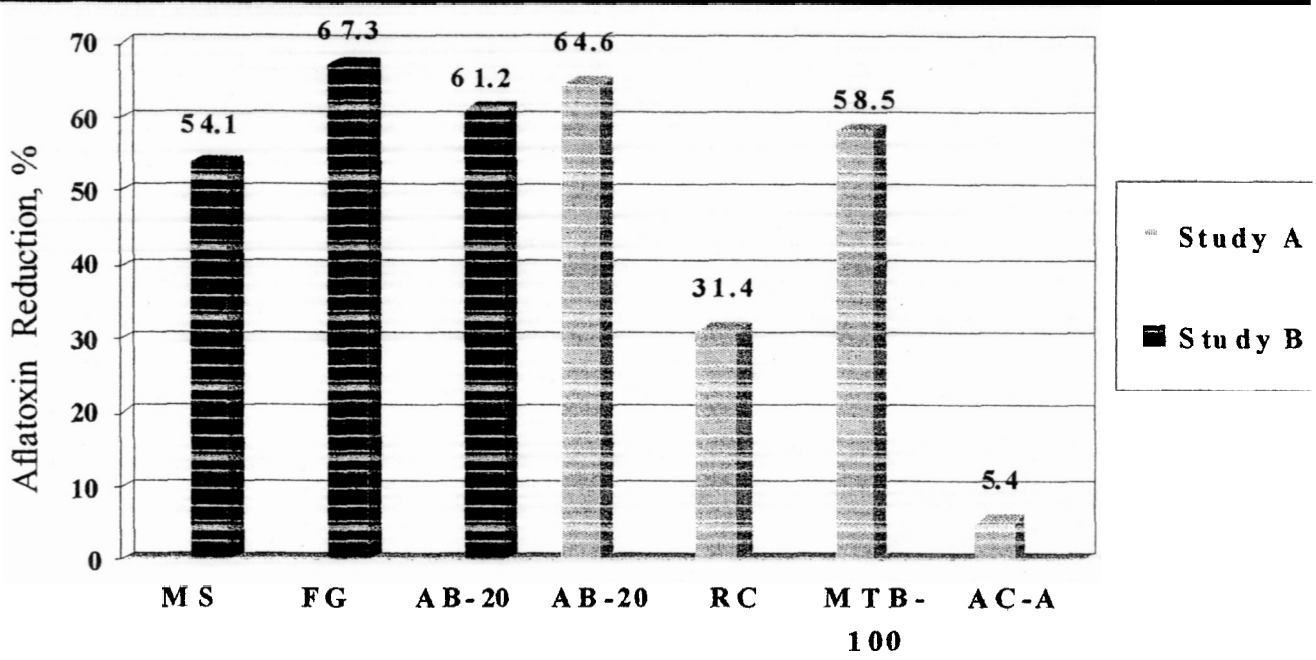
More information is needed about why mycotoxins occur, when to expect them, how to prevent their occurrence and how to deal with their presence. More data is needed about toxicity to dairy cattle, about interactions with other mycotoxins, with nutrients, and with stress factors such as disease organisms or environmental stress. Improved methods are needed for monitoring mycotoxin occurrence, for diagnosing toxicities, and for prevention and treatment.

Figure 1. Clearance and Appearance of Aflatoxin Associated With Consumption of Aflatoxin Contaminated Corn in Diets



With or Without Clay Products

Figure 2. Effect of Feed Additives on Reduction of Milk Aflatoxin Residues



MS, mycrosorb, a sodium bentonite fed at 1% of DM (American Colliod Co.) FG, flowguard, a sodium bentonite fed at 1% of DM intake (La Port Biochem.), **AB-20**, a sodium bentonite fed at 1% of DM intake (Prince AgriProducts, Inc.). **RC**, Red Crown, a calcium bentonite fed at 1% of DM intake (Prince AgriProducts, Inc.) and **MTB-100**, a modified glucomannan product fed at 0.05% of DM intake (Alltech, Inc.) significantly reduced ($P < .0001$) AFM1 residues in milk. **AC-A**, an activated charcoal fed at 0.25% of DM intake had no effect. Diaz, et al. 1999. Journal of Dairy Science 82:(Sol 14)838.

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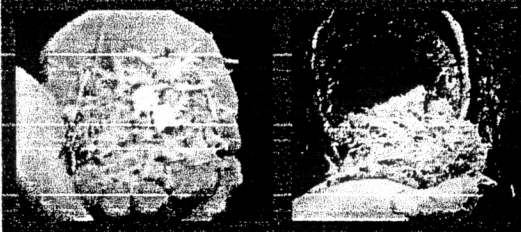
Optimizing Energy Intake in Ruminants Using Fibrolytic Enzymes

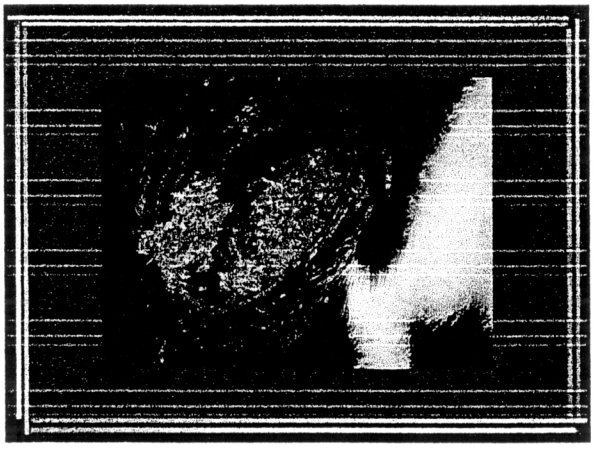


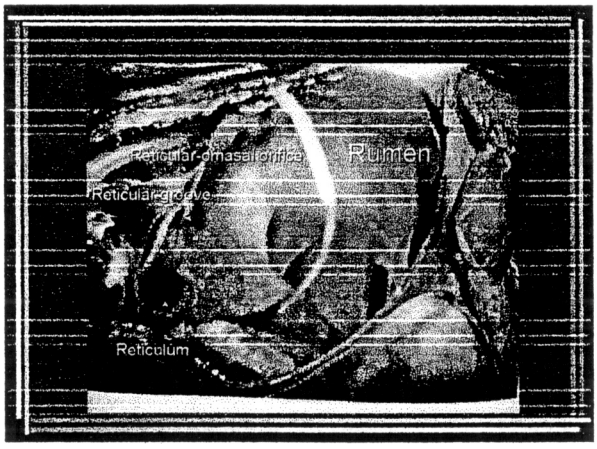
Richard Zinn
University of California, Davis

Extent of ruminal fiber digestion

- Passage rate, Kp
- Rate of digestion, Kd

$$\text{Dig, \%} = \frac{K_d}{K_d + K_p} \times 100$$





Fiber passage rate

- Initial forage particle size distribution
- Particle size reduction (<1.18 mm)
  - chewing, rumination
- Particle density
- Rate of digestion
- eNDF (Kp = 3.21 - .0161eNDF)

Effective NDF

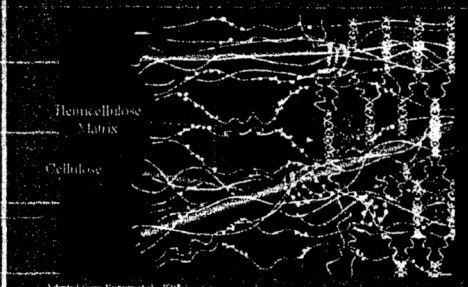
Feed	1.18-mm sieve
Sudangrass hay, long	98
Alfalfa hay, long	92
Alfalfa hay, 3" screen	67
Corn silage	48

(Adapted from Mertens, 1996)

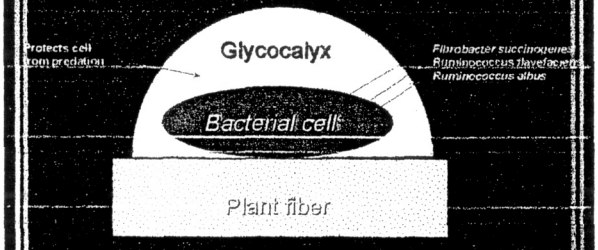
Rate of ruminal fiber digestion influenced by:

- Chemical-physical characteristics
- Ruminal digestive capacity
  - –Ruminal pH
  - –microbial distributions and adaptations

Plant cell walls are comprised of cellulose fibrils embedded in a hemicellulose matrix - like reinforced concrete the relationship between the two is intimate



Bacterial attachment



Major constraints:
- Surface matrix
- Substrate surface area
- Bacteria penetration into lumen

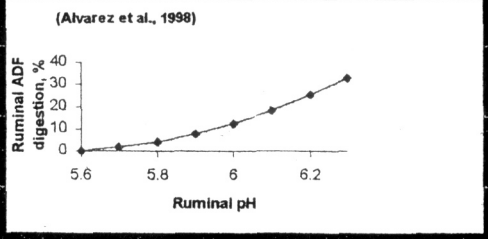
Rate of fiber digestion

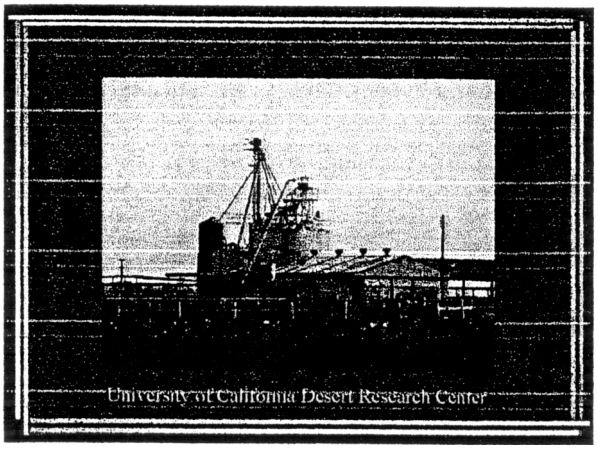
- Maximal rate of cellulose digestion is in the range of .05 to .08/h
- Normally rates are much lower
- 2 main limitations:
  - Substrate-enzyme accessibility
  - Ruminal pH
- Rate is not influenced by retention time

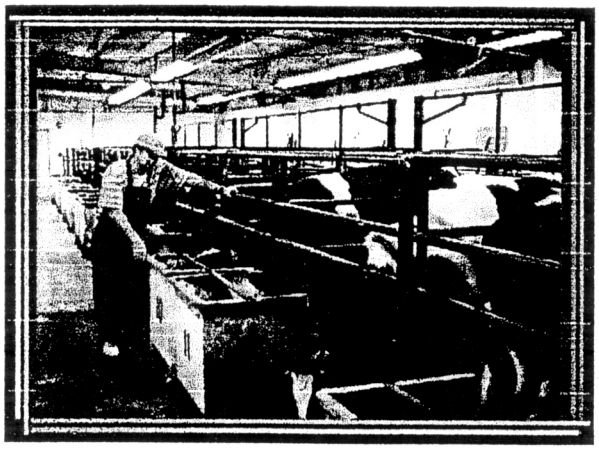
Ruminal pH fluctuates within the range of 5.5 to 6.5 with a mean of 5.97 (Allen, 1997)

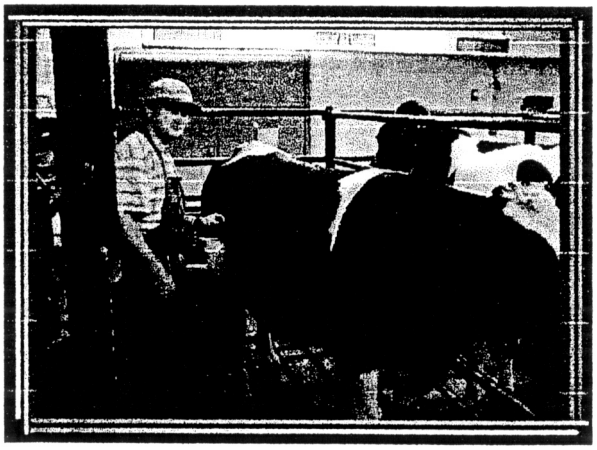
- **Cellulolytic bacteria do not grow at pH < 6.0** (Russell and Wilson, 1996)
  - **Toxicity due to loss of ability to regulate intracellular anion concentrations**
- **Specific growth rate decreases by roughly 14%/h for every .1 unit decrease in ruminal pH between 6.5 and 6.0**
- **Cellulose digestion is optimal at pH > 6.5**

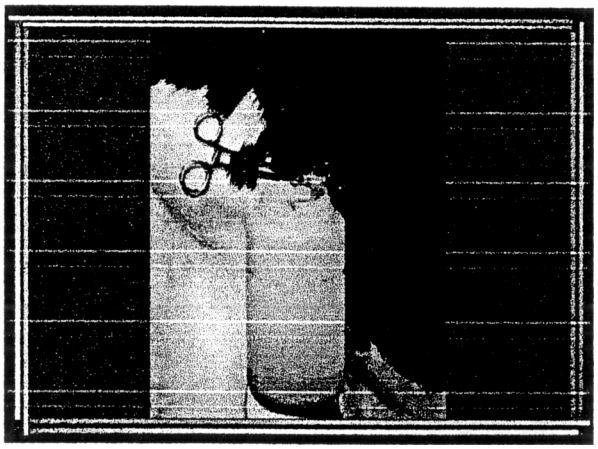
Ruminal pH and fiber digestion

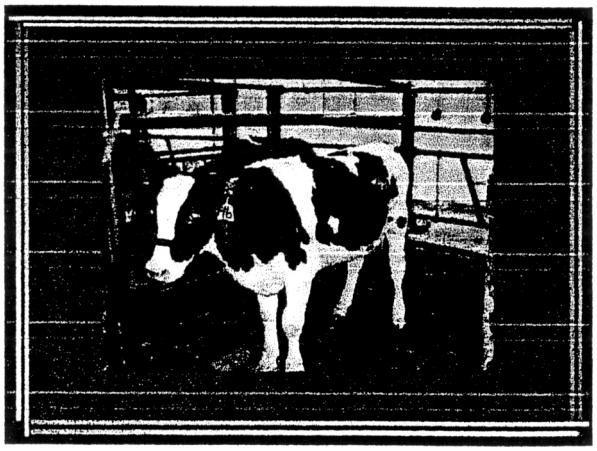



University of California Desert Research Center









Basal diet used in enzyme trials

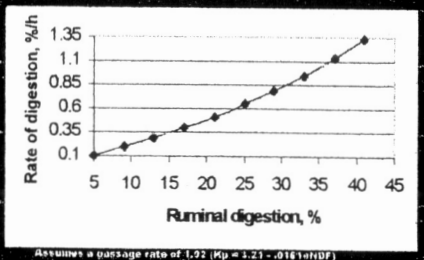
Item	%
Alfalfa hay	5.0
Sudangrass hay	17.0
Flaked corn	65.5
Limestone	1.0
Urea	1.1
TM salt	.4
Yellow grease	4.0
Cane molasses	6.0

Diet contained 19% NDF, 80% eNDF

Influence of Fibrozyme on ruminal digestion

Item	Control	Fibrozyme	
Replications	8	8	
Ruminal pH	6.44	6.41	
Ruminal digestion, %			
OM	61.0	63.2	
NDF	28.2	34.7	+ 23%
Starch	80.0	81.5	
Feed N	66.6	69.8	+ 5%
Microbial efficiency	24.0	23.8	

Rate vs extent of NDF digestion



Assumes a passage rate of 1.92 (Kp = 1.21 + .0161*eNDF)

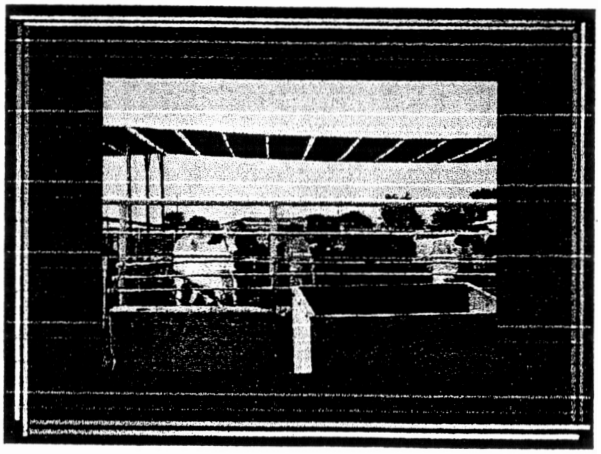
(265 kg steer, 19% dietary NDF, 80% e NDF)

Influence of cellulolytic enzymes on rate vs % ruminal NDF digestion

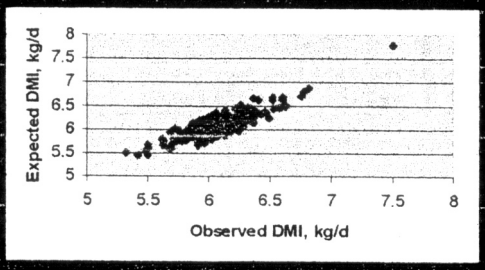
Item	Control	Fibrozyme	
Ruminal NDF digestion			
Kd, %/h	.75	1.04	+ 39%
Total, %	28.2	34.7	+ 23%

Influence of Fibrozyme on total tract digestion

Item	Control	Fibrozyme
Replications	8	8
Total tract digestion, %		
OM	76.7	76.8
NDF	39.4	39.9
Starch	98.5	98.4
N	63.7	63.9



Performance is a predictable function of energy intake



Variance in DMI for 205 close-outs

Generalized Maximal DMI For Feedlot Cattle Based on Dietary NDF

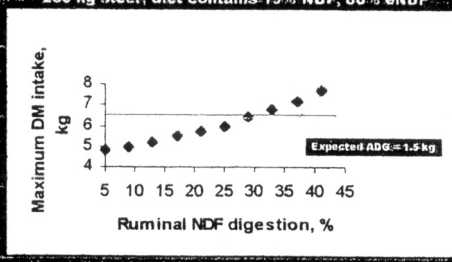
$$DMImax = \frac{(.098IW + 26.24) \times BW^{.75}}{(.01NDF \times (1 - .01RDNDF)) / ((.770 - .00385eNDF) \times (.042NDF - .027 - .00031NDF^2))}$$

where BW = IW + .0060353IW

DMImax = maximal dry matter intake, kg/d
IW = Initial body weight, kg
BW = Current body weight, kg
NDF = dietary NDF, %
eNDF = effective NDF, % total NDF
RDNDF = ruminal digestible NDF, %

Ruminal NDF digestion vs intake

265 kg steer, diet contains 19% NDF, 80% eNDF



Simulation based on generalized equation for feedlot steers (Zinn, 1999)

Expected maximal DMI for Control Steers based on Dietary eNDF

$$DMImax = \frac{(.098IW + 26.24) \times BW^{.75}}{(.01NDF \times (1 - .01RDNDF)) / ((.770 - .00385eNDF) \times (.042NDF - .027 - .00031NDF^2))}$$

IW = 180, kg
BW = 265 kg
NDF = 19%
eNDF = 80% of total NDF
RDNDF = 28.2 %

DMImax = 6.32 kg/d

Expected maximal DMI for Fibrozyme Steers based on Dietary eNDF

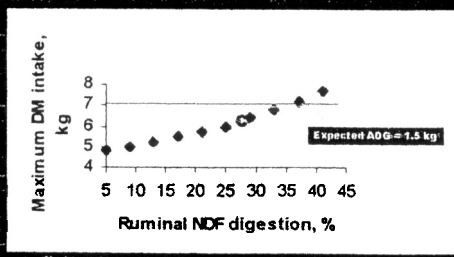
$$DMImax = \frac{(.098IW + 26.24) \times BW^{.75}}{(.01NDF \times (1 - .01RDNDF)) / ((.770 - .00385eNDF) \times (.042NDF - .027 - .00031NDF^2))}$$

IW = 180, kg
BW = 271 kg
NDF = 19%
eNDF = 80% of total NDF
RDNDF = 34.7%

DMImax = 7.07 kg/d

Ruminal NDF digestion vs intake

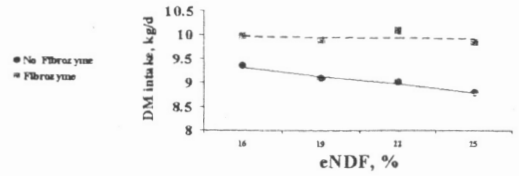
265 kg steer, diet contains 19% NDF, 80% eNDF



Influence of cellulolytic enzymes on steer growth-performance

Item	Control	Fibrozyme	
Replications	8	8	
Initial weight, kg	223	226	
Final weight, kg	308	317	
ADG, kg	1.33	1.41	+6.0%
DMI, kg	6.28	6.56	+4.6%
DMI/ADG	4.75	4.66	
Diet NEm, Mcal/kg	2.00	2.02	
Diet NEg, Mcal/kg	1.34	1.36	

Interaction of Fibrozyme and eNDF on DMI



Influence of Fibrozyme on DMI with a 35% forage diet

	Fibrozyme g / d		
	0	15	%D
DMI	9.05	9.95	9.9

Interactions of forage level and Fibrozyme on characteristics of ruminal digestion

	Forage level, %			
	33		66	
	Fibrozyme, g/d		Fibrozyme, g/d	
Item	0	15	0	15
OM intake, g/d[a]	3286	3295	9762	9809
Ruminal pH[b]	5.86	5.93	6.21	6.27
Ruminal digestion, %				
OM [cd]	72.3	74.4	72.5	72.4
NDF [ab]	46.3	51.0	62.0	59.3
ADF [cd]	32.4	46.5	58.6	53.9
Starch[ce]	90.8	94.2	93.6	93.6

[a] Forage level effect, P<.01. [b] Forage level x enzyme interaction, P<.05.
[c] Enzyme level effect (P<.10). [d] Forage level x enzyme interaction, P <.01
[e] Forage level x enzyme interaction, P <.10

Interactions of forage level and Fibrozyme on characteristics of total tract digestion

	Forage level, %			
	33		66	
	Fibrozyme, g/d		Fibrozyme, g/d	
Item	0	15	0	15
Total tract digestion, %				
OM [a]	81.9	83.1	79.9	80.3
NDF [ab]	62.0	65.2	68.0	68.8
ADF [cd]	52.3	58.8	64.4	62.6
N	76.7	79.6	74.9	76.8
Starch	99.5	99.7	99.6	99.5

[a] Forage level effect, P<.01. [b] Enzyme level, P<.05. [c] Enzyme level effect, P<.10
Forage level x enzyme interaction, P <.01

Influence of Fibrozyme and straw processing on digestion in Holstein cows

	Rice straw			
Item	Ground		Macerated	
Fibrozyme	0	15	0	15
OMI, g/d	10973	11256	10448	10923
Ruminal digestion, %				
OM [ab]	64.7	66.5	59	63
NDF [ab]	35.2	41.3	27.1	38.6
Starch	87.3	86.9	85.3	86.9
Feed N[c]	82.1	79.2	67.7	67.7
MN efficiency [d]	20.5	18.8	20.9	19.8
Kinetics of NDF digestion				
NDF Kp [a]	1.47	1.62	2.54	2.13
NDF Kd [be]	0.64	0.76	0.72	1.18

[a] Maceration effect P<.01. [b] Fibrozyme effect P<.05. [c] Maceration effect P<.05.
[d] Fibrozyme by maceration interaction, P<.10. [e] Fibrozyme level effect P<.10

Influence of Fibrozyme and maceration on digestion of rice straw in Holstein cows

	Rice straw			
	Ground		Macerated	
Fibrozyme	0	15	0	15
Total tract digestion, %				
OM [ab]	72.5	71.3	71.6	71.8
NDF [a]	42	41.2	44.8	46.5
Starch	98.7	98.7	99	98.7
N	66.8	67	64.4	63.9

[a] Maceration effect P<.05. [b] Maceration effect P<.01

Diet composition for trials 1 and 2

Item	Trial 1	Trial 2
Sorghum steam flaked	65.13	75
Limestone	1	1.45
Magnesium, oxide	0.2	0.13
Salt	0.4	0.39
Urea	1.1	1.07
Alfalfa hay, mid bloom	5	
Sorghum sudan, hay	17.1	11.41
Fat, yellow grease	4.01	3.06
Molasses, cane	6.14	6.99
Total	100	100

Influence of Fibrozyme supplementation on growth and finishing performance in feedlot steers

	Fibrozyme, g / d		
Item	0	15	%D
Phase 1, d 1 - 84			
Live weight gain, kg/d	1.49	1.58	6.0
DMI / ADG	5.88	5.62	4.4
Diet NEg, Mcal/kg[a]	1.33	1.38	3.7
Phase 2, d 84 - 145			
Live weight gain, kg/d	1.17	1.40	19.7
DMI/ADG	6.77	5.94	12.3
Diet NEg, Mcal/kg[a]	1.48	1.61	8.8
Overall			
Live weight gain, kg/d	1.40	1.54	10.0
DMI/ADG	6.06	5.68	6.3
Diet NEg, Mcal/kg[a]	1.37	1.46	6.8

Influence of Fibrozyme supplementation on DM intake (kg/d) in feedlot steers

	Fibrozyme, g / d		
Item	0	15	%D
Phase 1, d 1 - 84			
Observed, kg/d	8.75	8.92	1.9
Expected, kg/d	8.51	8.93	4.9
Observed/expected ratio	1.03	1.00	2.9
Phase 2, d 84 - 145			
Observed	7.95	8.33	4.8
Expected	7.79	8.80	13.0
Observed/expected ratio	1.02	.94	7.8
Overall			
Observed	8.52	8.75	2.7
Expected	8.19	8.80	7.4
Observed/expected ratio	1.04	.99	4.8

Conclusions

- **DM intake is positively associated with growth performance**
- **Increasing dietary NDF to regulate ruminal pH may depress DM intake**
- **Increasing ruminal fiber digestion by the addition of enzymes is an effective alternative for increasing DM intake**

Response to Fibrozyme may be greater when:

- Ruminal pH is less than 6.0 for a significant portion of the feeding interval
- Ruminal retention time of fiber is depressed
- Surface area of fiber is enhanced

Fibrozyme may enhance performance -- in a manner that is independent of its effects on fiber digestion.

???