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INSIDE THIS ISSUE:



## Silo Gas Alert

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(Preamble from Limin Kung, Jr., University of Delaware: In early September there was a death of a dairy farmer from silo gas poisoning in Juniata County, PA. Please read the following article and take extreme caution when opening or entering silos.)

One of the greatest risks of respiratory loss of life on dairy and livestock farms occurs during the ensiling process of corn and hay silage crops. That risk occurs from "silo gases" which form during the fermentation process in a silo. Silo gas can cause severe respiratory distress, permanent damage to lungs and even death. It is not something to be taken lightly.

Typically considered a problem with only corn silage crops, silo gas can and does occur during the fermentation of all silage/haylage crops. In fact, producers in Dauphin County commented earlier this year that when they were harvesting fourth cutting following the extreme drought conditions and the sudden and rapid rewetting of the fields in mid July that this cutting resulted in extremely high levels of silo gas, unlike any amounts ever noted from a haylage crop.

There are many different gases that form during the ensiling process including carbon dioxide and nitrogen oxide. The greatest risk from silo gas is from the nitrogen oxide which when exposed to air rapidly forms nitrogen dioxide (NO2). This highly toxic gas is characterized by a strong bleach-like odor and low lying yellow, red or dark brown fumes. Nitrogen dioxide, when inhaled into lungs mixes with moisture in the lungs to form nitric acid. This acid causes severe burning and irreversible scarring of the lungs and the respiratory system.

Even low level exposure to this deadly gas can create significant health problems. Death from silo gas can occur immediately or a farmer might breathe the gas without noticing any serious ill effects and then die in his/her sleep hours later from fluid that collects in their lungs. In many cases the victim suffers relapses with symptoms similar to pneumonia two to six weeks after the initial exposure. For these reasons it becomes extremely important for anyone who is exposed to this gas, even for a short period, to seek immediate medical attention.

Silo gas is heavier than air and because of this it will settle right on top of the silage or flow down the chute and collect in adjoining feed rooms or other low areas near the base of the silo. Gases may even flow out of the feed room and into the barn. Losses of livestock to silo gases have been reported. Good ventilation wherever possible around the silo is important while silage is fermenting.

Safety of your family is more important than livestock. Children should never be around feed rooms during the three weeks after filling when silo gas is of greatest risk. If needed keep doors to these areas locked or barricaded. Communicate these risks to all family members.

Approximately two to three weeks after filling it is unlikely more silo gases will be formed but hazards may still exist if the gas has not been able to escape the silo. If you must enter a silo to level off or set up the loader, do so immediately after the last load is in. The blower should always be running while you are inside. Leave silo doors open to allow gas to escape. If you must enter during the critical three week window after filling be sure to use a self-contained breathing apparatus. No respirator or dust filter can protect you from silo gas. You need a source of fresh air to breath.

## Energy Intake, Adiposity (body condition) and Average Daily Gains: Metabolic/Nutrient Interactions with Reproductive Function Driving Onset of Precocious Puberty in Heifers

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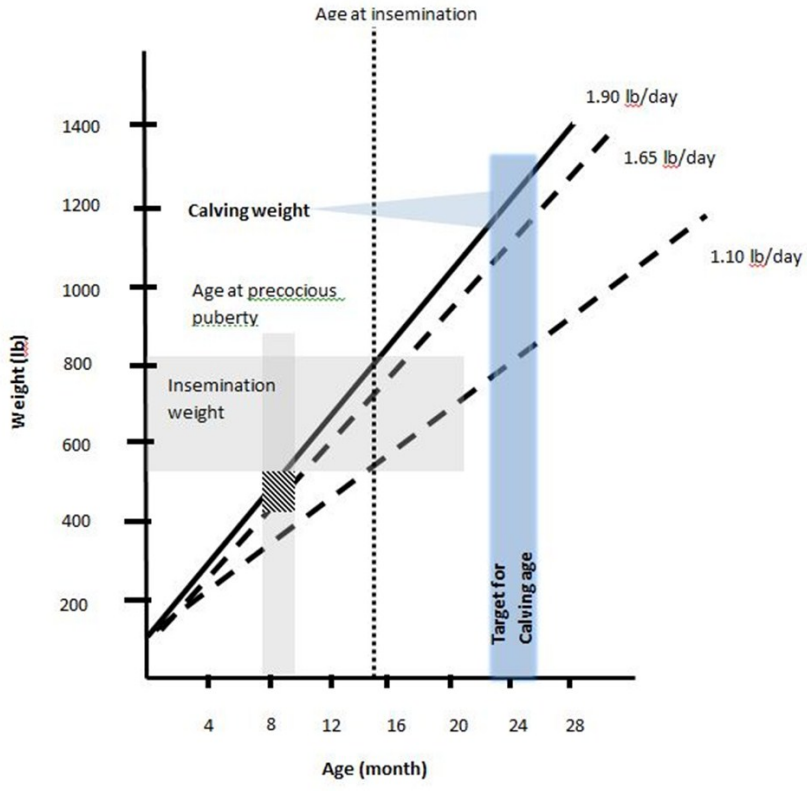
Age at the onset of puberty is highly related to prepubertal energy intake and average daily gains. Indeed, nutrient restriction during the often forgotten or neglected post natal period, stall growth, delays onset of reproductive maturity, and the onset of cyclicity and fertility in heifers. Important work over the past 4-5 years has begun to delineate pathways whereby centers sensing nutrient and metabolic status interact with higher centers in the brain orchestrating follicle growth, maturation and ovulation in the gonads (Amstalden et al., 2011). Work across a variety of animal species strongly indicates these higher centers of neurologic and endocrine function in the brain are anatomically integrated and impacted by nutrient status very early in the neonatal and prepubertal adolescent periods of growth. As a result, the onset of puberty in heifers is quite sensitive to nutrient and metabolic input very early in life. The nutrient effect can augment the onset of puberty before 300 days in heifers undergoing high rates of average daily gain (ADG) or delay the onset of puberty past 300 days in heifers experiencing lower ADG. Importantly, the data shows heifers can consume sufficient energy to drive different rates of gains in body weight but only those achieving *high rates* of ADG achieve precocious puberty. Growth *per se* is not the issue. Thus nutrient levels high enough to sustain highs rate of growth during the 3-7 months of prepubertal development is a key factor (Gasser et al., 2006a, 2006b, 2006c, 2006d).



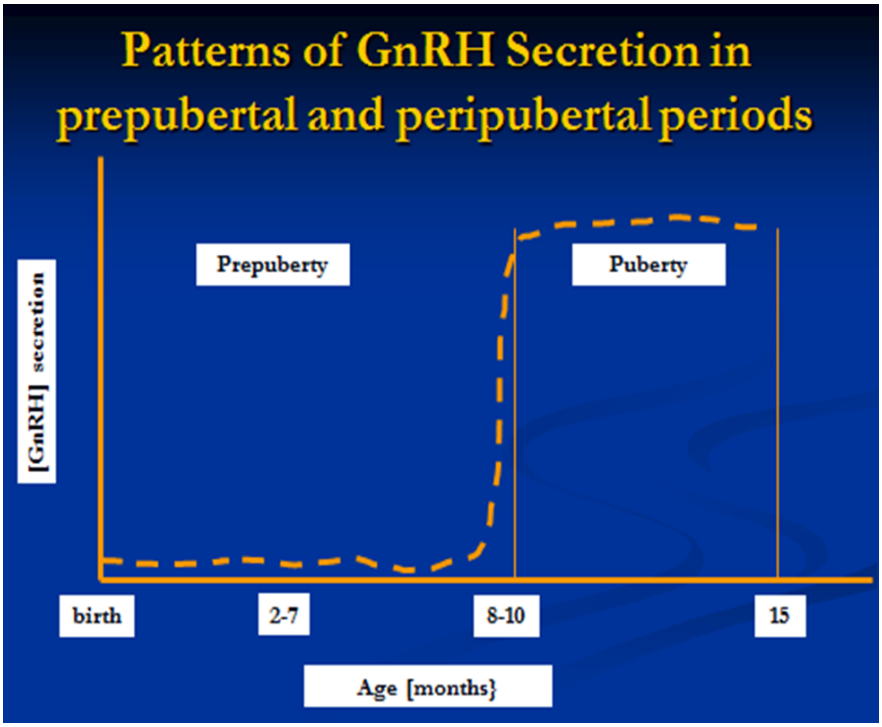
## Production implications of precocious puberty

The onset of reproductive maturity results from the integrated activities of the endocrine and nervous systems in heifers (Amstalden et al., 2011). Precocious puberty can be best defined as the onset of cyclicity with waves of follicle growth and maturation that generate ovulatory follicles prior to or by 150-180 days (5-6 months) of age. Onset of precocious puberty ensures heifers achieve sexual maturity and puberty at 40-45% (500-530 lb. body weight) and are inseminated at 60-65% mature body weight at 10-11 months of age. These targets enable heifers to progress through at least 3 estrous cycles prior to insemination to avoid insemination on the first estrus of puberty. This practice increases conception rates at the time of insemination by as much as 20% over conception rates on the first estrus of puberty. With a gestation length of 280 days (9-9.1 months) these goals allow replacement heifers to calve by an average age of 24 months at 85-90% of adult body weight. Entry into first lactation at 24 months also maximizes lifetime productivity and increases returns on the $1500 investment for each replacement heifer. Total investment in replacement heifers can be substantial in larger herds where annual culling rates (and therefore replacement animal requirements) average 30-35% of the adult herd population. Moreover, a 24 month goal for first calving reduces the number of animals that must be retained in the replacement heifer population when age at first calving becomes greater than 26 months of age.

Figure 1. Higher rates of average daily gain (ADG) in heifers during the prepubertal period (1.61-1.90) produce heifers that enter into puberty at a desirable age of 8-10 months (precocious puberty). Lower rates of ADG (1.10) do not promote adequate gains in body weight that support the onset of precocious puberty by the 8-10 month window. Weight and age for onset of precocious puberty.

What landmarks or objectives should producers expect to achieve that ensure early onset of puberty, conception at 14-15 months and entry into the lactating herd at 24 months of age? Assuming mature BW is 1250-1300lb, Holstein heifers need to achieve puberty at 40-45% (500-530 lb.) of mature BW, conceive at 60-65% mature BW (750-780 lb.) and then calve at 85-90% mature BW (figure 1). Reaching these goals requires ADG between 1.60-1.90lb/day. To ensure first parturition occurs at 24 months of age, heifers also need to enter precocious puberty between 8-10 months of age, and then conceive at 14-15 months of age. Figure 1 clearly shows heifers with low ADG will never achieve any of these milestones.

Ideally heifer weight gains, body condition score and height need to be carefully monitored in order to meet growth and age milestones in the prepubertal and post pubertal period of replacement development. Heifers growing too fast leave replacement pools early but may experience reduced first lactation milk yields (Sejrsen and Purup. 1997). Data is conflicting on whether these diminished milk yields in rapidly grown animals carry over into 2$^{nd}$ and later lactations. Ideally, rates of growth should be monitored to ensure replacements heifers calve at 1250-1300 lb. by 1$^{st}$ lactation. Discussions about high prepubertal ADG, BW and BCS always raise concerns about effects on mammary gland development because gland development during the prepubertal and peripubertal periods does impact milk yields. Normally, gland growth is proportional to gains in BW during the 1-2 month neonatal period and involves growth of ducts and supporting tissues. After 2 months, mammary growth surpasses gains in BW and involves ducts and fat tissues. Duct growth is critical for development of secretory tissue later during gestation. The onset of puberty (whether precocious (8-10 months or normal (11-12 months) normally slows mammary development. Although high rates of ADG enable earlier onset of puberty, a body of conflicting work (Daniels et al., 2009, Meyer et al., 2006, Davis-Rinker et al., 2006) exists about the effects of high ADG on mammary development in heifers. Some reports indicate planes of nutrition associated with growth rates of 1.5 lb-1.6lb/day may damage mammary gland growth and development, others show no effect of 2.0 lb./day gain on glandular development while still other reports of ADG as high as 2.3 lb./day hinder growth of mammary tissues. Most work indicates when development is diminished the effect is limited to the prepubertal period of mammary development (2-8 months). Higher rates of ADG consistently increase fat deposits in the mammary tissue leading to suggestions ration composition favoring excess fat deposition rather than high ADG *per se* diminish mammary development (Capuco et al., 1995, Silva et al., 2000). Interestingly, high rates of ADG attributable to non-adipose tissue deposition do not appear to impair mammary development (Siva et al., 2002). Altogether, data on higher planes of nutrition that support greater prepubertal ADG indicate there are great advantages stemming from enhance reproductive development but potential disadvantages due to diminished mammary development. The association of excess intramammary adipose tissue deposition on mammary development warrants producers pay particular attention to growth



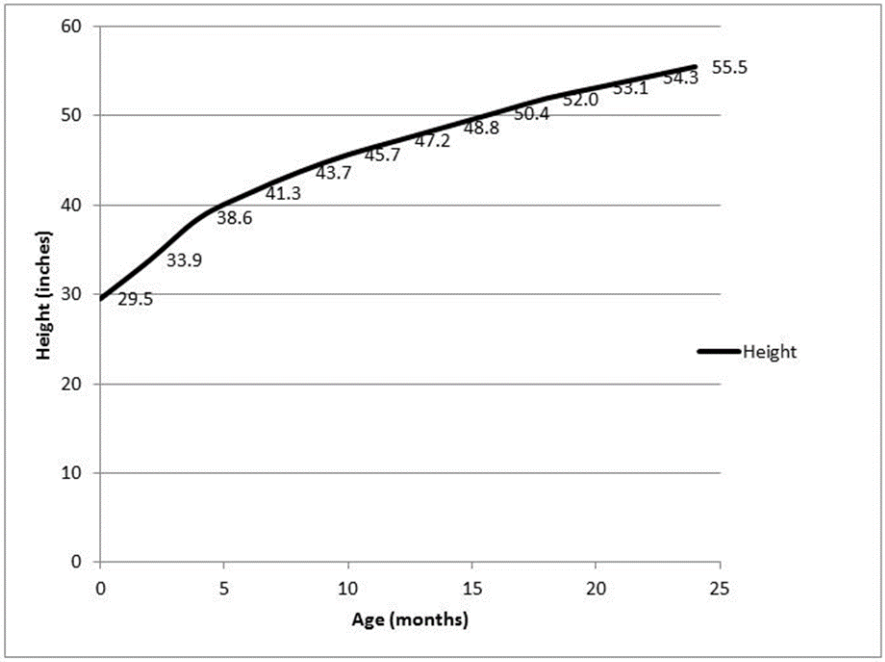

rates in conjunction with body condition scores in heifers. Since BCS is a reasonable estimate of adiposity, prepubertal heifers with BCS greater 2.5 may be at risk for under development of the mammary gland. Post pubertal rates of growth do not seriously impair mammary development.

**Effect of nutrition and ADG in the pre- and post-puberty periods on the onset of puberty**

Many studies have clearly established prepubertal and post pubertal energy intake and ADG impact the onset of precocious puberty (puberty at less than 300 days of age) (Radcliff et al., 1997, Schillo et al., 1992, Gasser et al. 2006a,b,c,d). Gasser (2006a,b,c,d) showed the effect of energy intake and ADG on onset of puberty was most pronounced in the early post-natal period (3-6 months of age) and had considerably less effective during the adolescent period immediately before the onset of puberty (6.5-13 months of age). Precocious puberty occurred on average at 9 months of age when heifers were fed high energy diets and showed higher ADG at 4-6 months of age. Heifers fed low energy diets and therefore achieving lower ADG during 4-6 months of age did not achieve puberty until 11 months of age. Delays in the onset of puberty in heifers on low energy diets during the 4-6 month prepubertal period were not corrected when these heifers were switched to higher energy diets 6.5-13 months immediately before the onset of puberty. The take home message is the onset of precocious puberty is driven by high energy intake and higher ADG in the 4-6 month post weaning period. Compensatory gains later in adolescent life cannot completely reverse the delay in onset of puberty associated with lower energy diets and poorer ADG at 4-6 months of age. The rate of growth after 5-6 months of age can be increased in poor gaining heifers at 3-6 months without much effect on the onset of puberty. Note, ADG was not the issue because all heifers gained weight and grew. The important concept is that only those energy levels great enough to support higher rates of ADG support earlier onset of puberty.

**Neuroendocrine factors supporting reproductive development and the onset of puberty**

Onset of puberty in heifers is initiated in higher centers in the brain that control release of gonadotropin releasing hormone (GnRH). Producers are very familiar with this hormone through its use in Ov Sync and, Pre Sync programs and treatment of cystic follicular degeneration in adult cows. Prepubertal increases in amplitude and frequency of pulsatile GnRH release from reproductive centers in the brain mark the onset of precocious puberty (Figure 2) at 8-10 months of age. Recently, a number of studies have begun to untangle the complex interactions between fat stores, metabolic/ nutrient sensing systems and the reproductive centers in cattle (Gasser et al., 2006a, 2006b, 2006c, 2006d, Allen et al., 2011, Armstalden et al., 2011, Redmond et al., 2011). These interactions integrate nutritional/metabolic status with the state of fertility (see figure 3). An important issue is recognition that fat synthesizes and secretes a hormone called leptin that signals the size of fat stores (therefore BCS) to nutrient/metabolic sensors in the brain. These nutrient/metabolic sensors regulate dry matter (appetite) and energy intake. In addition the metabolic/nutrient sensing centers are anatomically "hard wired" by nerves to neighboring centers in the brain that control reproductive development, cyclicity, follicle growth, follicle development and ovulation (figure 3). Higher energy intake increases fat stores (higher BCS) and therefore leptin signals to the metabolic/nutrient sensing centers. In the context of higher energy intake and therefore BCS (2.25-2.5), the metabolic/nutrient sensing centers increase positive signals while decreasing braking signals to reproductive centers. The net effect is to increase reproductive center output in the form of increased GnRH secretion that eventually drives ovarian functions such as folliculogenisis, ovulation, steroidogenisis, ovulation and the onset of puberty. Indeed, one endocrine manifestation of the onset of puberty is circulating levels of progesterone greater than 1 ng/ml se-



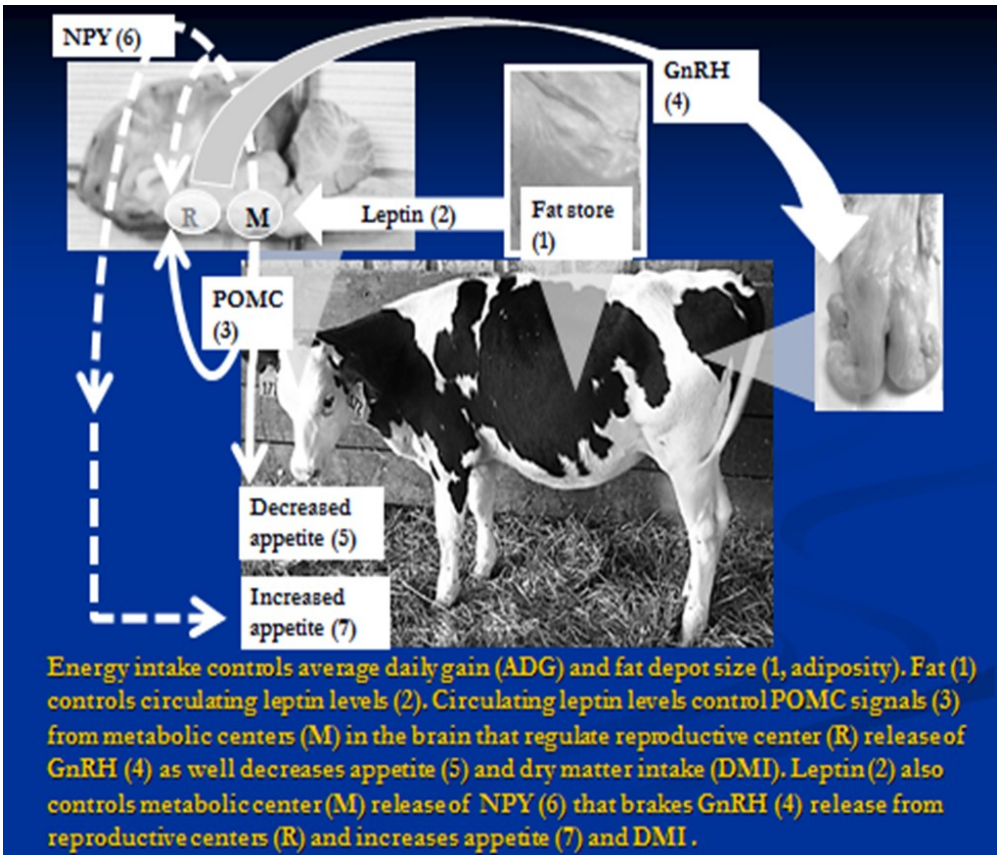

rum in heifers. Thus, growth rates, the size of adipose depots and BCS are closely related to the onset of puberty in heifers. The hard wiring between higher centers in the brain partially explains why growth restriction and lower ADG in prepuberty heifers delays the onset of puberty.

Figure 2. Low amounts of GnRH during the prepuberty period change to high amounts of hormone release at the onset of precocious puberty and cycling in heifers. Changes in GnRH are regulated by signals that integrate metabolic/ nutrient sensing centers with reproductive centers in the brain.

## Conclusion

Young stock can be nutritionally pushed into precocious puberty by feeding diets designed to achieve higher ADG and BCS. ADG and BCS however, need be monitored closely to ensure age-dependent changes in ADG and BCS are neither too little nor too big. Combined use of targets for whither height (figure 4) and body weight (figure 1) within each breed is an excellent method to guide pre- and post puberty growth and development.

Figure 4. Replacement heifer whither height by age (after Hoffman, 1997, Heinrichs and Hargove, 1987).




Excessive ADG and BCS may lead to underdevelopment of mammary glands and obesity at 24 months and first calving. ADG and BCS that are too low lead to delayed puberty, delayed conception and first calving past 24 months of age. In Holsteins, pre-pubertal ADG between 1.6-1.9 lb. /day and BCS 2.5-3.0 usher in precocious puberty. The reproductive goals are to achieve conception at 14-15 months of age in heifers at 60% of mature body weight and BCS of 3.0 (Heinrichs and G.L.Hoffman, 1987. Hoffman. 1997). Heifers should achieve precocious puberty at less than 300 days of age and 40-4% of body weight. Onset of puberty at this age enables breeding 3 or more estrous cycles after the first estrus of puberty. Higher growth rates after 6-6.5 months of age may not completely restore reproductive advantages to levels garnered by heifers with higher growth rates prior to 6-6.5

months of age. Calves experiencing the entire spectrum of neonatal and pre-pubertal disease problems show delayed gains and greater age at first calving. Poorer nutrition, disease problems and environmental factors that diminish ADG for longer periods of time in the prepuberty period can be expected to delay the onset of puberty and extend age at first calving.

Figure 3. Onset of puberty is controlled by communication between fat stores, higher centers in the brain that consist of nutritient/metabolism sensing elements (M) hard wired to reproductive (R) centers in the brain. The reproductive centers communicate with the reproductive tract by producing GnRH. Signals from nutrient/matabolic centers can increase or decrease GnRH release and therefore the onset of puberty by reproductive centers in the brain.

References upon request

## Factors That Negatively Affect Silage Quality and Animal Performance: Prevention and Remediation

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### Introduction

The primary goal of making silage is to preserve the maximum amount of DM and nutritive quality that is in the field at harvest so that it can be stored and retain its quality for feeding at a later date. However, many factors including the maturity at harvest, management at harvest affecting the resulting fermentation, time of ensiling, and management during storage and feed out can have negative effects on silage quality. This paper will review some of the major factors that negatively affect silage quality and briefly discuss potential ways to prevent their occurrence and cope with their effects.

### Quality Factor: Low NDF-Digestibility

The primary factor affecting NDF-digestibility (NDF-D) in silages is the stage of maturity at harvest. This is due to the fact that as forages mature, there is an increase in the concentration of lignin that hinders NDF-D (Smith et al., 1972). In general, normal silage fermentations do not directly affect the digestibility of fiber. However, excessive heat during fermentation is known to depress NDF-D especially in alfalfa harvested at high DM because proteins bind with fiber and form indigestible bonds (Yu and Veira, 1977).

### Prevention/Remediation for Low NDF-D

Preventing low NDF-D in silage is probably best managed by harvesting at suggested maturities for the specific crop. Preventing low NDF-D due to heat damage can usually be prevented by ensiling crops being stored in conventional silos before they reach 50-55% DM. An adequate length of chop and good packing density will aid in expelling excess amounts of air from the forage mass.

Remediation to increase NDF-D in silages has not yielded consistent results. For example, plant cell wall degrading enzymes (e.g., cellulase and hemicellulase enzyme complexes) have been used to improve the NDF-D of silages. However, in a review of these enzymes as silage additives, Kung et al. (2003) cited cases where their addition resulted in lower fiber digestibility of the resulting silage because treatment predigested the easily degradable cell wall fractions leaving the more difficult to digest material behind. They also cited publications where in some instances, treatment with these enzymes was overly effective, resulting in an undesirable increase in soluble effluent. Many current silage additives contain cell wall degrading enzymes but have not resulted in consistent positive effects on improving NDF-D.

A more reasonable method to improve NDF-D in silages with enzymes was suggested by Nsereko et al. (2008). They reported NDF-D in silage could be improved via the addition of strains of bacteria expressing activity for ferulic acid esterase (FAE). This application would be less likely to result in excess solubilization of fiber. Kang et al. (2009) reported improvement in NDF-D in one of two hybrids treated with an additive containing a strain of *Lactobacillus buchneri* with FAE activity. Using the same strain of bacteria, Hofherr et al. (2008) reported an improvement in NDF-D in 1 of 3 corn hybrids but it took 360 d of ensiling before the effect was noted in the one hybrid. Lack of an ability to measure increases in NDF-D using conventional in vitro digestion techniques in silages treated with this organism has been puzzling. Current efforts are underway to more accurately assess the effects of this specific additive via gas analyses. Future studies may use direct additions of FAE as a silage additive but this practice may be cost prohibitive. Improvements in NDF-D in silages by a variety of other microbial inoculants have been reported (Keady and Steen, 1995, Weinberg et al. 2007, Reich and Kung, 2010) but the exact modes of action were not clear and the repeatability of findings is uncertain.

Although Hallada et al. (2008) reported an increase in NDF-D with time of ensiling for corn silage, others have not been able to repeat this finding (Der Bedrosian et al., 2012; Snyder, 2011). We also did not find any effect of time in storage on NDF-D of alfalfa silage harvested at two DM contents and ensiled more than a year (Kung et al. 2010, unpublished data University of Delaware).

## Quality Factor: Low Starch Digestibility in Silages

Several physical and chemical factors are responsible for low starch digestibility (starch-D) in silage crops. First, starch-D is low if corn is not adequately processed because the pericarp presents a physical barrier to microbes and enzymes that need to access the starch granules. Second, starch-D in corn silages can be relatively low if the plant is harvested at an overly mature stage of maturity (~>45% DM compared to 35% DM) because of increased complexity of the prolamin-starch matrix (Hoffman et al., 2011). Studies have also shown that ruminal starch digestion is relatively low at harvest but increases with time in the silo for both corn silages (Philippeau and Michalet-Doreau, 1998, Allen et al., 2008) and high moisture corn (Benton et al. 2005). This finding may partially explain why many dairies experience a drop in milk when abruptly shifting from last year's corn silage to fresh forage that has not undergone several months of ensiling.

## Prevention/Remediation of Low Starch-D in Silages

Improvements in starch-D in corn silages can be easily obtained by breaking the pericarp via kernel processing at harvest and the effect is greater with advancing kernel maturity (Bal et al., 2000). (Post ensiling processing is not the norm but I have seen a custom-made kernel processor that could post-process enough corn silage for a 1000 cow herd in under 30 minutes on a farm in PA.) The corn silage processing score can be used to ensure an adequate degree of processing has been obtained (Mertens et al., 2002).

Because starch-D improves with time in the silo for corn silage and high moisture corn, storing these silages for 4-6 months before feeding has been recommended (Hallada, 2009; Behling, 2007; Ward and de Ondarza, 2008). However, this practice requires increased growing and storage capacities that are not options for many dairies. Because a proteolytic mechanism most likely explains the improvements in starch digestion found with time in the silo, Young et al. (2012) hypothesized that adding exogenous proteases at ensiling could be a method to improve ruminal starch digestion after only a short period of ensiling. In that study, addition of a high concentration of an exogenous protease at harvest resulted in corn silage with greater in vitro ruminal starch-D after 45 d of ensiling compared to untreated silage after 145 d of ensiling. Additional studies from my lab have shown that this effect is repeatable and also can be obtained in high moisture corn treated with proteases (several abstracts to be presented at the Annual Meeting for the American Dairy Science Association, Indianapolis, IN, July 8-12, 2103). Many unanswered questions remain on the potential use of proteases for this application. For example, there may be interactions between protease activity and a) the degree of processing (because the protease enzyme needs to have access to starch), b) DM of the crop (drier forages ensile slower), and c) temperature of storage (enzyme activity will probably be accelerated at high temperatures but reduced at low temperatures). However, development of a commercial additive is expected in the near future.

### Factor: Abnormal Amounts of Silage Fermentation Acids

There have been many attempts to correlate the end products of silage fermentation with animal performance. Although feedback from the field suggests that silages high in acetic acid depresses intake, data supporting this contention has been variable. Eisner et al. (2006) reported that the concentration of acetic acid in silage was negatively correlated with intake when silage and concentrates were fed separately. However, total acid concentration was the best factor that increased the fit of a model for the prediction of DM intake when animals were fed a total mixed ration. In a summary of research studies, Huhtanen et al. (2007) reported that only total acid and propionic acid concentrations were negatively correlated with intakes in lactating cows. In steers, Krizsan et al. (2007) reported that 71% of the variance of intake of 24 low DM grass silages was best explained by total acids, total volatile fatty acids, lactic acid/total acid ratio and propionic acid. Propionic acid is seldom found in well-fermented silages. It is more common to observe high levels of propionic acid (> 0.3 to 0.5%) in poorly fermented silages, especially because it can be an end product from some strains of clostridia. Overall, these studies tend to support the anecdotal reports from the field that intakes are depressed when lactating cows are fed wet silages that tend to have high concentrations of total acids. Other negative end products from silage organisms may also play a role in reduced intake from silages. For example, Figueiredo et al. (2007) reported finding at least 168 volatile compounds in red clover silage but the effect(s) of most of these compounds on DM intake in ruminants has not been studied.

### Prevention/Remediation of Abnormal Amounts of Silage Fermentation Acids

Total acid concentration in silages is negatively correlated to dry matter content of the crop. In drier silages, fermentation becomes restricted because water activity limits the growth of microbes responsible for fermentation. Thus, wilting to appropriate DM contents can prevent high levels of acid formation during ensiling. Neutralizing silages high in acid content with sodium bicarbonate prior to feeding (about 0.5 to 1% addition on DM basis) has been effective in some instances to improve intake (Shaver et al., 1984). Some strategies for managing silages with high (>5%) acetic acid, high ethanol (> 3-4%), high butyric acid (> 0.5%) include: reducing the amount of that silage fed, aerating the silage to volatilize the acids, and removing the silage and then gradually reincorporating it back into the diet over a 2–3 week period, but known of these practices have been studied in detail.

### Quality Factor: Clostridial Fermentations

Corn silages seldom undergo a clostridial fermentation but legumes in particular are highly susceptible because of their high buffering capacity. Clostridia can dominate a fermentation when there is an excessive amount of moisture, excessive amounts of air, a slow drop in pH, and when there is a lack of fermentable substrate. Both saccharolytic and proteolytic strains of these organisms have been identified in silages (Pahlow et al., 2003). These fermentations can be easily recognized by their foul smells. Clostridial fermentations are undesirable because of low recoveries of both energy (78%) and DM (66%) (Rooke and Hatfield, 2003). In addition, the end products of these fermentations can negatively affect animal performance. For example, clostridial silages often contain biogenic amines. Van Os et al. (1997) reported markedly lower intakes in unadapted sheep fed biogenic amines but this effect was slightly tempered by adaptation over a 14-d period suggesting ruminal adaptation to the amines. Phuntsok et al. (1998) reported significant ruminal metabolism of biogenic amines but still reported a linear decrease in ruminal motility, DMI, ruminal DM digestibility, and ruminal DM outflow as the amount of alfalfa silage with biogenic amines increased in the diet. Slipchenko (1994) reported that cows fed silage high in butyric acid produced less milk and prolonged periods of infertility.

Some clostridia found in silages also produce potent toxins that can affect animal performance. For example *Clostridium botulinum* produces one of the most potent classes of toxins known to man. The spores of these bacteria are wide spread in the environment but are dormant. Under anaerobic conditions and with the right nutrients, the spores can germinate and grow, thereby releasing toxins. There are eight known botulism toxins that are produced but there is some species specificity. For example, types A, B, C, and D toxins commonly affect livestock. The type B toxin is associated with poorly fermented legume or grass silages (especially ryelage) with high pH (> 4.5). There has also been an increased frequency of the disease with the advent of using big bales wrapped in plastic. Botulism toxins are not commonly found in corn silage because the pH (usually less than 4) prevents the growth of the clustridia. Type C botulism toxici-

ty is usually associated with decomposing carcasses. Type C toxicity has been reported with feeding of poultry litter to ruminants. In some instances, botulism toxicosis can be due to the consumption of silages that were contaminated with animal carcasses during ensiling or feeding (Anon. 1998).

**Prevention/Remediation for Clostridial Fermentations**

Several management practices may help to prevent a clostridial fermentation. First, clostridia are intolerant of high osmotic pressure and thus the recommendations to wilt forage above 30-35% DM have been made. Second, clostridia are relatively intolerant to low pH and thus any practice that stimulates a rapid acidification of the forage mass would be helpful. For example, wide swathing to minimize the time a crop wilts in the field, and fast and dense packing contribute to promoting a rapid drop in pH. Use of a good homolactic acid inoculant which hastens the drop in silage pH (Muck, 2010) and management in the field to minimize contamination from spores in the soil can also lower the incidence of clostridia in the forage.

Anecdotal improvements in intake have been reported when feeding clostridial silages if the silage is spread out thinly and allowed to aerate overnight prior to feeding, which results in volatilization of butyric acid and $NH_3$-N. Aeration does not pose a threat for increased aerobic spoilage because butyric acid is an extremely strong antifungal agent.

Oetzel (2007) suggested that there is no "safe" dose of butyric acid in silage for dairy but also offered the "divert, dilute or destroy it" options for dealing with these types of silages. For the first two options he suggested that such silages could be fed to late lactation cows, far off dry cows, or replacement heifers at no more than 50 g of butyric acid consumption per day.

**Quality Factor: Yeasts in Silages**

High numbers of yeasts are undesirable in silages for two reasons. First, under anaerobic conditions yeast can ferment sugars and produce ethanol resulting in large losses of dry matter (McDonald *et al.* 1991). Extremely high intakes of ethanol can cause off flavors in milk. Second, under aerobic conditions, lactate-assimilating yeasts are responsible for the initiation of aerobic spoilage. The degradation of lactic acid by yeasts causes a rise in silage pH to a level that allows opportunistic bacteria (e.g. *Bacilli*) and molds (e.g. *Aspergillus*, *Fusarium*, and *Pencillium*) to grow and further reduce silage quality (McDonald *et al.* 1991). Aerobic deterioration in silages probably accounts for more than half of the DM loss during storage of silages.

Complaints from the field about silages with high levels of yeasts causing problems in dairy herds are common. Several feed laboratories have suggested guidelines for feeding spoiled silages based on numbers of yeasts and molds. Anecdotal evidence from the field suggests that a reduction in animal performance occurs when the numbers of yeasts are more than $10^6$ (1,000,000)/g of silage. However, few studies have been conducted that directly implicate yeasts themselves as the primary problem. When animals consume spoiled silages with high numbers of yeasts, the exact causes of reduced intake and/or performance are not fully understood. Oxidation of nutrients reduces the nutritive value of spoiled silages and causes heating. However, detrimental yeasts in silage may also compete with rumen microbes for nutrients and may produce end products that might alter rumen fermentation. Recently, Santos et al. (2011) reported that adding high levels of *I. orientalis* to in vitro ruminal fermentations reduced NDF-D. Windle et al. 2013, (unpublished data University of Delaware) reported that heifers fed a spoiling TMR containing more than 66,000,000 yeasts per g of TMR ate less DM than heifers fed the same TMR that was fed fresh and contained only about 110,000 yeasts/g. Growth of molds in spoiled silage may result in the production of mycotoxins that could negatively affect animal performance. Effects on immune function may occur, but are not well documented. Spoiled silage may also be simply unpalatable to animals.

**Prevention/Remediation for Yeasts in Silages**

Proper silo management can help to prevent high numbers of yeasts in silages. Filling silos quickly with sufficient pack weight to maximize density and minimize porosity can reduce oxygen in a silo. Oxygen barrier plastics are also now available for use (Borreani et al., 2007). Such practices can reduce the number of yeasts in silages and improve

aerobic stability. Proper management for removal of silage from silos at the feed bunk can to slow the growth of yeasts in silage. Enough silage should be removed between facing to minimize aerobic spoilage. Removal of silage should be such to minimize disruption of the silage face and loose silage on the ground between feedings. Extreme care should be taken to prevent air from penetrating between the plastic and reaching the silage mass during feed out and storage and this can be accomplished by stacking tires, or lining gravel bags on the plastic at the leading edge of the feeding face.

Coupled with sound silo management, the addition or formation of sufficient quantities of antifungal end products in silage is probably the best way to prevent high numbers of yeasts in silages. For example, buffered propionic acid -based products are commonly used because of they are less corrosive and safer to handle than the straight acid. Potassium sorbate, benzoate and acetic acid are also commonly found as components of many antifungal formulations but are generally too expensive to be used alone in high concentrations. It is the undissociated form of organic acids that is responsible for their antifungal properties and its prevalence is dependent on pH. This fact, unfortunately, means that more acid is needed to be effective in crops that are naturally limiting in acids from silage fermentation (e.g. crops with more than 40% DM). At the pH of a standing crop of alfalfa (about 6) only about 1% of propionic acid is in the undissociated form whereas, at a pH of 4.8, about 50% of the acid is undissociated. The undissociated acid functions both by staying active on the surface of microorganisms and competing with amino acids for space on active sites of enzymes and by altering the cell permeability of microbes. Undissociated acids also can penetrate into microbial cells and disrupt cytosolic functions because of the release of $H^+$. Application of buffered propionic acid-based products ranges from about 1 to 6 lb/ton of forage depending on the specific situation. The efficacy of low application rates is questionable. For example, if 2 lb of a product that contained 65% propionic acid were added to 35% DM corn silage, this would increase the propionic acid content in that silage by 0.18% on a DM basis

Bacterial inoculants based on homofermentative lactic acid bacteria are commonly added to silages to improve fermentation and increase DM and energy recovery. However, most of these inoculants do little to inhibit growth of yeasts because they tend to maximize the production of lactic acid (poor antifungal activity) and decrease the accumulation of other organic acids that have good antifungal activity. Muck and Kung (1997) reported that a summary of the literature showed that treatment with classical homolactic acid-based inoculants improved aerobic stability about one third of the time, had no effect about one third of the time but made aerobic stability worse about one third of the time. A better additive to control the numbers of yeasts in silages is *Lactobacillus buchneri.* This organism is an obligate heterolactic acid bacterium that has been shown to suppress the numbers of yeasts in a variety of silages by converting moderates amount of lactic acid to acetic (Kleinschmit and Kung, 2006). Increases in acetic acid due to *L. buchneri* are typically the equivalent of 8 to 10 lb of acetic acid per ton of silage.

There are no known methods to remediate silages with high levels of yeasts. Spraying acids on the face of silos with spoiling silages is not an effective solution. Some have attempted to kill yeasts by mixing organic acids into the TMR prior to feeding, but success of this practice has not been studied under controlled conditions.

## Quality Factor: Mycotoxins in Silages

A variety of mycotoxins produced from fungi can be found in silages that are fed to dairy cattle (Driehuis et al., 2008). Their presence is undesirable because they have the potential to induce negative effects on the health of animals (CAST, 2003). The variety of mycotoxins present in silages is fairly wide including aflatoxins, zearalenone, trichothecenes, and the fumonisins. Their effects on animal metabolism are wide spread including some direct effects in the rumen but wide spread effects on the immune system probably explain many of their adverse reactions. Mycotoxins can accumulate on the plant in the field before harvest (Doerr, 2010), during storage or during processing or feeding (Whitlow and Hagler, 2008). Munkvold (2003) noted that the presence of mycotoxins on corn was dependent on susceptibility of the plant, environmental conditions that encouraged infections, and the presence of fungi capable of producing toxins. A common avenue of fungal infection occurs from damage to plants by insects (Dowd, 1998). This results in easy access to nutrients for fungal spores and encourages colonization because fungal hyphae are able to penetrate into the plant (Jouany, 2007). The thick waxy layer of the corn kernel pericarp is an important factor in limiting the accumulation of mycotoxins on kernels of corn. Sampietro et al. (2009) reported that the removal of kernel wax with chloroform increased the concentration of fumonisin on corn kernels.

## Prevention/Remediation for Mycotoxins in Silages

Methods to maintain optimal plant health during the growing season are the best way to prevent or minimize the occurrence of mycotoxins on forages in the field (Munkvold, 2003). Good practices including proper plant density, tillage, and crop rotations keep plants healthy and minimize the survival of toxigenic fungi in the environment. Prevention of toxin development during storage should include a program that emphasizes good silo management to prevent aerobic spoilage because the growth of yeasts under aerobic conditions precedes the development of molds capable of producing mycotoxins. During silage feed out, good management practices including the removal of adequate amounts of silage per day and maintaining a clean feeding face help to deter aerobic spoilage.

For forage crops, there is no successful way to lower the concentrations of mycotoxins in the silo. The ensiling process it self has had variable effects on the concentrations of mycotoxins during ensiling and thus should not be expected to consistently lower concentrations of mycotoxins coming in from the field. For example, Fink-Gremmels (2005) suggested that mycotoxins originating during pre-harvest and present before ensiling often remain stable and present in the preserved material for a long period of time. However, Mansfield et al. (2005) reported about a 50% reduction in the concentration of DON in fresh versus ensiled samples collected in the Northeastern US. Bourda and Morgavi (2008) reported as much as 100% disappearance of DON in low DM (28%) corn silage. In contrast, Teller et al. (2012) reported variable changes in the concentrations of mycotoxins between fresh and ensiled forages but in all instances the differences were relatively small.

If a definitive determination has been made that high levels of mycotoxins are present in silage, calculations should be made relative to the concentrations being fed to determine if total diversion is needed. The use of various binders to remediate silages with high levels of toxins has been discussed by Whitlow and Hagler (2010).

## Conclusions

Many factors can negatively impact the quality of silages. In most instances good management clearly is the best answer for preventing abnormal fermentations from occurring because methods of remediation are not always available or successful.

References upon request

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