

Subacute Ruminal Acidosis – Milk Fat Responses and Prevention by Nutritional Management

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This paper will discuss two aspects of subacute ruminal acidosis (SARA) in dairy herds. The first is the relationship of milk fat depression and SARA, and the second is a review of the nutritional principles for preventing SARA in dairy herds.

Defining Low Milk Fat Test Problems

The relationship between SARA and milk fat depression is inconsistent and complex. It can be surprisingly difficult, at times, to determine if a herd really has a low milk fat test or not. Normal milk fat percentage depends greatly on breed, season, and days in milk. Nutritional causes of milk fat depression (which may include SARA) only become a concern when these three major factors have already been taken into account.

The expected effect of season on milk fat percentage is presented in Figure 1. Milk fat test is normally about .25% lower in the summer than in the fall months. This effect is not completely understood, but could be mediated by increased risk for SARA. Cows are apparently at higher risk for SARA in the summer due to lack of ruminal buffering caused by heat stress, increased respiratory rate, respiratory alkalosis, and low blood bicarbonate concentrations. Other causes of increased SARA in the summer months could include atypical meal patterns in response to heat avoidance and ration formulation errors made when nutritionists attempt to compensate for reduced dry matter intake during heat stress by decreasing dietary fiber. This only makes cow performance and milk fat test worse.

Milk fat depression is broadly defined as herd average milk fat test below 3.2% in Holstein, Ayrshire, and Milking Shorthorn herds, below 3.4% for Brown Swiss herds, below 4.0% for Guernsey herds, and below 4.2% in Jersey herds. These definitions are clinical impressions only and are not based on rigorous epidemiological research.

Milk fat depression cannot be defined without first knowing the days in milk of the cows being evaluated. Herds typically do not have a wide variation in days in milk (unless they are seasonally calving). However, individual cows can have quite variable milk fat percentage due to days in milk alone. Days in milk can account for changes in milk fat percentage up to about .75% (see Figure 2).

Laboratory methods for milk fat testing are notoriously fallible. Most milk is tested for fat content by near infrared reflectance spectroscopy (NIRS). This test performs well compared to wet chemistry procedures (e.g., the Babcock test) when milk fat percentage is in the middle range. Thus, NIRS works well for bulk tank samples. However, it is not as accurate for very

high or very low milk fat percentage in individual cows. It also may be affected by somatic cell count or other changes in milk properties.

Milk fat (the cream) rises to the top of a milk sample that has not been agitated or homogenized. This can cause error in any type of milk fat sampling. Bulk tanks must be agitated before sampling for milk fat percentage. And individual cow samples must be shaken before testing in the lab.

Bulk tanks can be agitated too vigorously, which results butter formation within the tank. This could artificially lower milk fat percentage.

Milk fat percentage also varies dramatically from the start to the end of milkout. Thus, milk samples from individual cows must be collected with a proportioning device that meters out a representative portion of the entire milking. Strip samples from cows have no value whatsoever for milk fat testing.

DHI milk fat tests for the entire herd are determined by testing the milk fat percentage and milk volume from each cow. This is usually done for just one daily milking, and only once a month. The milk fat test for the herd is then calculated by a weighted average of the milk fat and milk volume contribution from each cow. Milk fat percentages are adjusted for AM or PM milkings, since the AM milking typically has higher volume and lower milk fat percentage. However, these corrections are estimates only, and become even less accurate when cows are milked more than twice daily. DHI milk fat test results are useful for looking at trends by days in milk and for variation in milk fat test. But they are not a particularly accurate measure of whole herd milk fat percentage. The milk fat test from the bulk tank is a much more accurate indication of whole herd milk fat percentage. Bulk tank milk fat percentages are also available on a daily or every other day basis. In contrast, DHI milk fat percentages usually represent only one milking per month.

Milk fat tests are typically interpreted as group or herd means. This is generally appropriate and is certainly convenient. However, group means may obscure outlier cows with very high or very low fat tests. And cyclic milk fat depression could be completely missed if individual cows within a group have milk fat depression at different and offsetting times. Thus, it can be helpful to interpret milk fat percentages as a proportion of cows with very high or very low test results. Very low milk fat tests (<2.5% for Holsteins) should not be present in more than about 10% of the cows tested on any single DHI test. The cows below 2.5% should typically only be cows between about 30 and 70 days in milk.

Milk fat : milk protein ratios are commonly used to evaluate milk fat depression. The often-accepted belief is that inversions of the milk fat: protein ratio (i.e., milk protein percentage > milk fat percentage) is a better reflection of milk fat depression than the milk fat percentage alone. This concept is not supported by science. Milk fat synthesis and milk protein synthesis are separate physiologic processes. Interpreting one only in light of the other most likely introduces even more error into the diagnosis of milk fat depression. It is interesting to note that the interpretation of milk fat: protein ratios did not appear to change after the basis for milk protein reporting was changed from crude protein to true protein in early 2000 in the US. I do

not recommend attempting to interpret milk fat: protein ratios when investigating milk fat depression problems.

Milk fat to milk protein ratios may have value in supporting a diagnosis of subclinical ketosis in early lactation cows. This is true because subclinical ketosis causes both an increase in milk fat percentage and a decrease in milk protein percentage. However, keep in mind that this is a completely different clinical issue than diagnosing milk fat depression.

Causes of Milk Fat Depression

If a diagnosis of milk fat depression is clearly made in a dairy herd, then there are three major causes to consider –over-feeding unsaturated fats, monensin feeding, and ruminal acidosis. All three causes share the same end pathway – the absorption of excessive amounts of certain *trans* fatty acids from the small intestine. More than one of these factors may be present in a herd, and their effects on milk fat depression are additive.

Excessive intake of dietary unsaturated fats is the most predictable and repeatable of all the causes of milk fat depression. Unsaturated fats cause milk fat depression when they are transformed and then incompletely biohydrogenated in the rumen. Some of the intermediate forms of these fatty acids (particularly *trans* 18:1 fatty acids) are then absorbed at the small intestine and taken up by the mammary gland (Figures 3 and 4). There they strongly inhibit milk fat synthesis, even at very low doses (5 grams or less per day).

The degree of escape of *trans* fatty acids from the rumen depends largely on the amounts of their unsaturated fat precursors (18:2 and 18:3 fatty acids) are present in the diet. High rates of passage also contribute to more escape of these fatty acids to the rumen. These fatty acids do not have detrimental health effects themselves; thus, it is possible for a herd to have milk fat depression without cow health problems.

The potential for plant source fats to cause milk fat depression depends on both their content of unsaturated fatty acids and on their rate of release in the rumen. Slowly released plant fat sources such as whole cottonseeds present much less risk for milk fat depression than do rapidly released plant fat sources such as wet distillers grains.

Animal source fats or ruminal by-pass fats pose little risk for incomplete biohydrogenation and milk fat depression (Figure 5). Rather, the risk with these fats can be reduced total tract digestibility as they by-pass not only the rumen, but the entire digestive tract as well.

Monensin supplementation may contribute to milk fat depression. Supplementing monensin on the high end of the dosage range (16 to 24 ppm – the same as 15 to 22 grams per ton) depresses milk fat percentage about .1 to .2%. The degree of depression may wane as the monensin is fed for a longer time period. Monensin feeding appears to interact with excessive feeding of unsaturated fats and ruminal acidosis to cause milk fat depression. Interestingly, monensin helps prevent ruminal acidosis (probably by inhibiting lactate producers and favoring lactate utilizers), yet still has the overall effect of reducing milk fat percentage. If a herd experiences noticeable milk fat depression soon after starting to feed monensin, it is better to

investigate and correct other causes of the milk fat depression rather than to reflexively remove the monensin from the diet.

Ruminal acidosis is the third major cause for milk fat depression. Ruminal acidosis does not depress milk fat percentage by reducing the proportion of propionate absorbed from the rumen, as was previously thought. Rather, it apparently causes milk fat depression by inhibiting bacteria responsible for fatty acid biohydrogenation in the rumen. Thus, more *trans* fatty acids are absorbed, even if the intake of unsaturated fatty acids was not necessarily high.

Clinical evidence suggests that the link between ruminal pH and milk fat depression is weak. Many herds with substantially depressed ruminal pH have no milk fat depression at all (Figure 6). This suggests that low ruminal pH probably has to interact with some aspect of dietary fat feeding or time before milk fat depression occurs.

Experimentally induced SARA for only day does not apparently cause milk fat depression, even when the ruminal pH depression is severe (Figure 7). This suggests that microbial responses to ruminal acidosis may be slow, and/or that multiple acidotic insults are necessary before ruminal biohydrogenation is inhibited enough to cause milk fat depression.

Milk fat test data has the potential to give us some information about whether we are over-feeding grain (and causing ruminal acidosis with subsequent health problems) or are under-feeding grain and losing profit. Unfortunately, the inference on ruminal pH provided by milk fat test is too often inaccurate and influenced more by other factors. A direct measurement of ruminal pH is vastly more useful.

Once a diagnosis of subacute ruminal acidosis (SARA) has been established in a herd, the cause of the acidosis must be determined before appropriate preventive measures can be instituted. Causes of ruminal acidosis can be grouped into three categories: excessive intake of rapidly fermentable carbohydrates, inadequate ruminal buffering, and inadequate ruminal adaptation to a highly fermentable diet.

Excessive Intake of Rapidly Fermentable Carbohydrates

This is the most obvious cause of ruminal acidosis in dairy cattle. Because of their relatively high dry matter intakes, dairy cattle cannot tolerate diets as proportionately high in concentrates as beef feedlot diets. An important goal of effective dairy cow nutrition is to feed as much concentrate as possible, in order to maximize production, without causing ruminal acidosis. This is a difficult and challenging task because the indications of feeding excessive amounts of fermentable carbohydrates (decreased dry matter intake and milk production) are very similar to the results from feeding excessive fiber (again, decreased dry matter intake and milk production). An important distinction is that even slightly over-feeding fermentable carbohydrates causes chronic health problems, while slightly under-feeding fermentable carbohydrates does not compromise cow health.

Chemical Fiber Requirements

Dairy nutritionists have carefully defined fiber requirements for dairy cattle in terms of acid detergent fiber (ADF) and neutral detergent fiber (NDF) (National Research Council, 2001). Nutritionists often go beyond the measures of carbohydrate nutrition defined by the National Research Council to include nutrients such as non-fiber carbohydrates (NFC), starch, effective NDF (eNDF), physically effective NDF (peNDF), (Mertens, 1997) and long fiber particles (Oetzel, 2000). Each of these nutrients looks at a slightly different aspect of carbohydrate nutrition.

Evaluating the dietary content for each of these nutrients is an important first step in determining the cause of SARA in a dairy herd. This requires a careful evaluation of the ration actually being consumed by the cows. A cursory evaluation of the “paper” ration formulated by the herd nutritionist is usually of little value. Ascertaining the ration actually consumed by the cows requires a careful investigation of how feed is delivered to the cows, accurate weights of the feed delivered, and updated nutrient analyses of the feeds delivered (particularly the dry matter content of the fermented feed ingredients). Careful bunk sampling and wet chemistry analyses of total mixed rations (TMR) may uncover unknown errors in feed composition or feed delivery. The total intake of rapidly fermentable carbohydrates is probably more important than the percentage of the carbohydrates in the diet (Oetzel and Nordlund, 1998). Herds or groups within herds with higher dry matter intakes will be at inherently higher risk for SARA and may need to be more conservative in carbohydrate nutrition than other herds or groups.

Diet Physical Form

The physical form of feed ingredients can be just as important as their chemical composition in determining how rapidly and completely they are fermented in the rumen. Grains that are finely ground, steam-flaked, extruded, and/or very wet will ferment more rapidly and completely in the rumen than unprocessed or dry grains, even if their chemical composition is identical. Similarly, starch from wheat or barley is more rapidly and completely fermented in the rumen than starch from corn. Corn silage that is very wet, finely chopped, or kernel-processed also poses a greater risk for SARA than drier, coarsely chopped, or unprocessed corn silage.

Particle size analysis of grains is a useful adjunct test when assessing the risk for SARA in a dairy herd. Very finely ground grains, especially if they are moist, will increase their rate of fermentation in the rumen and increase the risk for SARA.

High Corn Silage Diets

Feeding a large proportion of a lactation diet as corn silage puts cows at higher risk for SARA compared to diets containing more dry hay or hay crop silages. Corn silages vary considerably in their NDF digestibility, due to genetic design (e.g., brown midrib varieties) or due to growing / harvest conditions. Tests that estimate NDF digestibility can be very useful in identifying corn silages with unusually high rates and extents of ruminal fermentation. Unfortunately, most of these tests interfere with precise evaluation because they first require grinding the corn silage sample.

Corn silages also vary considerably in the extent of processing of the corn grain (e.g., kernel processing). Tests are being developed to help measure the extent of grain processing within corn silage. Combining these test results with digestibility data and particle length data furthers our ability to feed corn silages heavily without unnecessary risk for SARA.

Corn silage is also difficult to feed because it typically does not contribute enough long particles to a TMR. Very long chopping of corn silage is not recommended, because it impairs fermentation and increases the risk for sorting at the feedbunk. It is a common (and necessary) practice to add chopped long-stem dry hay or chopped dry straw to TMR containing a high proportion of the forage as corn silage. However, can be difficult to process the dry forage so that it distributes evenly throughout the TMR and so that the cows cannot sort it. Vertical mixers or prior tub grinding of the dry forage usually works the best.

Feed Delivery and Access

Dairy cattle groups are commonly fed for *ad libitum* intake (typically a 5% daily feed refusal) in order to maximize potential dry matter intake and milk yield. However, slightly limiting intake in dairy cattle at high risk for SARA would in theory reduce their risk of periodic over-consumption and SARA. Feed efficiency might also be improved. This approach has been successfully used in beef feedlots. However, dairy cow groups are much more dynamic than feedlot groups. This makes it considerably more challenging for dairy cattle feeders to slightly limit intakes without letting the feed bunks be without palatable feed more than about four hours a day. It can be done, but only with adequate bunk space and excellent feed bunk management. Perhaps *ad libitum* feeding with a 5% daily feed refusal is the best option for most dairy herds. This would especially apply to the pre- and post-fresh cow groups because they have rapid cow turnover and because individual cows have rapidly changing dry matter intakes during these time periods.

Meal Size

Our recent research studies with SARA suggest that meal size is an extremely important aspect of nutritional management of SARA. Cows are able to self-regulate their ruminal pH very effectively if they have continuous and predictable access to the same TMR every day. However, even modest bouts of feed restriction can cause cows to subsequently consume meals that are too large. Cows should always have 30 inches of bunk space per cow; this allows them all to eat at the same time. Good feedbunk management practices are critical SARA prevention - even when chemical fiber, particle length, and grain processing are optimal.

Special Considerations for First Lactation Heifers

Because first lactation heifers have lower dry matter intakes than older cows, it would seem that they should be a lower risk for SARA. However, clinical data from our herd investigations shows that first lactation heifers may actually be a higher risk (see Figure 8/). First lactation heifers sampled had a higher prevalence of SARA (28% vs. 17% in second or greater lactation cows) and also appeared to be at risk for SARA earlier in lactation than the older cows. These are observational data only, and should be interpreted with caution until they are validated (or

refuted) by results of controlled studies. It is possible that first lactation heifers have difficulty getting access to feedbunks when older cows are present, so they could be prone to eating large meals. It is also possible that first lactation heifers need time to learn to self-regulate their own ruminal pH after they begin consuming large amounts of fermentable carbohydrates after calving. Since first lactation heifers represent a largely uncullered population, it is possible that the heifers that are unable to learn to self-regulate their own ruminal pH have not yet been removed from the herd. However, they will be removed by the second or third lactation due to complications of SARA.

Inadequate Ruminal Buffering

Ruminant animals have a highly developed system for buffering the organic acids produced by ruminal fermentation of carbohydrates. While the total effect of buffering on ruminal pH is relatively small, it can still account for the margin between health and disease in dairy cows fed large amounts of fermentable carbohydrates (Firkins, 1997). Ruminal buffering has two aspects – dietary and endogenous buffering.

Dietary buffering is the inherent buffering capacity of the diet and is largely explained by dietary cation-anion difference (DCAD). Diets high in Na and K relative to Cl and S have higher DCAD concentrations, tend to support higher ruminal pH, and increase dry matter intake and milk yield (Sanchez, Beede, and Delorenzo, 1994) (Block and Sanchez, 2000). Optimal DCAD for early lactation diets is about +400 mEq/kg of $(Na + K) - (Cl + S)$ (Block and Sanchez, 2000). Mid-lactation cows have an optimal DCAD of about +275 to +400 mEq/kg. Formulating diets with a high DCAD typically requires the addition of buffers such as sodium bicarbonate or potassium carbonate. Alfalfa forages tend to have a higher DCAD than corn silage, although this depends considerably on the mineral composition of the soil on which they were grown. Concentrate feeds typically have low or negative DCAD, which adds to their already high potential to cause ruminal acidosis because of their high fermentable carbohydrate content.

Endogenous buffers are produced by the cow and secreted into the rumen via the saliva. The amount of physical fiber in the diet determines the extent of buffer production by the salivary glands. Coarse, fibrous feeds contain more effective fiber and stimulate more saliva production during eating than do finely ground feeds or fresh pasture (Bailey, 1961). Coarse, fibrous feeds also make up the mat layer of the rumen, which is the stimulus for rumination. Fiber particles need to be longer than about 1.5 inches in order to contribute to mat layer formation. Rumination promotes much chewing activity and therefore the secretion of large amounts of saliva into the rumen. As expected, ruminal pH increases noticeably during bouts of rumination (Allen, 1997).

The ability of a diet and feeding system to promote maximal amounts of ruminal buffering should be evaluated as part of the work-up of a herd diagnosed with SARA. Wet chemistry analysis of a carefully collected TMR bunk sample can be particularly effective in determining the actual DCAD of the diet delivered to the lactation cows. Diets with measured DCAD values below about +275 to +400 mEq/kg of $(Na + K) - (Cl + S)$ should be supplemented with additional buffers to provide more Na or K relative to Cl and S.

Endogenous buffering can be estimated by observing the number of cows ruminating (a goal is at least 40% of cows ruminating at any given time) and by measuring the particle length of the TMR actually consumed by the cows using the Penn State Forage Particle Separator (Lammers, Buckmaster, and Heinrichs, 1996; Oetzel, 2000).

Diets with less than 7% long particles put cows at increased risk for SARA, particularly if these diets are also borderline or low in chemical fiber content (Grant, Colenbrander, and Mertens, 1990) (Woodford and Murphy, 1988). Increasing chemical fiber content of the diet may compensate for short particle length (Beauchemin and others, 1994).

Diets with excessive (over about 15%) long forage particles can paradoxically increase the risk for SARA. This happens when the long particles are unpalatable and sortable. Sorting of the long particles occurs soon after feed delivery, causing the cows to consume a diet that is low in physically effective fiber after feeding. The diet consumed later in the feeding period is then excessively high in physically effective fiber and low in energy. Socially dominant cows are particularly susceptible to SARA in this scenario, since they are likely to consume more of the fine TMR particles soon after feed delivery. Cows lower on the peck order then consume a very low energy diet. Thus, cows on both ends of the social spectrum become thin and produce poorly. Limiting bunk space to less than 30 inches per cow exacerbates the effect of TMR sorting in a group of cows. Sorting of long particles during the feed-out period can be evaluated by conducting sequential analysis of the TMR bunk samples at differing times after feeding.

It is very difficult to quantitatively evaluate the extent that a TMR is sorted. The most rigorous approach is to gather representative samples of the TMR at approximately 2 hours after feeding and then do particle length analysis at each time point. Gathering representative TMR bunk samples is tedious (gather 12 or more representative along the length of the bunk, mix, and then shake down two six-cup subsamples), and repeating this procedure six to ten times over the course of a day is not very practical. A more reasonable approach is to first evaluate the particle length, coarseness of the long forage particles, and dry matter of the TMR. If the proportion of long particles is <15%, if the long particles are not coarse stemmy hay, and the TMR dry matter is below 50%, then it is probably unnecessary to do any further evaluation of TMR sorting. If there are problems in one or more of these areas, then it is practical to start by comparing particle lengths of TMR refusals to the particle lengths of the TMR offered. If the refusals contain no more than about 5 to 10% more total long particles than the TMR offered, then sorting is unlikely to be a major issue. For example, if the TMR offered contains 18% long particles and the TMR refusal is 24% long particles, then sorting is probably not a major issue. But if the TMR refusal contains >28% long particles, then this is cause for concern.

The most common cause of excessive TMR sorting is the inclusion of unprocessed, coarse, dry baled hay in a TMR. Despite the claims of manufacturers, most TMR mixers (except for some vertical mixers) are unable to adequately reduce the particle size of coarse dry hay. Processing this hay before adding it to the mixer is often necessary. In many cases, the dry hay can be eliminated from the TMR, provided there are adequate long particles from haylage and corn silage. It seems paradoxical, but the risk for SARA in a herd can sometimes be lowered by removing the baled hay from the TMR.

Inadequate Adaptation to Highly Fermentable, High Carbohydrate Diets

In theory, cows in early lactation should be particularly susceptible to SARA if they are poorly prepared for the lactation diet they will receive. Ruminal adaptation to diets high in fermentable carbohydrates apparently has two key aspects – microbial adaptation (particularly the lactate-utilizing bacteria, which grow more slowly than the lactate-producing bacteria) and ruminal papillae length (longer papillae promote greater VFA absorption and thus lower ruminal pH) (Dirksen, Liebich, and Mayer, 1985). Beef feedlots recognize the importance of gradually introducing steers to higher grain diets (Radostits, Blood, and Gay, 1994).

The known principles of ruminal adaptation suggest that increasing grain feeding toward the end of the dry period should decrease the risk for SARA in early lactation cows. However, a recent field study in TMR-fed herds found no effect of dry period feeding on early lactation ruminal pH. And ruminal pH in this study was unexpectedly lower in cows at 106 average days in milk compared to cows at 15 average days in milk (Garrett and others, 1997). These results suggest that high dry matter intake is a more important risk factor for SARA than ruminal adaptation problems in dairy herds. Also, a controlled study in component-fed cows found no positive effect of increased grain feeding during the dry period on early lactation ruminal pH or dry matter intake (Andersen, Sehested, and Ingvarsten, 1999). These results suggest that the practical impacts of ruminal adaptation may be small or even inconsequential in dairy herds - particularly when cows are fed a TMR after calving.

Prevention of Subacute Ruminal Acidosis in Dairy Herds

The basic principles of preventing SARA in dairy herds have been discussed above and include limiting the intake of rapidly fermentable carbohydrates, providing adequate ruminal buffering, and allowing for ruminal adaptation to high grain diets. However, I expect SARA to remain an important dairy cow problem even when these principles are understood and applied, because the line between optimal milk production and over-feeding grain is exceedingly fine. In many dairy herd situations, milk production can appear to be temporarily increased by over-feeding grain and causing SARA; however, the long-term health and economic consequences of this approach are devastating. Any additional nutritional interventions that might prevent SARA without limiting grain feeding are highly desirable. Several of these approaches are summarized below.

Enhancing Ruminal Lactate Utilizers

An important aspect of maintaining a stable rumen environment is maintaining a balance between lactate production and lactate utilization by bacteria that convert lactate to less dangerous VFA. Enhancing ruminal lactate utilizers reduces the risk for ruminal acidosis (particularly the acute form of ruminal acidosis). Supplementation with specific yeast strains may enhance lactate utilization within the rumen under certain dietary conditions (Dawson, 1995).

Preconditioning Microbes to Handle Lactate

Adding lactate to the diet or using feed ingredients high in lactate may improve the ability of the rumen to adapt to sudden increases in lactate production (Owens and others, 1998). Direct-fed microbials might also be used to provide a steady source of lactate in the rumen. A mixture of direct-fed microbials added to the rumen of dairy cows at the 1×10^5 dose increased corn digestibility and increased ruminal pH compared to higher doses of microbials (Nocek and others, 1999).

Selenomonas ruminantium is one of the bacteria that convert ruminal lactate to VFA. *S. ruminantium* is apparently stimulated to utilize lactate by malate (Martin and Streeter, 1995). Supplementing diets with malate as a feed additive may be cost-prohibitive; however, incorporation of forage varieties high in malate may allow for economical inclusion of malate in dairy diets (Callaway and others, 2000). Stage of maturity and variety affects malate concentrations in alfalfa (Callaway and others, 2000).

Supplementation with Ionophores

Feeding ionophores reduces ruminal lactate production; this effect appears to be caused by inhibition of lactate-producing bacteria, competitive enhancement of lactate utilizers, and possibly by reducing meal size (Owens and others, 1998). It appears that the benefits of monensin in preventing SARA are minor. It is probably more effective in preventing acute ruminal acidosis, which is consistently characterized by very high ruminal lactate concentrations.

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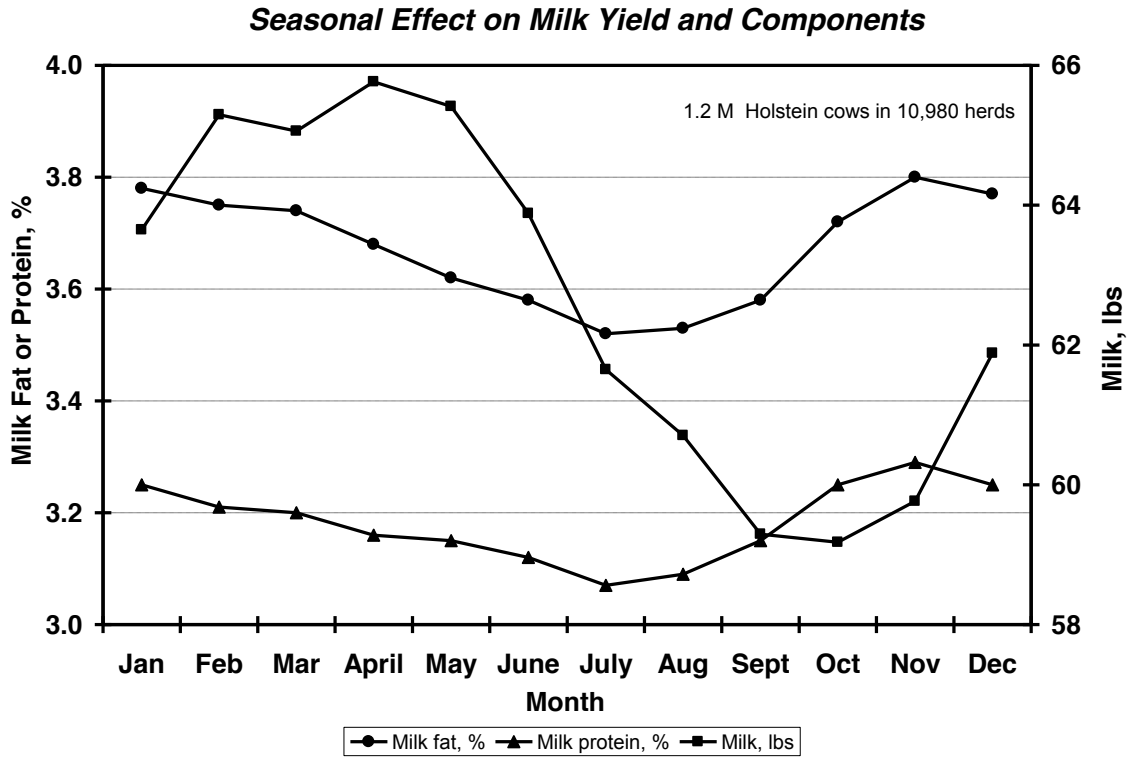


Figure 1. Milk yield, milk fat percentage, and milk protein percentage by month for Holstein cows. Data adapted from Hutjens, Hoards Dairyman 144(12):494, 1999.

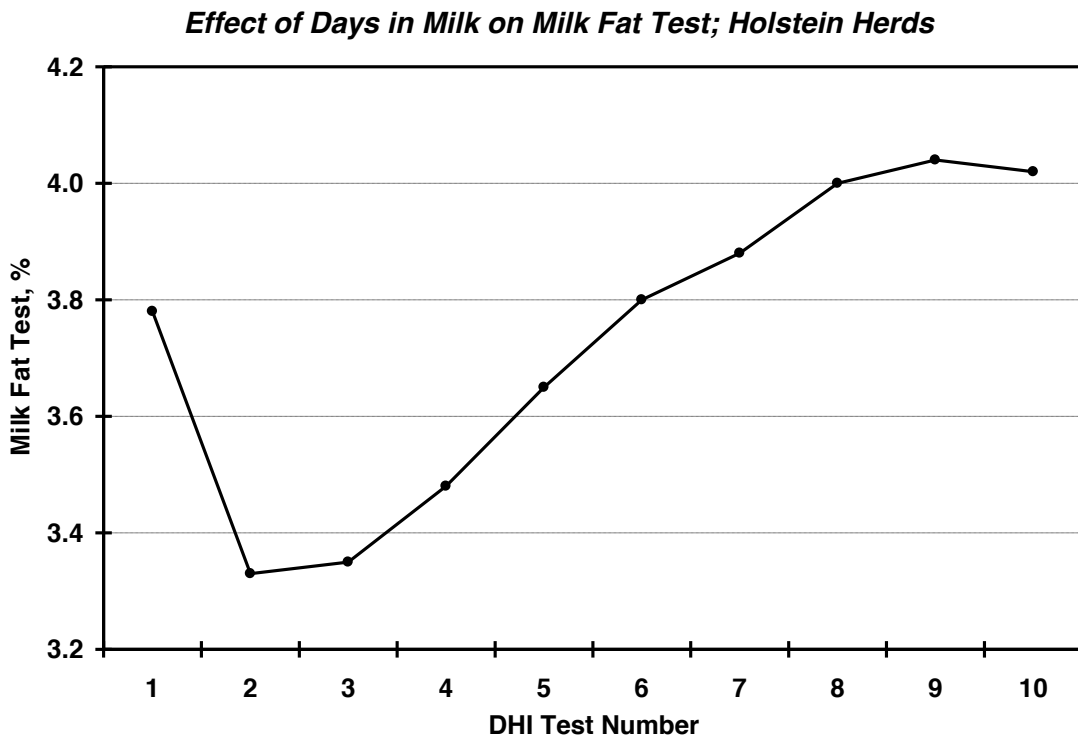


Figure 2. Average milk fat percentage by DHI test number.

Fat Digestion in the Rumen: Plant Source Fats

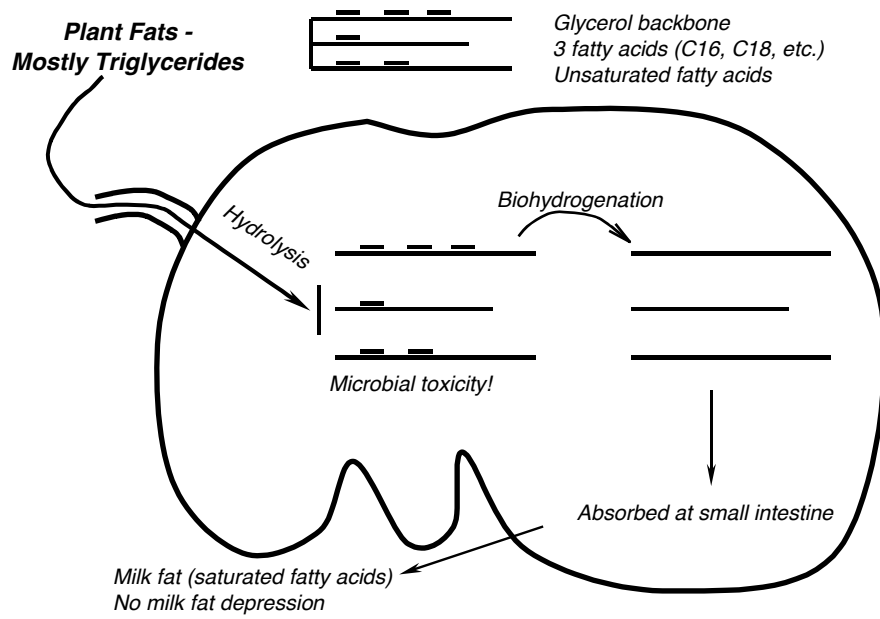


Figure 3. Normal digestion of plant source fats in the rumen.

Fat Digestion in the Rumen: Plant Source Fats

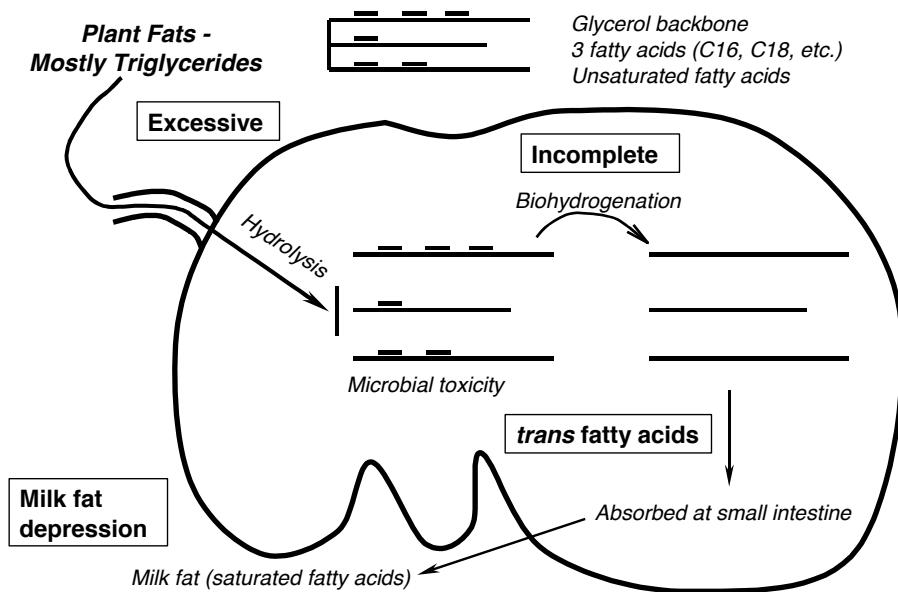


Figure 4. Milk fat depression following intake of excessive amounts of unsaturated plant fats.

Fat Digestion in the Rumen: Animal Source Fats

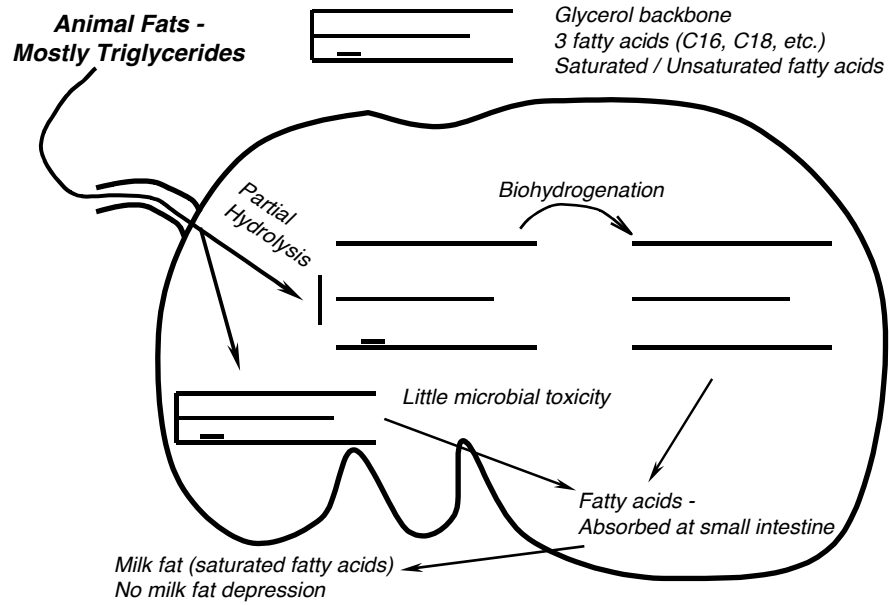


Figure 5. Digestion of animal fats in the rumen.

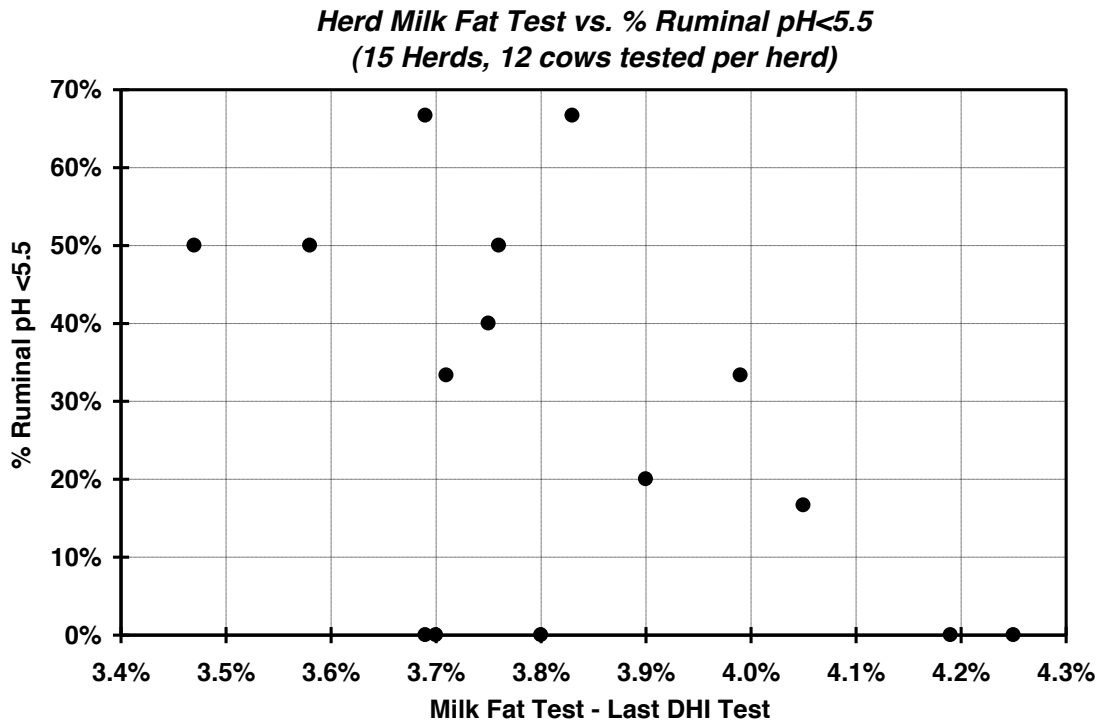


Figure 6. Milk fat test and percent of cows with low ruminal pH in 15 dairy herds.

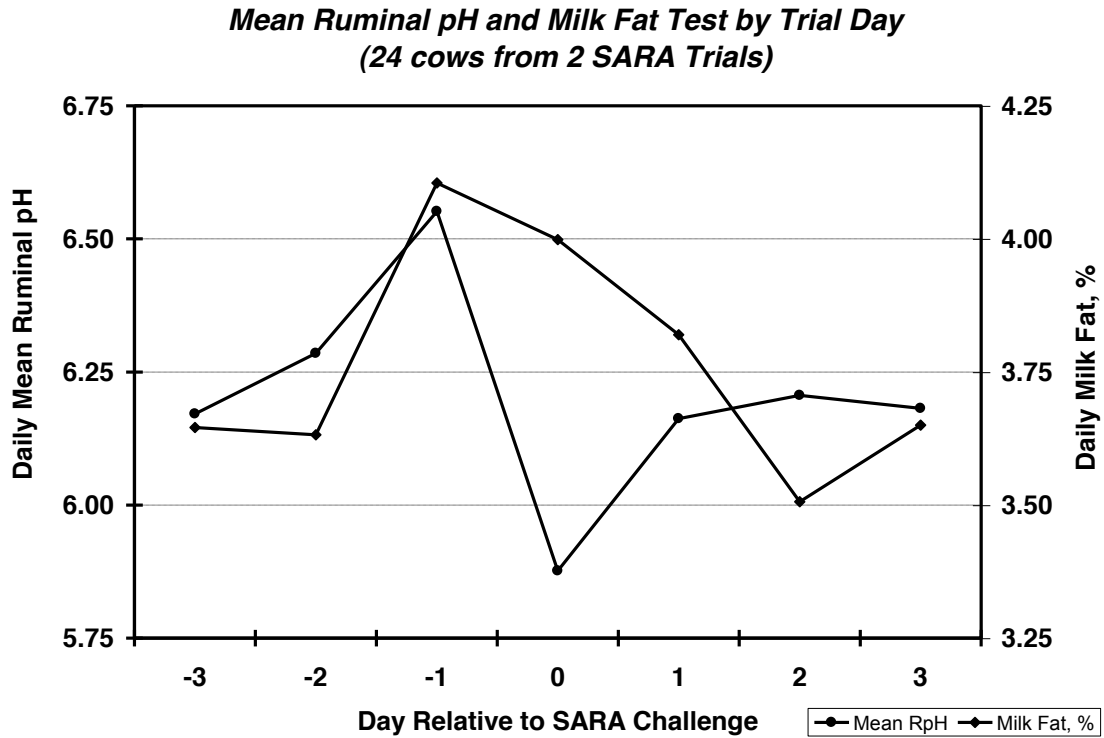


Figure 7. Daily mean ruminal pH and milk fat percentage in a short-term SARA challenge model.

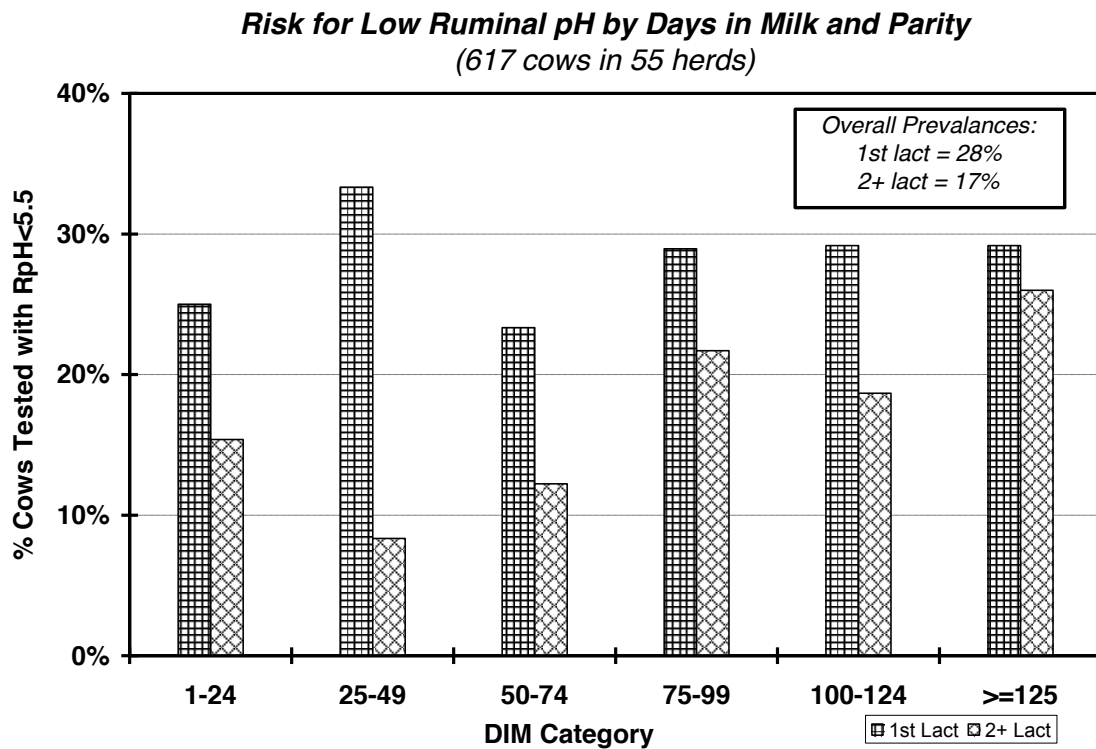


Figure 8. Incidence of low ruminal pH by days in milk category and by parity.