

# **2008 Southeast Dairy Herd Management Conference**



**November 12 & 13, 2008**

**Georgia Farm Bureau Building  
Macon, Georgia**

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# Southeast Dairy Herd Management Conference

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**Scott Angle, Dean and Director**

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# Southeast Dairy Herd Management Conference

## PROGRAM

Wednesday, November 12, 2008

### PCDART Workshop

9:30-Noon (Georgia Farm Bureau Building)

### First Session

- 11:00        **Conference Registration**  
Moderator- Dr. John Bernard
- 1:00        **Welcome** – Dr. Keith Bertrand Head, Animal & Dairy Science and Mr. Zippy DuVal, President GA Farm Bureau
- 1:15        **SE DHIA Update** - Dr. Dan Webb
- 1:45        **Dairy Cattle Contribution to Beef Industry**- Jim Collins
- 2:15        **The Application of Feed Efficiency on Dairy Farm**- Dr. David Casper
- 3:00        **Break**-  
–Sponsored by Zinpro Performance Minerals
- 3:30        **Economics of Post Partum Uterine Health**- Dr. Mike Overton
- 4:00        **The Georgia Mastitis Situation Cell Counts and Microbiology**  
- Dr. Warren Gilson.
- 4:30        **Assessing Milk Quality** - Dr. Stephen Oliver
- 5:30        **Reception**

Thursday, November 13, 2008

**Second Session**

- 8:00           **Conference Registration**  
Moderator- Dr. Warren Gilson
- 9:00           **Welcome**  
Dr. Keith Bertrand Head, Animal & Dairy Science, University of Georgia  
and  
Mr. Zippy DuVal, President of GA Farm Bureau
- 9:15           **CAES Intern Programs**  
Dr. Jean Bertrand  
University of Georgia
- 9:45           **Best Management Practices to Enhance Milk Quality**  
- Dr. Stephen Oliver
- 10:15          **The Influences of the Commodity Markets on the Costs of Forages  
and Feed** - Dr. David Casper
- 10:45          **Break-** Sponsored by Crystal Farms
- 11:15          **By-Product Feeding for Milk Production** – Dr. John Bernard
- 11:45          **Mycotoxins in Dairy Diets: Effects and Prevention** – Dr. Lon Whitlow
- 12:15          **Lunch** – Sponsored by Dairy Farmers of America  
Moderator- Dr. William Graves
- 1:15           **The Importance of Nutrient Management Plan Recording Keeping**  
- Melony Wilson
- 1:45           **Synchronization Programs Continue to Change** – Dr. William Graves
- 2:15           **Questions and Discussion**
- 2:30           **Adjourn**

## **Program Participants**

*Dr. John Bernard  
University of Georgia*

*Dr. Jean Bertrand  
University of Georgia*

*Dr. Keith Bertrand  
Head, Animal & Dairy Science  
University of Georgia*

*Dr. David Casper  
Agri-King, Inc.*

*Mr. Jim Collins  
Georgia Cattleman's Association*

*Mr. Zippy Duvall  
President, Georgia Farm Bureau*

*Dr. Warren Gilson  
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## **Contributors and Sponsors**

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**Dairy Farmers of America**

**Fort Dodge Animal Health**

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**West Central**

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*The above organizations provided support for the conference through financial contribution or by sponsoring a specific event. Express your appreciation to the representatives of these organizations.*

## **Southeast DHIA Update – Production and Management Trends**

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Data from DHIA herds in Alabama, Florida, Georgia, Mississippi, South Carolina and Tennessee were used to examine dairy production in the Southeastern United States. Herds with data in the DRMS database as of mid-October, 2008 included: 367 Holstein herds, 53 Jersey herds and 58 herds of other breeds. In addition, the all DRMS average from 15,466 herds located in 43 states was used for reference.

Milk production for all 480 Southeast herds averaged 18,016 pounds (rolling herd average) which was 477 pounds per cow below last year. The 2X-305-day mature equivalent average was 20,575 pounds. Average 150-day milk was 63 pounds. Average peak milk was 69 pounds for first lactations and 91 pounds for older cows.

Herd size of Southeast herds averaged 282 cows per herd, up 2 from last year with 37% milking in lactation 1. All DRMS herds averaged 144 cows, also with 37% first lactations. Herd turn-over rate was 35 and 34%, respectively. Death loss averaged 8% for Southeast herds and 6% for DRMS herds. Southeast herds averaged 274 calvings and had 77 calves per 100 cows on hand. Sixty-two percent of services were to proven AI sires. Southeast herds averaged 78% heifers with known sire identity, where the average DRMS herd was 86%. Average sire identity for adult cows was 55% for Southeast herds and 72% for DRMS herds. Average reported milk price was \$22.00.

Current month pregnancy rate (September), averaged 13% for Southeast herds and 11% for DRMS herds. Days to 1<sup>st</sup> service was 109 and first-service conception rate, 49%. Fifteen percent of cows were dry less than 40 days and 32% longer than 70 days.

Average somatic cell count was 426,000 compared to last year's 447,000. Forty-seven percent of cows had somatic cell score below 4.0.

In comparing performance among breeds, Jerseys had lower death loss, reduced herd exits for reproduction and notably higher pregnancy rates.

Differences among Southeastern states were few, but Florida herds were considerably larger and Tennessee herds smaller than the average.

Southeast herds had 7.3% of 1<sup>st</sup>-lactation cows with birth difficulty scores 4 or higher. DRMS herds had 5.8% of 1<sup>st</sup>-lactation cows above 4.

Table 1. Breed comparisons for Southeast States as of October, 2008

	<b>DRMS</b>	<b>Southeast</b>	<b>Southeast</b>	<b>Southeast</b>
	Holstein	Holstein	Jersey	Other Breeds
No. Herds	13304	367	53	58
No. Cows / Herd	149	313	150	210
No. 1st Lact	55	115	52	81
% 1st Lactation	37%	37%	35%	38%
Avg Days in Milk	193	211	181	204
% Left Herd	34	35	33	37
%died	6.1	8.3	6.2	7.8
%left Repro	6.0	6.5	4.0	6.5
Milk Price	19.10	21.90	22.50	22.30
Rolling HA Milk	20,974	18,797	14,202	16,338
Rolling HA Fat	784	688	651	625
Rolling HA Prot	643	575	499	518
Summit Milk 1st Lac	69	66	48	57
Summit Milk 3rd+	92	87	64	75
Peak Milk 1st Lac	76	73	53	62
Peak Milk 3rd+	101	96	70	82
Proj 305ME Milk	23,022	21,488	16,157	18,810
Std 150-day Milk	71	66	49	58
SCC Actual	327	434	383	420
SCC Score	3.1	3.5	3.4	3.7
SCC Score 1st Lact	2.6	3.1	3.1	3.2
SCC Score 2nd Lact	2.9	3.4	3.2	3.5
SCC Score 3rd Lact	3.5	4.0	3.9	4.2
% SCC Score <4	61	47	52	49
PregRate Current mo	14.1	10.6	16.5	13.9
Actual Calving Int	14.2	14.7	14.2	14.6
Days to 1st Serv	98	111	93	110
1st Serv Concep Rate	43	50	42	48
# Calvings	150	303	148	205
# calves per 100 cows	84	75	89	76
%Dry < 40 days	16	16	11	15
%Dry > 70 days	24	32	28	36
%Bred to Proven bulls	64	62	65	56
%Bred to non-AI	22	42	18	44
%Heifers with Sire ID	86	76	89	80
%Cows with Sire ID	71	51	87	56
% Births Difficulty > 4 for 1 <sup>st</sup> lactations	5.8	7.3	3.5	3.7
* Southeast - includes 6 southeastern states				
** DRMS - includes all herds processed by DRMS				

Table 2. Comparison by State 2008.

	Florida	Georgia	S. Car	Tenn	AI-Ms
No. Herds	54	132	31	116	35
Number of Cows/herd-All Lact	859	284	232	148	194
Number of Cows-1st Lact	312	105	96	54	62
Days in Milk	201	214	215	207	213
Cows Left Herd-All Lact, %	35.1	35.1	36.7	34.8	34.8
Cows Left Herd-1st Lact, %	14.8	16.4	15.9	19.2	20
Cows Died-All Lact, %	10.4	8	6.9	7.8	8.4
Cows Left Herd for Repro-All Lact, %	5.5	8.1	7	4.8	7.4
Rolling Milk	18205	18428	21058	18877	18952
Rolling Fat	629	661	778	715	657
Rolling Protein	533	566	658	576	573
Summit Milk 1st Lact	65.5	65.3	71.8	65.9	63
Summit Milk 3rd+ Lact	84.3	86.3	95.4	86	83.7
Peak Milk 1st Lact	74.3	71.4	79.5	71.8	70.1
Peak Milk 3rd+ Lact	96.1	94.3	106	94.9	93.3
Proj 305 Day ME Milk	20538	21252	23804	21787	20745
Standardized 150 Day Milk	62	65	73	68	64
SCC Actual	485	461	372	401	486
SCC Score	3.7	3.6	3.5	3.3	3.8
SCC Score for 1st Lact Cows	3.5	3.2	3.3	2.8	3.3
SCC Score for 2nd Lact Cows	3.6	3.5	3.5	3.1	3.6
SCC Score for 3rd+ Lact Cows	4.1	4.2	3.9	3.8	4.4
Cows (SCC of 0-3), %	48.8	49.7	52.1	53.7	43.9
Preg Rate-Current	8.3	10	11.1	11.2	12.1
Actual Calving Interval	14.4	14.8	14.5	14.8	14.9
Births 4+ Calving Diff-1st Lact, %	8.3	10	9.7	3.7	3.8
Days to 1st Serv-(%herd < VWP)	17.4	13.6	16.2	18.1	18.3
Days to 1st Serv-Total Herd	107.8	114.5	100	111.7	112.3
Con Rate for Past 12M-1st Serv	54.4	50.4	49.1	49.4	43.7
Calvings in Past Year	849	271	234	141	188
Dry Less Than 40 Days, %	16.4	16.5	12.2	15.9	14.6
Dry More Than 70 Days, %	34.2	31.7	26.2	33.1	30.8
%ile Rank of Proven AI Bulls	36.8	47.6	52.8	40.8	38.7
Herd Bred to Proven AI Bulls, %	62.5	64.3	55.2	62.2	63.9
Net Merit \$ for 1st Lact Cows	78.3	119.7	137.1	105.8	58.7
Net Merit \$ for All Cows	39.6	87.4	112.4	67	60.9
Net Merit \$ for Heifer	121.3	136.9	139.9	91.7	110.9
Heifers ID'd by Sire, %	66.7	75.9	86.3	78.8	74.7
Cows IDd by Sire, %	26.1	46.4	63.7	60.6	62.3
No.calves / 100 cows	53.9	66.7	92.4	87.6	90.1

Data from DRMS – Oct. 2008.Holstein Herds

Table 3. Comparison of Herds in Southeast to All DRMS Herds 2008.

<b>All Breeds</b>	<b>2008 Southeast *</b>	<b>2008 DRMS **</b>	<b>2007 Southeast *</b>	<b>2007 DRMS **</b>
No. Herds	480	15,466	498	15,574
No. Cows / Herd	282	144	280	139
No. 1st Lact	104	53	103	52
% 1st Lactation	37%	37%	37%	37%
Avg Days in Milk	205	191	209	193
% Left Herd	35	34	35	34
%died	8	6	8.5	9.5
%left Repro	6	5	6	9.5
Milk Price	22.00	19.20	23.10	21.50
Rolling HA Milk	18,016	20,308	18,493	20,309
Rolling HA Fat	676	768	687	764
Rolling HA Prot	559	629	573	626
Summit Milk 1st Lac	63	67	63	67
Summit Milk 3rd+	83	89	83	89
Peak Milk 1st Lac	69	73	70	73
Peak Milk 3rd+	91	98	92	97
Proj 305ME Milk	20,575	22,274	20,690	22,280
Std 150-day Milk	63	69	62	69
SCC Actual	426	328	447	335
SCC Score	3.5	3.1	3.6	3.1
SCC Score 1st Lact	3.1	2.7	3.2	2.7
SCC Score 2nd Lact	3.4	2.9	3.2	2.9
SCC Score 3rd Lact	4.0	3.6	4.1	3.6
% SCC Score <4	47	58	48	58
PregRate Current	11	14.4	13	14.8
Actual Calving Int	14.6	14.1	14.6	14.2
Days to 1st Serv	109	98	105	98
1st Serv Concep Rate	49	43	48	44
# Calvings	274	145	271	140
# calves per 100 cows	77	84	77	83
%Dry < 40 days	15	15	15	15
%Dry > 70 days	32	25	31	25
%Bred to Proven bulls	62	63	63	63
%Bred to non-AI	35	23	36	23
%Heifers with Sire ID	78	86	79	86
%Cows with Sire ID	55	72	56	71
* Southeast - includes 6 southeastern states				
** DRMS - includes all herds processed by DRMS				

## The Application of Feed Efficiency on the Dairy Farm

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### Introduction

Feed Efficiency (**FE**) or Dairy Efficiency (**DE**) has been a popular topic of observation and discussion on dairy farms the past few years. Many articles, both scientific and popular press, along with several conference proceedings (Atwell 2006a, 2006b, Britt et al., 2003, Britt and Hall, 2004, Casper et al. 2003, 2004, Hinders, 2005, Hutjens 2005, 2006, 2007, Linn et al. 2004) have been written on what **FE** is, how to measure and calculate **FE**, and factors affecting **FE** on the farm.

Monitoring feed efficiency is becoming a more common benchmark for monitoring the profitability of milk production relative to dry matter intake. In today's markets, feeds and commodities are becoming more costly, which is driving the requirement for more efficient utilization to maintain profitability. The goal of the dairy operation should be to maximize the efficiency of converting feed into milk, which adds the caveat of reducing manure production as well. How efficiently a dairy cow converts feed into milk can affect the dairy operation's bottom line, which during tough economic conditions, can be the difference between producing milk at a profit or a loss.

In this presentation to keep things simple, **FE** will be defined as an unit of milk produced per unit of dry matter (**DM**) consumed. This presentation will focus the discussion on those biological mechanisms that control the efficient utilization of feeds by the dairy cow, especially dry matter and fiber digestibility. The understanding of these fundamental mechanisms will enable management decisions on the dairy operation to be implemented that will further improve or enhance **FE**.

Other livestock industries, such as the poultry, swine and beef industries, have used **FE** as a benchmark for profitability. Many examples have been published demonstrating the economics of **FE** (Casper et al. 2003, Hutjens 2005, 2007). The interest in **FE** is due to it's relationship of reducing feed cost while increasing the profitability of milk production. Table 1 is a simple example of how improving **FE** can impact profitability. Both herds produced the same amount of milk, but the cows in Herd B consumed 7 lbs less dry matter than cows in Herd A. Assuming today that feed costs are \$0.10 per lb of DM (probably conservative), Herd B had a lower feed cost of \$0.70 per cow per day compared to Herd A to get the same amount of milk. This \$0.70 would be additional profit to the dairy operation. Thus, improving the **FE** will result in lower feed costs per unit of milk production while increasing profitability.

In addition, Figure 1 demonstrates the reductions in feed costs on a per cow per day basis as **FE** increases assuming constant milk production and a cost of \$0.10 per lb of **DM**. What is interesting about this graph is that the slope of this relationship is not linear but curvilinear. Thus, the biggest savings in feed costs can be realized by improving **FE** from 1.2 to 1.4, than improving **FE** from 1.6 to 1.8 (\$0.83 vs. \$0.63), respectively, however remember these savings would be additive if **FE** could be improved from 1.2 to 1.8. During periods of low milk prices, finding ways to improve the **FE** or maintaining a high **FE** can be the difference between producing milk at a profit or a loss.

The range in **FE** observed in the field or the scientific literature can be quite large. Table 2 contains a summary of 422 treatment means summarized from feeding studies conducted with Holstein dairy cows published in the scientific literature. Milk production across these treatment means averaged 72.9 lbs, but ranged from 41.0 to 103.0 lb/hd/d, while **DM** intake averaged 48.6 with a range of 30.0 lb/hd/d up to 67.9 lb/hd/d. The calculated **FE** observed in this data set averaged 1.51, but ranged from a low of 0.86 to a high of 2.30. Understanding why **FE** varies this dramatically across feeding studies will allow for management decisions to be made that can enhance **FE** in the future.

Agri-King has been monitoring **FE** for approximately 15 years because of our focus on improving the profitability of the dairy operations that we work with. Our first experience (Casper et al 2003) with increasing **FE** occurred when dairy herds were having high milk production on lower than expected **DM** intakes. Evaluating these dairy herds in depth indicated that the apparent reason(s) for these dairy cows achieving higher milk production on lower than expected **DM** intakes appeared to be related to the feeding of extremely highly digestible forages.

Many authors have published excellent reviews on factors influencing **FE**, such as days in milk, age, body weight, etc. (Atwell 2006a, Atwell 2006b, Linn et al. 2004, Hutjens 2005, Hutjens 2006, Hutjens 2007). However, our work (Casper et al. 2003, Casper 2004, Casper and Mertens 2007) has focused on identifying those basic fundamental factors that can be measured, manipulated, and managed to increase **FE**. This presentation will address what we believe to be some of the fundamental factor(s) influencing **FE** and that is the digestibility of nutrients from the feeds and forages fed to lactating dairy cows.

## Digestibility

The National Research Council (2001) demonstrates the greatest factor affecting energy availability to the lactating dairy cow is digestibility. In a small field study, Casper et al 2004 reported that nutrient digestibility had a direct effect on **FE**. Six dairy farms feeding a total mixed ration (TMR) were used to collect samples of TMR's and manure samples along with data on milk production, composition, and intake of **DM**. Nutrient composition of TMR's and manure samples were measured and nutrient digestibilities were calculated using acid insoluble acid (AIA) as an internal digestibility marker. Figure 2 shows that the **FE** responses of these dairy cows were directly related to the **DM** digestibility (**DMD**) of the ration ( $FE = 0.032 + 0.02 * DMD$ ,  $R^2 = .59$ ,  $P < .01$ ). In addition, Figure 3 demonstrates that as the **FE** increases the intake of **DM** was lower for these dairy cows. Indirectly, **FE** can be used as an indicator of ration digestibility, i.e. if **FE** is low than digestibility of the ration may be poor. Figure 3 demonstrates that dairy cows do not need to consume large amounts of **DM** in order to have high milk production. Supplying the required amounts of digestible nutrients in the ration is crucial to achieving high milk production. If that supply can be achieved by consuming less **DM** that is more digestible than milk production and **FE** should be improved.

Within this study, the range in digestibility of the forages explained most of the variation observed in digestibility of the ration by the lactating dairy cows. Thus, in most feeding situations, forages usually comprise the largest portion of the ration compared to other feed ingredients. Forages have much more variability in digestibility than grains or commodities. Therefore, forage quality and digestibility is going to have a major impact on **FE**. Tables 3, 4, and 5 demonstrate the ranges in forage quality and digestibility observed from samples submitted to our laboratory. As these tables demonstrate, the range in nutrient concentrations and the digestibility on a **DM** or NDF basis can be very large between samples within these forage categories. Submitting forage samples for measurement of digestibility of **DM** and NDF would be the first step towards improving **FE** on the dairy operation.

## Energy Metabolism Database

If **FE** is directly related to nutrient digestibility, then it follows that **FE** would be directly related to dietary energy density. One of the biggest databases in the world measuring the energy density of the ration is the Energy Metabolism Database from the Energy Metabolism Unit (**EMU**) of the United States Department of Agriculture – Agriculture Research Service (**USDA-ARS**). The **EMU** database, which was compiled by Casper and Mertens (2007), represents more than 40 years of studies measuring the energy and protein digestibility of dairy cattle fed diets that varied in forage types, grain sources, protein sources and fat supplements. Of the 3,018 individual energy and N digestion trials, only 1351 individual trials used lactating dairy cows of different breeds and stages of lactation.

The initial analysis of the **EMU** database indicated that ruminal acidosis may have occurred in many of the individual balance trials, which negatively affected nutrient digestibility. Thus, digestion trials conducted on lactating dairy cows having inverted fat and protein rations (acidosis criteria) were removed from the data analysis, which resulted in the final data set having 495 observations relating **FE** and nutrient digestion. These energy balance trials demonstrated that **FE** was directly related to the amount of absorbed **DM** consumed by the lactating dairy cow (Figure 4). ( $FE = .383 + .074 * DM \text{ absorbed g/d}$ ;  $R^2 = .44$ ,  $P < .01$ ). Therefore, lactating dairy cows having higher **FE** are those cows that are consuming rations containing more digestible **DM**.

Because dietary energy density is directly related to ration digestibility, it becomes apparent that **FE** is directly related to the net energy (**NE**) density of the diet (Figure 5;  $FE = -.01 + 1.25 * NE$ , Mcal/kg DM;  $R^2 = .60$ ,  $P < .01$ ). Since, absorbed **DM** is a function of both digestibility of the ration and intake of **DM** by the lactating dairy cow, it becomes apparent that improving **DM** digestibility has the potential to reduce the amount of **DM** needed to meet her nutrient requirements. Pushing dairy cows for maximum intake of **DM** may not always result in maximal or optimal milk production. Why push cows for high intakes of **DM** to get 80 pounds of milk when the same milk yield can be achieved with 50 pounds of **DM**? The extra feed cost is lost profit to the dairy operation.

## Acidosis

In the **EMU** database, feeding diets that resulted in lactating dairy cows having inverted fat and protein ratios (acidosis criteria) certainly had a negative effect on **FE**. Acidosis dramatically reduced the relationship of **FE** to absorbed **DM** ( $FE = 0.40 + 0.10 * DM \text{ absorbed, kg/d}$ ;  $R^2 = .28$ ,  $P < .01$ ). Acidosis, as expected, caused reductions in the digestibility of ADF and cellulose, which are the fiber fractions of the diet. Casper and Mertens also reported (2007) that acidosis increased the amount of heat produced per unit of digestible energy (51.4 vs. 54.6%), which resulted in a poorer conversion of digestible energy into net energy available for productive purposes. Acidosis negatively influences the energy metabolism of the lactating dairy cow along with affecting the health of the cow in a negative manner.

These data demonstrate that the biggest factor affecting energy availability to the lactating dairy cow is ration digestibility. This database analysis also demonstrates that by improving ration digestibility; the **FE** of the lactating dairy cow will increase as well. The corollary from an environmental standpoint is that improving ration digestibility will reduce manure output. In this data set, fecal energy output ranged from a low of 20% to more than 60% of gross energy intake. The data demonstrate that improving the nutrient digestibility of the diet to improve **FE** should result in more energetic efficient cows. Also, it stands to reason that using the best management practices of forage production to produce the highest quality forages or using feed additives that improve nutrient digestion, while preventing acidosis, have the greatest potential for improving **FE**.

## Silage Additives

Forages represent a major portion of the diet and the digestibility/quality of these forages will have a major impact on ration digestibility (Casper et al. 2004, Casper and Mertens, 2007). In this author's opinion, forage quality cannot be too good. Thus, producing or purchasing forages having the highest digestibility is going to result in the highest **FE** and the most economical milk production. The use of silage inoculants or silage fermentation aids during the ensiling process has increased in recent years to enhance the production of lactic acid along plus other benefits for the long term storage of forages.

The use of specific silage inoculants or silage fermentation aids (products) during the forage harvesting process that have been formulated with specific features and benefits have the potential to improve the digestibility of nutrients in ensiled forages. For example, we conducted a study (Ayangbile et al. 2000) evaluating the addition of a silage additive (Silo-King<sup>®</sup>, Agri-King, Inc., Fulton, IL) during the ensiling process at increasing rates to determine if the digestibility of alfalfa haylage could be enhanced. The additive was applied to alfalfa haylage at increasing applications rates (0.33, .67, and 1 lb/ton of alfalfa forage) at the time of ensiling. The ensiled alfalfa haylage was allowed to proceed through the ensiling process and was stored (> 60 days) before being fed to growing wethers. The experimental design was a replicated 4 x 4 latin square design using metabolism crates to measure the digestion and absorption of nutrients. Figures 6 and 7 demonstrated that application of the additive at increasing application rates resulted in increasing ( $P < .05$ ) the digestion and absorption of DM and NDF. Thus, improvements in **DM** and fiber (**NDF**) digestibility can be achieved by treating forages during the ensiling process. These improvements have the potential to improve the **FE** of lactating dairy cows through improvements in the digestibility of forages by the animal.

## Direct Fed Microbials (DFM) and Enzymes

This is an exciting area of research and product development being undertaken by several companies that holds great promise for improving **FE** by lactating dairy cows. Schingoethe et al. (2004) demonstrated that feeding enzymes resulted in an improvement in milk production. The stage of lactation and the cows' energy requirement will dictate the type of responses observed in **FE**.

For example, we have developed a product based on the combination of direct fed microbials (**DFM**) and enzyme technologies (Ru-Max<sup>®</sup>, Agri-King, Inc., Fulton, IL) that was evaluated using 1000 dairy cows split into 2 groups using a switchback trial design. Milk production (Figure 8) was similar ( $P > .10$ ) for both groups of cows, but the improvements in ration digestibility resulted in a 5.3 lb. decrease in intake of **DM**. Therefore, feeding the product resulted in an improvement in **FE** of 0.16 units (1.57 versus 1.73 for Control and Product, respectively). This resulted in a return on investment of 4.2 for every \$1 spent. These types of products hold promise in improving the **FE** of lactating dairy cows and the economics of producing milk

## Yeast and Yeast Cultures

Yeast and yeast cultures have been fed to dairy cattle for more than 60 years. Yeast culture has improved intake of **DM** and milk production in controlled studies (Miller-Webster et al. 2008, Schingoethe et al. 2004, White et al. 2008). Schingoethe et al. (2004) reported an increase in **FE** of 0.1 unit ( $P < .04$ ) when cows were fed yeast. This was the result of numerically greater ( $P > .10$ ) milk production and lower intake of **DM**. It is interesting to note that milk fat was numerically increased due to feeding yeast which would be hypothesized to occur from greater **DM** and fiber digestion. Miller-Webster et al. (2002) reported increases in **DM** digestibility of 2.4 and 5.0 percentage units when yeast products were evaluated using a continuous culture system. White et al. (2008) demonstrated a 3.2 percentage unit improvement in NDF digestibility by feeding cows yeast culture compared to cows receiving the same diet without yeast culture.

Using yeast as a feed additive has the potential to improve **FE** by approximately 0.1 units by improving rumen function and nutrient digestion.

It is the authors' field experience that reductions in intake of **DM** sometimes do not occur until cows are in a positive energy balance or gaining body weight. It is interesting to note that in the study by Schingoethe et al. (2004) that a numerical increase in body condition scores was observed with the reduction in DMI for cows fed the yeast containing ration.

## Conclusions

The greatest factor affecting nutrient availability to the lactating dairy cows is the digestibility of the ration. The **FE** potential of the dairy herd is directly related to the **DM** digestibility and energy density of the forages and feeds used in ration formulation. Producing or obtaining forages with the highest digestibility possible represents the greatest potential for improving **FE** and reducing the cost to produce 100 pounds of milk. Proper ration balancing to maximize fiber digestion and eliminating acidosis will improve **FE** and energetic efficiency of the dairy cow. The use of forage inoculants and feed additives (yeast cultures, live yeast, DFM, and enzymes) that improve ration digestibility can be used to further improve **FE**, however these improvements are not as dramatic as improving forage quality. Improving **FE** can increase the income over feed costs and reduce the cost to produce 100 pounds of milk. Tracking and improving **FE** on your dairy operation using those nutritional technologies that enhance digestibility and **FE** will improve profitability in good times and can be the difference between profit and loss in times of low milk prices.

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**Table 1.** Impact on feed costs in two herds with different feed efficiencies.

Measurement	Herd A	Herd B
Milk, lb/d	80	80
DMI, lb/d	57	50
Feed Efficiency	1.40	1.60
Milk Income @ \$16/cwt	\$ 12.80	\$ 12.80
Feed Costs @ \$0.10/lb DM	\$ 5.70	\$ 5.00
Income over feed costs	\$ 7.10	\$ 7.80
Cost to produce 100 lbs milk	\$ 7.13	\$ 6.25

**Table 2.** Milk production and composition, dry matter intake, and Feed Efficiency summarized from 422 treatment means published in the scientific literature.

Measurement	Average	Minimum	Maximum
Milk, lb/d	72.9	41.0	103.0
Fat, %	3.59	2.37	4.84
Protein, %	3.16	2.61	3.74
DMI, lb/d	48.6	30.0	67.9
Feed Efficiency, Milk/DMI	1.51	.86	2.30

**Table 3.** Nutrient concentrations, neutral detergent fiber digestibility (CWD), and digestibility of dry matter (DMD) of corn silage samples when ranked by DMD.

Item	CP	ADF	NDF	CWD	Lignin	Oil	NFC	Starch	DMD
Poor	8.0	30.8	51.1	46.8	3.29	1.94	21.1	22.2	55.5
Fair	8.5	29.3	50.1	50.1	3.06	2.29	36.4	22.9	67.8
Medium	8.4	24.5	42.9	52.0	2.44	2.70	43.8	30.4	72.7
Good	8.6	20.9	37.4	54.1	2.01	2.96	39.2	36.2	76.5
Excellent	9.0	16.5	30.7	55.2	1.58	3.25	55.8	43.9	80.9
Average	8.5	24.4	42.7	52.0	2.44	2.69	43.9	30.6	73.0

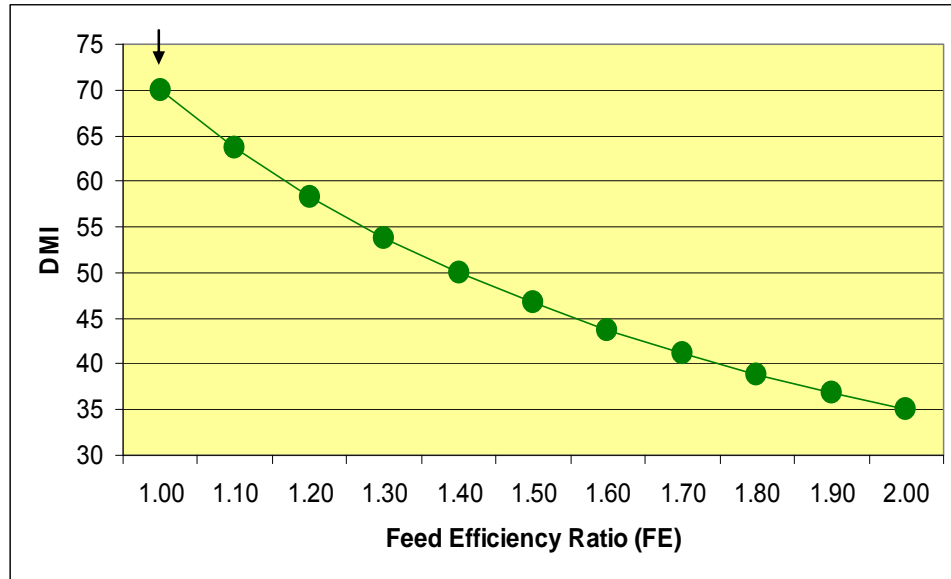
**Table 4.** Nutrient concentrations, neutral detergent fiber digestibility (CWD), and digestibility of dry matter (DMD) of ensiled haylage samples when ranked by DMD.

Item	CP	ADF	NDF	CWD	Lignin	NFC	DMD
Bad	12.3	47.5	66.0	46.0	12.2	17.5	43.2
Poor	13.9	42.7	61.6	52.3	8.5	19.6	56.6
Fair	18.3	36.1	50.9	57.6	6.9	23.8	66.4
Medium	21.1	31.4	43.7	60.0	5.9	27.2	72.4
Good	22.7	27.7	38.6	61.9	5.2	29.8	76.8
Excellent	24.3	23.8	33.3	65.2	4.4	32.8	81.5
Average	19.8	33.2	46.6	59.0	6.33	25.9	69.8

**Table 5.** Nutrient concentrations, neutral detergent fiber digestibility (CWD), and digestibility of dry matter (DMD) of hay samples when ranked by DMD.

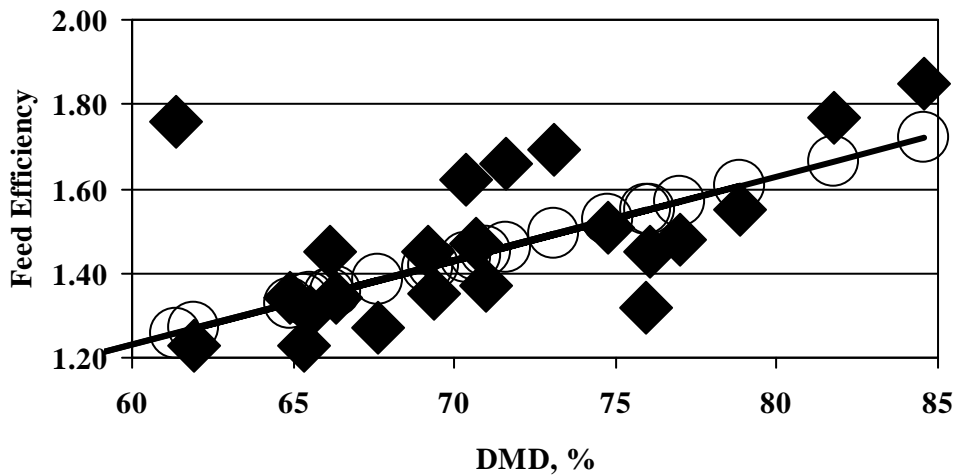
Item	CP	ADF	NDF	CWD	Lignin	NFC	DMD
Bad	8.71	45.9	71.7	41.6	7.2	15.9	45.9
Poor	12.2	40.6	63.8	48.8	6.3	19.9	55.7
Fair	18.1	34.9	51.7	54.4	6.5	24.8	65.7
Medium	21.3	29.8	42.3	57.1	6.0	29.5	72.4
Good	23.2	25.8	35.4	58.5	5.3	33.1	76.9
Excellent	24.9	21.8	29.2	62.1	4.6	36.2	81.4
Average	18.7	33.2	48.8	54.6	6.14	26.5	67.4

**Figure 1.** Change in feed costs as the feed efficiency ratio improves and dry matter intake (DMI) declines for producing 70 pounds of milk at a cost of 10 cents per pound of dry matter.



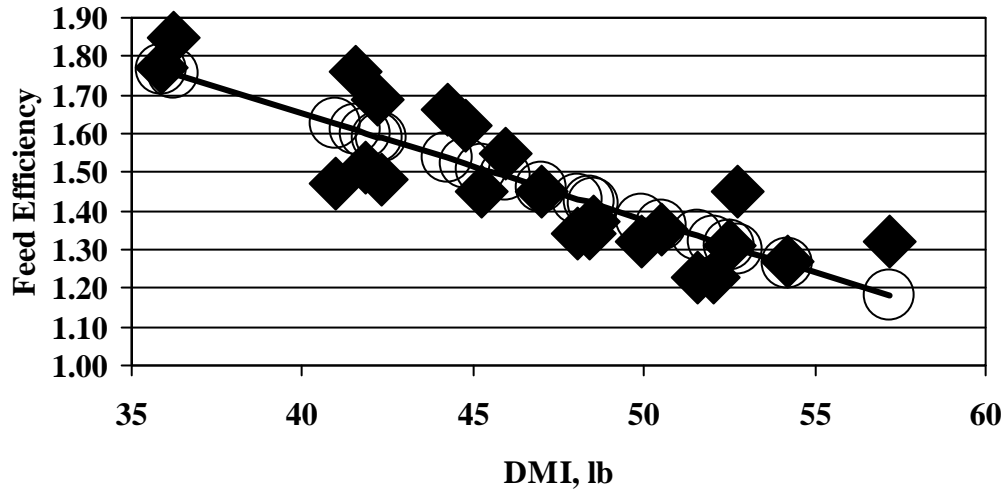
**Figure 2.** The relationship between Feed Efficiency and ration dry matter digestibility (DMD) by lactating dairy cows.

**Feed Efficiency=0.032+0.02\*DMD, R<sup>2</sup>=.59, P< .01.**

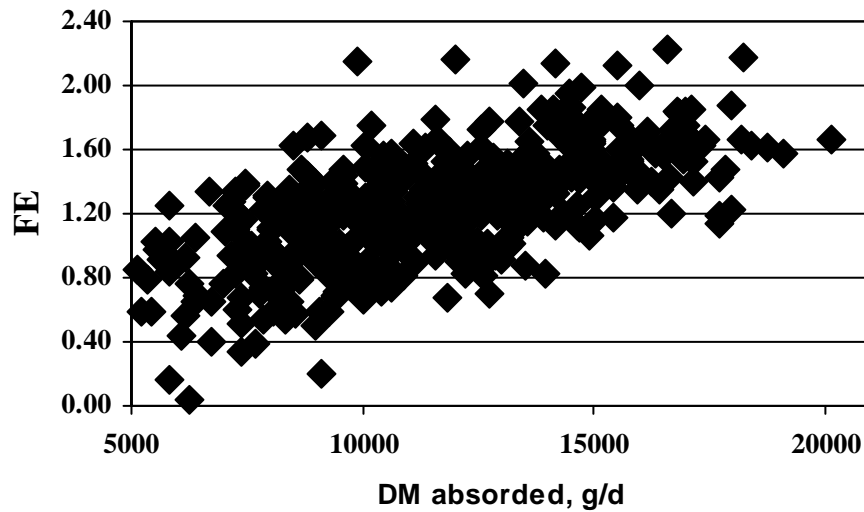


**Figure 3.** The relationship between Feed Efficiency and dry matter intake (DMI) by lactating dairy cows.

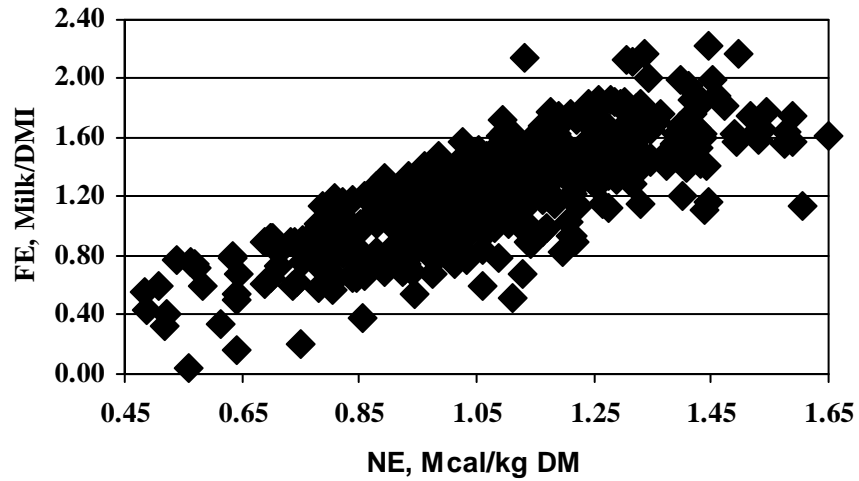
$\text{Feed Efficiency} = 2.76 - 0.028 * \text{DMI}, R^2 = .72, P < .01.$



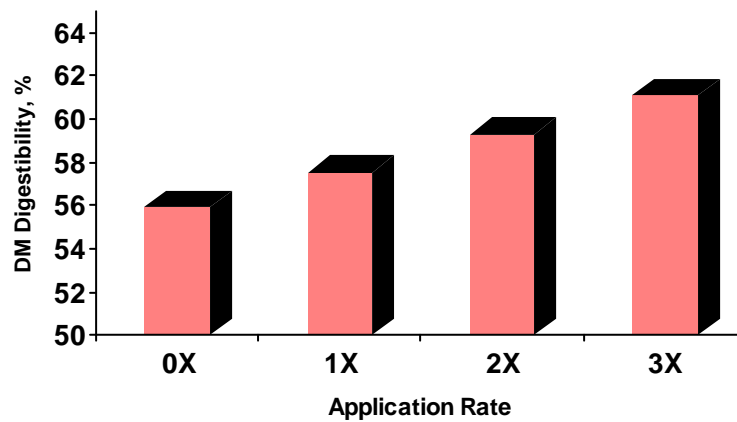
**Figure 4.** The relationship of Feed Efficiency (FE) to the amount of dry matter (DM) absorbed by lactating dairy cows.



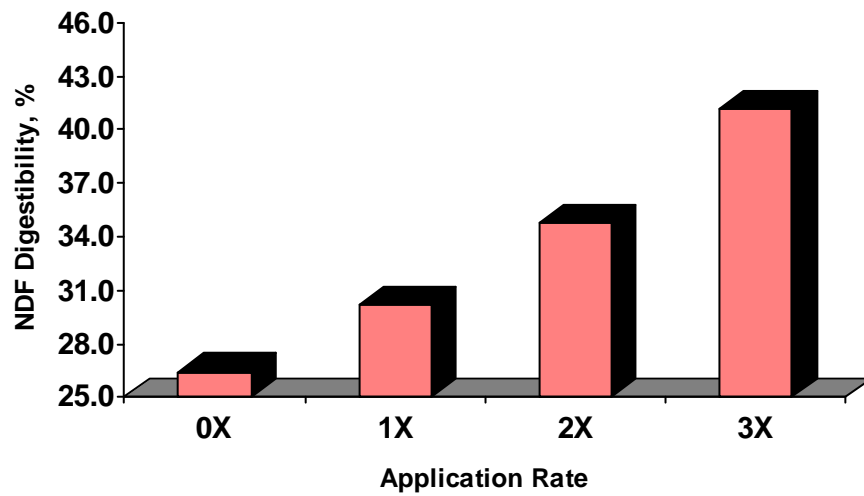
**Figure 5.** The relationship of feed efficiency (FE) to the net energy content (NE) of the diet fed to lactating dairy cows.



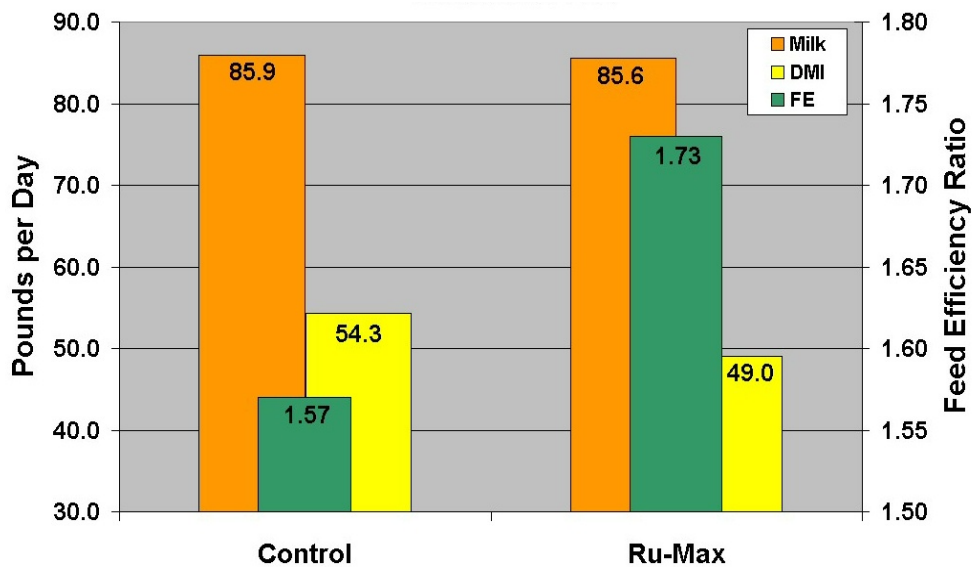
**Figure 6.** Effect of Silo-King<sup>®</sup> application rate on dry matter (DM) digestibility of alfalfa haylage by growing wethers.



**Figure 7.** Effect of Silo-King® application rate on neutral detergent fiber (NDF) digestibility of alfalfa haylage by growing wethers.



**Figure 8.** Milk production, dry matter intake (DMI) and feed efficiency ratio when lactating dairy cows are fed the same ration without (Control) or with Ru-Max.



## Economics of Postpartum Uterine Health

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### Introduction

The transition period has been identified as a critical time in a dairy cow's life due to the major physiological changes that are occurring then (Goff and Horst 1997, Drackley 1999). Special management attention should be devoted to improving the feeding, housing and care of animals during this periparturient period due to its impact on early lactation milk production, risk of postparturient disease and overall herd profitability. Postparturient diseases and metabolic issues such as hypocalcemia, ketosis, retained placenta, metritis and abomasal displacement are often directly linked to preparturient management. These and other diseases that occur during the early postparturient period are detrimental because they decrease milk production, increase treatment costs, and increase mortality and culling risk. Indirectly, these diseases affect profitability by increasing the risk of other disease problems. In addition, these problems negatively impact fertility both directly by damaging the reproductive tract and oocytes and indirectly by impacting energy balance and by interfering with the normal hypothalamic-pituitary-ovarian hormonal control system.

Metritis and endometritis, unfortunately, is a very common disease complex observed in postparturient cattle, with a median lactational incidence risk of approximately 10%, but with many herds in the 20 – 30% range (Kelton et al. 1998). Numerous studies have demonstrated both direct and indirect negative impacts of uterine disease on overall dairy herd performance and profitability (Borsberry and Dobson 1989, Lee et al. 1989, Rajala and Grohn 1998, Fourichon et al. 1999, LeBlanc et al. 2002, Gilbert et al. 2005). California researchers found that cows with metritis averaged 4.9 lbs/ day less milk over the first 120 days of lactation compared to normal herdmates (Deluyker et al. 1991). Others have found lower levels of milk loss. Rajala and Grohn reported a loss of 6 lbs/ day for cows with metritis, but only for a period of about two weeks (Rajala and Grohn 1998). Still, others have reported no effect of metritis on milk yield (Bartlett et al. 1986).

Reproductive performance is also negatively affected by metritis that occurs within the first three weeks in milk. Most commonly, the depression in fertility is reported as a change in average days open (typically about 18) or in median days open (range of 13 – 28)(Bartlett et al. 1986, Lee et al. 1989, Fourichon et al. 2000). Perhaps a more appropriate way to examine the fertility impact is to examine the effect on the daily probability of conception for the herd through the use of survival analysis (time-to-event analysis). This is the foundation of the concept of 21-day pregnancy rate. Using this approach, two of the previously cited references determined that metritis lowered the 21-day pregnancy rate by 16 – 30%. In other words, if the normal cows had a 21-day pregnancy rate of 20%, the cows with metritis would have a pregnancy rate of 14 - 16.8%, an absolute reduction of about 3 – 6 units of pregnancy rate performance.

Surprisingly, there is very little peer-reviewed information available that fully evaluates both the direct and the indirect costs of metritis. A complete cost estimate would ideally include the estimated financial losses from decreased milk production, depression in pregnancy rate, increased attributable culling risk, and any treatment costs. The report by Bartlett, et al attempted

to look at both direct and indirect costs associated with metritis and found the total cost per lactation with metritis was \$106 in 1986 (Bartlett et al. 1986). The goal of this paper is to estimate the total cost of metritis based on information from a large dairy herd using previously collected production, reproduction and culling data.

### Economic Model and Background

A spreadsheet model was built to estimate the total expected cost due to acute puerperal metritis. The data used to estimate milk loss, culling risk, and reproductive performance changes attributable to metritis was adapted from work by M. W. Overton and W. M. Sischo in a single, large dairy herd in California and included 500 cows diagnosed with metritis within the first 10 DIM. Metritis was defined as the presence of an atonic uterus, a malodorous, watery vaginal or uterine discharge, and a fever of 39.4°C (103°F) or greater within the first 10 DIM. Cows experiencing metritis were compared to a randomly selected group of normal cows (not diagnosed with metritis) that were also monitored daily for the first 10 DIM. The overall lactational incidence risk for metritis was 22%. The normal group was a randomly selected group of cows that had been monitored but were not diagnosed with metritis. Milk production information was collected using daily milk meters. Culling and reproductive information was obtained from the on-farm record system (DairyComp 305).

Cows experiencing metritis in the first 10 DIM had a different culling risk (proportion of animals that calved that were later sold or died on-farm) than normal cows. Instead of modeling the cost of culling for each group and then examining the difference, we utilized the attributable risk, calculated by subtracting the risk of culling for the normal cows from the risk of culling for the cows with metritis. The attributable risk for being sold and for dying within the first 60 DIM was calculated by parity group.

Many models will assign a “cost” of the cull by subtracting the salvage value from replacement cost. This is the cash cost of the cull but does not account for varying levels of depreciation that occur as cows go through successive lactations. Using this incorrect approach, a first lactation animal that falls and breaks her leg at one DIM would “cost” the same as a sixth lactation animal that died at one DIM due to severe hypocalcemia. In order to more accurately assess the cost of the cull, one has to determine the expected value of that animal at that given time. The model calculates the cow’s current, depreciated value at the start of her current lactation. Subtracting the salvage value, if any, from this calculated value is a better estimate of the real cost of her removal from the herd.

Within the model, parity-specific attributable culling risks for the first 60 DIM are used to calculate culling losses due to metritis as shown in Figure 1. The salvage value for first lactation animals is \$460 and for lactation two and above, it is \$621, based on differences in body weight at the time of culling. Culling losses are stratified into losses due to animals that were sold and animals that died. Patterns of culling are very similar between metritis and normal cows from 60 DIM until the end of the breeding period. Culling differences within the breeding period are accounted for within the reproduction model. In this herd, given the assumptions used, the estimated cost of culling within the first 60 DIM is \$85 per case of metritis.

**Figure 1. Cost of Premature Culling (Sold and Died) From Herd Due to Metritis**

Cost of Premature/ Excess exits from herd due to Metritis									
	Avg Value at Start of Lactation	Salvage Value if Sold	Proportion of Total Metritis Cases	Attributable Culling Risk (Sold)	Culling Loss	Weighted Cost of Culls (Sold) Due to Metritis	Attributable Culling Risk (Dead)	Dead Cow Losses	Weighted Cost of Culls (Dead) Due to Metritis
lact 1	\$ 2,262	460	33%	4.2%	\$ 1,802	\$ 25	0.5%	\$ 2,262	\$ 4
lact 2	\$ 1,863	621	34%	1.1%	\$ 1,242	\$ 5	6.5%	\$ 1,863	\$ 41
lact 3	\$ 1,551	621	18%	2.6%	\$ 930	\$ 4	0.3%	\$ 1,551	\$ 1
lact 4	\$ 1,304	621	16%	5.0%	\$ 683	\$ 6	0.1%	\$ 1,304	\$ 0
			100%			\$ 39			\$ 46
total loss to excess culling and death (per cow in metritis case population)									<b>\$ 85</b>

Milk production differences from the data set were incorporated into the model as follows: a) data from the herd showed that cows with metritis that were culled during the first 30 DIM produced 15.1 lbs less milk per day and had a median days-to-exit of 10, b) cows with metritis that were culled during 31 - 60 DIM produced an average of 9.1 lbs less milk per day and had a median days-to-exit of 42, and c) cows with metritis that survived past 60 DIM experienced an average of 6.2 lb loss per day over the first 110 DIM and then no difference over the rest of lactation as compared to the normal cows. A marginal milk value of \$0.13 per lb was used, assuming a baseline milk price of \$18/ cwt and an expected 2.5 lbs of marginal milk produced per pound of marginal feed consumed. As a consequence, the total estimated milk loss (weighted average) attributable to metritis was \$83/ case of metritis as shown in Figure 2.

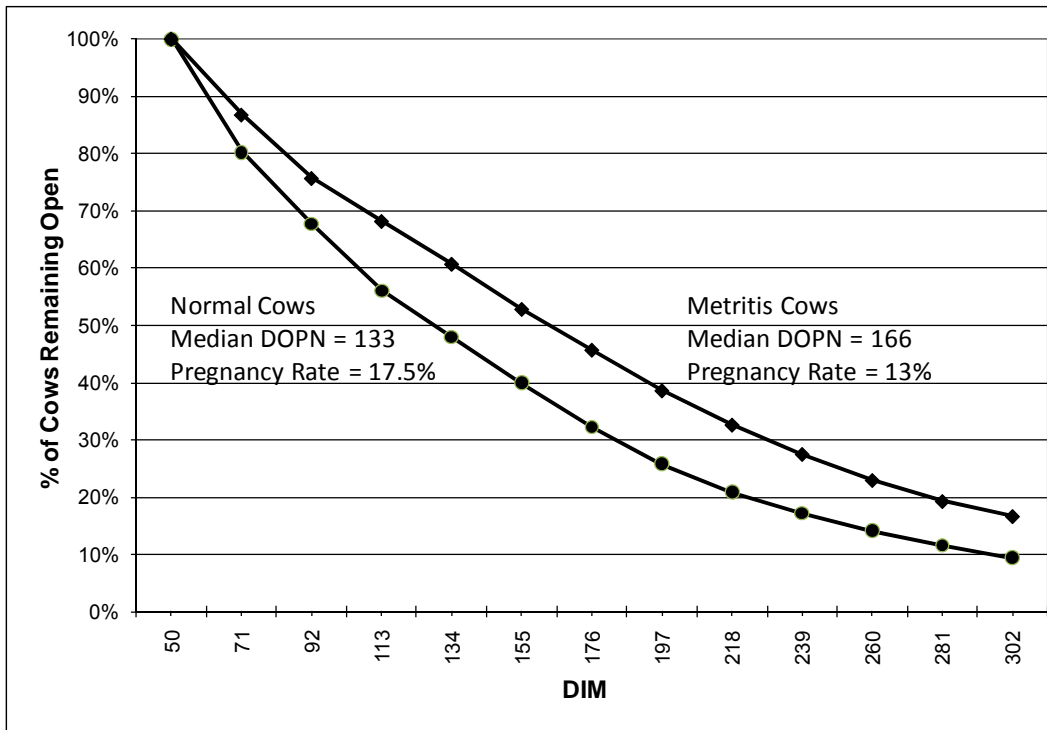
**Figure 2.** Cost Associated with Reduced Milk Production Due to Metritis

<b>Production Losses (Reduced Milk Production)</b>	
Marginal value of lost milk/ metritis case culled (1st 30 DIM)	\$ (20)
% of metritis cases culled/ dead 1st 30 DIM	6%
Marginal value of lost milk/ metritis case culled (31 - 60 DIM)	\$ (49)
% of metritis cases culled/ dead 2nd 30 DIM	4%
Marginal value of lost milk/ cow with metritis retained past 60 DIM	\$ (89)
% of metritis cases retained past 60 DIM	90%
<b>Total milk loss/ case (weighted avg)</b>	<b>\$ (83)</b>

The metritis-related cost associated with reduced reproductive performance was estimated using a modified version of Overton's previously described economic model (Overton 2001, Overton and Galvao 2004, Overton 2006a, Overton 2006b). The reproductive performance data was fit in the model in order to generate a simulated Kaplan-Meier survival plot that approximated the original study data and to estimate the 21-day pregnancy rate for each subgroup by modifying the insemination and conception risk for each 21-day period. Animals that failed to conceive within 12-21day breeding cycles were assumed culled as non-pregnant cows. In addition, variable culling risks were applied within each 21-day period to mimic the dairy's real results.

A total of 73% of normal cows that calved became pregnant and survived for the entire lactation compared to only 59% for the cows with metritis. The modeled survival plot reveals an attributable culling risk of 8% due to metritis-associated infertility. Combining this 8% risk with the attributable culling risk during the first 60 DIM (5.3%) yields a total that approximates the 14% from the actual data (73% – 59%). Average and median days open were 16 and 33 days longer, respectively, for the metritis group. The predicted 21-d pregnancy rate was 17.5% for the normal cows and 13% for the cows experiencing metritis. As a consequence of the combined effects of excess culling and the costs of extra insemination and breeding program efforts, the predicted monetary loss was approximately \$121 per cow in the breeding pool, or \$109 per case of metritis, once the total was adjusted to account for the earlier culls. These values were calculated using a replacement cost of \$2,200, herd level 305ME of 25,000, a milk price of \$18, salvage value of \$621, and an interest rate of 8% as inputs in the reproduction model.

**Figure 3.** Time-to-Event Plot for Normal vs. Metritis Cows



The final area of consideration is the direct treatment cost associated with metritis. The model considers two different antibiotic choices for systemic therapy, ceftiofur (Excenel®RTU, Pfizer) and ampicillin (Polyflex®, Fort Dodge), and assumes no therapeutic advantage for one vs. the other. We assumed that one would use the Excenel as per label (1 mg/ lb (2 ml/ 100 lbs) IM or SQ once daily for 5 days) and that Polyflex would also be used once daily for 5 days (5 mg/ lb or 2 ml/ 100 lbs of rehydrated product). The cost per 100 ml bottle was assumed to be \$58 for Excenel and \$29 for Polyflex based on current market prices for each. Excenel does not require a milk withdrawal and as long as no other therapy that requires a withdrawal is used, the treated cow does not have to enter the hospital pen. Polyflex, on the other hand, has a withdrawal of 48 hours following the last treatment. The model does not consider any supportive therapy or escape therapeutic options since these are assumed to be the same for each drug.

The model allows the user to toggle between the drugs and to also select how the discarded milk is handled if Polyflex is used. If Excenel is used, the estimated cost is \$81. If Polyflex is used and the milk is fed as waste milk, the cost is \$53 after accounting for the opportunity cost of the discarded milk that is utilized as calf feed. On the other hand, if Polyflex is used and the milk is discarded, the cost is \$109, accounting for the lost opportunity cost of the discarded milk.

By adding each of the components of the model together, the total estimated cost of a case of metritis may be determined. The total cost due to culling in the first 60 DIM as a consequence of metritis is \$85 per case, the total milk loss due to metritis is \$83 per case and the losses due to reproductive issues is \$109 per case. The actual treatment cost varies from \$53 to \$109 depending upon drug used and the utilization of any withheld milk. Using the aforementioned definition of metritis and actual data derived from the farm, the total estimate cost

per case of metritis is \$358 if Excenel is used, \$329 if Polyflex is used and the milk is used to feed calves, or \$386 if Polyflex is used but the withheld milk is discarded.

### **Discussion and Conclusion**

The total estimated cost of metritis in this model is significantly higher than the previous estimate cited (\$106 by Bartlett et al, 1986). However, a few major differences in herd performance and approach used in the models should be recognized between the two estimates. In the Bartlett paper, which relied on monthly DHIA data, no impact on milk production due to metritis was found. In the current paper, significant losses were identified that were very similar to those reported by Deluyker et al, 1991. Both of these studies used daily milk weights and perhaps there was a greater ability to measure differences with more frequent (and presumably more sensitive) measurements. Other possibilities to explain the discrepancies include a differing level of milk production for the herds investigated and potentially, a difference in the definition for metritis used between the studies. If disease misclassification occurs, the tendency would be to bias the results toward finding no difference due to the inclusion of normal cows in the abnormal group and vice versa.

Bartlett et al reported a very low culling risk overall and an attributable culling risk due to metritis of only 6.1%. In this paper, the attributable culling risk was 5.3% within the first 60 DIM alone, and when combined with the breeding period, the total attributable culling risk was 14%. The difference in attributable culling risk alone accounts for approximately \$100 of the large difference between estimates.

Reproductive losses were handled in different ways as well. In Bartlett et al, the total cost due to reproductive failure was estimated to be only \$18.89, based primarily on the value of differences in days open. In the current study, the approach used to estimate the reproductive losses used an existing reproductive model to account for changes in expected milk production as a consequence of change in reproductive performance, a difference in number of inseminations, and differences in culling due primarily to reproductive failure. Using commonly cited values for the cost of a day open, the current data set would have a reproductive cost of approximately \$48 if only average days open was the criteria used, but this approach underestimates the true cost of the reduced reproductive performance.

It is difficult to address the differences in treatment costs between the two studies. Exact treatment used was not reported by Bartlett et al but they did state that the medication cost per treated cow was only \$2.74 as compared to at least \$53 in the current study. Milk that was withheld as a consequence of treatment was estimated to cost the dairy \$23.85 based on their estimate that approximately half of the milk that was assumed to have been withheld was fed to calves. The price of milk and level of production is likely very different between the two datasets. In summary, the total cost of metritis in this large herd was estimated to be approximately \$358 per diagnosed case, despite aggressive systemic antibiotic therapy. The magnitude of this estimate may surprise some, but the reality is that metritis is an expensive disease problem. If the results of this model are applied to a herd of 1,000 milking cows, and the lactational incidence for metritis is 22% as found with this modeled herd, the total cost would be approximately \$79,000 per year using the previously mentioned assumptions. Of course, individual herd costs are likely to vary depending upon treatments utilized, definition of metritis and detection methods used, cow comfort potential, season, nutritional support, etc. Regardless of the exact cost, the authors of this paper suggest that there may be significant financial returns to made by improving transition management in an effort to reduce the risk of developing metritis.

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# The Georgia Mastitis Situation: Cell Counts and Microbiology

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## Introduction

Consumers are increasingly becoming interested in purchasing locally produced foods. They also demand a quality product at a competitive price. Therefore it is important that we provide the highest quality product. One aspect of quality is the somatic cell count which directly impacts the shelf life of the finished product.

Mastitis is the most costly disease in the dairy industry and control has been a major emphasis of the industry for over 50 years. This emphasis has resulted in significant improvement in control.

Georgia dairy producers have made significant strides in controlling mastitis over the past decades and have been effective in reducing the prevalence of some organisms. This has been for a variety of reasons, including the application of recommended practices such as teat dipping, dry cow therapy and proper milking procedures. Other factors such as quality premiums and regulatory mandates have also had an effect.

We need to know how others are doing if we are to fully understand how we are doing. This can be accomplished by looking at the values for other herds throughout the country. We can achieve this by evaluating the DHIA values available through the Dairy Records Management System (DRMS).

## Somatic Cell Counts

Somatic cell count scores (SCCS) for the past 6 years for surrounding states are illustrated in Figure 1. One can easily see that the SCCS averages about 3 (2.98) with little variation among the years. There is a slight increase in SCCS during the months of July, August and September; however, the cell counts remain rather constant during the remaining months.

Figure 2 illustrates the same information for Georgia. You can see that the values for Georgia are about one-half score higher (3.41). This equates to a loss of about 200 pounds of milk per cow per lactation than for the region. This may not seem like much but when you apply that value to an entire herd it becomes pretty significant.

There has been a slight improvement in SCCS over the years; however, it continues to lag behind the average for other states. This puts Georgia at a competitive disadvantage compared to many states.

The University of Georgia dairy herd has been on the somatic cell count program since its inception. This allows us to look at the herd's performance over several years and evaluate the effect of changes in management.

The herd had been bedded with sawdust until November, 2005 when the bedding material was gradually changed to sand as the barn was remodeled and adapted for sand. The complete change took several months so the effect is moderated.

Figure 3 shows the SCCS for the herd for the past 4 years. The cell counts were higher than we would have liked and we attribute much of this to the housing available. You will notice that the

cell counts immediately dropped following the introduction of sand. They continued to drop for the next several months as more cows were bedded on sand. Cell counts reached a low in June of 2006 and then rose during the subsequent months. They have fluctuated primarily between 2.6 and 3.0 in subsequent months. There have been some blips in the cell counts when they rose higher than desired. These can be directly attributed to the stalls becoming contaminated due to a lack of sand availability.

The percentage of cows considered to be uninfected (SCCS 0 – 3) is illustrated in Figure 4. One can easily see that the percentage has increase significantly since sand bedding was introduced. Conversely, Figure 5 presents information on the percentage of cows which are considered to be infected (SCCS 7-9). This category has seen a significant decrease.

## **Microbiology**

For the past 4 years we have been culturing all cows at dry off, at freshening and day 10 after calving. Clinical cases and cows with high cell counts are also cultured to identify the causative organisms.

The good news is that we have not cultured any *Streptococcus agalactiae* (Str. ag.). This does not mean that it has been totally eliminated but has been at least reduced to a minimal level.

The bad news is that we have cultured a large number of *Staphylococcus aureus* (S. aureus) in some herds. This is a contagious and very destructive organism and doesn't respond as well to therapy as many other organisms. It can and does cause significant losses for dairy herds through milk quality problems, potential loss of market and increased culling rates.

Coagulase negative staphylococci (CNS) are the primary organisms generally cultured. These organisms are ubiquitous. Many of these infections are subclinical and remain subclinical throughout the lactation. However, they can cause a significant increase in somatic cell counts causing quality problems.

Environmental streptococci (ES) have been cultured sporadically. These also cause significantly elevated cell counts although they remain subclinical. Coliforms have also been cultured sporadically and the incidence in the University of Georgia dairy herd has decreased significantly since the introduction of sand bedding.

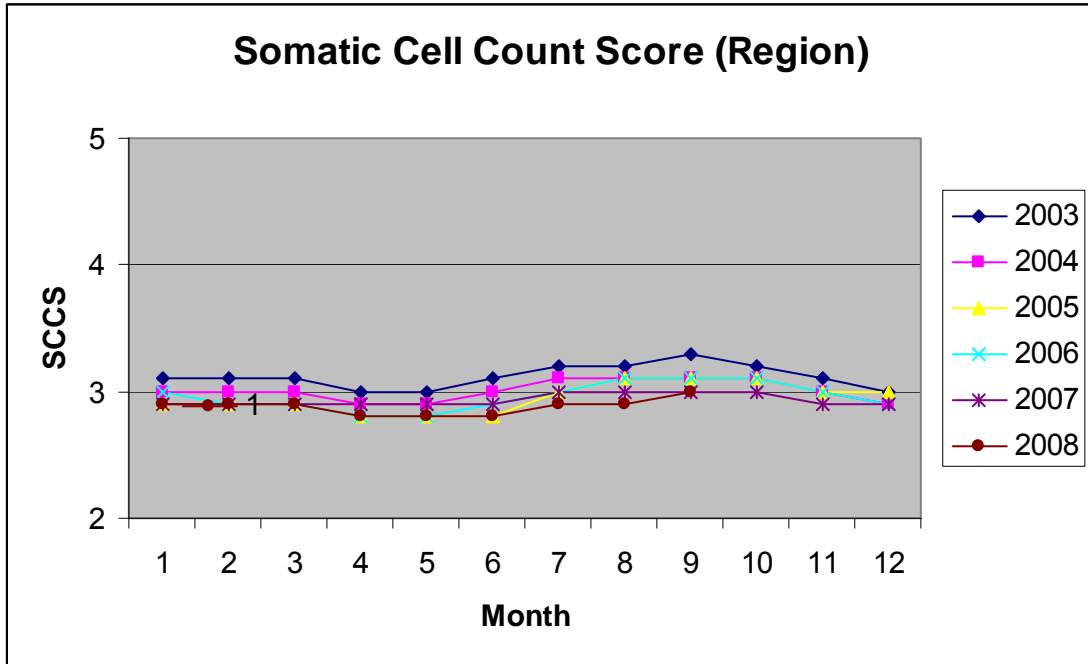
## **Conclusions**

Milk quality is one way in which Georgia dairy producers compete in the marketplace with producers from other states. Georgia has seen a slight improvement in cell counts over the past few years. However, there is still room for improvement.

*Streptococcus agalactiae* does not appear to be a problem but *Staphylococcus aureus* does. Coagulase negative staphylococci are the primary organism causing clinical mastitis and elevated somatic cell counts. Other organisms may be a problem in isolated instances.

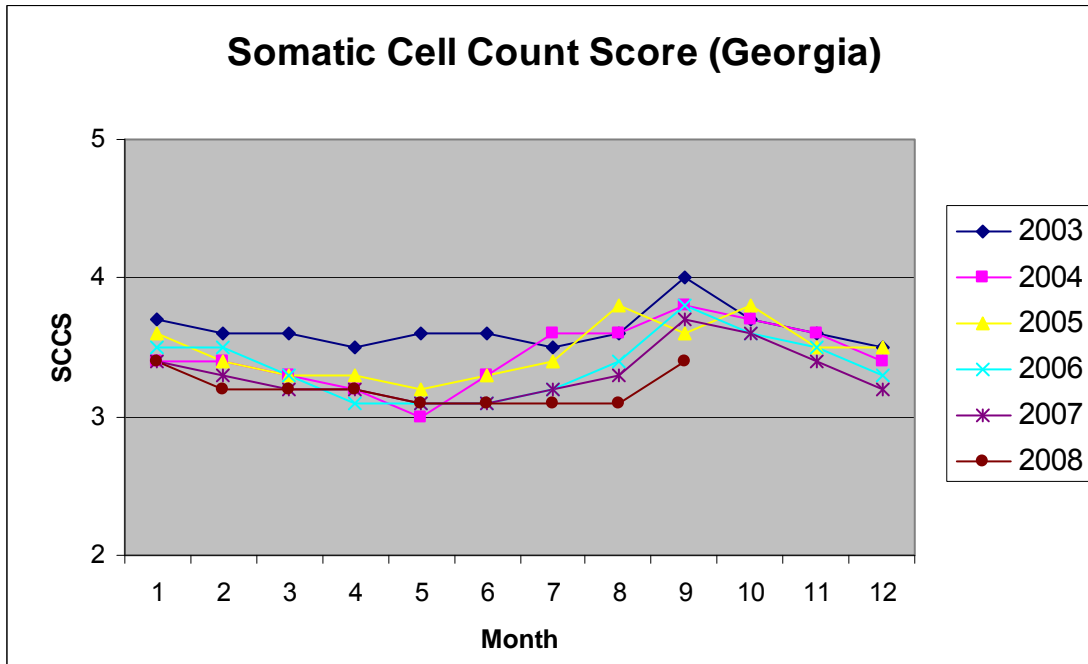
Sand is an effective bedding material for helping to reduce somatic cell counts and the incidence of mastitis. It must be kept clean otherwise the incidence of mastitis will increase.

Figure 1. Monthly Average Somatic Cell Count Score for Region (2003-2008)



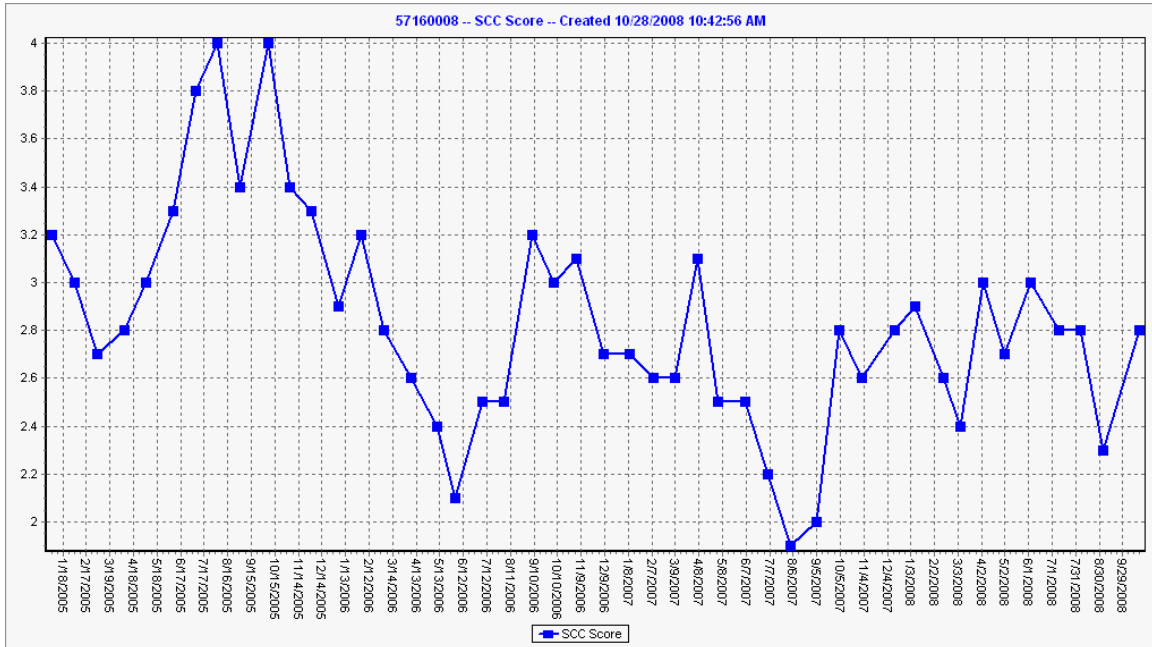
Source: DRMS, Raleigh, NC

Figure 2. Monthly Average Somatic Cell Count Score for Georgia (2003-2008)



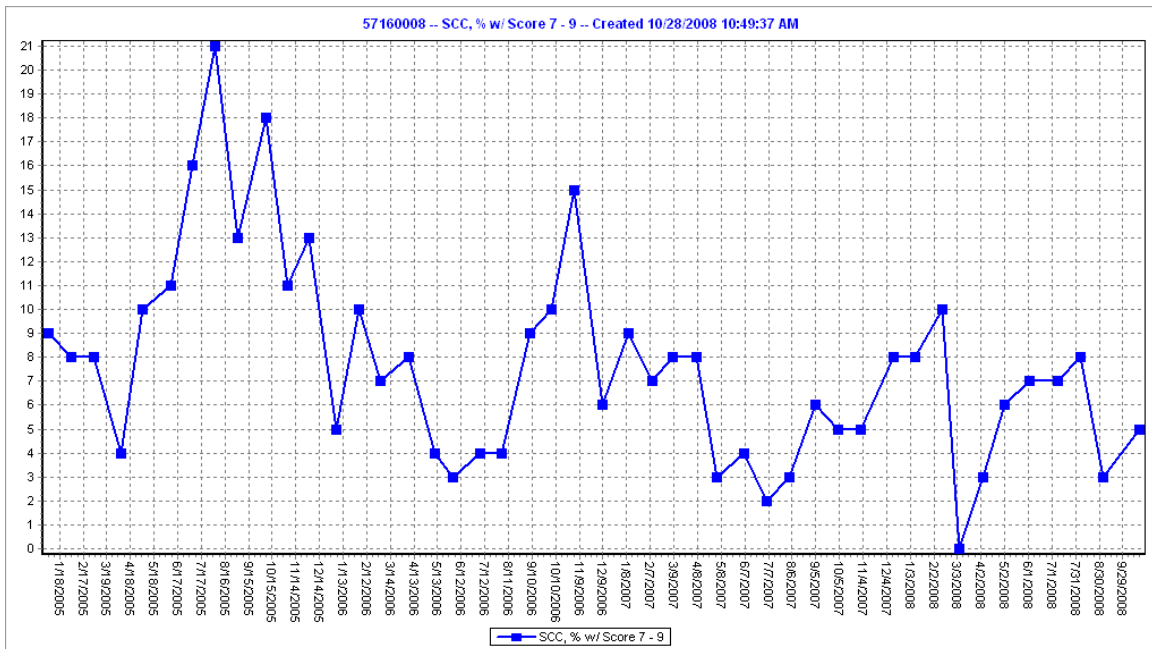
Source: DRMS, Raleigh, NC

Figure 3. Monthly Average Somatic Cell Count Score for University of Georgia dairy herd (Jan, 2005 – Sep, 2008)



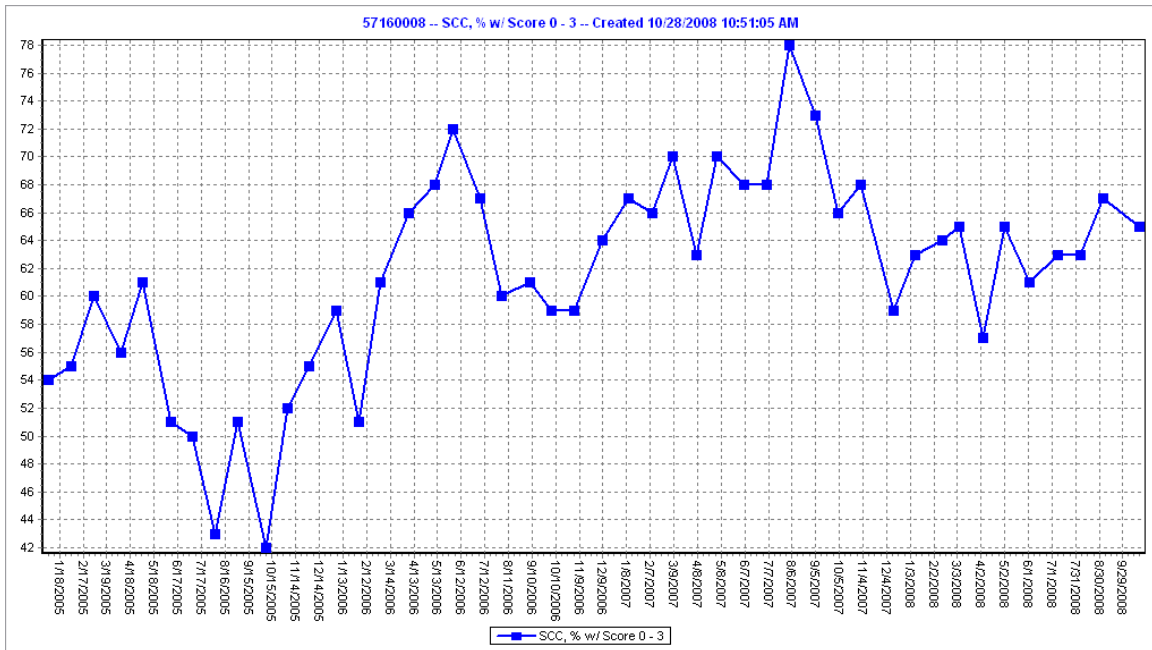
Source: DRMS, Raleigh, NC

Figure 4. Monthly Percentage of Cows with Somatic Cell Count Score 7-9 for the University of Georgia Dairy herd (Jan, 2005 – Sep, 2008)



Source: DRMS, Raleigh, NC

Figure 5. Monthly Percentage of Cows with Somatic Cell Count Score 0-3 for the University of Georgia Dairy herd (Jan, 2005 – Sep, 2008)



Source: DRMS, Raleigh, NC

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## Assessing Milk Quality

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**Introduction:** Production of maximum quantities of high quality milk is an important goal of every dairy operation. On the other hand, poor milk quality affects all segments of the dairy industry, ultimately resulting in milk with decreased manufacturing properties and dairy products with reduced shelf-life. How is milk quality determined? Several different methods are used to assess milk quality (Standard Methods for the Examination of Dairy Products, 2004). Some methods such as the somatic cell count (SCC) and standard plate count (SPC) are mandated by the Grade A Pasteurized Milk Ordinance (revised in 2007), which is a document that specifies safety standards of Grade A milk. Other methods, while not mandated, are useful to monitor milk quality and to help diagnose potential on-farm problems/deficiencies associated with abnormally high counts and poor quality milk. The following is a brief description of the primary methods used to assess raw milk quality.

**Somatic Cell Count:** The number of somatic cells in milk, referred to as the somatic cell count or SCC, is used throughout the world as an indicator of milk quality. The current regulatory limit for somatic cells in milk in the U. S. defined in the Grade A Pasteurized Milk Ordinance is 750,000/ml. For a variety of very good reasons, there is continuing pressure from animal health advocacy groups to reduce the regulatory limit for somatic cells in milk from the current 750,000/ml to 400,000 or less.

Poor quality milk has a high number of somatic cells, and is an inferior product with reduced processing properties resulting in dairy products with a reduced shelf-life (Barbano et al., 2006; Ma et al., 2000). On the other hand, high quality milk has a very low number of somatic cells, has a longer shelf-life, tastes better, and is more nutritious. One characteristic feature of cows with mastitis is a significant elevation in the number of somatic cells in milk. Milk from uninfected mammary glands contains < 100,000 somatic cells per milliliter. A milk SCC > 200,000/ml suggests that an inflammatory response has been elicited, that a mammary quarter is infected or is recovering from an infection, and is a clear indication that milk has reduced manufacturing properties. It is not uncommon for milk from cows with subclinical and/or clinical mastitis to contain several hundred thousand and even millions of somatic cells/ml of milk. Thus, an increase in the SCC of milk is a good indicator of mastitis or inflammation in the udder. Infection of the udder by mastitis pathogens alters milk composition and reduces milk yield. Most studies that evaluated the influence of mastitis on the composition of milk used SCC as the basis for determining the infection status of udders and for determining the degree of inflammation.

The bulk tank SCC (BTSCC) can be used to gauge the udder infection status of a dairy herd, and also gives a good indication of the loss in milk production in a herd due to mastitis. As the BTSCC increases, the percent of mammary quarters infected increases and the percent production loss increases. Small increases in SCC can impact production. Most herd milk contains between 200,000

to 500,000 somatic cells/ml of milk (Miller et al., 2008). These herds are losing at least 8% in potential milk production. Thus, methods of mastitis control that reduce SCC will not only improve milk yield and composition but will also decrease economic losses due to mastitis.

A recent report published by the USDA Animal Improvement Program Laboratory (Miller et al., 2008) summarized SCC data from all herds in the United States enrolled in the Dairy Herd Improvement (DHI) testing program for 2007. The good news is that the national SCC average for 2007 was 276,000 cells/ml of milk, which is 12,000 cells/ml lower than in 2006 (Miller et al., 2007). The bad news, however, was that 3.5% of herds in the U. S. had SCC's in excess of 750,000 and 24% of the national dairy herd had SCC > 400,000. In 2006, almost 4% of herds in the U. S. had SCC > 750,000/ml and 25% of the national dairy herd had > 400,000 SCC/ml. The SCC of milk produced by dairy farms in the Southern Region of the U. S. over the last 10 years was about 35% higher than the U.S. average with a yearly range of approximately 30% higher in 2000 to almost 41% higher than the U. S. average in 2003. These data demonstrate quite clearly that there is much room for improving milk quality in the U. S., and this is particularly the case for milk produced on dairy farms in the Southeast.

**Standard Plate Count (SPC):** The SPC is an estimate of the total number of viable aerobic bacteria present in raw milk. This test is done by plating milk on a solid agar, incubating plates for 48 hours at 32°C (90°F) followed by counting bacteria that grow on plates. The SPC is used to monitor progress since consistent application of proper milking system cleaning practices, proper milking practices, udder hygiene and good mastitis prevention and control practices should allow dairy producers to produce milk with a low SPC (< 5,000 colony forming units (cfu) of bacteria/ml). Federal regulations defined in the Pasteurized Milk Ordinance mandate that the milk SPC should not exceed 100,000 cfu/ml. However, most segments of the dairy industry feel that more stringent standards (SPC ≤ 10,000 cfu/ml) will result in higher quality milk. Though it is impossible to eliminate all sources of bacterial contamination of milk; milk from clean, healthy cows that has been properly collected generally has a SPC < 1,000 cfu/ml. Consistent application of proper milking practices, udder hygiene and good mastitis prevention and control practices should allow dairy producers to produce milk with a SPC of ≤ 5,000 cfu/ml, while most farms can produce milk with counts of < 10,000 cfu/ml. High bacterial counts (> 10,000 cfu/ml) suggest that bacteria are entering milk from a variety of possible sources (Gillespie et al., 2007; Gillespie et al., 2008; Jayarao et al., 2004). The most frequent cause of high SPC's is poor cleaning of milking systems. Milk residues on equipment surfaces provide nutrients for growth and multiplication of bacteria that contaminate milk of subsequent milkings. Cows with mastitis (streptococcal and coliforms), soiled cows, unclean milking practices, failure to cool milk rapidly to < 4.4°C (40°F), failure of the water heater, and extremely wet and humid weather can also contribute to high SPC's in raw milk (Figure 1). Some limitations of the SPC method include: 1) no indication of the bacterial types present, 2) no indication of the specific source of high counts, and 3) the SPC does not give a complete count of all bacteria as some bacteria only grow at lower temperatures.

**Preliminary Incubation Count (PI count):** The PI count is an estimate of the number of psychrotrophic (cold-loving) bacteria in milk. The PI count is not a regulatory test and results of this test are interpreted as a general reflection of milk production practices on the farm and are used as a tool to identify inadequate on-farm sanitation practices and holding temperature of milk in the bulk tank. The PI count is conducted by holding milk at 55°F for 18 hours. Bacteria that grow under refrigerated conditions are enumerated using the SPC method described above. PI counts are generally higher than SPC's. Selection of a PI count cut-off and interpretation of PI count results are difficult because variability in PI counts negatively influences repeatability (Boor et al., 1998; Murphy and Boor, 2000; Jayarao et al., 2004). Some milk plants use a specific cut-off number while others use PI counts in relation to SPC's. PI counts < 10,000 cfu/ml are considered low, while PI counts > 20,000 cfu/ml are considered high (Table 1, Jayarao et al., 2004). A PI count 3 - 4 fold higher than the SPC is suggestive of potential problems related to cleaning and sanitation of the milking system or poor udder preparation before milking (Figure 1). Failure to cool milk rapidly, marginal cooling, prolonged storage times, milking cows with wet teats, and/or extremely wet and humid weather conditions may also result in high PI counts (Gillespie et al.,

2007; Gillespie et al., 2008). A PI count equal or slightly higher than a high SPC (> 50,000 cfu/ml) may suggest that the high SPC is possibly due to mastitis (Figure 1). The PI count has been used by some as an indicator of the shelf-life of processed dairy products. However, research conducted at Cornell University (Boor et al., 1998; Murphy and Boor, 2000) and Penn State University (Jayarao et al., 2004) has shown that the PI count alone can not be directly correlated with the flavor quality of raw milk OR quality OR shelf-life of processed dairy products. PI counts are most useful with data from other tests and additional information such as farm observations and inspections.

**Laboratory Pasteurization Count (LPC):** The LPC, also known as the thermoduric count, is an estimate of the number of bacteria that can survive laboratory pasteurization at 62.8°C (143°F) for 30 minutes. This process destroys most of the mastitis causing pathogens, selecting for those bacteria that can survive pasteurization temperatures (thermoduric bacteria). This is not a regulatory test required by state or federal agencies; however, some milk processors perform this test to ensure quality of the final product. Bacteria not killed by pasteurization are enumerated using the SPC method. LPC's are generally much lower than SPC's (Gillespie et al., 2007; Gillespie et al., 2008; Jayarao et al., 2004). An LPC of > 200 cfu/ml is considered high (Table 1). A high LPC is most often seen with persistent cleaning problems; faulty milking machine or worn out parts such as leaky pumps, old pipe line gaskets, inflations and other rubber parts; and milkstone deposits. Significant contamination from soiled cows can also contribute to high LPC's.

**Coliform Count (CC):** The CC is a test that estimates the number of bacteria that originate from manure or a contaminated environment. Milk samples are plated on Violet Red Bile Agar or MacConkey's agar and incubated for 48 hours at 32°C (90°F), after which typical coliform colonies are counted. Coliform counts reflect hygiene and sanitation practices followed on the farm. Coliforms enter the milk supply as a consequence of milking dirty cows or dropping the milking claw into manure during milking. Coliform counts > 100 cfu/ml suggest poor milking practices, dirty equipment, contaminated water, dirty milking facilities, and/or cows with subclinical or clinical coliform mastitis (Jayarao et al., 2004).

**Conclusions:** Production of maximum quantities of high quality milk is an important goal of every dairy operation. Poor quality milk affects all segments of the dairy industry, ultimately resulting in milk with decreased manufacturing properties and dairy products with reduced flavor quality and reduced shelf-life. Several different methods are used to assess milk quality. Some methods such as the SCC and SPC are mandated by the Grade A Pasteurized Milk Ordinance. Other methods, while not mandated, are useful to monitor milk quality and to help diagnose potential on-farm problems/deficiencies associated with abnormally high counts and poor quality milk.

Recently, some milk buyers/dairy processing plants have made changes to their milk quality requirements for incoming raw milk. These changes have occurred, in part, by demands from retailers and major food service companies requiring milk with a higher quality to achieve a longer shelf-life. SCC and SPC limits for raw milk to be acceptable at dairy processing plants may decrease to levels much lower than they are now, making it increasingly problematic for dairy producers to meet these higher standards. In addition, some processing plants use PI counts and LPC's in addition to SCC and SPC's to assess milk quality.

Production of higher quality milk will place a much greater emphasis on management strategies to minimize contamination of raw milk such as cow and equipment cleanliness, and sanitation procedures; and management strategies for the prevention and control of mastitis to reduce the number of somatic cells in milk. Effective milking-time hygiene, proper milking machine function, pre- and post-milking teat disinfection, lactation therapy, antibiotic dry cow therapy and culling of chronically infected cows are time-tested management strategies for controlling mastitis and are used extensively throughout the world. Providing and maintaining a clean, dry, comfortable environment for heifers, lactating cows **AND** dry cows will reduce/minimize problems associated with environmental contamination of raw milk while also reducing mastitis caused by environmental mastitis pathogens.

A safe, wholesome, abundant and nutritious milk supply should be the goal of every dairy producer in the world. Safety and quality of dairy products start at the farm and continue throughout the processing continuum. One thing is certain....it is impossible to transform a low quality raw milk product into a high quality finished dairy product! To meet increased raw milk quality standards, producers must adopt production practices that reduce mastitis and reduce bacterial contamination of bulk tank milk. Use of effective management strategies to minimize contamination of raw milk and proven mastitis control strategies will help dairy producers achieve these important goals.

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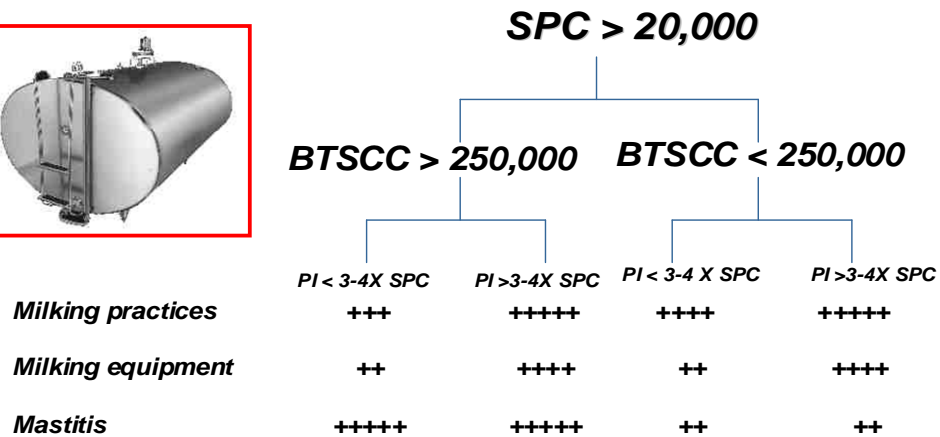
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# Table 1. Interpretive Criteria for Bulk Tank Milk Monitoring

Parameter	Low	Medium	High
Bulk tank SCC	<200,000	200,000 - 400,000	>400,000
Standard Plate Count (SPC)	<5,000	5,000 - 10,000	>10,000
Preliminary Incubation Count (PIC)	<10,000	10,000 - 20,000	>20,000
Lab Pasteurized Count (LPC)	<100	100 - 2000	>200
Coliform Count	<50	50 - 100	>100

Adapted from Jayarao et al. J. Dairy Sci. 2004

## Figure 1. Troubleshooting Sources of Bulk Tank Milk Contamination



From Jayarao, Penn State Univ



**College of Agriculture and Environmental Sciences  
Office of Academic Affairs Update, Fall, 2008**

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**Enrollment**

Enrollment in UGA's College of Agriculture and Environmental Sciences (CAES) for the fall, 2008 semester is at an all-time high. A total of 1588 undergraduates is 9.4% higher than the fall, 2007 enrollment of 1451 students and tops the all-time high enrollment of 1532 students set in 1978. In addition, graduate enrollment grew by 13.4% over the 2007 enrollment of 365 students to 414 students. The percentage of minority students increased from 12% to 3%, and the percentage of female students increased from 50% to 53% over the last year.

Table 1 presents the number of undergraduate students in each major in CAES. The two largest majors in the College are Biological Sciences (283) and Animal Science (257). These are followed by Agricultural Engineering (135), Agribusiness (124), Animal Health (99), and Biological Engineering (98).

<b>Table 1. Undergraduate enrollment in the majors in the College of Agricultural and Environmental Sciences, fall, 2008.</b>					
<b>Major</b>	<b>Campus</b>	<b>No. of Students</b>	<b>Major</b>	<b>Campus</b>	<b>No. of Students</b>
Agribusiness	Athens	124	Biol. Sciences	Athens	283
Agribusiness	Griffin	3	Biological Sciences	Griffin	5
Ag. & Appl. Econ.	Athens	31	Dairy Science	Athens	3
Ag. Communication	Athens	32	Env. Chemistry	Athens	15
Agricultural Education	Athens	37	Env. Econ. & Mgmt.	Athens	79
Agricultural Education	Tifton	14	Entomology	Athens	9
Ag. Engineering	Athens	135	Env. Resource Sci.	Griffin	16
Ag. & Env. Systems	Athens	12	Food Ind. Mgmt. & Adm.	Athens	1
Ag. & Env. Systems	Tifton	25	Food Science	Athens	57
Animal Health	Athens	99	Horticulture	Athens	70
Animal Science	Athens	257	Landsc. & Grds. Mgmt.	Athens	5
Applied Biotechnology	Athens	37	Poultry Science	Athens	26
Avian Biology	Athens	32	Turfgrass Management	Athens	38
Biological Engineering	Athens	98	Water and Soil Resources	Athens	20
			Unspecified	Athens	25

Table 2 presents the number of graduate students in each department in CAES. The Department of Food Science and Technology has the largest graduate program with 81 students. The Department of Animal and Dairy Science (ADS) has 37 graduate students.

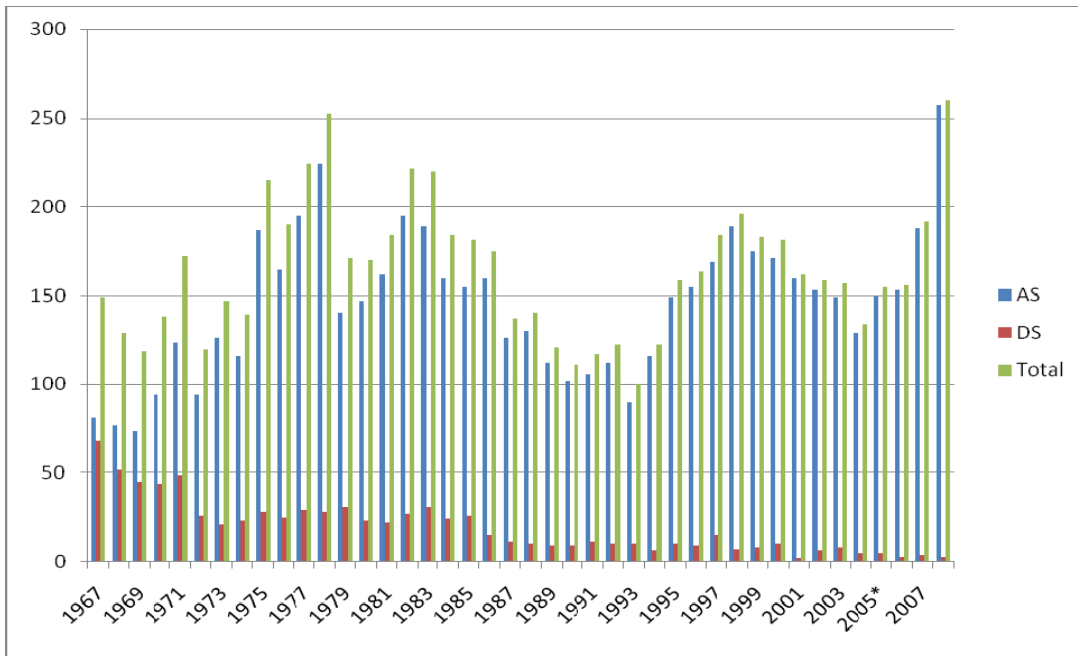
<b>Department</b>	<b>Number of Students</b>
Agricultural and Applied Economics	38
Animal and Dairy Science	37
Agricultural Leadership, Education, and Communication	46
Biological and Agricultural Engineering	48
Crop and Soil Science	53
Entomology	45
Food Science and Technology	81
Horticulture	18
Plant Pathology	29
Poultry Science	19

Most students enrolled in CAES, 96%, attend the Athens campus (Table 3). Twenty-four undergraduate students attend the Griffin campus majoring in Environmental Resource Science (16), Biological Sciences (5) or Agribusiness (3). Thirty-nine students attend the Tifton campus and major in either Agricultural Education (14) or Agricultural and Environmental Systems (25).

<b>Campus</b>	<b>No. Students</b>
Athens	1525
Griffin	24
Tifton	39

Enrollment in ADS is also at an all-time high and increased 37% from fall, 2007 (Figure 1). A major recruiting effort was launched as part of the summer orientation process that targeted pre-vet, pre-med, and pre-law students. Of the 257 Animal Science majors, 65 have chosen the emphasis area of Animal Biology, 26 have chosen Equine Science Management, 31 have chosen Production and Management, and 136 have not yet declared an emphasis area. While there are only three students majoring in Dairy Science, there are an additional nine students with double majors in Dairy Science and Animal Science, and one student is double majoring in Dairy Science and Poultry Science.

**Figure 1. Enrollment trends for Animal Science majors, Dairy Science majors, and total in the Animal and Dairy Science Department.**



Another bright spot for students in CAES is the starting salaries. According to the most recent survey of UGA graduates conducted by the UGA Career Center, graduates from CAES have the third highest starting salaries on campus. Students that graduated from CAES in 2007 had an average starting salary of \$39,000. This is only behind the Terry College of Business at \$42,000 and the College of Environment & Design at \$40,300 (Table 4).

**Table 4. Median starting salaries of UGA 2007 graduates.**

College	No. of Responses	Median Starting Salary, \$
Business	197	\$42,000
Environment & Design	10	\$40,300
<b>CAES</b>	<b>44</b>	<b>\$39,000</b>
Education	65	\$35,900
Social Work	26	\$33,500
Family & Cons. Sci.	71	\$32,000
Forest Resources	9	\$32,000
Public Health	7	\$31,500
Public & International Aff.	35	\$30,000
Journalism	59	\$30,000
Arts & Sciences	264	\$30,000
<b>UGA</b>	<b>765</b>	<b>\$34,500</b>

Source: UGA Career Center, [www.career.uga.edu](http://www.career.uga.edu).

One of the goals of CAES has been to increase the number of students participating in Study Abroad programs and rise to the average level of participation at UGA, which is one of the highest in the country at about 30%. Great success was achieved in the 2007-08 academic years with a participation rate of 33%, an increase from 22% in 2006-07.

## **Dean's Promise**

When Dean Scott Angle came to CAES in 2005, he started a new program called 'The Dean's Promise' that promises all students a meaningful out-of-classroom experience. This includes participation in activities such as internships, study abroad, leadership, service-learning, and undergraduate research.

There are several outstanding internship programs offered through the College and many more offered through the departments. On the College level, the Brussels Internship is a collaboration between CAES and the Georgia Department of Agriculture. One student is selected each year to spend the summer at the Georgia Department of Agriculture's Office in Brussels, Belgium promoting Georgia agricultural products in the European Union.

The Congressional Agricultural Fellowship program is a collaboration between Georgia congressional offices in Washington, DC and CAES. This past summer, students worked in the offices of Saxby Chambliss, Johnny Isakson, Sanford Bishop, Jack Kingston, Jim Marshall, and John Barrow. Through the years, a number of these students have gone on to work full-time in legislative offices.

The UGA Cooperative Extension Internships match students with an agent to work on agriculture and natural resources, family and consumer science, and 4-H programming. The objective of this program is to recruit students into careers with the UGA Cooperative Extension Program.

The Georgia Farm Bureau Legislative Internship is a new program and holds the opportunity for college students or recent graduates to become involved in farm policy development, education and implementation with the Georgia Farm Bureau. The Rural Caucus Legislative Internship is also new and provides the opportunity for students to become involved in issues and legislation that impact agriculture and rural communities.

New for the summer, 2008 was the New Zealand Dairy Grazing Internships. Nine students spent the summer on dairy farms in New Zealand and another student is spending the summer and fall semester there. They learned about rotational grazing and management of dairy cows on grass. This program was started by Allen Titchmarsh and Richard Watson, New Zealanders who are investing in Georgia farmland and developing grazing dairies. They hope to grow a management force to assist with the management of their Georgia dairies. In 2009, they will accept 4 students in summer slots and up to ten students from July to December. Interested students should contact Lane Ely ([laneely@uga.edu](mailto:laneely@uga.edu)) or Jean Bertrand ([jeanbert@uga.edu](mailto:jeanbert@uga.edu)).

## Best Management Practices to Improve Milk Quality

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**Introduction:** Production of maximum quantities of high quality milk is an important goal of every dairy operation. Poor milk quality affects all segments of the dairy industry, ultimately resulting in milk with decreased manufacturing properties and dairy products with reduced shelf-life. Mastitis is the most important factor associated with reduced milk quality. Mastitis is an inflammation of the udder that affects a high proportion of dairy cows throughout the world. Mastitis differs from most other animal diseases in that several diverse bacteria are capable of infecting the udder. These pathogens invade the udder, multiply there and produce harmful substances that result in inflammation, reduced milk production and altered milk quality. Because mastitis can be caused by many different pathogens, control is extremely difficult and economic losses due to mastitis can be immense. The NMC, formerly referred to as the National Mastitis Council, estimates that mastitis costs dairy producers in the United States over two billion dollars annually. Thus, mastitis continues to be one of, if not, the most significant limiting factor to profitable dairy production in the United States and throughout the world. Objectives of this paper are to discuss the importance of high quality milk, and how dairy producers can produce high quality milk by controlling mastitis using proven methods of mastitis prevention and control.

**Why Should We Be Concerned About Milk Quality?** The quality of milk has been and continues to be a topic of intense debate. One important measure of milk quality is the number of somatic cells in milk, referred to as the somatic cell count (SCC). Milk with a high SCC is produced by cows with mastitis and is of inferior quality. In the United States, the current regulatory limit for somatic cells defined in the 2007 Grade A Pasteurized Milk Ordinance (PMO) is 750,000/ml of milk. Recently, California lowered their state SCC regulatory standard for legal milk to 600,000 cells/ml. There is continuing pressure from a variety of advocacy groups to reduce the regulatory limit for somatic cells in milk from the current 750,000/ml to 400,000 or less to be competitive in the global dairy marketplace. Global standards are considerably lower (400,000 somatic cells/ml), and as low as 150,000 to 200,000 somatic cells/ml in some of the Scandinavian countries. Thus, this disparity in SCC makes it difficult, if not impossible; to export United States produced milk/milk products to other developed countries.

A recent report was published by the USDA Animal Improvement Program Laboratory (Miller et al., 2008) on SCC data from all herds in the United States enrolled in the Dairy Herd Improvement (DHI) testing program for 2007 (Table 1). The good news is that the national SCC average for 2007 was 276,000 cells/ml of milk, which is 12,000 cells/ml lower than in 2006. The bad news was that 3.5% of herds in the U.S. had > 750,000 SCC/ml and

**Table 1.** Characteristics of DHI herd test days for milk yield and SCC by State during 2007.

State	Herd test days <sup>1</sup> (No.)	Cows <sup>2</sup> per herd (No.)	Avg daily milk yield (Pounds)	Average SCC (Cells/ml X 1000)	% Herd test days <sup>3</sup> with SCC greater than			
					750,000 cells/ml	600,000 cells/ml	500,000 cells/ml	400,000 cells/ml
Alabama	238	122.3	50.6	407	4.2	10.1	22.7	42.9
Arizona	264	1451.6	69.6	257	0.0	0.8	1.9	7.6
Arkansas	329	108.4	55.1	441	15.8	24.9	35.6	53.5
California	9,327	702.0	73.9	253	2.4	4.8	7.7	13.7
Colorado	343	689.0	69.3	268	1.2	3.5	7.9	14.3
Connecticut	807	92.6	67.5	285	3.6	6.7	11.5	20.0
Delaware	251	116.2	68.3	320	2.4	4.4	8.8	20.3
Florida	222	755.6	69.0	333	8.6	18.5	27.0	50.9
Georgia	1,134	134.7	61.2	422	6.7	17.8	31.7	51.8
Idaho	1,718	684.9	75.5	255	1.7	3.8	8.1	14.7
Illinois	4,427	86.0	69.5	294	3.0	7.4	13.9	26.7
Indiana	3,621	84.8	69.1	306	4.6	9.6	15.6	28.6
Iowa	8,918	91.9	71.0	304	4.2	9.6	16.3	29.3
Kansas	2,021	95.7	66.6	360	6.8	14.4	22.8	37.9
Kentucky	1,721	79.8	63.3	354	6.0	14.3	23.8	39.7
Louisiana	448	106.2	51.2	446	13.6	29.0	42.4	60.7
Maine	1,208	72.3	63.7	267	3.1	6.5	12.7	21.5
Maryland	3,616	80.3	66.3	284	3.3	7.4	12.3	22.2
Massachusetts	831	79.2	68.2	276	2.0	5.2	9.7	17.9
Michigan	7,678	151.4	78.1	247	2.3	4.8	8.6	16.5
Minnesota	25,131	78.6	69.9	320	4.7	10.3	18.1	31.3
Mississippi	336	162.6	64.9	337	2.4	10.4	18.2	41.1
Missouri	3,399	65.5	58.3	356	6.9	13.7	21.8	36.5
Montana	400	115.9	75.2	200	0.0	0.5	2.0	6.3
Nebraska	1,591	127.0	68.1	331	6.2	12.5	21.5	36.0
Nevada	116	540.5	78.3	306	7.8	7.8	11.2	12.9
N Hampshire	873	84.5	69.9	245	1.8	4.2	8.4	17.1
New Jersey	565	63.8	65.8	344	4.4	10.3	18.6	33.3
New Mexico	255	1391.4	70.8	289	5.5	7.1	12.9	19.2
New York	20,265	112.4	71.2	258	2.4	5.8	11.0	20.5

State	Herd test days <sup>1</sup>	Cows per herd	Avg daily milk yield	Average SCC	% Herd test days <sup>3</sup> with SCC greater than			
	(No.)	(No.)	(Pounds)	(Cells/ml X 1000)	750,000 cells/ml	600,000 cells/ml	500,000 cells/ml	400,000 cells/ml
N Carolina	1,644	125.8	68.2	324	2.0	6.1	12.9	26.9
North Dakota	384	88.2	69.3	320	2.1	4.2	10.7	22.4
Ohio	8,534	89.1	68.8	317	3.6	8.1	14.9	26.7
Oklahoma	582	123.6	57.9	343	6.7	15.1	27.5	44.3
Oregon	2,255	154.4	67.6	228	2.8	4.6	7.6	12.0
Pennsylvania	42,727	59.4	69.4	296	3.0	7.2	13.2	24.1
Puerto Rico	956	110.5	36.1	499	18.5	30.3	45.5	63.4
Rhode Island	41	73.0	62.6	160	0.0	0.0	0.0	9.8
S Carolina	538	161.2	62.8	355	2.0	5.2	12.3	33.1
South Dakota	1,372	154.3	71.2	288	5.4	12.7	22.2	36.2
Tennessee	1,525	86.7	59.6	418	5.2	14.8	27.6	49.6
Texas	1,563	396.4	61.5	318	2.8	6.5	12.5	26.2
Utah	1,397	163.8	69.0	242	2.6	4.8	8.3	16.7
Vermont	3,478	101.0	67.6	230	1.5	3.5	6.7	13.6
Virginia	4,034	107.3	68.5	309	2.0	5.6	10.9	23.8
Washington	1,842	240.5	74.4	237	2.0	2.9	4.6	8.7
West Virginia	409	84.8	60.1	324	3.7	8.3	18.8	33.0
Wisconsin	52,264	80.9	74.5	258	3.3	6.7	11.2	19.8
Wyoming	28	162.5	70.9	320	0.0	0.0	0.0	7.1
<b>United States</b>	<b>227,626</b>	<b>125.1</b>	<b>71.4</b>	<b>276</b>	<b>3.5</b>	<b>7.6</b>	<b>13.4</b>	<b>24.0</b>

<sup>1</sup>All herd test days with usable records. This includes records missing sire identification but having acceptable information in other field.

<sup>2</sup>Cows with usable records (less than total cows on test).

<sup>3</sup>Herd test days with  $\geq 10$  usable records.

From Miller et al. (2008).

24% of the national dairy herd had > 400,000 SCC/ml. Variation among States was large. State average SCC's were often lower than the national average in the Northeast, Upper Midwest, and the far West and higher in the Southeast, Mid-Atlantic and Central states; a finding consistent with previous reports. The Southern Region had the poorest quality milk of all regions of the United States; an average of 37% higher than the national average. In 2007, six states had average SCC's > 400,000/ml, and all were in the Southern Region (Table 1).

The SCC of milk produced by dairy farms in the Southern Region over the last 10 years is presented in Table 2. The average SCC during this period was about 35% higher in the Southern Region than the U.S. average with a yearly range of approximately 30% higher in 2000 to almost 41% higher than the U. S. average in 2003. Texas and Virginia consistently had the lowest annual average SCC and Oklahoma, N. Carolina, Kentucky, and S. Carolina had average SCC's

< 400,000. On the other hand, Florida, Louisiana, Tennessee, Alabama, Puerto Rico, Arkansas, Mississippi, and Georgia had the highest average annual SCC from 1998 – 2007 that were generally > 400,000/ml and sometimes in excess of 500,000/ml. Data in Table 2 also demonstrate that dairy producers in many states in the Southern Region are making progress towards lowering SCC's. For example, the average SCC decreased substantially in Tennessee over the last two years from 504,000/ml in 2005 to 418,000/ml in 2007. This coincides with when the Tennessee Quality Milk Initiative, a science-based comprehensive program to enhance milk quality and thus improve the profitability and sustainability of dairy farms in Tennessee via an educational, research and outreach approach, was launched. However, data in Table 2 also demonstrate quite clearly that there is much room for continued improvement as a high proportion of herds in the Southeast still have cell counts in the 400,000 to 600,000 range.

**Table 2.** DHI SCC in Southern Region from 1998 - 2007.

State	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	Avg.
Alabama	420	427	441	444	485	517	455	433	432	407	446
Arkansas	410	448	427	486	436	387	404	448	457	441	434
Florida	508	533	504	548	529	633	475	473	319	333	486
Georgia	429	411	409	407	432	479	418	433	428	422	427
Kentucky	405	376	370	413	412	419	383	392	395	354	392
Louisiana	455	454	476	479	525	498	449	416	456	446	465
Mississippi	450	456	448	442	498	480	425	386	368	337	429
N. Carolina	377	366	370	364	371	414	365	358	355	324	366
Oklahoma	392	387	396	483	403	356	357	363	333	343	381
Puerto Rico	408	423	475	412	471	441	459	429	443	499	446
S. Carolina	423	389	379	404	389	448	390	387	383	355	395
Tennessee	501	446	420	413	463	476	469	504	463	418	457
Texas	297	288	294	342	316	364	308	346	282	318	316
Virginia	355	329	338	333	330	374	336	320	331	309	336
<b>SE avg</b>	<b>416</b>	<b>410</b>	<b>411</b>	<b>426</b>	<b>433</b>	<b>449</b>	<b>407</b>	<b>406</b>	<b>389</b>	<b>379</b>	<b>413</b>
<b>U.S. avg</b>	<b>318</b>	<b>311</b>	<b>316</b>	<b>322</b>	<b>313</b>	<b>319</b>	<b>295</b>	<b>296</b>	<b>288</b>	<b>276</b>	<b>305</b>
<b>% difference</b>	<b>30.8</b>	<b>31.7</b>	<b>29.9</b>	<b>32.3</b>	<b>38.3</b>	<b>40.8</b>	<b>38.0</b>	<b>37.3</b>	<b>35.0</b>	<b>37.3</b>	<b>35.4</b>

Adapted from USDA/ARS Animal Improvement Program Laboratory reports on Somatic Cell Counts of Milk from DHI Herds published from 1998 – 2007. Information from all states can be found at <http://aipl.arsusda.gov/publish/dhi/scc.html>.

Another important milk quality issue relates to human health. Opponents claim there is no human health risk associated with high bulk tank SCC milk, therefore the SCC limit in the PMO should not be lowered. However, milk with a high SCC is associated with a higher incidence of antibiotic residues in milk (Ruegg, 2005), and the presence of pathogenic organisms and toxins in milk (Oliver et al., 2005b). Last, but certainly not least, is the fact that poor quality milk is an inferior product with reduced processing properties resulting in dairy products with a reduced shelf-life (Barbano et al., 2006; Ma et al., 2000). Thus, milk with a high SCC is associated with indirect health risks to the consumer and is an inferior quality product. Good quality milk lasts longer, tastes better, and is more nutritious. These issues are the basis for animal health advocacy groups to lower the SCC regulatory limit.

A mandated reduction in the number of somatic cells in milk via regulatory intervention may not be necessary because in the near future milk buyers may only purchase milk of excellent quality. Recently, some dairy processing plants have made changes to their milk quality requirements for

incoming raw milk. These changes have occurred, in part, by demands from retailers and major food service companies for milk with a higher quality with a longer shelf-life. Eventually, changes in SCC limits and perhaps even requirements for raw milk to be free of specific bacteria could be implemented. Thus, SCC limits for raw milk to be acceptable at dairy processing plants may decrease to levels much lower than they are now, making it increasingly problematic for dairy producers to meet these higher standards. Production of better quality milk will place a much greater emphasis on strategies for the prevention and control of mastitis to reduce the number of somatic cells in milk.

**What is Mastitis?** Mastitis, an inflammation of the mammary gland caused by bacterial infection, trauma, or injury to the udder, remains the most common and most expensive disease affecting dairy cattle throughout the world. Mastitis is caused by several different bacteria that can invade the udder, multiply there and produce harmful substances that result in inflammation. Mastitis reduces milk yield and alters milk composition. The magnitude of reduced milk yield and alterations in milk composition is influenced by the severity of the inflammatory response, which in turn is influenced by the mastitis pathogen causing the infection (Oliver and Calvino, 1995). Clinical mastitis is characterized by abnormal milk and/or visible abnormalities of the udder such as hot and swollen udders. However, subclinical mastitis (often referred to as hidden mastitis), the most common form of mastitis, is not readily apparent because there are no visible signs of the disease.

Cows with clinical mastitis have more dramatic changes in milk yield and composition than cows with subclinical mastitis. Results of studies published thus far support the contention that alterations in milk composition associated with mastitis are due to several factors including impaired milk synthesis and secretion, mammary epithelial cell death and degeneration, and transport of substances from blood to milk and from milk to blood. The most notable changes in milk composition associated with mastitis are decreased concentrations of fat, lactose, casein and calcium; and increased concentrations of albumin, sodium and chloride. Concentrations of lipases, proteases, oxidases, plasmin and plasminogen increase, which may adversely influence milk stability, milk flavor, and processed dairy products. In addition, factors not normally found in milk such as inflammatory mediators and bacterial enterotoxins and endotoxins have been detected in milk from cows with mastitis. From a dairy manufacturing perspective, mastitis decreases concentrations of desirable components and increases concentrations of undesirable components all of which influence milk shelf-life and taste.

The measurement used most commonly to detect subclinical mastitis is the SCC of milk. One characteristic feature of mammary gland inflammation is an elevation in the number of somatic cells in milk. Milk from uninfected mammary glands contains <100,000 somatic cells per milliliter. A milk SCC >200,000/ml suggests that an inflammatory response has been elicited, that a mammary quarter is infected or is recovering from an infection, and is a clear indication that milk has reduced manufacturing properties. Thus, an increase in the SCC of milk is a good indicator of inflammation in the udder. Infection of the udder by mastitis pathogens alters milk composition and reduces milk yield. Most studies that evaluated the influence of mastitis on the composition of milk used SCC as the basis for determining the infection status of udders and for determining the degree of inflammation.

The bulk tank SCC (BTSCC) has been used to gauge the udder infection status of a dairy herd, and also gives a good indication of the loss in milk production in a herd due to mastitis. As the BTSCC increases, the percent of mammary quarters infected increases and the percent production loss increases. Similarly, as the percent of cows with a SCC >800,000 increases, rolling herd production decreases. Small increases in SCC can impact production. Most herd milk contains between 200,000 to 500,000 somatic cells/ml of milk. These herds are losing at least 8% in potential milk production. Thus, methods of mastitis control that reduce SCC will improve milk yield and composition and decrease economic losses due to mastitis.

**Prevention & Control of Mastitis:** Mastitis is a difficult disease to control because many different bacteria are capable of infecting the udder and producing the disease. Microorganisms that most frequently cause mastitis can be divided into two broad categories: contagious pathogens, which are spread from cow to cow primarily during the milking process; and environmental pathogens, which are found throughout the habitat of dairy cows.

**Contagious Mastitis Pathogens:** Contagious mastitis is caused primarily by *Staphylococcus aureus* and *Streptococcus agalactiae*. *Mycoplasma bovis* and other *Mycoplasma* species have been increasingly reported as important contagious mastitis pathogens. The primary source of these organisms is the udder of infected cows. Contagious mastitis pathogens spread from infected cows to uninfected cows primarily at milking. Some characteristics of herds with a contagious mastitis problem include: (1) a high prevalence of intramammary infection (IMI) during lactation, (2) a high BTSCC, (3) infections of long duration, (4) low proportion of infections result in clinical mastitis (infections mostly subclinical), and (5) a low prevalence of infection during the dry period.

**Environmental Mastitis Pathogens:** Environmental mastitis is caused primarily by environmental streptococci including *Streptococcus uberis*, *Streptococcus dysgalactiae* subsp *dysgalactiae*, and coliforms including *Escherichia coli* and *Klebsiella* species. The primary source of environmental mastitis pathogens is the environment of the cow. Infections generally occur between milkings and during the milking process. Some characteristics of herds with an environmental mastitis problem include: (1) a low prevalence of IMI during lactation, (2) a low BTSCC, (3) infections of short duration, (4) many IMI result in clinical mastitis, and (5) a high prevalence of infection during the dry period.

**Proven Methods of Mastitis Control:** The NMC recommended mastitis control program (NMC, 2006a) is as follows:

### **1. Establishment of Goals for Udder Health**

- ✓ Set realistic herd targets for average SCC or linear score and clinical mastitis rate.
- ✓ Review goals on a timely basis, with input from the Herd Udder Health Advisory Team (veterinarian, producer, herdsman, milking personnel and advisors).
- ✓ Prioritize management changes to achieve stated goals.

### **2. Maintenance of a Clean, Dry, Comfortable Environment**

- ✓ Ensure proper stall usage by assessing adequacy of stall size and design.
- ✓ Ensure proper stocking density in facilities.
- ✓ Maintain clean, dry, and comfortable stalls through appropriate bedding management.
- ✓ Keep cow lots and traffic areas clean and dry.
- ✓ Ensure ventilation system is functioning properly.
- ✓ Control detrimental environmental influences (i.e. heat stress, frostbite, stray voltage, etc.).
- ✓ Ensure that cows remain standing after milking (i.e. provide fresh feed and water).

### **3. Proper Milking Procedures**

- ✓ Wear clean gloves during the milking process to limit spread of contagious pathogens.
- ✓ Apply pre-milking teat disinfectant that completely covers the teat skin and allow it to remain on teats for at least 30 seconds.
- ✓ Examine foremilk to facilitate early detection of clinical mastitis and proper milk letdown.
- ✓ Dry teats using a properly washed and disinfected cloth towel for use on one cow, or a single service paper towel.
- ✓ Attach teat cups squarely and level with the udder within 90 seconds of udder preparation.

- ✓ Adjust cluster during milking to prevent liner slips and squawks.
- ✓ With manual removal, avoid machine stripping and shut off vacuum to the claw before removing cluster.
- ✓ Apply teat disinfectant immediately following teat cup removal, and assure complete coverage of teats.
- ✓ To optimize mastitis control and reduce costs, teat dipping is preferred to spraying for method of disinfectant application.
- ✓ Pre- and post-milking teat disinfectants should be selected based on documented efficacy data which can be found on the NMC website (<http://www.nmconline.org/info.htm>).
- ✓ Milk cows with confirmed contagious intramammary infections last.

#### **4. Proper Maintenance & Use of Milking Equipment**

- ✓ Service, maintain, and regularly evaluate equipment function according to manufacturer's guidelines, using dynamic evaluation methods and an appropriate record form.
- ✓ Ensure milking system is adequately sized to handle milk and air flow, according to NMC Airflow Guidelines.
- ✓ Replace inflations and other rubber and plastic parts regularly, according to manufacturer's guidelines.
- ✓ Replace broken or cracked inflations and short milk tubes immediately.
- ✓ Sanitize equipment prior to each milking and thoroughly wash and sanitize equipment after each milking.

#### **5. Good Record Keeping**

- ✓ For each case of clinical mastitis, record cow identification, date detected, days in milk, quarter(s) infected, number and type of treatments, outcome of treatments (i.e. return to normal milk, time to discard milk) and the causative bacterial pathogen if a sample was cultured on-farm or in a laboratory.
- ✓ Use a computerized or manual record system to manage information, such as individual cow SCC data, on the prevalence and incidence of subclinical mastitis.
- ✓ Keep all maintenance and purchase records for 5 years.

#### **6. Appropriate Management of Clinical Mastitis During Lactation**

- ✓ Develop and implement a herd clinical mastitis treatment protocol with the Herd Udder Health Advisory team.
- ✓ Carefully consider the economic ramifications of therapy decisions.
- ✓ Collect a pre-treatment milk sample aseptically for microbiological culture.
- ✓ Clearly identify all treated cows and record all treatments in a permanent record
- ✓ Prior to infusion, disinfect the teat with a germicide and scrub the teat-end with an alcohol swab.
- ✓ Use an appropriate therapeutic regimen; use drugs according to the protocol, or as recommended by the health advisors.
- ✓ For infusion of intramammary antibiotics, use a single-dose, regulatory approved product by the partial insertion method.
- ✓ Observe the correct withdrawal period for the antibiotic used, as stated on the label. If extra-label drug use is necessary, follow regulatory guidelines under the supervision of a veterinarian (e.g. in the systemic treatment of coliform mastitis).
- ✓ Do not treat chronic non-responsive infections.
- ✓ Always follow recommended drug storage guidelines and observe expiration dates.

## **7. Effective Dry Cow Management**

- ✓ Decrease the energy density of the ration during late lactation to reduce milk production before dry-off.
- ✓ Dry cows off abruptly and dry treat each quarter immediately following the last milking of lactation.
- ✓ Disinfect teats and scrub the teat-end with an alcohol swab before infusing.
- ✓ Use the partial insertion method of dry treatment infusion.
- ✓ Treat all quarters of all cows with a commercially available approved long-acting dry cow antibiotic.
- ✓ Disinfect teats immediately following infusion with an external or internal teat sealant with any approved post dip.
- ✓ Provide adequate dry cow nutrition to enhance immune system function.
- ✓ Maintain a clean, dry, comfortable environment for dry cows. Dry cow environmental management is important to minimize exposure to pathogens.
- ✓ In situations of high environmental pathogen exposure, use an internal or external teat sealant for dry cows.
- ✓ In herds with coliform mastitis problems, vaccinate with a core antigen endotoxin vaccine following manufacturer's directions.
- ✓ Clip flanks and udders to remove excess body hair. Udder singeing may be useful to ensure hair removal.

## **8. Maintenance of Biosecurity for Contagious Pathogens & Marketing of Chronically Infected Cows**

- ✓ Request bulk tank and individual cow SCC data, and use CMT for decisions prior to purchasing new cows.
- ✓ If possible, obtain aseptically collected milk samples for bacteriological culture from cows prior to purchase.
- ✓ Isolate recently purchased cows, and milk separately, until there is assurance of the absence of intramammary infection.
- ✓ Segregate cows with a persistently high SCC or linear score (e.g. SCC greater than 300,000 or linear score greater than or equal to 5.0 for several months) and observe response to dry treatment or other recommended therapy.
- ✓ Cull or permanently segregate cows persistently infected with *Staphylococcus aureus* or other non-responsive microbial agents (*Mycoplasma*, *Nocardia*, *Pseudomonas*, or *Arcanobacterium pyogenes*).
- ✓ Consider udder health status of first-calf heifers for herd biosecurity.

## **9. Regular Monitoring of Udder Health Status**

- ✓ Enroll in an individual cow SCC program or use some other monitor of subclinical infections.
- ✓ Use CMT as a cow-side monitor of inflammation in cows suspected of infection and in high-risk periods (i.e. early lactation).
- ✓ Monitor distributions of high SCC cows, and rates of change to elevated SCC.
- ✓ Conduct milk bacteriological culture of clinical cases and high SCC cows regularly.
- ✓ Monitor udder health for the herd using reports from the regional regulatory agency or milk marketing organization and DHI.
- ✓ Calculate clinical mastitis rates and distributions on a regular basis.
- ✓ Use SCC and clinical mastitis records to evaluate protocols, and to make treatment and marketing decisions.

## 10. Periodic Review of Mastitis Control Program

- ✓ Obtain objective evaluations from veterinarian, industry field person or extension representative.
- ✓ A step by step approach to the review, and a standard evaluation form are useful.
- ✓ Make use of the entire Herd Udder Health Advisory Team: veterinarian, producer, herdsman, milking personnel, and advisors.

**Mastitis Control Strategies:** Current mastitis control programs are based on hygiene and include teat disinfection, antibiotic therapy and culling of chronically infected cows. Acceptance and application of these measures throughout the world has led to considerable progress in controlling mastitis caused by contagious mastitis pathogens such as *Strep. agalactiae* and *Staph. aureus*. However, as the prevalence of contagious mastitis pathogens was reduced, the proportion of IMI caused by environmental pathogens such as *E. coli* and *Strep. uberis* has increased markedly (Oliver and Mitchell, 1984). Therefore, it is not surprising that mastitis caused by coliforms and environmental *Streptococcus* species has become a major problem in many well-managed dairy farms that have successfully controlled contagious pathogens.

Controlling mastitis is not simply a matter of doing just one thing. Rather, the control of mastitis involves a number of steps that constitute a control program. Mastitis control programs should have the following characteristics: (1) practical, (2) economical, (3) subject to easy modification, and (4) effective under most management conditions. Two different approaches are outlined regarding mastitis management. The first approach is aimed at herds that have a serious problem and where immediate action is necessary. The second more comprehensive approach is the preferred strategy that should be applicable to the majority of dairy herds (Philpot and Nickerson, 1991).

Herds with a high SCC will likely need to adopt a short-term goal of reducing SCC as quickly as possible so that milk can meet standards as set forth in the PMO. This will require extensive use of highly trained personnel and laboratory facilities and consequently is an expensive approach. Some goals would be to confirm the extent of infection, identify bacteria causing mastitis and identification of cows to be treated or culled from the herd. One excellent method of making some of these important decisions is through a SCC program. This is relatively inexpensive and SCC data can be used by dairy producers, veterinarians, extension personnel, and dairy consultants for making educated decisions regarding: (1) cows to be sampled for microbiological culture, (2) cows to be culled, (3) milking order of cows, and (4) cows to be dried off early. Withholding milk from a few cows with high SCC can have a DRAMATIC impact on the BTSCC.

A more comprehensive strategy is preferred for controlling mastitis for the following reasons: (1) it is a more practical approach, (2) advocates adoption of management practices applicable to most herds without knowledge of specific pathogens or prevalence of mastitis in herds, (3) this strategy involves conscientious application of only a few basic practices, and (4) success of this strategy has been well documented. This approach is geared towards reducing the rate of new infection and shortening the duration of existing infections. **The success of this program has been proven repeatedly and documented extensively throughout the world and consists of effective milking hygiene, proper milking machine function, pre- and post-milking teat disinfection, lactation therapy, antibiotic dry cow therapy and culling.**

Contagious mastitis pathogens are controlled effectively by procedures that prevent spread of bacteria at milking time, which include good udder hygiene, and premilking and postmilking teat disinfection with effective teat disinfectants. In the U. S., the general recommendation is that all quarters of all cows be infused with antibiotics approved for use in nonlactating cows after the last milking of lactation to eliminate existing infections and to prevent new infections during the early dry period which is a time that the udder is highly susceptible to new infection. It may be necessary to cull chronically infected cows.

Control of environmental mastitis pathogens is best achieved by maintaining a clean, dry environment for lactating **AND** nonlactating cows. Premilking and postmilking teat disinfection are recommended. Antibiotic dry cow therapy is recommended also. Dry cow therapy helps control new infections during the early dry period caused by environmental streptococci. However, dry cow therapy has little effectiveness in controlling coliforms and is not effective in preventing new infections that occur near calving. Vaccines to reduce the severity and duration of coliform mastitis are available and are useful in herds with environmental mastitis.

**Current Methods of Mastitis Prevention & Control:** Because of the large number of pathogens capable of causing mastitis and the fact that these pathogens behave quite differently, a one size fits all approach to mastitis management is not feasible. Paying attention to the small details described above will continue to be important in every mastitis control program. Since pathogenic bacteria gain entrance into the mammary gland through the teat canal, the greater the bacterial load at the teat end, the greater the probability of an infection occurring thus emphasizing the importance of maintaining a clean dry environment and udder hygiene at milking time. Any procedure that reduces the number of bacteria to which the teat end is exposed will likely be beneficial. Proper milking hygiene and good milking practices consist of the following elements: (1) milk in a clean stress-free environment, (2) check foremilk and udder for signs of clinical mastitis, (3) minimize use of water in the milking parlor, (4) wash teats with warm sanitizing solution, if necessary, (5) apply premilking teat disinfection, (6) dry teats thoroughly 30 to 45 seconds after premilking teat disinfectant application, (7) attach teat cups within one minute after cleaning, (8) provide stable vacuum at claw during peak milk flow, (9) avoid squawking or slipping of teat cup liners during milking, (10) adjust milking units as necessary, (11) shut off vacuum before removing machine, and (12) apply postmilking teat disinfectant shortly after milking machine removal.

**Premilking Teat Disinfection:** Premilking teat disinfection has been adopted by several dairy producers and is intended to combat environmental pathogens that may have been transmitted to the teat at some point after the last milking. Studies have shown that premilking teat disinfection in combination with postmilking teat disinfection was more effective in preventing new infections than postmilking teat disinfection only. Premilking teat disinfection appears to be effective against environmental pathogens and may also influence contagious pathogens (Oliver et al., 1993; 1994). Dairy producers using this mastitis control procedure must make sure that the premilking teat disinfectant is removed from teats before milking to prevent contamination of milk. There are several good teat disinfectants on the market. However, when choosing a teat disinfectant, require the sales representative to provide evidence that the product is safe, effective and registered. Furthermore, make sure that manufacturer's recommendations are followed. Finally, do not assume that all postmilking teat disinfectants would be effective as a premilking teat disinfectant. The NMC publishes a summary of peer-reviewed publications on efficacy of premilking and postmilking teat disinfectants that is updated annually that provides information that may be useful to dairy advisors and producers when making decisions on teat disinfectants (NMC, 2008). This information is available online at [www.nmconline.org](http://www.nmconline.org).

**Postmilking Teat Disinfection:** Postmilking teat disinfection has been shown repeatedly to be an effective technique for preventing new IMI during lactation. This procedure destroys mastitis pathogens on teats after milking. In general, effective postmilking teat disinfectants reduce the rate of new infection by 50% or more when used in conjunction with other components of mastitis control. This has certainly been the case in studies conducted at The University of Tennessee (Oliver et al. 1989; 1990b, 1999). Postmilking teat disinfection has been adopted widely in major milk-producing countries throughout the world as an essential part of mastitis control programs. However, postmilking teat disinfection is generally not as effective in preventing new IMI by environmental pathogens such as coliforms and *Strep. uberis*. This may be due to decreased germicidal activity in the period between milkings. For this reason, efforts have been made to examine premilking teat disinfection and to develop barrier-type teat dips to prevent new IMI by environmental pathogens during the intermilking interval.

**Barrier Teat Dips:** Barrier-type teat dips were developed with the goal of reducing exposure of teat ends to environmental pathogens during the intermilking period. Barrier dips are generally more viscous. However, their efficacy for prevention of environmental mastitis pathogens is equivocal. The incidence of new IMI actually increased with some barrier-type teat disinfectants when evaluated under conditions of experimental challenge with *Strep. agalactiae* and *Staph. aureus* (Nickerson and Boddie, 1995).

Persistent barrier-type dips have also been used to prevent mastitis during the early dry period and near calving when cows are at high risk for new IMI (Timms, 2000). One problem has been persistence of the barrier on teat ends.

**Antibiotic Therapy of Clinical Mastitis:** Despite mastitis control measures such as pre- and postmilking teat disinfection and good milking time hygiene, mastitis does occur and often requires antibiotic treatment. Antibiotic therapy of clinical mastitis involves: (1) detection of the infected quarter, (2) prompt initiation of treatment, (3) administration of the full series of recommended treatments, (4) maintaining a set of treatment records, (5) identification of treated cows, and (6) making sure the milk is free of antibiotic residues before adding to the bulk tank.

There has been and continues to be concern over the low efficacy of antibiotic mastitis therapy against certain mastitis pathogens. This is due to bacterial factors, pharmacologic and pharmacokinetic limitations, and pathobiologic circumstances of the infected mammary gland. Many of these factors appear to be beyond human manipulation for improved therapeutic efficacy, but there are some areas where work could be done to enhance selection of appropriate antibiotics for therapy.

Efficacy of mastitis therapy is extremely low for chronic *Staph. aureus* infections;  $\beta$ -lactamase production may be partly responsible for the low cure rate. However, even with antibiotics to which the bacteria were sensitive in vitro, the cure rate was still low (Owens et al., 1997). This suggests the presence of some other mechanisms that interfere with therapy such as formation of microabscesses in mammary tissues and internalization into phagocytic and epithelial cells (Almeida et al., 1996). Most antibiotics used in mastitis therapy do not penetrate into the infected area and have poor intracellular penetration. Pirlimycin has been studied extensively to treat cows with chronic *Staph. aureus* IMI because of its lower minimum inhibitory concentration and its tissue-penetrating property. Extended therapy with pirlimycin greatly improved the cure rate against chronic *Staph. aureus* IMI during lactation (Belschner et al., 1996; Deluyker et al., 2001). Results from our laboratory have shown that extended therapy with pirlimycin is an effective procedure for treatment of chronic environmental *Streptococcus* species (*Strep. uberis* and *Strep. dysgalactiae*) IMI in lactating dairy cows (Gillespie et al., 2000). We have also had much success with extended therapy using ceftiofur hydrochloride for treatment of cows with naturally-occurring subclinical mastitis and experimentally induced clinical *Strep. uberis* mastitis (Oliver et al., 2004a, 2004b).

It is still controversial whether to treat or not treat cows with coliform mastitis. Clinical signs of coliform mastitis are mainly due to the effects from endotoxin. There are few antibiotics suitable for treating cows with coliform mastitis, however, ceftiofur hydrochloride has good in vitro activity against a wide variety of Gram-negative mastitis pathogens, and could prove useful for intramammary treatment of cows with clinical mastitis due to Gram-negative mastitis pathogens.

When treating cows with clinical or subclinical mastitis, dairy producers must recognize that administration of antibiotics in a manner inconsistent with the label instruction is considered extra-label use, and MUST be carried out under the supervision of the herd veterinarian. Furthermore, milk and meat for human consumption from antibiotic-treated cows must be free of drug residues.

**Dry Cow Antibiotic Therapy:** The importance of the dry period in the control of mastitis in dairy cows has been recognized for more than 50 years. A classic study by Neave et al. (1950) demonstrated that udders were markedly susceptible to new IMI during the early dry period. The rate of new infection during the first 21 days of the dry period was over 6 times higher than the rate observed during the previous lactation. Studies have also shown that udders are highly susceptible to new IMI near calving (Smith et al., 1985a; 1985b; Oliver 1988a; 1988b; Oliver and Mitchell, 1983; Oliver and Sordillo, 1988). Increased susceptibility to new IMI is likely associated with physiological transitions of the mammary gland either from or to a state of active milk production. Many IMI that occur at this time persist throughout the dry period and are often associated with clinical mastitis after calving. Thus, the early dry period was identified as an extremely important time for the control of mastitis in dairy cows.

Since the early work by Neave et al. (1950), procedures were developed to control infections during the dry period. Most dairy advisors recommend that all quarters of all cows be infused with antibiotics approved for use in dry cows following the last milking of lactation. The objectives of dry cow therapy are twofold: (1) to eliminate infections present during late lactation, and (2) to prevent new infections during the early dry period when mammary glands are highly susceptible to new IMI.

Antibiotic therapy at drying off plays an important role in the control of mastitis during the dry period. Dry cow therapy is particularly effective against streptococci and to a lesser extent against *Staph. aureus*. Smith et al. (1985a, 1985b) demonstrated that antibiotic therapy at drying off reduced the rate of new environmental streptococcal infection during the early dry period only and that the rate of new coliform IMI was not affected at all. Thus, two significant limitations of present antibiotic formulations used for dry cow therapy are: 1) ineffectiveness against coliform bacteria, which can cause a high proportion of IMI during the early dry period and near calving, and 2) ineffectiveness in preventing new IMI by a broad spectrum of mastitis pathogens during the period near calving when mammary glands are highly susceptible to new infection (Oliver, 1988a; 1988b; Oliver and Sordillo, 1988; 1989).

Dry cow antibiotic preparations are formulated primarily to maintain persistent activity during the early dry period and most likely provide little protection during the late dry period. Oliver et al. (1990a), using the *Bacillus stearothermophilus* disc assay to detect antibiotic residues, demonstrated that dry cow antibiotics persisted for only 14 to 28 days after infusion, and some persisted for shorter periods. Thus, based upon present methods of formulation, it would appear that antibiotic preparations currently available for use in dry cows will not control IMI that occur during the late dry period based on a dry period length of 6 to 8 weeks.

Experimental evidence suggests that dry cow therapy is effective in controlling IMI due to *Strep. agalactiae* and somewhat effective against *Staph. aureus*. However, dry cow therapy appears to be less effective against streptococci other than *Strep. agalactiae* and ineffective against coliform bacteria (Smith et al., 1985a; 1985b). Differences in effectiveness of dry cow antibiotic therapy to prevent new IMI are most likely related to several factors. *Strep. agalactiae* and *Staph. aureus* are thought to be transmitted primarily during the milking process, and transmission can be controlled by hygiene and antibiotic therapy. The sources of these two organisms are infected mammary glands, colonized teat ducts, and teat lesions. Extramammary sources of contagious mastitis pathogens have been identified but appear to be relatively unimportant in the pathogenesis of infection. Thus, exposure of mammary glands to contagious pathogens during the dry period is reduced in the absence of regular milking and therapy at drying off tends to control these pathogens effectively.

**Heifer Mastitis:** Mastitis in breeding age and pregnant heifers is much higher than previously thought. A review on this topic was published recently (Oliver et al., 2005a). Many IMI in heifers can persist for long periods of time, are associated with elevated somatic cell counts (SCC), and may impair mammary development during gestation and affect milk production after calving. Presence of mastitis before calving increased the risk of infection during lactation, increased the

risk of clinical mastitis in the first week after calving, and increased the risk of further cases of mastitis and culling during the first 45 days of lactation.

Mastitis in heifers can be a significant problem for dairy producers. Prepartum intramammary antibiotic infusion of heifer mammary glands was shown to be an effective procedure for eliminating many IMI in heifers during late gestation and for reducing the prevalence of mastitis in heifers both during early lactation and throughout lactation (Oliver et al., 2005a). Data are equivocal regarding the influence of antibiotic treatment of heifers before or near calving on milk production in the subsequent lactation. Some studies reported that prepartum antibiotic-treated heifers produced significantly more milk than control heifers (Owens et al. 1991; Oliver et al. 2005a; Sampimon and Sol, 2005). Conversely, other studies have shown that antibiotic treatment of heifers before or near calving reduced IMI but did not increase milk production or lower SCC in the subsequent lactation (Borm et al. 2005; Middleton et al. 2005). Reasons for this are unclear and need to be delineated. One potential explanation for differences or lack thereof in milk production following prepartum antibiotic therapy could be due to the prevalence of infection in the herds evaluated. In support of this contention, Sampimon and Sol (2005) indicated that prepartum antibiotic treatment of heifers was beneficial on high prevalence farms but not on low prevalence farms. This study was conducted in 13 Dutch dairy farms where 196 heifers were treated with cloxacillin 8 to 10 weeks before expected calving and another 196 heifers served as untreated controls. Farms with <15% of heifers with a cow SCC >150,000 cells/ml at the start of the trial were considered low prevalence (LP) while farms with >15% were considered as high prevalence farms (HP). Expected 305-day milk production was significantly higher (496 L) in antibiotic-treated heifers from HP farms in comparison with untreated animals but this difference was only 77 L (not significant) in heifers from LP farms. In both groups of farms, cow SCC was significantly lower in antibiotic-treated heifers compared to untreated controls. An IMI had a significant influence on milk production and cow SCC in the treated and also in the untreated group in comparison to animals without an IMI. Authors concluded that treatment of heifers is beneficial on HP but not on LP farms. Thus, treatment of heifers in a high prevalence herd may be more advantageous from a milk production perspective than in lower prevalence herds. However, high and low prevalence herds still need to be defined.

While much has been learned about mastitis in heifers, many issues remain unanswered such as: (1) identification of herds where this strategy would be most advantageous and cost effective, (2) should all heifers in the herd be treated or only certain heifers? (3) are there certain bacteria that are more problematic than others? and (4) identification of key risk factors that could have a significant impact on prevention of heifer mastitis so that antibiotic treatment could be minimized. Additional studies are needed to address these fundamentally important questions.

Use of antibiotics in heifers and cows at times when udders are infected or most susceptible to new IMI is a sound management decision and a prudent use of antibiotics on the farm. Strategies involving prudent use of antibiotics encompass identification of the pathogen causing the infection, determining the susceptibility/resistance of the pathogen to determine the most appropriate antibiotic to use for treatment, and a long enough treatment duration to ensure effective concentrations of the antibiotic to eliminate the pathogen. It is clear that the goal of mastitis therapy should be to eliminate the pathogen causing the infection. Currently, many dairy producers evaluate treatment success based on return of milk and/or the udder to normal. If pathogens causing IMI are eliminated, the opportunity for that pathogen to develop antimicrobial resistance is eliminated.

**Internal Teat Sealants:** Use of internal teat sealants is a relatively new concept and much of the early data came from studies conducted in New Zealand (Woolford et al., 1998). Results of those studies showed that internal teat sealants were effective in preventing new IMI during the dry period. A total of 528 cows in late lactation with SCC <200,000 cells/ml were identified in three commercial herds. Of these, bacteriological examination showed 482 cows were uninfected in all four quarters and 46 were infected in only one quarter. At drying off, uninfected quarters were allocated randomly to the following treatments: no infusion (negative controls), infusion with a

bismuth subnitrate based teat sealer, infusion with teat sealer plus antibiotic, or infusion with a cephalonium-based dry cow antibiotic (positive control). New infections were identified during the dry period by periodic udder palpations and at calving by bacteriological culture. All three treatments reduced the incidence of new IMI due to *Strep. uberis*, both during the dry period and at calving, by about 90%. The majority of infections were due to *Strep. uberis*. For all treatments, a 50% lower incidence of clinical mastitis over the first 5 months of the ensuing lactation was reported by farmers. X-ray imaging of 19 teats showed that the teat sealer material was retained, at least in part, in the lower teat sinus over about 100 days of the dry period. The internal teat sealant was as effective in reducing new dry period infections as the infusion of a long-acting dry cow antibiotic formulation. The lower incidence of new infections in the ensuing lactation among the infused quarters implies that fewer subclinical infections persisted from the dry period. Use of teat sealers at drying off appears to offer the same prophylactic efficacy as the dry cow antibiotic approach. Other studies reported similar results (Huxley et al., 2002; Godden et al., 2003). Internal teat sealants are apparently quite popular in organic dairy herds.

However, internal teat sealants do not contain antimicrobials and therefore will not eliminate IMI that are present during late lactation. The internal teat sealant in combination with antibiotics would be necessary if cows are infected during late lactation. There have also been some problems reported about sealant residues in milk following calving which apparently can impact cheese production.

**Advances in Mastitis Vaccine Research:** Given today's public health and food safety concerns regarding antimicrobial resistance and antibiotic residues in dairy products associated with treatment of diseases like mastitis, approaches to enhance the cow's immunity to prevent disease and thus minimize use of antibiotics has gained considerable attention. Yet, for a variety of reasons, vaccines developed for the prevention and control of mastitis have achieved only limited success. The multiplicity of pathogens capable of causing mastitis, and knowledge of mammary gland immunology, bacterial virulence factors, and mechanisms of pathogenesis are factors that have hindered development of effective mastitis vaccines. However, some progress has been made in these areas in the last decade or so.

**Staphylococcus aureus:** Most of the early vaccine research focused on *Staph. aureus* and vaccines were based on bacterins derived from in vitro grown bacteria. As our knowledge of bacterial virulence factors increased, different approaches to vaccine formulation have been attempted. Watson et al. (1996) developed a *Staph. aureus* mastitis vaccine consisting of killed bacteria bearing pseudocapsule and toxoided exotoxins. A large field trial involving 1819 cows and heifers from 7 dairy herds was conducted. The vaccine was administered to pregnant heifers twice during the last trimester of pregnancy and to cows at the end of lactation and again 4 to 6 weeks later. Differences in the incidence of clinical mastitis and prevalence of subclinical mastitis between vaccinated and controls animals were not significant for the whole population of cows and heifers. However, the vaccine was efficacious in reducing the incidence of clinical mastitis and prevalence of subclinical mastitis in one herd that had a serious staphylococcal mastitis problem. Nordhaug et al. (1994) tested a vaccine containing whole-inactivated *Staph. aureus* with pseudocapsule, and  $\alpha$ - and  $\beta$ -toxoids in heifers. Results of that study showed a potential protective effect on general udder health of this vaccine during the entire first lactation period. Nickerson et al. (2000) suggested a positive effect of vaccination with a polyvalent *Staph. aureus* vaccine by increasing antistaphylococcal antibody titers and in preventing new *Staph. aureus* infections when the program was initiated at an early age in heifers from a herd with a high exposure to *Staph. aureus*. More recently, Nickerson et al. (2008) reported that the percentage of heifers with *S. aureus* IMI at calving was significantly lower in heifers vaccinated with a commercially available vaccine containing a lysed culture of polyvalent *S. aureus* somatic antigens containing 5 phage types than in unvaccinated heifers. SCC's were also lower in vaccinated heifers during the first week of lactation.

At USDA, Guidry and O'Brien randomly sampled the national herd and found three *Staph. aureus* capsule serotypes were responsible for 100% of bovine *Staph. aureus* mastitis in the U.S. They

formulated a vaccine, using the 3 serotypes, and tested its ability to cure chronic *Staph. aureus* infections. In preliminary field trials, the trivalent *Staph. aureus* vaccine with antibiotics was as effective as the autogenous vaccine with antibiotics for curing chronic *Staph. aureus* infections (Sears et al., 2000). This would allow for treatment of cows chronically infected with *Staph. aureus* without the necessity of preparing a herd-specific vaccine. Further testing is being conducted to determine the effect of duration of infection on cure rate.

***Escherichia coli*:** An interest in vaccines against environmental mastitis pathogens has been growing. Results obtained with bacterins prepared from the J5 mutant strain of *E. coli* (O111:B4), referred to as *E. coli* J5 vaccine, have been encouraging. This mutant is an epimerase-negative strain in which a terminal sugar is absent from the lipopolysaccharide moiety of the cell wall and the lipid A determinant is thus exposed. Trials in California showed that the J5 vaccine reduced clinical coliform mastitis by up to 80% during the first 100 days of lactation (Gonzalez et al., 1989). Hogan et al. (1992a) reported that *E. coli* J5 vaccine did not prevent IMI but did reduce severity of clinical symptoms following experimental challenge with a heterologous *E. coli* strain. In a field trial, Hogan et al. (1992b) reported that percentage of quarters infected at calving with Gram-negative bacteria did not differ between vaccinated and control cows. However, the vaccine reduced incidence of clinical mastitis; 67% of Gram-negative infections detected at calving in control cows resulted in clinical mastitis during the first 90 days of lactation compared with 20% in vaccinated cows. These data indicate that this vaccine does not prevent new Gram-negative IMI, but does reduce the severity of the disease.

***Streptococcus uberis*:** *Streptococcus uberis* is an important cause of mastitis in dairy cows, particularly during the dry period, the period around calving, and during early lactation that is not controlled effectively by current mastitis control practices (Jayarao et al., 1999; Oliver et al., 1998). Many *Strep. uberis* IMI that originate during the nonlactating period and near calving result in clinical and subclinical mastitis during early lactation. Control programs for reducing *Strep. uberis* IMI should focus on periods adjacent to the nonlactating period where opportunities exist to develop strategies to reduce the impact of *Strep. uberis* infections in the dairy herd (Oliver et al., 1998).

Research from our laboratory has focused extensively on development of in vivo and in vitro models to study host-pathogen interactions, and on identification and characterization of virulence factors associated with the pathogenesis of *Strep. uberis* mastitis and other environmental streptococci. Use of molecular biology tools such as proteomics, genomics and bioinformatics has led to the discovery of a novel protein produced by *Strep. uberis* referred to as *Streptococcus uberis* Adhesion Molecule or SUAM (Almeida et al., 2006; Luther et al., 2008). The SUAM DNA sequence was deposited in GenBank in 2005 (Luther, D. A., R. A. Almeida, H. M. Park, M. J. Lewis, M. E. Prado, and S. P. Oliver. 2005. *Streptococcus uberis* adhesion gene, *sua*, encoding *Streptococcus uberis* adhesion molecule. GenBank Accession Number DQ232760). ***Our hypothesis is that SUAM plays a critical role in the pathogenesis of streptococcal mastitis by facilitating bacterial adherence to bovine mammary epithelial cells. Streptococcus uberis expresses SUAM and uses a protein found in cows' milk and/or on the epithelial cell surface to adhere to mammary epithelial cells.*** Results of studies thus far demonstrate that SUAM plays a critical role in the pathogenesis of streptococcal mastitis and appears to be a promising vaccine candidate for the prevention of mastitis in dairy cows (Almeida et al., 2006; Prado et al., 2008). Vaccination of dairy cows at drying off, during the mid-dry period and near calving with recombinant SUAM (rSUAM) increased antibody titers in serum at times when the udder is highly susceptible to mastitis. Serum antibodies to rSUAM blocked adherence to and internalization of the homologous and heterologous strain of *Strep. uberis* into bovine mammary epithelial cells. Anti-SUAM antibodies were also found in colostrum of vaccinated cows (Prado et al., 2008). Results suggest that vaccination of dairy cows with rSUAM induced specific antibody capable of blocking and/or interfering with the early pathogenic processes of *Strep. uberis* IMI. We are currently optimizing methods for the production and purification of rSUAM to conduct proof of concept studies in cows to determine the potential of SUAM as a vaccine for the

prevention of *Strep. uberis* during the nonlactating period.....STAY TUNED! Much of this research has been supported by USDA/NRI/CSREES grant 2004-35204-14739.

**Culling of Chronically Infected Cows:** Culling is an extremely important component of every mastitis control program. Cows not responding favorably to treatment that continue to flare-up with clinical mastitis should be culled. In addition, cows with consistently high SCC should be monitored closely. Their continued presence in the herd likely results in other cows becoming infected, especially if cows are chronically infected with contagious mastitis pathogens such as *Staph. aureus*.

**Issues Associated With Antimicrobials:** Antibiotics are used extensively in food-producing animals to combat disease and to improve animal performance. On dairy farms, antibiotics such as penicillin, cephalosporin, streptomycin, and many others are used for treatment and prevention of mastitis caused by a variety of Gram-positive and Gram-negative bacteria. Antibiotics are often administered routinely to entire herds to prevent mastitis during the nonlactating period. Benefits of antibiotic use include decreased pathogen loads, a lower incidence of disease, and a better quality product for human consumption. In contrast to these benefits, however, are suggestions that agricultural use of antibiotics may be partly (largely) responsible for the emergence of antimicrobial resistant bacteria, which in turn may decrease the efficacy of similar antibiotics used in human medicine to treat diseases of humans. In addition, the risk of antibiotic residues in raw milk is not only a public health issue, but an important economical factor for the producer who gets penalized for adulterated milk, and for the milk processing plant which jeopardizes the manufacture of dairy foods by processing adulterated milk.

**Dairy Food Safety Issues:** One area that up until recently has received little attention but is extremely important is pre-harvest dairy food safety. Milk and products derived from milk of dairy cows can harbor a variety of microorganisms and can be important sources of foodborne pathogens (Rohrbach et al., 1992; Jayarao, 1999; Oliver et al., 2005b). The presence of foodborne pathogens in milk is due to direct contact with contaminated sources in the dairy farm environment and to excretion from the udder of an infected animal. Most milk is pasteurized, so why should the dairy industry be concerned about the microbial quality of bulk tank milk? There are several valid reasons including: (1) outbreaks of disease in humans have been traced to the consumption of unpasteurized milk and have also been traced back to pasteurized milk, (2) unpasteurized milk is consumed directly by dairy producers, farm employees and their families, neighbors, and raw milk advocates, (3) unpasteurized milk is consumed directly by a large segment of the population via consumption of several types of cheeses manufactured from unpasteurized milk, (4) entry of foodborne pathogens via contaminated raw milk into dairy food processing plants can lead to persistence of these pathogens in biofilms, and subsequent contamination of processed milk products and exposure of consumers to pathogenic bacteria, (5) pasteurization may not destroy all foodborne pathogens in milk, and (6) inadequate or faulty pasteurization will not destroy all foodborne pathogens. Furthermore, pathogens such as *Listeria monocytogenes* can survive and thrive in post-pasteurization processing environments thus leading to recontamination of dairy products. These pathways pose a risk to the consumer from direct exposure to foodborne pathogens present in unpasteurized dairy products as well as dairy products that become re-contaminated after pasteurization. Current data supports the model in which the presence of pathogens depends on ingestion of contaminated feed followed by amplification in bovine hosts and fecal dissemination in the farm environment. The final outcome of this cycle is a constantly maintained reservoir of foodborne pathogens that can reach humans by direct contact, ingestion of raw contaminated milk or cheese, or contamination during the processing of milk products. Isolation of bacterial pathogens with similar biotypes from dairy farms and from outbreaks of human disease substantiates this hypothesis.

**Tennessee Quality Milk Initiative (TMI):** The purpose of TQMI is to develop a science-based comprehensive program to enhance milk quality and thus improve the profitability and sustainability of dairy farms in Tennessee via an educational, research and outreach approach. The TQMI website (<http://www.tqmi.utk.edu>) continues to be a work in progress and contains information on a variety of topics for producers, industry representatives and trainers.

In 2007, the educational phase referred to as the Tennessee Quality Milk Producer (TQMP) program was launched (Campbell et al., 2008). The TQMP Program is a fee-based comprehensive program designed to motivate and educate dairy producers on aspects of production that affect milk quality. The goal of the TQMP Program is to deliver available knowledge and recent research findings on reducing mastitis and bacteria levels to maintain high bulk tank milk quality. Educational materials are developed by Extension, research and industry experts and presented in a module format. Each module has a unique central theme, and producers receive a reference manual containing educational information presented with each module. At the completion of each session, producers are given an examination or asked to complete certain activities. A passing grade (70% or higher) or completion of activities will award producers with a level certification. Extension Agents and dairy industry representatives are trained to deliver the program and assist with individual on-farm situations. Since the program was launched in October 2007, 208 dairy producers or ~ 40% of Tennessee's dairy industry have completed three modules of the TQMP Program. The following is a brief description of each module.....

Module One: *Understanding the Basics* is a 4 hour training session. This session begins at the very basics of mastitis and milk quality. The goal of Module One is to ensure producers have a complete understanding of this disease and milk quality issues before trying to solve problems. Topics include: an overview of the TQMI program and objectives; environmental vs. contagious pathogens plus unique aspects of individual pathogens; mastitis control programs based on the NMC's recommended practices; the economics of mastitis including formulas to estimate production and profit losses and quality premiums; and definition, importance and use of various bulk tank quality parameters (standard plate count, preliminary incubation count, laboratory pasteurized count and coliform count).

Module Two: *Troubleshooting Mastitis and Bacteria Counts* is a 4.5 hour training session. The second session connects the knowledge gained in the first session to on-farm situations. Producers can use the knowledge and tools gained from this session immediately and begin the process of improving milk quality. Topics include: general steps to troubleshooting mastitis; using SCC information to pinpoint problem areas; mastitis culturing programs and proper sampling technique; practical ways to troubleshoot bacteria count problems; and milking machine function, cleaning, sanitizing, maintenance and evaluations.

Module Three: *Milking for Quality* is a 3 hour training session that is specifically geared to milkers, whether it be producers or hired employees. The goal of Module Three is to link udder anatomy and physiology, milk letdown reflex, animal handling and milking procedures to mastitis infections and bulk tank bacterial contamination.

In addition to educational activities, a comprehensive analysis of bulk tank milk quality of approximately 30% of Tennessee dairy farms is being conducted in partnership with milk buyers in Tennessee and the Tennessee Department of Agriculture. All samples have been collected and analyzed and our goal is to evaluate data and prepare materials for distribution by the first quarter of 2009. Results from this study will allow us to determine the influence of bulk tank milk SCC on parameters of bulk tank milk quality including standard plate count; preliminary incubation count; lab pasteurized count; and coliform, streptococcal and staphylococcal counts. We are also evaluating bulk tank milk samples for the presence of *Mycoplasma* species.

We also plan to conduct on-farm research and demonstrations on commercial dairy farms in Tennessee. We will select small, average and large dairy farms producing high quality, average quality and poor quality milk based on SCC and bulk tank milk quality data obtained from the bulk tank milk study. A four-stage approach will be used: (A) pre-trial evaluation of dairy farm management practices and development of an objective mastitis control and milk quality plan, (B) implementation of the mastitis control and milk quality plan, (C) evaluation of the mastitis control

and milk quality plan, and (D) analysis of data and development of science-based educational and outreach materials to be disseminated throughout Tennessee and the Southern Region.

We are partnering with other universities in the Southern Region (The University of Georgia, the University of Florida, Virginia Tech and perhaps others) to take our quality milk initiative beyond the borders of Tennessee to other southeastern states. Our goal is to develop a science-based comprehensive program to enhance milk quality and thus improve the profitability and sustainability of dairy farms in the Southern Region via an educational, research and outreach approach. Collaborative projects will be developed and submitted to different USDA competitive grant programs. If funded, we will continue to develop and disseminate new educational modules to interested dairy producers, and conduct different on-farm demonstration/research trials in commercial dairy herds in several Southeastern states.

**Conclusions:** Production of maximum quantities of high quality milk is an important goal of every dairy operation. On the other hand, poor milk quality affects all segments of the dairy industry, ultimately resulting in milk with decreased manufacturing properties and dairy products with reduced shelf life. One important measure of milk quality is the number of somatic cells in milk. Milk with a high SCC is produced by cows with mastitis and is of inferior quality. SCC limits for raw milk to be acceptable at dairy processing plants may decrease to levels much lower than they are now, making it increasingly problematic for dairy producers to meet these higher standards. Production of better quality milk will place a much greater emphasis on strategies for the prevention and control of mastitis to reduce the number of somatic cells in milk. Effective milking-time hygiene, proper milking machine function, pre- and post-milking teat disinfection, lactation therapy, antibiotic dry cow therapy and culling of chronically infected cows are time-tested management strategies for controlling mastitis and are used extensively throughout the world. Advances in biotechnology have brought exciting new technologies that can/will be used to solve complex problems confronting animal agriculture. New developments; approaches; strategies; and advances in mastitis diagnosis, treatment, and prevention will dramatically improve dairy herd health programs and result in production of maximum quantities of high quality milk at lower costs. A safe, wholesome, abundant and nutritious milk supply should be the goal of every dairy producer in the world. Use of effective mastitis control strategies will help dairy producers achieve these important goals.

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# The Influences of the Commodity Markets on the Costs of Forages and Feeds

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## Introduction

Commodity prices are changing almost on a daily basis and the markets have been anything but calm (Alexander, 2008). From the time of the initial request to actually writing this paper, grain and protein prices have dropped dramatically (Table 1). However, many people are expecting these declines in prices to be short lived or temporarily. So, the expectation of prices rising should be factored into your decision making process.

We are in volatile times due to a number of reasons affecting ingredient prices and uses (ethanol, bio-diesel, export, international value of dollar). We are expecting to see a lot more up the ups and downs (volatility) in these markets in the future. Volatility can bring both opportunity and risk. With shrinking margins on the dairy operation, due to other costs increasing as well (fuel, electric, supplies, etc.), managing volatility is a key way to protect, if not improve, your profit margins.

The supply of nutrients required by the dairy cow for a given level of milk production does not change with the price of forages, feeds, or commodities. These feed ingredients supply nutrients to the dairy cow in order for her to make milk and milk components. In addition, remember that the supply of nutrients required by the dairy cow does not change with the price of milk either. Our dairy cows are oblivious to economics. Obviously, the profitability of the dairy operation is dictated by the difference between the milk income and the cost of producing that milk (i.e. income over feed costs).

Reducing the supply of nutrients to the lactating dairy cow perhaps can reduce feed costs, however, in most situations milk production and milk income more than likely will be reduced to a greater extent, thereby, negating any benefits of reducing feed costs. Thus, the dairy producer ends up with a greater lost in milk income than the money saved in feed costs, which negatively affects profitability. Specific examples are known and been experienced every time feed costs skyrocket or milk prices decline dramatically (Hutjens, 2008). In addition, herd health and reproductive efficiency can be lost as part of these situations, which only exacerbates the lost revenues versus the attempted feed cost savings. Keep in mind that sometimes these reductions in performance can take some time to develop, i.e. body condition and reproductive efficiency.

The thought process is usually too supply these nutrients to the dairy cow to meet here nutrient requirements for maintenance, health, reproduction and milk production through grain, proteins and/or the use of commodity byproducts (distillers, whole cottonseed, soyhulls, hominy, etc.). One of the benefits in today's world of computer models is that breakeven prices of ingredients can be determined very quickly by commodity brokers. These models can easily be used to calculate the breakeven or replacement prices of commodities relative to the price of corn, soybeans, and other ingredients listed on the Chicago Board of Trade (CBOT) or with your local commodity brokers. Therefore, in the short term (day to a week), good buys of a particular commodity might be available, but in the long term, the price of commodities will move relative to the price of other commodities, i.e. corn, soybean meal, etc. Thus, if typical commodities are not available at prices that are economically justifiable in the ration for supplying nutrients to reduce feed costs, what other options does a Dairy Person have available to maintain profitability?

One area that has received little emphasis until recently is in the area of forage quality. Forages can represent from 40 to over 70% of the ration dry matter. Improving forage quality will improve the nutrient supply to the animal. What has not been done is to evaluate the value of forages relative to the value of commodities to supply nutrients. This is the focus of the remainder of this paper.

### **Value of Forage Quality**

The range in quality of forages can be quite large. In the companion paper presented at this conference on “The Application of Feed Efficiency on the Dairy Farm” (Casper, D. P. 2008), are 3 Tables listing the nutrient composition of corn silage, haylage, and hay for quality groups based on dry matter digestibility. The subjective grades of bad to excellent for haylage and hay and poor to excellent for corn silage are based on ranges of dry matter digestibility of the samples run through our laboratory. The reason for measuring the digestibility of every forage sample going through the laboratory is that the biggest factor affecting nutrient availability to the dairy cow is digestibility (Casper and Mertens) of the dry matter, energy, etc. Also, the basis of improving Feed Efficiency is to use forages with higher digestibilities of dry matter, fiber, and energy in the ration.

The digestibility of dry matter, energy, and fiber (NDF) of the forage is going to dictate the amount of digestible nutrients supplied by forage. Thus, as the digestibility of the forage increases or improves, the greater the nutrient supply to the animal will be from that forage. Thus, higher quality forages are better able to meet the nutrient requirements for high milk production. Therefore, the breakeven or best buy price for forages with different digestibilities can be calculated relative to the supply of nutrients from commodities and their value. Thus, forages of higher quality should be more valuable for supplying nutrients to the ration.

Dr. Norman St.-Pierre (2005) developed the concepts and a software program (Sesame) for calculating break-even prices of feedstuffs based on their nutritional composition and market prices using a maximum likelihood method. The concepts developed in this program are more appropriate to accurately evaluate the price of nutrients and contribution to determining the value of ingredients relative to each other. Then when the value of the nutrients have been determined, those values can be use to calculate the breakeven value of other feed ingredients. These concepts are valid for calculating the breakeven values of forages in combination with commodities. We have adapted these concepts into an Excel spreadsheet to calculate the break-even prices of commodities. However, using the nutritional composition and market prices of several commodities (Table 1) allows for calculating the breakeven prices of forages based on their nutritional compositions as forage quality improves.

Tables 2, 3, and 4, contain the calculated break-even prices for the corn silage, haylage, and alfalfa hay based on the ingredient prices given in Table 1. In addition, break-even prices were calculated based on quality with in each forage type based on two time points approximately 4 months apart, i.e. June versus October. June 2008, was approximately the time of the highest commodity prices seen this year and they have dropped dramatically since then (Table 1). The point is that evaluating forage value when commodity prices fluctuate demonstrates the fluctuation in the value of forages, as would be expected. Thus, as commodity prices increase, forages become more valuable as a source of nutrients.

These tables also demonstrate that the improving the quality of the forage produced on the farm or purchased results in the higher quality forages being more valuable. If the Dairy Producer can produce or source forages with higher quality (digestibility) it will result in the opportunity to reduce ration costs while still meeting the nutrient requirements for that level of milk production. Thus, if you can produce or purchased forages for less cost than given for a particular quality level, then the opportunity exists to produce the same amount of milk at less cost. In addition, the higher the quality of the forage, the higher the forage content of the ration can be which will have additional benefits to the dairy cow in addition to reducing ration cost.

## **Conclusions**

Certainly good buys can be found for various commodities at various times. However, over longer periods of time, these commodities will be priced and valued relative to other commodities in the market place, thereby minimizing the availability of really great buys. Thus, they may or may not continue to be good buys to reduce ration costs. In order to reduce ration costs over the long term the focus needs to be placed on producing or sourcing forages with the highest quality (digestibility) possible. In the opinion of this author, forage quality can not be too good.

The value of forages and their quality can and should be evaluated relative to commodity prices for supplying nutrients to the ration. Higher quality forages are more valuable as a source of nutrients in the rations and ultimately will reduce ration cost. This allows for improving feed efficiency and reducing the cost to produce milk by the dairy operation.

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**Table 1.** The price of various commodities (\$/ton) at two time points during 2008.

Feed Ingredient	June 30, 2008	October 20, 2008
Corn	\$ 256.43	\$ 139.64
48 soybean meal	\$ 432.00	\$ 248.00
Soyhulls	\$ 200.00	\$ 185.00
Whole cottonseed	\$ 435.00	\$ 293.00
Cottonseed meal	\$ 345.00	\$ 265.00
Linseed meal	\$ 305.00	\$ 195.00
Tallow	\$ 690.00	\$ 580.00
Corn gluten feed	\$ 165.00	\$ 109.00
Hominy	\$ 172.00	\$ 131.00
Corn distillers	\$ 185.00	\$ 133.00
Wheat midds	\$ 145.00	\$ 153.00
Beet pulp	\$ 460.00	\$ 510.00
Molasses, wet	\$ 180.00	\$ 170.00
Corn gluten meal 60	\$ 605.00	\$ 470.00
Canola meal	\$ 307.00	\$ 170.00
Blood meal, pork	\$ 1150.00	\$ 1025.00
Fish meal	\$ 975.00	\$ 975.00

Based on Chicago or Minneapolis prices by Feedstuffs<sup>®</sup> for these dates.

**Table 2.** The breakeven value of different qualities (digestibility) of corn silage (35% dry matter) at two time points based on commodity prices published by Feedstuffs<sup>®</sup>.

Corn Silage Quality	June 30, 2008	October 20, 2008
Poor	68.78	57.30
Fair	71.09	59.15
Medium	73.81	61.13
Good	76.52	63.18
Excellent	79.96	65.84

**Table 3.** The breakeven value of different qualities (digestibility) of haylage (35% dry matter) at two time points based on commodity prices published by Feedstuffs®.

Haylage Quality	June 30, 2008	October 20, 2008
Bad	83.93	72.74
Poor	91.73	78.97
Fair	110.14	94.83
Medium	126.03	108.36
Good	132.79	113.99
Excellent	139.41	119.55

**Table 4.** The breakeven value of different qualities (digestibility) of alfalfa hay at two time points based on commodity prices published by Feedstuffs®.

Alfalfa Hay Quality	June 30, 2008	October 20, 2008
Bad	135.63	115.16
Poor	172.49	147.56
Fair	234.26	201.25
Medium	260.25	223.33
Good	271.98	232.71
Excellent	287.06	245.58



## By-product Feeding for Milk Production

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The cost of corn and soybean meal has increased substantially over the last two years. To control or reduce feed cost, producers and nutritionists are looking at by-product feeds to help control or reduce feed cost. Since the price of by-product feeds is based on the market price of corn and soybean meal, the cost of by-product feeds has increased as well. There are few if any by-product feeds that may be a bargain relative to their nutrient content, but there are still opportunities to reduce total feed cost. One of the keys for successfully using by-product feeds is an understanding of the nutrient characteristics and limitations of each by-product feed. Properly used, by-product feeds will maintain milk yield or body weight gain as well as keep animals healthy when rations are properly balanced. As by-products are used to replace corn and soybean meal, rations should be adjusted to maintained desired concentrations of nutrients that will maintain or improve ruminal fermentation. This presentation will examine the characteristics of select by-product feeds and how this impacts their value and consideration's producers should take into account when feeding combinations of by-product feeds.

### ***Economic Value of By-Products***

There are several methods for comparing the prices of byproduct feeds. Many ration formulation programs calculate the value of each feed ingredient based on the nutrient requirements of the diet and the nutrients available from ingredients offered. This method provides specific information for that particular situation, but many producers do not have the software to perform these calculations.

More commonly, producers use programs specifically designed to compare the value of several feeds compared to a reference feed such as corn and soybean meal. One program that is commonly used is the FEEDVAL program available from the University of Wisconsin (<http://www.wisc.edu/dysci/uwex/nutritn/spreadsheets/FEEDVAL-Comparative.xls>). This program calculates the value (\$/ton) based on the dry matter (DM), crude protein (CP), total digestible nutrients (TDN), calcium (Ca), and phosphorus (P) concentrations of each by-product feed compared with the test feeds (shelled corn, 48% CP soybean meal, limestone, and dicalcium phosphate). The nutrient composition of the feeds can be changed to match the products available in your area as well as the percentage feed loss.

An example of the results from FEEDVAL is presented in Table 1. Prices used for this analysis were obtained from October 20, 2008 issue of *Feedstuffs*. An additional \$30/ton was added to the market price for freight. From this example analysis there are several by-product feeds that offer opportunities for savings based on the current price of corn and soybean meal whereas the market price of other by-product feeds is higher than the value of the nutrient s provided. Because of differences in actual freight rates from the source of the by-product feed and changes in the market, this type of analysis should be run periodically as the potential value will change.

The decision to actually purchase one of these feeds should also take into consideration feeds that are already in inventory, what is needed to balance the ration, and the effect of shrinkage, storage and handling, and processing issues have on the final cost of the by-product feed. It is critical to know the nutrients needed to balance the ration to produce a diet that will not only support the animal performance desired, but also maintain ruminal function and animal health. For example, many of the by-products included in the example price evaluation also contain digestible fiber which may be as important as total energy and protein if forage is limited or you are feeding a corn silage based ration and need some digestible fiber. If forage is limited, the value of high-fiber by-products may be more as they would help maintain a healthy ruminal environment.

### ***Nutrient Content and Considerations***

The nutrient content of several commonly used by-product feeds is presented in Table 2. The actual nutrient content of these by-product feeds may vary from these values because of differences in raw materials and processing methods. Because of this, all by-product feeds should be sampled routinely to determine actual nutrient content before they are fed.

Consideration should be given to the concentration of each nutrient and its form as this will affect ruminal fermentation and how the by-product feed should be fed. When replacing corn in the diet, we frequently look for a source of starch or sugar that will provide a source of rapidly available energy. Although there isn't a specific requirement for starch, limited amounts of starch provides rapidly fermentable energy that can improve microbial fermentation and protein synthesis. Examining the list of byproduct feeds in Table 2, only bakery byproducts, hominy feed, and molasses have any appreciable amount of starch and sugar. Citrus pulp contains pectin (included in soluble fiber) which is also extensively fermented. Low starch diets can be fed that will support milk yield and animal growth. This is the basis of many of the build-in-roughage (BIR) or one-shot rations that have been successfully used for years in the region. Rations can be properly formulated with starch levels much lower than typically fed.

Many by-product feeds have above average concentrations of fat. The fat in these by-products is primarily vegetable fat which is reactive in the rumen. The total dietary fat from basal ingredients plus these reactive fats should not exceed 5% of the dietary DM to avoid potential negative effects on ruminal fermentation and milk fat depression. These fats contain high concentrations of polyunsaturated fatty acids which reduce ruminal fermentation of fiber through either inhibition of fibrolytic microorganisms or through physically coating fiber which prevents the enzymes from attaching. These fats also produce more trans-fatty acids which have been linked to milk fat depression. Too much reactive fat in the diet can occur when multiple by-products are used to replace corn and soybean meal. For example, if you are feeding whole cottonseed and substitute hominy feed for corn and corn distiller's grains with solubles for a portion of the soybean meal, the total fat content in the diet increases from 4.3% to 5.5% of the DM. To avoid this type of problem, the amount of each by-product feed may need to be limited to keep the total fat content of the ration below 5%.

Many of the by-product feeds contain significant amounts of digestible fiber. Citrus pulp, corn gluten feed, and soy hulls have been used to replace the starch from corn grain and improve ruminal fermentation in many cases, especially when diets are based on corn silage. Soybean hulls are also a good source of digestible NDF as discussed at this conference last year by Dr. Grant.

Another aspect to consider is the type and quality of protein contained in these by-product feeds. By-products from corn have low concentrations of lysine, an essential amino acid which is considered to be one of the most limiting amino acids for supporting milk production and growth. Diets based on corn silage and supplemented with corn and corn by-product feeds typically have a lysine deficiency. This not only may limit milk yield, but also decreases the efficiency of protein utilization. Protein contained in by-product feeds differs in the degree to

which it is degraded in the rumen. By-products such as brewers grain and distiller's grains have a greater proportion of undegradable protein than the other protein by-products. Heat or chemical treatment of canola meal or soybean meal reduced degradability which is useful in diets formulated for high producing dairy cows. The protein provided by several by-product feeds such as bakery corn gluten feed and wheat middlings is very degradable. These by-products are good sources of degradable protein and work well with corn silage or grass hay, but their use to supplement diets containing moderate to high protein haylage would result in excessive nitrogen loss through the urine.

There are other aspects that should be considered when using by-product feeds. For example peanut skins contain tannins which binds some of the protein in the diet. When diets are formulated with more than 16% peanut skins, DM intake, milk yield and milk fat percentage will decline. Because of this, peanut skins should be limited to 16% or less of the diet DM. Wheat middlings can be used up to 20% of the dietary DM, but feeding more reduces DM intake and performance. Also, the quality of protein from wheat middlings is not as desirable as other protein sources because of the amino acid balance.

### ***Less Common By-product Feeds***

Occasionally there are opportunities for producers to purchase by-products that are not as common such as candy, speciality bakery or food items, vegetables, etc. Each of these by-products provides unique combinations of nutrients as well as limitations that may not be specifically defined for the by-product. For example there are candy by-products available for feeding which have high concentrations of sugar, so the amount fed is limited to less than 5% of the DM because of the sugar is rapidly fermented and increases the production of lactic acid which could cause ruminal acidosis short term and increased foot problems longer term.

Unsweet chocolate and chocolate by-products from the production of chocolate from the coca bean contain theobromine which is toxic when consumed in very small quantities (less than 3% of DM). The quantity of theobromine in milk chocolate has been diluted (1/8 to 1/10 of unsweet chocolate) and doesn't pose a problem, but the fat and sugar content of chocolate candy limits' intake to less than 5% of the DM.

Vegetable and fruit by-products provide a good source of nutrients and digestible fiber, but availability and handling issues complicate their use. Some of these products contain high concentrations of specific nutrients which may limit their use for certain animals. For example apple pomace contains high concentrations of potassium and should not be fed to close-up dry cows. Many of the vegetable by-products have very low DM concentrations, so transportation cost limits the economic opportunities if the supply is not close by. These products will also spoil quickly and have high shrinkage losses.

If you are in the position to purchase some of the odd by-products, you should get an analysis of the product so your nutritionist can properly formulate the ration and determine the value of these products. Do not forget to ask about any special compounds that may naturally be in the product or introduced during production that could pose problems when consumed. Many of these by-product feeds have special handling issues because of the packaging that must be addressed before committing to the supply.

### Summary

By-product feeds can be successfully incorporated into diets fed to replacement heifers or lactating dairy cows to maintain performance and control or reduce feed cost. To successfully incorporate by-product feeds into rations, knowledge of their nutrient content is essential to avoid any potential negative effects on ruminal fermentation or milk composition. When using multiple by-products in a diet, the amount of each by-product may need to be limited because of the total amount of fat, form of protein, or unique compounds that could affect animal performance. Properly formulating rations will allow by-products to be fed in place of corn and soybean meal and maintain acceptable rates of gain or milk yield.

Table 1. Calculated value (\$/ton) of select by-product feeds.

Item	Calculated Value <sup>1</sup>	Market price	Difference Calculate - Market
		----- \$/ton -----	
Bakery byproduct	227	245	-18
Brewers grains, wet	58	40	18
Citrus pulp	170	225	-55
Hominy feed	195	200	-5
Molasses, cane	142	215	-73
Soy hulls, pelleted	192	175	17
Canola meal	273	240	33
Cottonseed meal	280	295	-15
Cottonseed, whole	269	275	-6
Corn gluten feed, dry	239	180	59
Corn distillers dried grains with solubles	251	182	69

<sup>1</sup>Values were calculated using FEEDVAL and are based on \$196/ton ground corn, \$305 soybean meal (48% CP), \$7.00/cwt feed grade limestone, and \$55.00/cwt dicalcium phosphate.

Table 2. Chemical composition of common by-product feeds used as primary sources of energy and protein<sup>1</sup>.

	DM	CP	ADF	NDF	NFC	Sugar	Starch	Soluble Fiber	Fat	Ash	Ca	P	NE <sub>i</sub>
	%		----- % of DM -----					-----					Mcal/ lb
Energy by-products													
Corn	88.0	9.0	4.0	9.0	77.1	1.5	74.8	0.8	4.2	1.6	0.04	0.30	1.09
Bakery by-product	94.8	13.0	3.2	7.4	70.6	12.3	56.3	2.1	9.0	3.3	0.33	0.26	1.13
Citrus pulp	88.6	7.0	19.9	23.9	62.5	26.9	1.3	34.4	3.1	6.4	1.82	0.11	0.87
Hominy feed	88.4	11.0	6.0	19.0	64.6	2.6	54.3	7.8	4.9	3.0	0.11	0.35	1.01
Molasses, cane	73.0	5.8	0	0	82.0	69.8	0	8.2	1.0	11.0	1.00	0.10	0.94
Peanut skins	90.0	14.0	18.0	34.0	27.4	4.1	21.9	1.4	20.0	6.0	0.19	0.20	1.16
Soy hulls	91.0	12.0	47.0	66.3	17.4	0.7	1.0	13.0	2.6	5.0	0.64	0.18	0.80
Wheat middlings	89.0	18.4	12.2	38.0	32.8	3.9	19.0	9.8	5.0	6.5	0.15	1.24	0.80
Protein by-products													
Soybean meal, 48% CP	90.0	55.0	6.0	10.0	27.2	10.9	2.2	14.1	2.8	6.7	0.29	0.71	1.01
Brewers grains, wet	24.0	29.0	23.0	47.0	21.5	1.7	12.0	6.9	6.5	4.4	0.34	0.68	0.79
Canola meal <sup>2</sup>	90.0	36.0	20.7	30.2	32.4	12.3	14.3	5.8	5.7	7.3	0.75	1.24	0.75
Corn DDGS <sup>3</sup>	88.8	30.3	17.8	32.2	25.9	3.4	12.2	1.0	14.4	5.9	0.04	0.93	0.91
Corn gluten feed, dry	90.0	24.0	10.7	34.7	32.4	13.1	14.3	5.1	4.2	7.9	0.07	1.40	0.88
Cottonseed meal	92.0	42.0	20.2	29.9	21.7	8.2	1.7	11.7	6.1	6.9	0.24	1.24	0.80
Whole cottonseed	90.1	21.0	40.1	50.3	7.3	3.3	0.4	3.7	19.3	6.9	0.18	0.58	0.85

<sup>1</sup>Values are the default values included in CPM-Dairy.

<sup>2</sup>Mechanically extracted.

<sup>3</sup>Distillers dried grains with solubles from the ethanol industry.



## **Mycotoxins in Dairy Diets: Effects and Prevention**

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### ***Mycotoxins***

Mycotoxins are toxins produced by toxigenic filamentous fungi that cause an undesirable effect (mycotoxicosis) in exposed animals. Exposure is usually by consumption of contaminated feeds, but may also be by contact or inhalation. Biological effects include liver and kidney toxicity, central nervous system effects, immune suppression and estrogenic effects. There are hundreds of mycotoxins which are chemically diverse. Only a few have been extensively researched and even fewer have routine methods of analysis available. The primary classes of mycotoxins are aflatoxins, zearalenone, trichothecenes, fumonisins, ochratoxin A and the ergot alkaloids.

### ***Molds Can Cause Disease***

A mold (fungal) infection resulting in disease is referred to as a mycosis. Of recent concern, *Aspergillus fumigatus* is known to cause mycotic pneumonia, mastitis and abortions and has been proposed as the pathogenic agent associated with mycotic hemorrhagic bowel syndrome (HBS) in dairy cattle (Puntenney et al., 2003). It is theorized that with a mycosis, mycotoxins produced by the invading fungi can suppress immunity; therefore increasing the infectivity of the fungus. Feeding a commercial mycotoxin adsorbent with anti-fungal properties stimulates immunity and reduced HBS (Puntenney et al., 2003).

### ***Mold growth, mycotoxin formation***

Many species of mold produce mycotoxins in feedstuffs, yet feed can be moldy without the presence of mycotoxins. Feeds can also appear normal, but contain significant amounts of mycotoxins. Molds grow and mycotoxins can be produced pre-harvest or post-harvest during storage, transport, processing or feeding. Mold growth and mycotoxin production are related to plant stress caused by weather extremes, insect damage, inadequate storage practices and faulty feeding conditions. Molds grow over a temperature range of 10-40°C (50-104°F), a pH range of 4-8 (*Penicillium* grows at a low pH) and above 0.7 aw (equilibrium relative humidity expressed as a decimal instead of a percentage). Molds can grow on feeds containing more than 12-15% moisture. In wet feeds such as silage, moisture helps exclude air, but molds will grow if sufficient oxygen is present.

### ***Mycotoxin occurrence***

Mycotoxins occur frequently in a variety of feedstuffs and are routinely fed to animals. Occurrence and concentrations are variable by year, because of the annual variation in weather conditions and resulting plant stresses. Worldwide, approximately 25% of crops are affected by mycotoxins annually (CAST, 1989). Table 1 provides mycotoxin analyses of feed samples submitted by North Carolina farmers over a nine-year period indicating that mycotoxins in feeds including corn silage and corn grain occur commonly at unsuitable concentrations (Whitlow et al., 1998).

Table 1. Occurrence of five mycotoxins in corn silage, corn grain and in all feed samples submitted for analysis by producers in North Carolina over a nine year period.					
Mycotoxin	Feedstuff	Number of samples	Positive above limits, %	Mean	Standard deviation
Aflatoxin, >10 ppb	Corn Silage	461	8	28	19
	Corn Grain	231	9	170	606
	All Feeds	1617	7	91	320
Deoxynivalenol, > 50 ppb	Corn Silage	778	66	1991	2878
	Corn Grain	362	70	1504	2550
	All Feeds	2472	58	1739	10880
Zearalenone, > 70 ppb	Corn Silage	487	30	525	799
	Corn Grain	219	11	206	175
	All Feeds	1769	18	445	669
T-2 toxin, > 50 ppb	Corn Silage	717	7	569	830
	Corn Grain	353	6	569	690
	All Feeds	2243	7	482	898
Fumonisin, > 1 ppm	Corn Silage	63	37	--	--
	Corn Grain	37	60	--	--
	All Feeds	283	28	--	--

### ***Mycotoxin effects***

Mycotoxins, in large doses, can be the primary agent causing acute health or production problems in a dairy herd. A more likely scenario is to find mycotoxins at lower levels interacting with other stressors and contributing to chronic problems including a higher incidence of disease, poor reproductive performance, or suboptimal milk production. To the animal producer, these chronic losses are of greater economic importance than losses from acute effects, and more difficult to diagnose.

Mycotoxins exert their effects through several means including 1) reduced intake or feed refusal; 2) reduced nutrient absorption and impaired metabolism; 3) altered endocrine and exocrine systems; 4) suppressed immune function; 5) altered rumen microbial growth, and 6) cellular death.

Ruminal degradation of mycotoxins helps to protect the cow against acute toxicity, but may contribute to chronic problems, associated with long term consumption of low levels of mycotoxins. Ruminal degradation of mycotoxins may mask mycotoxin effects in dairy cows. In recent years, as production stresses increased, the dairy industry has placed more attention on management details and the significance of chronic mycotoxin effects has been more widely recognized (Jouany and Diaz, 2005).

Symptoms of a mycotoxicosis vary depending on the mycotoxins involved and their interactions with other stress factors. Symptoms result from a progression of effects, and may reflect those of an opportunistic disease. Cows may exhibit few or many of a variety of symptoms. The more stressed cows, such as fresh cows, are most affected; perhaps because their immune systems are already suppressed. Symptoms may include: reduced production; reduced feed consumption; intermittent diarrhea (sometimes with bloody or dark manure); reduced feed intake; unthriftiness; rough hair coat; and reduced reproductive performance including irregular estrous

cycles, embryonic mortalities, pregnant cows showing estrus, and decreased conception rates. There generally is an increase in incidence of early lactation diseases such as displaced abomasum, ketosis, retained placenta, metritis, mastitis, and fatty livers. Cows do not respond well to veterinary therapy.

### **Toxicity of Individual Mycotoxins**

#### **Aflatoxin**

Aflatoxins are extremely toxic, mutagenic, and carcinogenic compounds produced by *Aspergillus flavus* and *A. parasiticus*. Aflatoxin B1 is secreted in milk in the form of aflatoxin M1. The FDA limits aflatoxin to no more than 20 ppb in lactating dairy feeds and to 0.5 ppb in milk. A thumb rule is that milk aflatoxin concentrations equal about 1.7% (range from 0.8 to 2.0%) of the aflatoxin concentration in the total ration dry matter. Cows consuming diets containing 30 ppb aflatoxin can produce milk containing aflatoxin residues above the FDA action level of 0.5 ppb. Aflatoxin appears in the milk rapidly and clears within three to four days (Diaz et al., 2004).

Symptoms of acute aflatoxicosis in mammals include: inappetance, lethargy, ataxia, rough hair coat, and pale, enlarged fatty livers. Symptoms of chronic aflatoxin exposure include reduced feed efficiency and milk production, jaundice, and decreased appetite. Aflatoxin lowers resistance to diseases and interferes with vaccine-induced immunity (Diekman and Green, 1992). Production and health of dairy herds may be affected at dietary aflatoxin levels above 100 ppb, which is higher than the 30 ppb that is expected to produce illegal milk residues. Guthrie (1979) showed when lactating dairy cattle in a field situation were consuming 120 ppb aflatoxin, reproductive efficiency declined and when cows were changed to an aflatoxin free diet, milk production increased over 25%. Aflatoxin is more often found in corn, peanuts and cottonseed grown in warm and humid climates.

Table 2. Action levels for total aflatoxins in livestock feed, (Henry, 2006)

<b>Class of Animal</b>	<b>Feed</b>	<b>Aflatoxin Level</b>
<b>Finishing beef cattle</b>	<b>Corn and peanut products</b>	<b>300 ppb</b>
<b>Beef cattle, swine or poultry</b>	<b>Cottonseed meal</b>	<b>300 ppb</b>
<b>Finishing swine over 100 lb.</b>	<b>Corn and peanut products</b>	<b>200 ppb</b>
<b>Breeding cattle, breeding swine and mature poultry</b>	<b>Corn and peanut products</b>	<b>100 ppb</b>
<b>Immature animals</b>	<b>Animal feeds and ingredients, excluding cottonseed meal</b>	<b>20 ppb</b>
<b>Dairy animals, animals not listed above, or unknown use</b>	<b>Animal feeds and ingredients</b>	<b>20 ppb</b>

#### **Deoxynivalenol (DON) or Vomitoxin**

Deoxynivalenol is a *Fusarium* produced mycotoxin, commonly detected in feed. Surveys have shown DON to be associated with swine disorders including: feed refusals, diarrhea, emesis, reproductive failure, and deaths. The impact of DON on dairy cattle is not established, but clinical data show an association between DON and poor performance in dairy herds (Whitlow et al., 1994). Dairy cattle consuming diets contaminated primarily with DON (2.5 ppm) have responded

favorably (1.5 kg milk,  $P < .05$ ) to the dietary inclusion of a mycotoxin binder, providing circumstantial evidence that DON may reduce milk production (Diaz et al., 2001).

The presence of DON may serve as a marker, indicating that feed was exposed to a situation conducive for mold growth and possible formation of several mycotoxins. Like other mycotoxins, pure DON added to diets, produces less toxicity than does DON from naturally contaminated feeds, perhaps due to the presence of multiple mycotoxins in naturally contaminated feeds.

Table 3. Advisory levels for deoxynivalenol (vomitoxin) in livestock feed, (Henry, 2006)

<b>Class of Animal</b>	<b>Feed Ingredients &amp; Portion of Diet</b>	<b>DON Levels in Grains &amp; Grain By-products and (Finished Feed)</b>	
<b>Ruminating beef and feedlot cattle older than 4 months</b>	<b>Grain and grain by-products not to exceed 50% of the diet</b>	<b>10 ppm</b>	<b>(5 ppm)</b>
<b>Chickens</b>	<b>Grain and grain by-products not to exceed 50% of the diet</b>	<b>10 ppm</b>	<b>(5 ppm)</b>
<b>Swine</b>	<b>Grain and grain by-products not to exceed 20% of the diet</b>	<b>5 ppm</b>	<b>(1 ppm)</b>
<b>All other animals</b>	<b>Grain and grain by-products not to exceed 40% of the diet</b>	<b>5 ppm</b>	<b>(2 ppm)</b>

### **T-2 Toxin (T-2)**

T-2 toxin is a very potent *Fusarium* produced mycotoxin that occurs in a low proportion of feed samples (<10%). Russell et al. (1991) found 13% of Midwestern corn grain contaminated with T-2 toxin in a survey of the 1988 drought damaged crop. In dairy cattle, T-2 has been associated with gastroenteritis, intestinal hemorrhages (Petrie et al., 1977; Mirocha et al., 1976) and death (Hsu et al., 1972 and Kosuri et al., 1970). Dietary T-2 toxin at 640 ppb for 20 days resulted in bloody feces, enteritis, abomasal and ruminal ulcers, and death (Pier et al., 1980). 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 estrous cycles in cows exposed to T-2. Serum immunoglobulins and complement proteins were lowered in calves receiving T-2 toxin (Mann et al., 1983). Gentry et al. (1984) showed a reduction in white blood cell and neutrophil counts in calves. McLaughlin et al. (1977) found that the primary basis of T-2 reduced immunity is reduced protein synthesis. Guidelines for T-2 toxin are not established, but avoiding levels above 100 ppb may be reasonable. Diacetoxyscirpenol, HT-2 and neosolaniol may occur along with T-2 toxin and cause similar symptoms. The FDA has established no guidelines for T-2 toxin in feedstuffs.

### **Zearalenone (ZEA)**

Zearalenone is a *Fusarium* produced mycotoxin that has a chemical structure similar to estrogen and can produce an estrogenic response in animals. Zearalenone is associated with ear and stalk rots in corn and with scab in wheat. A controlled study with non-lactating cows fed up to 500 mg of ZEA (calculated dietary concentrations of about 25 ppm ZEA) showed no obvious effects except that corpora lutea were smaller in treated cows (Weaver et al., 1986b). In a similar study with heifers receiving 250 mg of ZEA by gelatin capsule (calculated dietary concentrations of about 25 ppm ZEA), conception rate was depressed about 25%; otherwise, no obvious effects were noted (Weaver et al., 1986a). Field reports have related ZEA to estrogenic responses in ruminants including abortions (Kallela and Ettala, 1984; Khamis et al., 1986; Mirocha et al., 1968; and Mirocha et al., 1974). Symptoms have included vaginitis, vaginal secretions, poor reproductive performance, and mammary gland enlargement of virgin heifers. In a field study, (Coppock et al., 1990) diets with about 660 ppb ZEA and 440 ppb DON resulted in poor consumption, depressed milk production, diarrhea, increased reproductive tract infections, and total reproductive failure. New Zealand workers (Towers et al., 1995) have measured blood ZEA and metabolites ("zearalenone") to estimate ZEA intake. Dairy herds with low fertility had higher levels of blood "zearalenone". Individual cows within herds examined by palpation and determined to be cycling had lower blood "zearalenone" levels than did cows that were not cycling. In this study, reproductive problems in dairy cattle were associated with dietary ZEA concentrations of about 400 ppb. The FDA has established no guidelines for zearalenone in feed, such that any contamination issue is dealt with on a case by case basis (Henry, 2006).

### **Fumonisin (FB)**

A USDA, APHIS survey of 1995 corn from Missouri, Iowa, and Illinois found that 6.9% contained more than 5 ppm fumonisin B1. Fumonisin was prevalent in Midwestern corn from the wet 1993 season as well. Corn screenings contain about 10 times the fumonisin content of the original corn.

Fumonisin B1 produced by *F. verticillioides*, was first isolated in 1988. It causes leukoencephalomalacia in horses, pulmonary edema in swine, and hepatotoxicity in rats. It is carcinogenic in rats and mice and may be a promoter of esophageal cancer in humans (Rheeder et al., 1992). Fumonisins are structurally similar to sphingosine, a component of sphingolipids, which are in high concentrations in certain nerve tissues such as myelin. Fumonisin toxicity results from blockage of sphingolipid biosynthesis and thus degeneration of tissues rich in sphingolipids.

While FB1 is much less potent in ruminants than in hogs, it has now been shown toxic to sheep, goats, beef cattle, and dairy cattle. Osweiler et al. (1993) showed that 148 ppm of resulted in mild liver lesions in beef calves and a trend for lower weight gains. Dairy cattle (Holsteins and Jerseys) fed diets containing 100 ppm fumonisin for approximately 7 days prior to freshening and for 70 days thereafter produced less milk (6 kg/cow/day) which was explained primarily by reduced feed consumption (Diaz et al., 2000). Serum enzyme concentrations suggested mild liver disease.

**Table 4. Guidance levels for total fumonisins in animal feeds, (Henry, 2006)**

<b>Class of Animal</b>	<b>Feed Ingredients &amp; Portion of Diet</b>	<b>Levels in Corn &amp; Corn By-products</b>	<b>Levels in Finished Feeds</b>
<b>Equids and Rabbits</b>	<b>Corn and corn by-products not to exceed 20% of the diet**</b>	<b>5 ppm</b>	<b>1 ppm</b>
<b>Swine and Catfish</b>	<b>Corn and corn by-products not to exceed 50% of the diet**</b>	<b>20 ppm</b>	<b>10 ppm</b>
<b>Breeding Ruminants, Breeding Poultry and Breeding Mink*</b>	<b>Corn and corn by-products not to exceed 50% of the diet**</b>	<b>30 ppm</b>	<b>15 ppm</b>
<b>Ruminants &lt;3 Months Old being Raised for Slaughter and Mink being Raised for Pelt Production</b>	<b>Corn and corn by-products not to exceed 50% of the diet**</b>	<b>60 ppm</b>	<b>30 ppm</b>
<b>Poultry being Raised for Slaughter</b>	<b>Corn and corn by-products not to exceed 50% of the diet**</b>	<b>100 ppm</b>	<b>50 ppm</b>
<b>All Other Species or Classes of Livestock and Pet Animals</b>	<b>Corn and corn by-products not to exceed 50% of the diet**</b>	<b>10 ppm</b>	<b>5 ppm</b>

\* Includes lactating dairy cattle and hens laying eggs for human consumption.

\*\* Dry weight basis.

#### **Ergot alkaloids, including fescue toxicity**

One of the earliest recognized mycotoxicoses is ergotism caused by a group of ergot alkaloids. They are produced by several species of *Claviceps*, which infect the plant and produce toxins in fungal bodies called sclerotia or ergots, which are small black colored bodies similar in size to the grain. Ergotism primarily causes a gangrenous or nervous condition in animals. Symptoms are directly related to dietary concentrations and include reduced weight gains, lameness, lower milk production, agalactia and immune suppression (Robbins et al., 1986). Sclerotia levels above 0.3% are related to reproductive disorders.

Fescue grass infected with *Neotyphodium* or *Epichloe* can contain ergot alkaloids and cause "fescue toxicity" (Bacon, 1995). Animal symptoms are lower weight gains, rough hair coat, elevated body temperature, agalactia, reduced conception, and gangrenous necrosis of the extremities such as the feet, tail and ears. Fescue is a major pasture grass throughout the lower Midwest and upper South and over half is thought to be infected. More than 20% of US beef cattle graze fescue, making this a serious problem for cattle producers.

## PR toxin

PR toxin is one of the several mycotoxins produced by *Penicillium* molds. *Penicillium* grows at a low pH and in cool damp conditions and has been found to be a major contaminant of silage. PR toxin, produced by *P. roquefortii*, is highly toxic and has been suggested as the causative agent associated with moldy corn silage problems (Seglar 1997 and Sumarah et al., 2005). Surveys of grass and corn silage in Europe have found an occurrence of *P. roquefortii* in up to 40% of samples (Auerbach, 2003) and associated with cattle disorders (Boysen et al., 2000). PR toxin, caused acute toxicity in mice, rats and cats by increasing capillary permeability resulting in direct damage to the lungs, heart, liver and kidneys (Chen et al., 1982) and was the suspected vector in a case study with symptoms of abortion and retained placenta (Still et al., 1972). Other *Penicillium* produced mycotoxins in silages, such as roquefortine C, and mycophenolic acid have been associated with herd health problems (Auerbach, 1998; Scudamore and Livesay, 1998, and Sumarah et al., 2005).

## Prevention and Treatment

Adapted crop varieties with resistance to fungal disease or to insect damage (Bt hybrids) have fewer field produced mycotoxins. Positive field factors are irrigation, timely harvest, avoidance of harvesting lodged or field damaged materials and avoiding kernel damage.

Harvested grains should be dried to below 15% moisture and preferably to <13% help to compensate for non-uniform moisture concentrations throughout the grain mass. Because high temperatures increase the amount of free moisture (water activity), grain should be drier when stored at high temperatures. Storage must be sufficient to eliminate moisture migration, moisture condensation and leaks. Aeration helps reduce moisture migration and non-uniform moisture concentrations. Commodity sheds should protect feedstuffs from rain and have a vapor barrier in the floor to reduce moisture. Bins, silos and other storage facilities should be cleaned to eliminate sources of inoculation. Check stored feed at intervals to determine if heating and molding are occurring. Organic acids can be used as preservatives for feeds too high in moisture for proper storage.

Mold will grow in moist hay, but it is sometimes difficult to achieve adequate dry down which is related to moisture at harvest, air movement, humidity, air temperature, bale density and the storage facility. Rate of dry down can be improved by ventilation, creation of air spaces between bales, reduced size of stacks, alternated direction of stacking and avoidance of other wet products in the same area.

Production of mycotoxins in silage can be reduced by following accepted silage making practices aimed at preventing deterioration primarily by quickly reducing pH and eliminating the oxygen. Accepted silage making practices emphasize ●harvesting at the proper moisture content; ●chopping uniformly at the proper length, ●filling the silo rapidly; ●packing the silage sufficiently to exclude air; ●using an effective fermentation aide; and ●covering completely and well. Infiltration of air after ensiling can allow growth of acid tolerant microorganisms, an increase in the pH and then mold growth. *Penicillium* molds are acid tolerant and can grow if any air is present. Microbial or other additives that enhance fermentation and rapidly reduce pH can reduce mold growth and mycotoxin formation. Chemical treatments that inhibit microbial growth have also been used effectively. Ammonia, organic acids, sulfates, urea and nitrates are shown to be at least partially effective at inhibiting mold growth. Organic acids have been used to treat the entire silage mass, or to selectively treat the outer layers of the silo. Organic acids are sometimes used during feedout to treat the silo feeding face in an effort to reduce deterioration of the feeding face. Treatment of the TMR with organic acids can reduce heating in the feed bunk. Silo size should be matched to herd size to insure daily removal of silage at a rate faster than deterioration. In warm weather, it is best to remove a foot of silage daily from the feeding face. The feeding face of silos should be cleanly cut and disturbed as little as possible to prevent aeration into the silage

mass. Silage (or other wet feeds) should be fed immediately after removal from storage. Spoilage should not be fed and feed bunks should be cleaned regularly.

High moisture grains or wet byproduct feeds must be stored at proper moisture content to exclude air and stored in a well maintained and managed structure. Wet feeds must be handled in quantities which allow them to be fed out rapidly. Organic acids can help prevent mold and can extend storage life.

Nutritional factors such as increasing nutrients such as protein, energy and antioxidants may be advisable. Animals exposed to aflatoxin show marginal responses to increased protein. In some situations, poultry respond to water soluble vitamins or to specific minerals, but data is lacking for cattle. Acidic diets seem to exacerbate effects of mycotoxins, and therefore adequate dietary fiber and buffers are recommended. Because a robust rumen fermentation can help destroy mycotoxins, cows may benefit from feed additives that enhance rumen function. Feeding management to encourage intake may be helpful. Dry cows, springing heifers and calves should receive the cleanest feed possible. Transition rations can reduce stress in fresh cows and reduce effects of mycotoxins. Strategic use of mold inhibitors may be beneficial.

### **Mycotoxin Adsorbents (Binders)**

The addition of mycotoxin binders to contaminated diets may be the most promising dietary approach to reduce effects of mycotoxins. A binder decontaminates mycotoxins in the feed by binding them strongly enough to prevent toxic interactions with the consuming animal and to prevent mycotoxin absorption across the digestive tract. Therefore, this approach is often considered as preventative rather than a therapy.

Potential absorbent materials include activated carbon, aluminosilicates (clay, bentonite, montmorillonite, zeolite, phyllosilicates, etc.), complex indigestible carbohydrates (cellulose, polysaccharides in the cell walls of yeast and bacteria such as glucomannans, peptidoglycans, and others), and some synthetic polymers.

Overall, the benefits are variable by type and amount of binder, specific mycotoxins and their amounts, animal species, and interactions of other dietary ingredients. Several of these adsorbent materials are recognized as safe feed additives (GRAS) and are used in diets for other purposes such as flow agents or pellet binders. However, no adsorbent product is approved by the FDA for the prevention or treatment of mycotoxicoses.

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## Importance of Nutrient Management Plan Record Keeping

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Nutrient management planning is a valuable management tool for livestock operations. In Georgia, any facility that has 300-1000 dairy cows must have a state land application system (LAS) permit; and facilities with more than 1000 dairy cows must have an national pollution discharge elimination system (NPDES) permit. A requirement of both of these permitting systems is a nutrient management plan for the operation. The goal of nutrient management planning is twofold: efficiently use nutrients to produce desired agronomic yields while protecting water quality. Nutrient management planning is a dynamic process that changes over time as management practices change. Record keeping is an essential component of nutrient management planning to determine profitable from unprofitable practices. Record keeping is also valuable to demonstrate environmental compliance.

What records should be kept as part of a nutrient management plan? Soil and manure test reports should be collected as part of the plan writing process and should be kept as part of an operation's records. Each field in the nutrient management plan will have recommended application rates of manure. When manure is applied, the following information should be recorded:

- Date of application
- Weather conditions the day previous, day of, and day following application
- Field name
- Manure type
- Application method
- Amount applied
- Total nitrogen and phosphorus applied

Additional records that should be kept include, equipment calibration records, daily rainfall records, off-farm transfers (date of transfer, name and address of recipient, amount transferred) and, monitoring well test results. Documentation of facility and lagoon inspections along with any corrective actions taken should be recorded. Additional information that can be helpful for management decisions include crop planting date, harvest dates, crop yields, and crop nutrient value.

In what format should records be kept? Records can be kept in several different formats. There are many different computer programs available but are not necessary to keep adequate records. Hand written records in notebooks, on calendars, or custom forms are sufficient to keep sufficient records. Records should be kept at the operation and be made available upon request to inspectors.

Although it takes some extra time and effort to keep good records the benefits can greatly outweigh the inconvenience. Good records can help determine not only what is working well on an operation but also what isn't economically viable. With increased demand on limited water resources it is more important than ever before to demonstrate good environmental stewardship. Record keeping is the most valuable tool available to producers to defend their operations and demonstrate environmental compliance.



## Synchronization Programs Continue to Change

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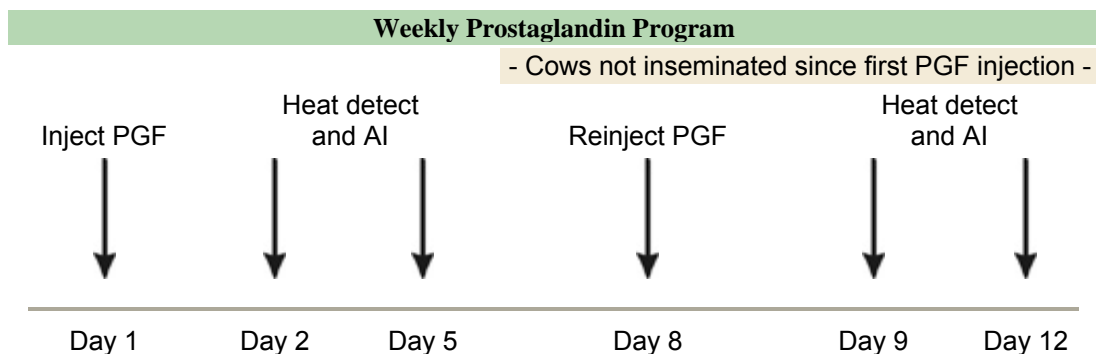
Since Federal labeling of prostaglandin in the mid seventies, many schemes have been investigated to synchronize cattle for breeding purposes. The objective of this presentation is to discuss where we are and where we are going with current synchronization programs, not to review what has been done in the past thirty years.

Prostaglandins have proven to be helpful in bringing groups of animals into heat. Animals must be cycling and heat detection must be efficient for prostaglandin programs to be successful. These programs can be used on cows and heifers, unlike the ovulation synchronization programs (that will be discussed later), which work best on cows.

Remember, heifers are the most fertile animals in your herd and should be bred artificially to genetically superior bulls. Select those bulls known to produce the fewest difficult births. This is a good place to think about using gender selected semen as well. With the new genomics tests, a record number of new sires will be available after the January bull proofs are released.

Prostaglandin procedures require detecting heat. Cows only stand a total of 3 to 5 minutes at a time during their heat period. Heat detection must be done routinely and accurately. Watch for heat three times a day for 15 to 20 minutes each. Heat periods only last 8 to 15 hours and can begin any time throughout the day or night. Animals on concrete are not as active as those on dirt or pasture, and activity is lower while milking or feeding.

Weekly or bi-weekly controlled prostaglandin (PGF) breeding programs are an economical way to use heat synchronization programs. Prostaglandins require a functional corpus luteum (CL) on the ovary for the animal to respond. If the animal is between days 6 and 16 of her cycle, she will generally come into heat 36 to 72 hours after injection of the drug.



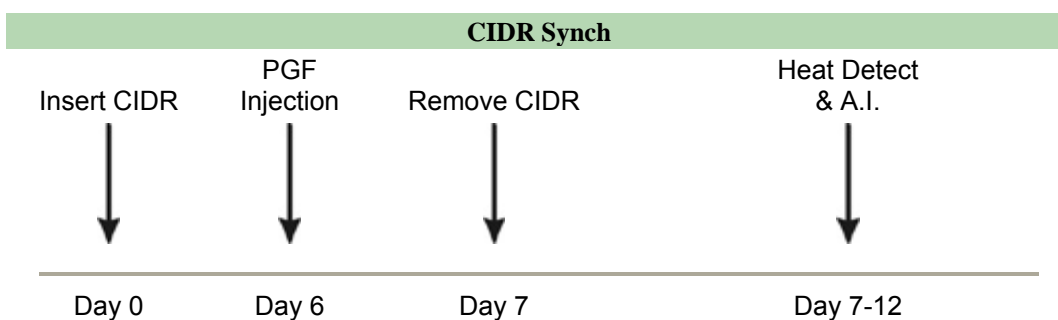
One of the most popular programs is the Monday Morning Program (Pfizer-Pharmacia Animal Health), which recommends you begin with a 30-day postpartum examination as part of a monthly herd health program. All healthy cycling cows 50 days postpartum are candidates. The producer selects a day of the week, usually Monday.

On Monday morning, any cows that are 50 days or greater postpartum are given prostaglandin and checked for heat the remainder of the week. A cow observed in heat during the week is inseminated 8-12 hours later. Most cows will come into heat by Friday. A cow not seen in heat is re-injected the following Monday morning and the same procedure is followed. A cow not observed in heat and inseminated after three weeks of injections is recommended for a reproductive examination.

The benefits of this program are that animals come into heat at a predetermined time, thus aiding in heat detection efficiency. Animals also come in heat in groups, increasing estrus activity and, hopefully, heat detection efficiency. Remember that animals must be cycling for prostaglandins to work.

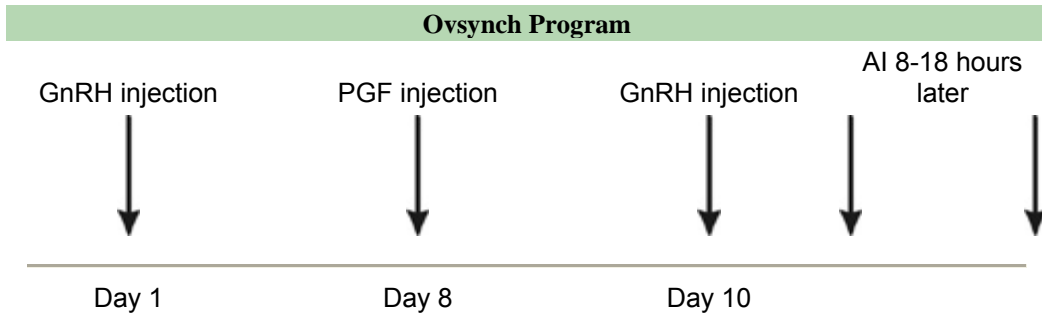
CIDR™ (Pfizer Animal Health) are available as an intravaginal progesterone releasing device. CIDR stands for *controlled internal drug release*. CIDRs have a nylon case with a silicone rubber cover and are designed to deliver natural progesterone slowly over a seven day period to prevent heat expression. CIDRs are approved for both dairy heifers and lactating cows.

These are T-shaped inserts and are placed into the vagina with an applicator that collapses the wings for insertion. An injection of prostaglandin can then be used to bring animals into heat before removing the inserts. CIDRs are easy to apply and remove and have excellent retention rates.



Through the use of ultrasonography, studies examining follicular development have resulted in a method for the synchronization of ovulation (Ovsynch). Many Georgia producers refer to this procedure as "C-L-C." This is based on the trade names of the hormones used (Cystorelin-Lutalyse-Cystorelin). Two injections of Gonadotropin-Releasing Hormone (GnRH) 7 days before and 2 days after prostaglandin (PGF<sub>2a</sub>), will effectively synchronize ovulation in up to 90 percent of lactating cows treated. Time of ovulation occurs 24 to 32 hours after the second injection of GnRH.

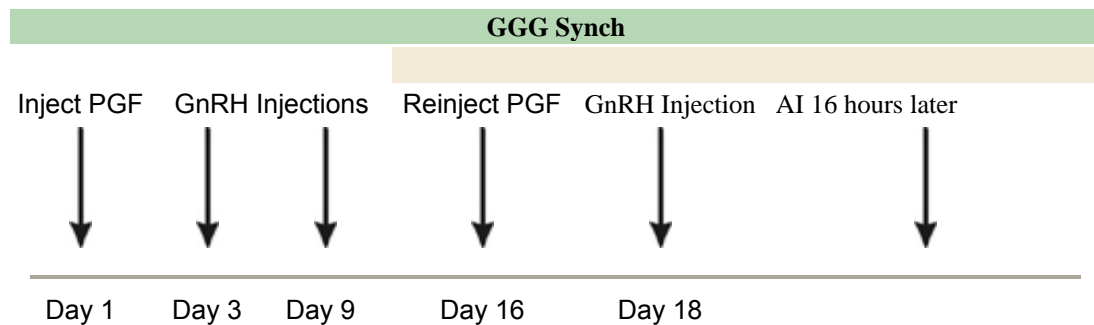
Using this technique provides us the opportunity to breed all animals treated at a designated time. Animals should be bred 16 hours after the second GnRH injection. Note, there is a clear advantage to administering GnRH at 56 hours, 16 hours before a 72 hour AI. Animals between day 5 to 12 of their cycle respond best to Ovsynch. Heifers do not respond as well to this treatment because of possible differences in follicular waves.



Administering two injections of PGF 14 days apart and 12 days prior to initiating the Ovsynch protocol has been shown to improve pregnancy rates. This is referred to as the Presynch program.

Also, a 50 µg dose of GnRH has been shown as effective as 100 µg. This will lower costs. It is important when trying the lower dosage to use a 20 gauge 1½ inch needle with the GnRH and get the entire dose in the animal.

The newest synch developed is the G6G Ovsynch. An injection of PGF is first given, followed 2 days later by an injection of GnRH, then 6 days later another GnRH. In 7 days PGF is injected, then 2 days another GnRH is given, and finally at 16 hours timed AI. Over 15% more pregnancies have been reported using G6G Ovsynch versus Presynch.



Costs of Ovsynch programs are generally higher than the PGF programs. However, if you look at total pregnant animals with lower days after the first service, Ovsynch type programs are the most efficient and lowers the daily demands of heat detection and inseminating animals. The benefit of the program is that 100 percent are inseminated at a set time after calving. CIDR programs are also a little more costly, but provide better results in many cases.

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