Student Clinical Reports

A Practical Approach to the Diagnosis of a Dairy Herd Problem: A Case Report

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Summary

A 390 cow dairy presented in August of 1994 with a complaint of chronic respiratory disease in their milking herd. The history involved numerous management changes over the previous 2 years, including 3 changes in nutritionists, purchase of 37 new heifers from 2 outside sources and construction of a new milking parlor.

An investigation of the herd established that the rolling herd average had declined nearly 6000 lb in the previous year and a half. The dairy had a cumulative incidence of cystic ovaries in a 3 month cohort of cows of 46% and body condition scores were well below desirable goals for the whole herd.

The major problems of decreased milk production and loss of body condition were the focus of the investigation. Although the total mixed ration formulation calculated and recommended by the current nutritionist was deemed to be adequately balanced for each milking string, analysis of the cow rations revealed an energy deficit of 6.03 Mcal/cow/day for the top milking string and comparable deficits for the lower strings.

It was discovered that the auger used to meter out grain for the total mixed ration was miscalibrated. The auger was delivering 22% less grain into the ration than was believed. The producer had the auger calibrated to correct the problem.

A follow up investigation was done nearly 2 years later, in May of 1996, to evaluate the changes in milk production, services per conception, body condition scores and other parameters over a 6 year period. Both rolling herd average and body conditions had improved markedly from the time of the original investigation. This case was used as a general example of how to approach the diagnosis of a herd problem.

Introduction

A 390 cow purebred Holstein dairy in western Washington presented with the complaint of chronic respiratory disease in cows. The cull rate had recently increased due to low milk production and/or suboptimal reproduction. The dairy had lost 4 cows in 2 weeks and they seemed to have no correlation with freshening. Several newly fresh heifers were lame and the owner reported an increased number of lamenesses in adult cows as well.

The clinical syndrome included fever, mucoid nasal discharge, increased respiratory effort and inappetance. Of the 5 adult cow necropsies done in the previous 30 days, one showed lesions consistent with traumatic reticuloperitonitis while the other 4 showed varying degrees of lung consolidation and emphysema. Previous diagnostic work had demonstrated blood selenium levels within normal limits but blood copper levels were not known.[1] No problems were noted in calves or growing heifers.

In 1993, a variety of management changes were implemented. The dairy had changed nutritionists 3 times in the past 2 years (Jan 92, Jan 93 and Aug 93). They stopped feeding a probiotic to all lactating cows in February but continued feeding it to dry cows. At that time, body condition scores dropped dramatically and the cows developed rough hair coats, droopy ears and had underdeveloped udders at time of freshening. The dairy changed nutritionists in March and began bedding with ground gypsum in April. In May the cows reportedly began experiencing increased lamenesses and leg problems. The dairy also began construction of a new milking parlor that month. The parlor was complete and in use by June, 37 new heifers were brought in from 2 outside sources in July and they began feeding the probiotic to all cows again. In August the dairy changed from a 45 to a 60 day dry period and changed nutritionists again in September.

In 1994, the year of the investigation, they stopped bedding with gypsum in January and stopped feeding probiotic in March. First and second strings were started back on probiotic in April and in May there was another reported increase in lamenesses and foot problems. Milking frequency was decreased from 3 to 2 times per day in June. In August, heel pain was observed in several newly fresh heifers and an investigation was done.

At the time of the investigation, the following management practices were in effect.

1. Placement in milking strings was done as follows: All newly fresh cows and heifers were placed in the hospital pen for 3 days. First lactation heifers were then moved to the second string. Second and greater lactation cows were placed in the second string for a 5 day transition period then bumped up to the first string until their milk production dropped to <90 lb/d. The third string consisted of lower producing cows of all ages. The fourth string consisted of cows > 120 days in milk and not confirmed pregnant as well as all "do not breed" cows.
2. Milking cows were fed a total mixed ration (TMR) consisting of rolled barley, haylage and silage formulated to contain the computed values shown in Table 1. Feedstuffs were kept constant from day to day except that the amount of silage fed varied daily depending upon TMR consumption.

Table 1. Rations for June 1994

			Net Energy Lactation (Mcal/kg)	
String	Milk (Lb)	% Fat	Computed	Actual
1	100	3.5	1.78	1.38
2	75	3.8	1.77	1.37
3	60	4	1.68	1.29

3. Cows were fed twice daily with the goal that a small amount of feed be left over at the next feeding; feed was pushed up to within cow reach a minimum of twice daily.
4. Second string received 50 lb TMR and free choice alfalfa hay.
5. Refused feed from milking strings was swept up each morning and fed to dry cows.
6. Far off dry cows were fed 5 lb of rolled barley and 1 lb of vitamin supplement/cow/day plus free choice hay (25% alfalfa:75% grass) and mineral package.
7. Close up dry cows were fed third string TMR and 1 lb/cow/day of vitamin supplement plus free choice grass hay and low Ca:high P mineral package.
8. All haylage and silage were produced on the farm with domestically grown ingredients.
9. Cows and heifers were bred using artificial insemination and a clean up bull was used in some cases. The bull in pasture was noted to be lame in one forelimb and one hind limb.
10. Regular foot care and trimming was done at 3 month intervals.

Both DHIA records and records from the dairy's computer system and nutritionist were collected for analysis. From Figure 1, steady long-term decline in milk production was evident although the dairy manager reported a relatively constant dry matter intake (DMI) and total digestible nutrients (TDN). Services per conception (Figure 2) had steadily increased over the previous 2 years. The risk group appeared to be all cows and heifers presently milking, suggesting a whole herd rather than a point source problem.

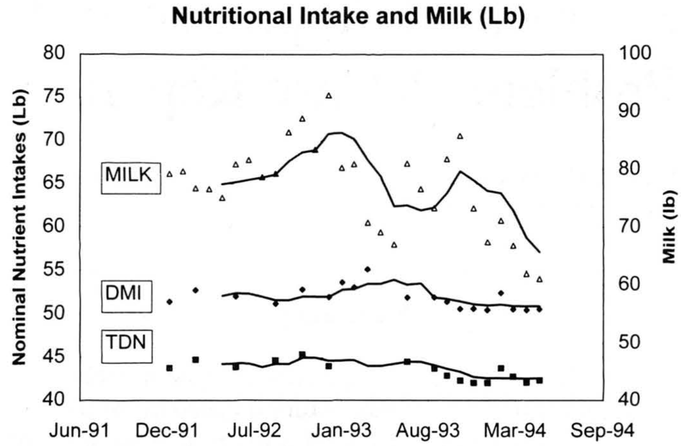

Figure 1. Milk production, dry matter intake (DMI) and total digestible (TDN) in pounds from 1991-1994. Five month rolling averages are indicated by trend lines.

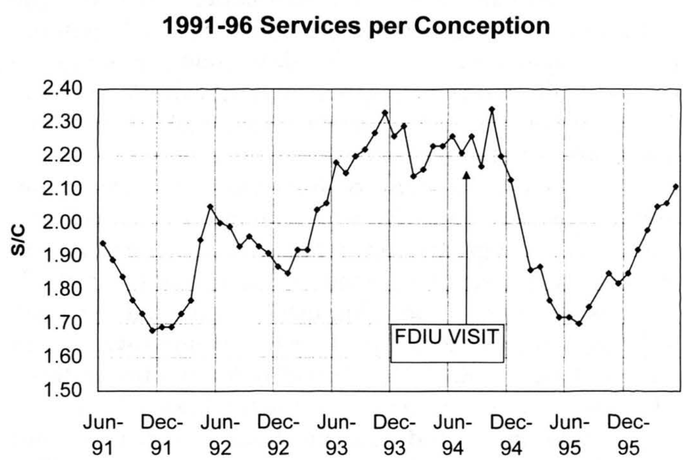

Figure 2. Average services per conception.

Peak milk production of cows in third or greater lactation (Figure 3) had decreased dramatically over the previous year. Dry cows (Figure 5) were freshening at an average body condition score (BCS) of 3, a full half point lower than desired.[1] In addition, cows in later stages of lactation were also displaying suboptimal body condition. [2,3] Dairy records indicated the Jan-Mar 94 calving cohort (n=100) experienced a cumulative incidence of cystic ovaries of 46%.

The major problems identified were decreased milk production and excessive loss of body condition in early lactation. Although the 46% cumulative incidence of cystic ovaries, increased incidence of lameness and chronic respiratory disease could have had separate causes, it seemed likely they were at lease partly related to the primary problems.

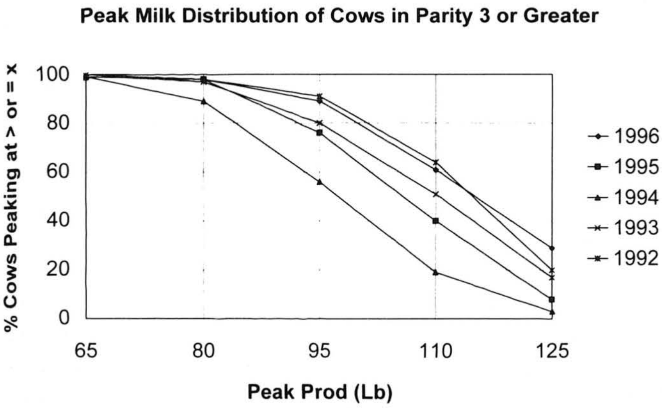

Figure 3. Cumulative percent of cows in parity 3 or greater whose peak milk production fell above 65, 80, 95, 110 and 125 pounds.

In general, differential diagnoses for decreased milk production include inadequate nutrition, reproductive problems, mastitis, inferior genetics and equipment failures. Inferior genetics was easily ruled out because 306 of the 392 cows in the herd had artificial insemination sires with projected transmitting abilities of 967. Somatic cell counts for the preceding year did not indicate the presence of a significant mastitis problem nor did the milking parlor appear to be experiencing equipment failures. Additionally, the decrease in milk production had begun months prior to completion of the new milking parlor. Primary reproductive problems would not have explained the large drop in milk production or the large drop in body condition. Inadequate nutrition, therefore, became the most likely cause.

Due to the paradoxical decline in milk production in the face of reportedly constant DMI and TDN, the peak milk production findings and the suboptimal BCS, a tentative diagnosis of inadequate energy intake was made. Differential diagnoses included a TMR mixing error, an improperly balanced ration and an actual DMI lower than calculated.

The results of samples submitted from two necropsies done during the investigation were non-specific and unrewarding.[3] The nutritionist's recommended rations were deemed adequate so samples of first and second string TMRs were submitted for analysis.[2] For the high string TMR, analysis revealed a net energy lactation (NEL) of 1.38 Mcal/kg instead of the 1.72 Mcal/kg recommended by NRC and much less than the 1.77 Mcal/kg targeted by the nutritionist. The high string ration was reportedly balanced for a 1350 lb cow producing 100 lb of 3.5% fat milk. NRC recommendations for such a cow are 41.05 Mcal and yet the ration provided only 35.02 Mcal. So the cows were being fed a ration which was energy deficient by 6.03 Mcal/cow/day.

It was immediately recommended that the producer check the calibration on equipment used for measuring/weighing TMR components and that he review how to use the mixing sheets and equipment with all personnel involved in making the TMR. The producer discovered that an auger used to meter out the grain was miscalibrated such that TMR was 22% deficient in grain components.

A follow-up investigation done in May 1996 found that services per conception improved by 0.5 (Figure 2) within 10 months following the initial investigation but began rising again and were back at 2.1 by May of 1996. Peak milk distribution in cows parity >2 (Figure 3) steadily improved and the percent of those peaking at >110 lb increased by 20% each year. Figure 4 demonstrates the improvement in body condition of cows in 1996 over 1994. BCSs were back to within recommended ranges for the critical dry and early lactation periods.[2,3]

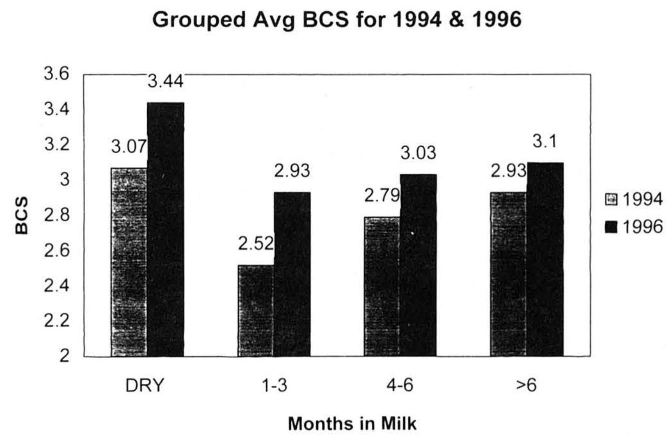

Figure 4. Average body condition scores for dry cows and for cows in 1-3, 4-6 and >6 months of lactation in 1994 and 1996.

Milk production and rolling herd average (RHA) for a 6 year period are shown in Figure 5. Calving trends are evident as seasonal decreases in milk/cow/day. Care should be taken not to misinterpret these seasonal nadirs as resulting from poor management practices when they are actually due to a seasonal calving pattern common in most herds.

Findings in this case strongly suggest that producers and veterinarians not depend solely on calculated nutritional intakes but use those tools readily available to independently assess the nutritional status of the herd. Body condition scoring, peak milk production and TMR feed analysis can be combined to paint a useful picture of nutritional status of a dairy herd.

This case serves as an excellent example of the stepwise procedure required for a definitive diagnosis of any herd problem. Although steps are similar to working up an individual animal case, important differences exist. Initial steps include establishing signalment and

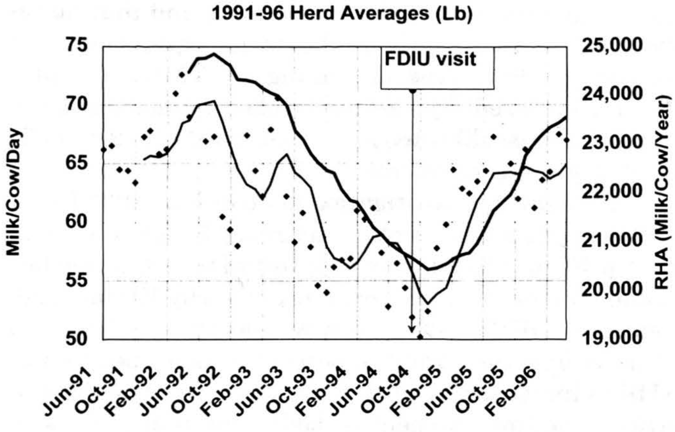

Figure 5. Avg milk production and rolling herd avg over six years. Trend line for milk/cow/day is a 5 month rolling avg.

owner complaint. As in this case, the presenting complaint (chronic respiratory disease) is occasionally not reflective of the major problem (decreased milk production). Compilation of a thorough history is a must, including identification of risk groups within the herd (e.g. parity, lactation stage, geographic location on the farm).

The next step is to do a "physical exam" on the herd. This requires data for the examination of trends over time (e.g. percent distribution of peak milk production by parity, rolling herd averages, body condition scores at different months in milk). A differential list can then be compiled keeping in mind that rule outs = hypotheses = critical control points. Stated differently, rule outs must be management practices which can be changed, not a list of etiologic agents. Diagnostic testing can then be used to specifically target the rule outs. Indiscriminate sampling and testing is a losing proposition since the practitioner must know exactly which questions the tests will answer prior to submitting samples.

When arriving at a definitive diagnosis, caution must be used when multiple problems are present simultaneously. In crisis situations, such as the case in point, the primary problem should be identified and pursued as the top priority, leaving secondary problems to be dealt with later. Happily, the secondary problems often resolve to some degree with correction of the primary problem.

In individual animal cases, the veterinarian often does the treatment but in herd cases, where diagnoses are equivalent to management practices, it is the producer who implements the changes to correct the problem. The veterinarian should make economically sound recommendations and offer expert advice regarding management changes but, ultimately, the "treatment" is in the hands of the producer. The veterinarian's role is critical, however, in future evaluation of trends to determine whether the problems identified begin to resolve and continue in a favorable direction. Practitioners must follow up these herd cases by collecting further data, extending charts and graphs out over time and confirming that trends are improving.

Footnotes

[1]Phoenix Central Laboratory, Everett, WA
[2]Ohio Cooperative Extension Service, Ohio Agricultural Research and Development Center, Research and Extension Analytical Laboratory, The Ohio State University, Columbus, OH
[3]Washington Animal Disease Diagnostic Laboratory, Washington State University, Pullman, WA

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