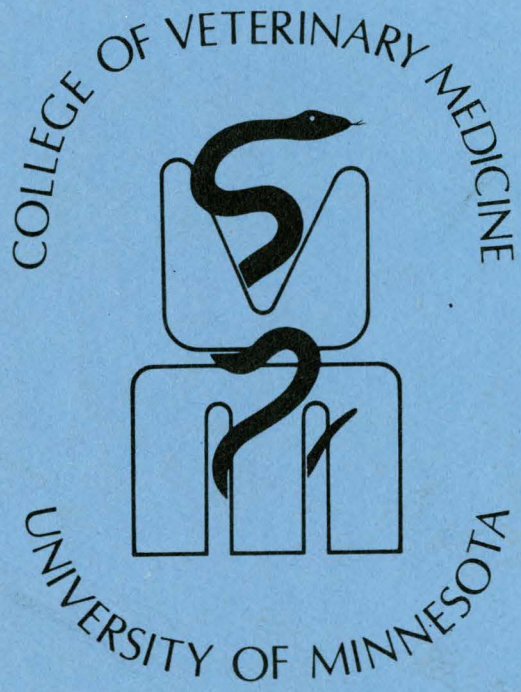
DAIRY HERD HEALTH PROGRAMMING CONFERENCE



JUNE 1 AND 2, 1983

TABLE OF CONTENTS

Identifying Herd Problems

by William G. Olson

Whether a client is on a continuing herd health program or on a call basis, early identification of a herd problem as opposed to individual animal problems will save the client considerable economic loss. Once a herd problem is considered and the causes(s) identified corrective action can be taken. With the emphasis on prevention of disease problems, the first step is to initiate conversation about problems that have been occurring and/or review any records that can document the severity or impact of the problems (i.e., DHI records, milk check stubs, bulk tank wall chart, veterinary invoices or billings, sales of cull cows vs. breeding stock sold, etc.). Intuitive observations and knowing what the usual incidence of various diseases or problems may be (i.e.,milk fever is commonly 1-5% annually and over 10% is cause for concern; all cows don't conceive on first service), are very important in this process. What are the client's disease and production goals?

At this stage you have listed all of the complaints or problems and are synthesizing a presumptive diagnosis. The task now is to determine cause(s) and formulate a corrective or preventive plan(s).

The major problems in dairy cows today occur in the pre- and early post-partum period up to breeding.

No single disease problem is usually present in "herd health situations."

Our major concerns here today are in the area of nutrition, metabolic disease, management, and productivity.

The noninclusive list of problems to consider are as follows:

A. Locomotion - foot rot - swollen joints, sole abscesses
B. Respiratory disease problems
C. Metritis mastitis retained placenta
D. Milk fever, downer cows
E. Ketoses fatty liver or fat cows syndrome
F. Displaced abomasum - milk fat depression
G. Poor production
H. BVD and/or other systemic infectious diseases

Herd reproduction or mastitis problems are not being discussed here but certainly they must be considered in any herd problem solving exercises.

Where do you start in identifying herd problems.

1. Data available
   - DHI or other computer records
   - DMSCC
   - Reproductive records
   - Health records -- vet bills and records and diagnostic lab reports
   - Nutritional information
   - Vaccination program a lack of or what should it be

2. Survey buildings and lots

   Observe building lots pens and how they are being managed

   - ventilation
   - sanitation
   - animal movements
   - pen and stall sizes
   - behaviors and attitude of animals including body condition and size compared to breed standards

Nutritional guidelines which any program should fulfill.

One of the most difficult problems a veterinarian can get into is to say "must be the feed." Some farmers are quick to pick up on any suggestion that the feed is the source of their problem. Certainly feed problems are encountered and can be the cause of production or disease problems. Nutritional problems with disease interactions are also a potential problem and/or may be coincidental.

To evaluate or assess any nutritional program you must be familiar with the NRC guidelines and recommendations. I use the following set of thumb rules and suggest that you can effectively evaluate any recommendations to see if they fall in the ballpark.

Thumb rules to determine if the cows are getting what they are programmed to receive. "Are they getting the recommended ration?"

Energy	40-60% of DM	1 lb. grain to 2.5 lbs of milk 20-25 lbs. of grain/cow/day ave. Ear corn shelled corn (high moisture), barley oats "Too much energy" "Is the bunk kept full?"
Protein		0-8 lbs of SBOM/cow depending on milk production 13-16% protein in total dry matter intake depending on milk production Availability - heat damage - ADIN
Phosphorus and Calcium		4 oz of Dical or its equivalent per cow/day e.g., 100 cow herd - 25 lbs minimum per day
Trace Minerals		1 source either in protein, mineral or salt
Salt		100 cow herd - 20 lbs. per day, 0.5 to 1% of grain mix

Vitamins 50,000 I.U. A and 10,000I.U. D/cow/day

Other Additives - Follow manufacturer's directions (are they logical?)

No matter whose program you are evaluating, these are minimal goals of feeding that must be met for high milk production. Feeding at a greater rate may be beneficial as long as all nutrients and minerals are kept in balance. For the highest producing cows, perhaps balances or ratios of nutrients are even more critical.

Herd Health Management Program Criteria and Goals

by Dr. Jack Cote
from AABP Newsletter, April 1983

	Nos.	%	Goals
I. REPLACEMENTS			
A. Mortality			
1. Birth = calves dead at birth / calves born =	_____	_____	5%
2. 0-30 Days = calves died 0-30 days / calves born =	_____	_____	5%
3. 1-24 Months = calves died 1-24 mos / calves born =	_____	_____	2%
4. Total = calves died birth to 14 mos / calves born =	_____	_____	10%
B. Breeding			
1. Age = interval birth to first service / total heifers =	_____	_____	15 mo.
2. Weight = total weights at 1st service / total heifers =	_____	_____	800 lbs.
3. Height =	_____	_____	50"
C. Calving			
1. Age = interval birth to parturition / total heifers =	_____	_____	24 mo.
2. Weight = total weight at parturition / total heifers =	_____	_____	1250 lbs.
3. Height =			56"
II. REPRODUCTIVE EFFICIENCY			
A. Interval to 1st estrus = calving to 1st estrus / total cows =	_____	_____	45 D

B. Interval to 1st service = calving to 1st service / total cows = _____ _____ 60 D

C. Days open = days calving to conception / total cows = _____ _____ 100 D

D. Calving interval = days calving to calving / total cows = _____ _____ 380 D

E. Services per conception = services in all cows / total conceptions = _____ _____ 2.0

III. **Periparurient Condtions**

A. Milk fever = cows milk fever / total parturitions = _____ _____ 3%

B. Ketosis = cows ketosis / total parturitions = _____ _____ 3%

C. Displaced abomasum = cows displacements / total parturitions = _____ _____ 3%

D. Mastitis = cows mastitis / total cows = _____ _____ 10%

E. DMSCC = _____ _____ 150,000

WISPLAN Computer Ration Formulation Increases Feed Efficiency and Dairy Farm Profit

W.T. Howard, Scott Hendrickson, Richard Lcroix and Al Wollenzein

Introduction

The WISPLAN dairy ration formulation programs began in 1971 with the use of the Michigan TELPLAN Least Cost Dairy ration program in Wisconsin. The Michigan program was used via a touch tone phone and the Michigan computer that responded with all input and output in a voice generated by the computer. That voice generator cost nearly a quarter of a million dollars and now can be obtained in a better version for a few hundred dollars. Rations were formulated for 22 farms in the early part of 1971. The cooperating farms reported a 5 to 6% reduction in feed costs. In 1983 terms their results translates to a $0.50 to 0.60 cost/100 lb milk reduction in costs to produce milk. In 1972 several Wisconsin counties were equipped to access the Michigan TELPLAN programs. The dairy rations gained in acceptance from farmers and continued to demonstrate the value to dairy farm operators found in the original sample of farmers that consented to try the programs rations in their herds. Use of the Michigan TELPLAN system continued until 1978 when the phone costs and other factors resulted in the development of the program now known as the WISPLAN LCD or Least Cost Dairy ration formulation program.

In the late 1960's and early 1970's the University of Wisconsin Soil and Plant analysis laboratories developed and encouraged feed and forage analyses for use in ration formulation. Feed and forage analyses progressed to several thousand samples by early 1972 at the Madison and Marshfield labs. At a meeting called by Professor Leo Walsh, Soils Department (now Dean of the College of Agricultural and Life Sciences) concern about the

apparent failure of follow through in farmers using the feed analyses after having them tested was expressed. As a result of that meeting the Badger Balancer was developed and introduced in Wisconsin in January 1973 for use by Wisconsin dairy farmers. The Badger Balancer was run on the large computer located on the UW-Madison campus using computer punch cards. The Badger Balancer program as all the WISPLAN programs are now available on a dial-access basis to all the Wisconsin County Extension offices. In recent months the availability of the WISPLAN Computing System on a direct access external user basis has increased the potential for more use by Wisconsin dairy farmers and agri-business firms. Currently WISPLAN external users include feed dealers, veterinarians, farmers, a large potatoe grower and processor, Vocational Agriculture departments in high schools, credit agencies, VTAE districts and others.

The use of the WISPLAN LCD and BADGER BALANCER programs have progressed to over 500 rations formulated per month for the Wisconsin diary industry. These programs have proven their worth to the dairy farmers time after time and continue to play an effective role in helping maintain dairy farm profits.

Professor-Dairy Science, Associate Professor-Manitowoc Agricultural Agent
Associate Professor-Fond du lac Dairy Agent,
Associate Professor-Dane County Dairy and Livestock Agent, University of Wisconsin-Extension

Results of Computer Formulated Ration Use

1971 User Response Survey

The first 22 farmers that tried the computer formulated rations were asked to evaluate the results in their herds. These farmers were in many

instances skeptical about the idea of using the computer to make feeding recommendations. Their fears were generally overcome during the trial period. the flexibility of the first program to fit the feeding program to the farmers needs and desires left a lot to be desired. However, the cost of the dairy rations were reduced by about 5 to 6%. The increased milk production realized on these farms was about 2.5 times that of the average Wisconsin dairy herd in the Wisconsin DHI program. One third of the farms in the first group of 22 farmers had increased feed costs due costs due to the need to purchase additional protein and mineral supplements needed to balance their herd's rations. These farmers were where some of the greatest increases milk production and profit increases were realized. This helped dispel the idea that least cost ration formulation meant use of cheap or poor quality feeds that did not maintain milk yield. The computer has only helped demonstrate that "You Can't Starve A Profit From A Good Cow or Feed A Profit From A Poor Cow".

1975-1976 User Results

By 1975 several county agents were using the TELPLAN Least Cost Ration program and the Wisconsin Badger Balancer program to assist farmers in increasing dairy farm profits and utilize feed and forage analyses. The results of the survey of users of the Badger Balancer rations had DHI herd average increases of 720 lb milk/year while the DHI state average increased by 442 lb milk/year. Of the 76 respondents to the survey 73.7% indicated increased milk/cow while only 26.3% indicated decreased production. Nearly half of the respondents with lower production indicated that no ration changes were made as per the recommendations. Garry Blomberg, Green County Agricultural Agent at that time, documented changes in income above feed

cost on a sample of 8 Green county farms. Their income above feed costs increased by $100 to $320 per month for each herd.

1975-1976 Least Cost Ration Use Survey

Nearly one third of the Forest-Florence county dairy farmers had Least Cost Rations formulated by Scott Hendrickson, Agricultural Agent (now Agricultural Agent-Manitowoc County). These farmers realized an average decrease in feed costs of $35 per cow/year. They all reported either increased or maintained production levels. The $35/cow represented over $0.30 reduced costs per 100 lb milk or an increased profit of over $1200 per farm. Dennis Nehring (Walworth Agricultural Agent-now Rock Agricultural Agent) and Al Wollenzein (Sheboygan Dairy and Livestock Agent-now Dane County Dairy and Livestock Agent) both had outstanding reports from their participating farmers. Milk production increases ranged from 350 lb/cow/year to over 2000 lb/cow/year as measured by their DHI or O.S. herd records.

1978 Ration Use Survey

Following the 1976 drought the Wisconsin Legislature funded a project starting in mid-1977 to assist farmers to recover from the effects of the drought. In 1978 dairy farm operators who used the LCD and Badger Balancer rations were again surveyed as to the effect of use of the computer rations and forage testing.

More than 80% of all respondents indicated that use of computer ration formulation helped establish feed and forage testing as a routine part of their management practices. Nearly 90% indicated that it also helped them to recognize the need to change rations as forage type and quality changed.

Eighty eight percent of the farm managers said they followed entirely or in part the computer ration feeding program.

Table 1. Effect of Computer Ration Balancing on Changing Dairy Farm Enterprise EFficiency

	----------% of All Survey Responses----------		
Farm Profit	84	4	12
Milk Yield/Cow	67	2	31
Purchased Feed	30	51	19

It is important to note that 84% with increased profits was obtained with only 67% having increased milk production. Since only 30% had increased purchased feed costs it was apparent that adopting balanced rations for the entire herd caused a redistribution of grain and supplements in order to effect the changes reported. The LCD program was more effective in causing a reduction in feed costs, 67% with reduced purchased feed costs versus the Badger Balancer users with 40% having reduced purchased feed.

Table 2. Yield and Profit Changes by Users of LCD and Badger Balancer Rations

Ration Type	Production Change**	Profit Change**
Badger Balancer	1040 lb/Cow/Year	72/Cow/Year

These results once again confirmed the value of the computer formulated rations and ration balancing in general. Numerous farms reported with pride and sometimes amazement the small amount of protein supplement needed when they had the high quality legume forage in their feeding programs. One of the most satisfied user of the rations had been able to develop an excellent alfalfa production program but had not changed the feeding program. That farm realized a $1500/month decrease in purchased feed for their 80 cow dairy herd.

Summary and Conclusions

1. The WISPLAN LCD and Badger Balancer programs have demonstrated a clear and definite potential to increase dairy farm profits. The 1978 survey results indicated a cost reduction of more than $0.50/100 lb milk produced. A significant number of the users of the WISPLAN computer formulated rations increased profit without increasing milk yield.
2. The WISPLAN ration programs are available in all Wisconsin counties via phone dial-access and printing computer terminals for improved availability and turn-around time. This reduces the waiting for rations following analyses. Hopefully, the Near Infra Red Analyses equipment coupled with the WISPLAN programs can more efficiently serve the Wisconsin dairy industry.
3. Educators on an external user basis will further increase the use of feed analyses and ration balancing and thereby increase dairy profits.
4. Milk prices are expected to drop by $0.50 to $1.00 per 100 lb during the the next year. Dairy farm managers and the agri-business firms serving these farms must work together to produce milk as efficiently as possible. The dairy industry must stress profit per cow and strive diligently to find strategies to maintain profit without increasing total milk production.
5. The following actions can help maintain farm profit without increasing total farm milk production and may result in equal profits from less cow
   (1) If not on DHI or O.S. test get started now!!! Failure to have good records prevents efficient feeding and culling of cows not contributing to dairy profits.
   (2) Striving for production of high yields and high quality legume forage must be the number 1 crop production priority of all dairy

managers. Efficient milk production cannot be achieved from poor quality forages. Farms able to produce, store and feed high quality forages will be those able to withstand the possible drop in milk prices without threatening the very existence of the farm.

(3) Dairy managers must use feed and forage analyses to be able to accurately and efficiently balance dairy rations.

(4) Regular use of ration balancing such as available through University of Wisconsin-Extension's WISPLAN and many of the feed companies may be the difference between profit and loss during these next few trying years in the dairy industry.

(5) The ability of dairy farmers in the upper midwest to produce high quality legume forage and grains will serve to make them among the most profitable and efficient farms in the United States and the world.

References

1. Howard, W.T. and D. Rohweder, 1978. Forage tests and computerized balanced rations really play. Ext. Bull. A7780109.U.W.-Ext.

2. Howard, W.T., D. Mehring, A. Wollenzein, S. Hendrickson, D. Drost and D. Olson. 1978. Using computerized ration balancing to assist dairy farm operators during a drought emergency. Paper P-913. Presented at 73rd Annual Meeting of the American Dairy Science Assn. July 10-13, 1978. Michigan State University, East Lansing.

3. Howard, W.T. and John R. Schmidt. 1981. Paper P-60 Presented at the 1981 American Dairy Science Assn. Annual Meeting. June 1981. Baton Rouge, La.

Minnesota Dairy Ration Balancer

Minnesota Dairy Ration Balancer

Extension Folder 292—Revised 1977
J.G. Linn and G.R. Steuernagel

The Minnesota Dairy Ration Balancer is a computerized method of calculating a nutritionally balanced ration for your dairy cows and growing heifers based on forage quality, amount of various forages fed, and available grains on your farm.

WHAT DO YOU GET?

A balanced grain mixture for the level of milk production, fat test, and body weight you specify.

A grain feeding guide for production of from 10 to 100 pounds of milk (high moisture corn, top dressed protein, two-grain feeding program, or one-grain mix).

A balanced grain ration including energy, protein, mineral, and vitamin needs for lactating and dry cows and growing heifers.

Calculation of DHI (Dairy Herd Improvement) forage energy codes and other economic and nutritional information about your computerized grain and forage ration.

WHAT DOES IT COST?

One computerized ration costs $2.00. If you want to consider another alternative, it will cost an additional $1.00 per ration. Be sure your check, which should be made payable to the University of Minnesota, is included with the form. You can request as many as you want (just attach the information).

WHERE DO YOU SEND IT?

Be sure all the information on the input sheet is complete. Mail the input sheet with your payment to:

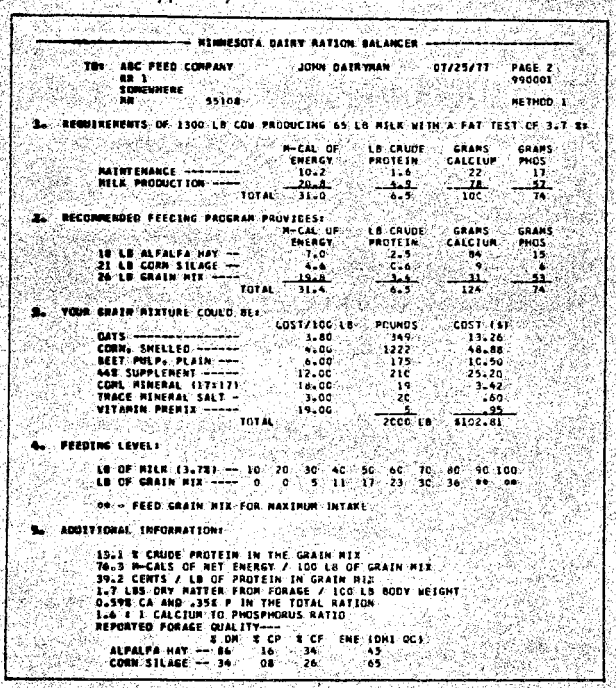
Dairy Extension,
Dept. of Animal Science
101 Haecker Hall
University of Minnesota
St. Paul, MN 55108

FILLING OUT THE FORM

The input form has been revised to make the program easier to use and complete. The form is on the back side. BE SURE TO COMPLETE THE REQUIRED SECTIONS.

Complete the identification section; be sure your zip code is entered. The extra identification blank allows you to add a specific name or number. Below are comments to help complete the form. For each ration requested, complete a column indicating changes desired (add additional columns if more than two rations are wanted).

1. **Type of ration.** Circle one class of dairy animal for which a ration is desired.
2. **Body weight.** Enter average weight of animals.
3. **Milk production.** Enter pounds of milk and fat test (add 10 pounds above the herd average).
4. **Forage quality.** Enter the wet pounds of each forage consumed. An estimation of quality is needed: a) If forage is tested, list the name or type and attach a copy of the forage test results. b) If forage is not tested, list type of forage (½ alfalfa and ½ brome grass hay silage) and stage of maturity at cutting (1/10 bloom).
5. **Protein Supplement.** Circle the method or way protein is fed. List the percent protein and price per hundred (cwt) of your protein supplement. Attach a feed tag if available.
6. **Grain Mixture.** Enter the percent of each grain or commercial mixture used. (Do not include protein supplement, minerals, or vitamins.) Describe the type of grain (oats, ear corn, shelled corn, beet pulp, molasses, urea, etc.) and price per hundred pounds (indicate if high moisture grains are used). Attach any feed tags. The percentages must add up to 100.
7. **Optional Group Fed Grain.** This refers to situations where grain or supplements are blended with forage, spread on top of forage, fed in a bunk, consumed off a lick wheel, or a magnetic grain feeder is available to all cows. List the pounds per cow, dry matter, percent crude protein (C.P.) on a 100 percent dry matter (D.M.) basis, and type (high moisture ear corn, liquid urea-molasses, etc.).
8. **Optional Calcium-Phosphorus Mineral.** This allows you to enter your mineral (commercial or base ingredient, i.e. dicalcium phosphate). List the percent calcium and phosphorus, name, and price per 100 pounds. Again attach a feed tag. Only one mineral is allowed. The computer will select the correct mineral type if you do not enter one.



MINNESOTA DAIRY RATION BALANCER

TO: ABC FEED COMPANY — JOHN DAIRYMAN — 07/25/77 — PAGE 2
RR 1 — 990001
SOMEWHERE
MN 55108 — METHOD 1

1. REQUIREMENTS OF 1300 LB COW PRODUCING 65 LB MILK WITH A FAT TEST OF 3.7 %

	M-CAL OF ENERGY	LB CRUDE PROTEIN	GRAMS CALCIUM	GRAMS PHOS
MAINTENANCE	10.2	1.6	22	17
MILK PRODUCTION	20.8	4.9	78	57
TOTAL	31.0	6.5	100	74

2. RECOMMENDED FEEDING PROGRAM PROVIDES:

	M-CAL OF ENERGY	LB CRUDE PROTEIN	GRAMS CALCIUM	GRAMS PHOS
10 LB ALFALFA HAY	7.0	2.5	84	15
21 LB CORN SILAGE	4.6	0.6	9	6
26 LB GRAIN MIX	19.8	3.4	31	53
TOTAL	31.4	6.5	124	74

3. YOUR GRAIN MIXTURE COULD BE:

	COST/100 LB	POUNDS	COST ($)
OATS	3.80	349	13.26
CORN, SHELLED	4.00	1222	48.88
BEET PULP, PLAIN	6.00	175	10.50
44% SUPPLEMENT	12.00	210	25.20
CORL MINERAL (17:17)	18.00	19	3.42
TRACE MINERAL SALT	3.00	20	.60
VITAMIN PREMIX	19.00	5	.95
TOTAL		2000 LB	$102.81

4. FEEDING LEVELS:

LB OF MILK (3.7%)	10	20	30	40	50	60	70	80	90	100
LB OF GRAIN MIX	0	0	5	11	17	23	30	36	**	**

** = FEED GRAIN MIX FOR MAXIMUM INTAKE

5. ADDITIONAL INFORMATION:

15.1 % CRUDE PROTEIN IN THE GRAIN MIX
76.3 M-CALS OF NET ENERGY / 100 LB OF GRAIN MIX
39.2 CENTS / LB OF PROTEIN IN GRAIN MIX
1.7 LBS DRY MATTER FROM FORAGE / 100 LB BODY WEIGHT
0.59% CA AND .35% P IN THE TOTAL RATION
1.6 : 1 CALCIUM TO PHOSPHORUS RATIO
REPORTED FORAGE QUALITY—

	% DM	% CP	% CF	ENE (DHI OC)
ALFALFA HAY	86	16	34	43
CORN SILAGE	34	08	26	65

Sample output you will receive.

INPUT SHEET -- MINNESOTA DAIRY RATION BALANCER

- Send to: Dairy Extension, 101 Haecker Hall
  University of Minnesota
  St. Paul, MN 55108
- Enclose a check ($2 for one ration and $1 for each additional ration requested). Complete a new column for each additional ration. Make checks payable to the University of Minnesota.
- Attach feed tags and forage test results.

Name_____

Address_____

_____
Zip Code

County_____ Phone_____

Extra Identification_____

Sample

1) Type of ration (circle) — Ration 1: Milking cow / Dry cow / Heifer

2) Body weight: 1300 lb

3) Milk production (leave blank for dry cows and heifers): Lb Milk 65; % Fat 3.7

4) Forage quality and type

Name	Quality	
ALFALFA HAY	1/4 bloom (June 5)	18 lb
CORN SILAGE	Pulident, normal	21 lb
		lb
		lb

5) Protein supplement (circle): Topdress / mixed with grain
Name SOYBEAN MEAL; % CP 44; Price/cwt 12.00

6) Grain mixture

Name	Price	
OATS	3.80/cwt	20 %
Shelled CORN	4.00/cwt	70 %
BEET PULP (plain)	6.00/cwt	10 %
		%
		%
		%

7) Optional group fed grain: Name_____ lb/cow; % D.M._____ % C.P._____

8) Optional calcium-phosphorus mineral: Name SUPER 17-17; Price/cwt 18.00; % Ca 17; % Phos 17

	Ration 1	Ration 2
1) Type of ration (circle)	Milking cow / Dry cow / Heifer	Milking cow / Dry cow / Heifer
2) Body weight	_____ lb	_____ lb
3) Milk production (leave blank for dry cows and heifers)	Lb Milk_____	Lb Milk_____
	% Fat_____	% Fat_____
4) Forage quality and type		
Name _____ Quality _____	_____ lb	_____ lb
Name _____ Quality _____	_____ lb	_____ lb
Name _____ Quality _____	_____ lb	_____ lb
Name _____ Quality _____	_____ lb	_____ lb
5) Protein supplement (circle)	Topdress / Mixed with grain	Topdress / Mixed with grain
Name_____	% CP_____	% CP_____
	Price/cwt_____	Price/cwt_____
6) Grain mixture		
Name _____ Price _____	_____ %	_____ %
Name _____ Price _____	_____ %	_____ %
Name _____ Price _____	_____ %	_____ %
Name _____ Price _____	_____ %	_____ %
Name _____ Price _____	_____ %	_____ %
7) Optional group fed grain		
Name_____	_____ lb/cow	_____ lb/cow
% D.M._____ % C.P._____		
8) Optional calcium-phosphorus mineral		
Name_____		
Price/cwt_____		
% Ca_____ % Phos_____		

NUTRITION: "FANTACIES AND FALLACIES"

James W. Crowley
University of Wisconsin-Extension
Department of Dairy Science

Two words that should not be used in biological processes are "always" and "never." There are exceptions to the most proven fact and eventually the most unexplained will happen. Therefore in thinking about the general subject of "fantacies" and "fallacies," it is essential to emphasize that there are very few absolute fantacies or fallacies in nutrition. Certainly veterinarians are aware of the exceptions that occur between the basic textbook fact and what happens on the farm. "If anything can go wrong, it will." There are fantacies and fallacies in veterinarian medicine and there are common fantacies and fallacies in nutrition. Also exceptions and lack of total knowledge does not justify burning the veterinary textbooks or the nutrition books. Well trained veterinarians and well trained nutritionists offer the best hope for improving current disease and nutrition problems. Future research may prove some present recommendations wrong. But using the best information available is a lot better than following the fantacies and fallacies which will even more likely be proven wrong.

Nutrition is a Science. For more than 150 years chemists, biologists, physiologists, nutritionists, bacteriologists and veterinarians have been studying and researching the general subject of nutrition. To even list all the important discoveries or milestones would require more time and space than we have available. With each major discovery one or more fantacies or fallacies are usually proven wrong. Frequently the discovery of a new requirement is only the beginning of hundreds of additional research studies. Vitamin D for example was first discovered in 1922 but was the subject of numerous research studies in the last year. Availability from various sources, toxic effects of excessive amounts, exact requirements of different animals and made of specific activity still leave room for fantacies and fallacies. However, the basic knowledge and experience has been incorporated into sound feeding standards such as National Research Council. Using the suggestions given by the committee is the best way to avoid a fallacy. New research as well as experience should be used to modify standards slightly but dramatic changes are not likely. In using feeding standards read the text and explanation as well as the tables. In some cases allowances for variation in feed composition and extra for insurance against deficiencies are included in the table. For other species extra may not be included. The text will also indicate if additional amounts are needed for various conditions.

Nutrition, One Link in the Chain. Animal performance is a combination of genetics, management, disease control, parasite control, nutrition, environment and other factors. Diagnosing the cause of a problem is the most essential step to a solution. The greatest fallacy in nutrition is using feed changes or additions to solve management deficiencies. Poor observations and inadequate records are the most common cause of lack of heat in cows. But changing the ration is often the cure that is tried.

Feeding will not correct a low production caused by using genetically poor bulls.

The Major Nutrients of 1st Importance. Protein, energy (digestible organic matter and fat), and undigested fiber account for about 97% of the nutrient requirement of the high producing cow. Major minerals account for about 2.8 percent and trace minerals and vitamins 0.2 - 0.3%. One of the fallacies is forgetting to look at the forest instead of the little trees.

Important Nutrients Have Limits. Most rations are either dificient in several nutrients or adequate in all. Very seldom do we find a ration that is deficient in one specific trace mineral or vitamin that accounts for a specific problem. If a nutrient is missing expect several general symptoms including poor milk production, low growth rate, unthrifty appearance and poor appetite. One or more of these problems plus a specific problem might be helped with a specific nutrient. However, do not promise too much. Each new nutrient discovered often looks like the final missing link but so far each has failed to cure-all. A Missouri biochemist used vitamin A to illustrate the life cycle of nutrients. Briefly each goes through five stages. First is the discovery of a needed compound. Second is over-expectation of its importance. Third is realistic and routine use in the ration. Fourth, it is so well known, accepted and used that it is forgotten. Fifth, it is rediscovered and the cycle repeats.

If a Little is Good - More is Better. A common fallacy that has received much attention recently concerns excess nutrients rather than deficiencies. Iodine, selenium and copper are examples of trace minerals that are essential in small amounts but toxic at high levels. Vitamins A and D are similar examples for vitamins. Excess calcium especially for dry cows can be more harmful than slight deficiency. Even excess energy and excess protein have been shown to be a detriment. Shortages or excesses of required nutrients can be avoided by using balanced ration and allowing extra only in moderation.

If a Lot is Bad a Little is Bad. This fallacy is just the opposite of the previous one but is primarily of concern for drugs or contaminants rather than an essential nutrient. We have all read stories about compounds such as P.C.B. and steroids causing cancer in mice. The assumption is that if a lot causes cancer a little might. Although it may be pointed out that you need to eat several tons of feed to get the dose proven harmful there is still the fantacy for perfection.

For animals, examples include the proven safety of recommended levels of urea, nitrates, antibiotics and ionophores. Yet there is the fallacy of avoiding these and other products because of fear.

SUMMARY - The best solution to nutrition is a ration balanced in all known nutrients. If problems still exist the cause might be an unknown nutrient but is more apt to be some other biological cause.

The Periparturient Sick Dairy Cow Complex

O.M. Radostits, June 1983

The peri-parturient sick dairy cow complex occurs in cows within a few days before but more commonly after calving and is characterized by inappetence to complete anorexia and a variable reduction in milk yield. Sometimes the cause of the illness is obvious such as severe mastitis. However, often the cause is not obvious or there may be more than one abnormality present and it may be difficult to decide which abnormality, if any, is the cause of the illness. For example, there may be a retained placenta, a mild clinical mastitis and a 2^+ ketonuria. Does the cow have metritis, mastitis, or some other abnormality which is not obvious? It is usually difficult to determine the contribution of each abnormality to the illness or to priorize the abnormalities. Furthermore, not all abnormalities are easily detectable clinically (i.e. fatty liver, abomasal displacements, peritonitis).

The clinician is faced with a difficult diagnostic problem. Only by repeated thorough clinical examinations can he identify and distinguish from each other the abnormalities which contribute to the illness.

The table below summarises the salient features of the diseases which occur commonly in the dairy cow around the time of parturition.

Based on clinical experience, the abnormalities causing illness in the periparturient dairy cow will likely be present in the following body systems:

1. Mammary gland

   Acute and peracute mastitis.

2. Alimentary tract

   (a) Rumen-Simple indigestion.
   (b) Reticulum-Traumatic reticuloperitonitis.
   (c) Abomasum-Left-Side displacement, right side dilatation, displacement and torsion.
   (d) Liver-Fatty infiltration and fatty degeneration.
   (e) Intestines.

3. Reproductive Tract

   Retained placenta.
   Toxemic Metritis.

4. Body as a Whole

   Hypocalcemia
   Acetonemia

Abnormalities causing the illness are unlikely to be present in the following body systems:

Cardiovascular

Respiratory

Nervous

Musculoskeletal

Skin

Urinary

If a complete clinical examination does not reveal the abnormalities which are causing the illness, the animal should be re-examined with an emphasis on the body systems which are likely to be affected. Those body systems which are found to be normal can be ruled out and those which are abnormal can be re-examined again. Some simple field tests may assist in the detection of certain abnormalities such as ketonuria and proteinuria.

The Differential Diagnosis of the Peri-Parturient Sick Dairy Cow Complex

Name of Disease	Salient Epidemiological and Clinical Findings	Diagnosis		Prognosis and Treatment
		In The Field	In The Laboratory	
1. Toxic Metritis	Toxemia, fever (but not always), anorexia, rumen stasis, tachycardia, copious amounts, of foal-smelling liquid brown to seronsanguinous uterine contents usually with a retained placenta uterus enlarged and flaccid. Diarrhea due to toxemia may occur. Weakness and recumbency may occur.		Marked degenerative leftshift in leukon.	Prognosis usually favourable with parenteral and intra-uterine antibiotics and supportive fluid & electrolyte therapy.
2. Peracute & Acute Mastitis	Toxemia, anorexia, tachycardia, fever initially (but may be normal 12 to 18 hours after onset), one or more quarters of mammary gland enlarged, warn and firm, may be gangrenous, contains grossly abnormal secretions (watery, serous, purulent, gas, bloody, with flakes). Weakness and recumbency are common with peracute coliform mastitis. Diarrhea due to toxemia may occur. Experimentally, a severe toxemia occurs 10 hours following the introduction of E. coli into the gland. The milk still appears grossly normal at 10 hours and changes are only recognizable at 15 hours.	Strip cup with black surface. California mastitis test is 4+ before gross changes in milk are visible.	Marked degenerative leftshift in leukon. Culture milk.	Prognosis variable depending on duration of mastitis before treatment begins. In early stages the prognosis is usually good. When cow is weak and recumbent, prognosis is poor.

Name of Disease	Salient Epidemiological and Clinical Findings	Diagnosis		Prognosis and Treatment
		In The Field	In The Laboratory	
3. Fat Cow Syndrome (fatty liver)	Depressed but not toxemic. Inappetence to complete anorexia. Marked drop in milk production. Body score 4 or 5 out of 5. Marked Ketonuria (4^+). Rumen stasis, scant feces, occasionally small volume soft feces. Heart rate 80-90/ min; respiratory rate 40-60/min. Weakness and recumbency may occur in 2 to 4 days after onset.	Ketonuria 3 - 4^+.	Liver biopsy. Stress leukon. Serum liver enzymes (SGOT SDH). Blood glucose. May be subnormal, normal or higher than normal.	Prognosis unfavourable in cows which are completely anorexic. Those cows which are inappetent will usually recover if offered good hay and treated for Ketosis with glucose and propylene glycol.
4. Left side displacement of abomasum (L.D.A.)	Moderate state of ketosis in a cow few days postpartum. Inappetence usually. Rarely complete anorexia. Characteristic fundings associated with LDA; rumen sounds not clearly audible, "ping" by percussion & auscultation over upper half of 9th to 12th rib.	Ketonuria	Ketonuria Decreased blood glucose Laboratory findings of fatty liver may be present if secondary to L.D.A.	Omentopexy and treatment for secondary ketosis.
5. Traumatic Reticulo-peritonitis	Inappetence to complete anorexia. Mild fever 39.0 - 39.5°C. Rumen movements markedly decreased or absent. Size of rumen gas cap may be increased. Grunt on deep palpation of xiphoid sternum or pinching of withers. Amount of feces reduced.		Regenerative left shift or may be normal white blood cell count and differential. Paracentesis for peritoneal fluid.	Prognosis is good, about 80% will recover following antimicrobial therapy daily for 3-5 days along with immobilization in a stanchion to assist healing. A magnet is also recommended.

Name of Disease	Salient Epidemiological and Clinical Findings	Diagnosis		Prognosis and Treatment
		In The Field	In The Laboratory	
6. Chronic reticulo-peritonitis	Inappetence. Some milk production. Normal temperature, heart and respiratory rates normal. Rumen movements decreased in frequency and amplitude. Feces scant. Usually no grunt on deep palpation. Ketonuria due to secondary ketosis may be present.	Nothing useful.	Neutrophilia may be present but leukon may be normal. Paracentesis for peritoneal fluid may be difficult to obtain in chronic peritonitis.	Treat with broad spectrum antimicrobials daily for 5 days.
7. Vagus Indigestion	Inappetence to anorexia dehydration, progressively enlarging abdomen, enlarged rumen hyperactive or later atonic, scant feces.	Nothing useful.	Leukon variable. Metabolic alkalosis with hypokalemic hypochloremic alkalosis.	Slaughter for salvage. Exploratory laparotomy and/or rumenotomy.
8. Right Side Dilatation, Displacement of the Abdomen	Usually occurs several days to a few weeks after calving. Marked drop in milk production. Anorexia, inappetence, rumen movements decreased, reduced amount of feces, ping over right paralumbar fosso, maybe able to palpate distended abomasum in right lower quadrant rectally. Temperature normal, heart rate normal to increased to 80/min. Dilatation and Torsion Phase: Much more severe. No milk production. Heart rate 90 to 120/min, peripheral circulatory failure, size and loudness if ping have increased, right paralumbar fossa may be distended, rectally can usually feel tense distended abomasum in middle third of right flank.	Urine pH may be acid due to paradoxic aciduria	Serum biochemistry indicates metabolic alkalosis with hypochloremia, hypokalemia	Prognosis depends on severity. Heart rate over 100 per minute and a serum chloride conc. of less than 79 mEq/L. indicates a poor prognosis. Recovery is excellent following surgical correction in the early stages. Early cases with small amounts of gas will recover spontaneously in a few days.

Name of Disease	Salient Epidemiological and Clinical Findings	Diagnosis		Prognosis and Treatment
		In The Field	In The Laboratory	
9. Abomasal Ulcers	Abomasal ulcers may be bleeding or non-bleeding in high-producing cows within few days after calving. Anorexia, no milk production, rumen stasis, dehydration, scant black tarry feces, or normal colored feces, ballottement of right flank may reveal fluid splashing sounds.	Feces are usually black and tarry when ulcer bleeding. Hemetest tablets for occult blood in feces when ulcers not bleeding grossly.	Hemmorrhagic anemia.	No specific treatment. Prognosis poor for bleeding ulcers.
10. Simple Indigestion	Inappetent or anorexic. No systemic abnormalities. Rumen full but movements decreased or absent. Reduced amount of normal range feces passed and in rectum.	Nil	Nil	Usually recover in 2 days. Administer Mg OH or mineral oil orally.
11. Subclinical Hypocalcemia	Anorexic. Temperature normal to slightly subnormal. Heart rate normal range but amplitude of sounds decreased. Rumen static. No feces passed but dry mucus coated feces may be present in rectum. May be recumbent but will usually stand if urged.	Nil	Serum levels of calcium are decreased.	Prognosis usually excellent following treatment with calcium borogluconate.
12. Primary Acetonemia	Usually in first 2-4 weeks after parturition. Inappetent. Milk production decreased by ½ to one third. Temperature, pulse and respiratory rates normal. Amplitude and frequency of rumen movement decreased, dry mucous coated feces.	Ketonuria 2+-4+	Same as field test. Blood Ketones. Blood glucose.	Dextrose 50%. 500-1000 ml I.V. Propylene glycol orally. Corticosteroids occasionally

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13. Downer Cow	May be a complication of delayed treatment for milk fever or muscle injury which occurred from fall caused by weakness associated with milk fever. Most are systemically normal and eat and drink and creep; some do not move or try to get up. Those which are anorexic for several days usually die.	Proteinuria due to muscle damage.	Serum enzymes CPK and SGOT are elevated.	Deep bedding non-slip floor provide palatable feed and water with added electrolytes

VIRAL DIARRHEA OF CALVES, LAMBS, KIDS, PIGLETS AND FOALS

Otto M. Radostits
College of Veterinary Medicine
University of Saskatchewan

There is now firm evidence that some viruses can be the primary cause of acute diarrhea in any of the newborn farm animal species. The rotavirus and coronavirus are most commonly involved, but other viruses have also been isolated from young diarrheic farm animals.

Etiology

A group of viruses known as the rotaviruses have been isolated from calves, lambs, piglets and foals and are considered to be primary causes of diarrhea in those species. All members of this group share a common morphology and were previously known as the reovirus-like viruses. The rotaviruses isolated from calves, piglets, lambs and foals readily infect kidney cell cultures. The details of the virological aspects of the rotaviruses are available. Comparative studies have confirmed that the rotaviruses of human infants, calves, pigs and foals are morphologically indistinguishable from each other and from the virus of infant mice. In addition, the lamb rotavirus is similar to the calf and pig viruses.

Coronavirus-like viruses are also considered as causative agents of acute diarrhea in calves and piglets. The coronavirus-like virus in pigs is similar to, but distinct from the virus of transmissible gastroenteritis (TGE) and is thought to be the cause of porcine epidemic diarrhea type II. A coronavirus has been isolated from adult cows with diarrhea which suggests a causative agent for winter dysentery. The physiochemical and biological properties of some neonatal calf coronaviruses have been compared.

While the rotavirus and the coronavirus-like virus are considered to be the most important causes of viral diarrhea in newborn farm animals (other than TGE of piglets), an adenovirus, a parvovirus, and an enterovirus have also been isolated from diarrheic calves, but are currently not considered as common primary pathogens. Small viruses resembling astroviruses and calciviruses have also been isolated from calves and piglets with acute enteritis, but their entiological significance is unknown. The viruses produced diarrhea in gnotobiotic calves.

Multiple mixed viral infections are being recognized more frequently as diagnostic techniques are improved. The rotavirus and coronavirus may occur in the same diarrheic calf with or without the presence of enterotoxigenic E. coli. A concurrent rotaviral and transmissible gastroenteritis infection has occurred in a newborn piglet and calicivirus-like particles have been isolated along with the rotavirus from young pigs with diarrhea.

Epidemiology

Rotavirus infections occur characteristically in young animals. The rotavirus is ubiquitous and has been isolated in outbreaks of diarrhea in calves, pigs, lambs and foals. Serological surveys indicate that up to 90% and more of adult cattle, sheep, horses and pigs have antirotaviral antibody.

The intestinal tract is the site of multiplication of rotavirus and virus is excreted only in the feces. Infected feces may contain as many as 10 10/g virus particles. Because rotaviruses are stable in feces and relatively resistant to commonly used disinfectants it is extremely difficult to prevent gross contamination of animal housing once infection has been introduced. The mature animals is considered to be the source of infection for the neonate.

The factors which influence rotavirus infection and its clinical severity include the age of the animal, immune status of the dam and absorption of colostral antibody, ambient temperature, degree of viral exposure, the occurrence of weaning, and the presence of other enteropathogens. The mortality is highest in the youngest animals which have received insufficient colostrum and are subjected to severe weather conditions. Under experimental conditions, the mortality rate in gnotobiotic piglets inoculated with the rotavirus is 100% at birth, 5-30% at 7 days and only a mild disease at 304 weeks of age.

An important epidemiological characteristic of rotavirus infection in newborn farm animals is that protection against disease is dependent on the presence of specific rotavirus colostral antibody in the lumen of the intestine of the newborn. Colostral serum antibody in the newborn does not protect animals against clinical disease. The protective effect of colostrum depends on its antibody titer and on the amount ingested. The daily oral administration of colostrum containing specific antibody of hyperimmune serum to lambs will protect them against experimental challenge. The protection is against clinical disease and not necessarily infection. Calves, lambs and piglets may still excrete virus in the feces while they are protected against clinical disease by the presence of colostral antibody in the intestinal lumen. The protection lasts only as long as colostral antibody is present which explains why rotaviral diarrhea occurs commonly after 5-7 days of age. Survival from rotavirus diarrhea in calves is probably dependent on a high level of serum colostral immunoglobulin.

The rotaviruses from one species can infect members only of some other species. Experimental infection of pigs, calves and lambs with human rotavirus have been described. The calf rotavirus can infect pigs. However, the significance of interspecies infection under field conditions has not been evaluated. Cross-infection between species is not a property shared by all rotaviruses.

The susceptibility of the lamb rotavirus to disinfectants has been examined. An iodophore preparation at 4% will partially inactivate virus in feces-contaminated material. Lysol at 5% and formol saline at 10% are more effective, while a 3% solution of sodium hypochlorite with 11% available chlorine is ineffective.

Calves

Many of the epidemiological characteristics of neonatal calf diarrhea caused by the rotavirus and coronavirus which have been reported, must be considered in the context of 'acute undifferentiated diarrhea of newborn calves' because rarely would an outbreak of diarrhea in newborn calves be caused by the virus alone.

The reovirus-like virus (rotavirus) was first isolated in the United States in 1969 and thought to be the cause of outbreaks of diarrhea in beef calves in Nebraska. Since then the virus has been recovered from calves affected with diarrhea in many countries including Canada, the United Kingdom and Australia, which suggests that it may have a worldwide distribution.

While the rotavirus has been most commonly associated with outbreaks of diarrhea in beef calves raised in groups outdoors, it has also been recovered from diary calves raised together in large groups in large dairy herds. The morbidity rate in beef herds has varied from herd to herd and from one year to another. In some herds the disease started at a low rate to 50-10% in the first year, increased to 20-50% in the second, and to 50-80% in the third year. In other herds, explosive outbreaks affecting 80% of the calves have occurred in the first year. The case fatality rate has also been variable, in some herds as low as 5%, while in others it has been as high as 60%. The mortality rate probably depends upon the level of colostral immunity in the calves, the incidence of enteric colibacillosis, and the level of animal husbandry and clinical management provided in the herd.

A unique epidemiological characteristic of rotavirus infection in calves is the short-term nature of the immunity provided by colostrum. Newborn calves are protected from the effects of the rotavirus only during the first few days after parturition, during which time the colostrum contains specific rotavirus antibody which is active in the lumen of the intestine. The serum immunoglobulins acquired by the calf from the colostrum do not provide protection against infection in the intestine. This correlated well with the peak incidence of rotavirus diarrhea which is 5-7 days of age, which would coincide with a marked drop in colostral immunoglobulins by the third day after parturition and allow for an incubation period of 18-24 hours for the disease to occur. The levels of serum and colostral antibody are lower in first-calf heifers which may explain the higher morbidity and mortality in their calves.

The virus is excreted by both calves and adult cattle in large numbers (up to 10 10/g of feces) and excretion may last for up to 3 weeks. Even under open-range conditions, there is a rapid spread of the virus throughout calves which come in frequent contact with each other,

particularly during the calving season. It would appear that calves are contaminated after birth from the dam's feces or from other infected diarrheic calves. There is no evidence that the virus crosses the placental barrier and infects fetuses in utero.

There is evidence that subclinical infection is common. Antibody to rotavirus and coronavirus can be found in the serum of 90-100% of young adults, and clinically normal calves with high serum antibody may excrete the virus. The explanations include (a) calves may be protected from disease but excrete the virus during the colostral feeding period; (b) calves may be infected at a subclinical level but still excrete the virus although they may have been denied colostral antibody. The excretion of the virus by adult cattle may serve as a source of the virus for calves.

The calf rotavirus can be experimentally transmitted to piglets and has been isolated from natural outbreaks of diarrhea in piglets. The isolation of a rotavirus from neonatal deer affected with diarrhea in a zoo in Australia raises some interesting epidemiological possibilities. The bovine rotavirus has been found in the feces of dogs, and dogs have been experimentally infected which suggests that dogs may play a role in the dissemination of the virus.

The occurrence and distribution of the virus in diarrheic and normal calves have been studied. The rotavirus has been found before, during and after the onset of diarrhea. It has been found along with the coronavirus and adenovirus in diarrheic calves. This serves to emphasize that while the virus can be considered as a primary pathogen in calves the results of field and laboratory investigations indicate that multiple mixed pathogen infections are probably more common than single pathogen infections. There may also be differences in the virulence between isolates. Earlier work suggested that most cases of naturally occurring rotavirus diarrhea in calves were also infected with E. coli. However, no attempt was made to determine if the E. coli possessed the virulence characteristics described under colibacillosis.

The coronavirus has been isolated from field outbreaks of diarrhea in calves in the United States, Canada, the United Kingdom and Denmark. The morphological and antigenic characteristics of the viruses from the United Kingdom, the United States and Denmark have been compared and cultivated in tissue culture. The viruses have been serially passed in gnotobiotic calves which developed diarrhea. The physicochemical and biological properties of some coronaviruses isolated from calves are described.

Lambs

The rotavirus has been isolated from the feces of lambs with diarrhea under 3 weeks of age. The disease appears to be sporadic in lambs and no particular epidemiological characteristics have been described. The experimental disease in lambs is mild and characterized by milk diarrhea, abdominal discomfort and recovery in a few days. The mortality in lambs is much higher when both the rotavirus and enteropathogenic E. coli are used.

Pigs

In piglets, rotaviral diarrhea occurs from 1 to 8 weeks of age, but is most common in pigs which are weaned under intensive management conditions at 3 weeks of age. The disease resembles what has been referred to as milk-scours, or 3-week scours of piglets. The morbidity may reach 80% and the case fatality rate ranges from 5 to 20% depending on the level of sanitation. The early weaning of piglets at a few days of age or at 3 weeks of age results in the removal of the antibody supplied by the sow's milk.

Outbreaks of diarrhea caused by a coronavirus-like virus may occur in pigs of all ages. The mortality is high in piglets under 7 days of age while most of the adult pigs recover.

Diarrhea in unweaned piglets has been associated with a combined infection of rotavirus and coccidia.

Foals

Rotaviral diarrhea occurs in foals from 5 to 35 days of age. Outbreaks of the disease occur on horse farms with a large number of young foals where the population density is high. Serological surveys indicate the presence of rotavirus antibody in almost all of the mares whose foals are infected with the virus. The available evidence suggests that the rotavirus is a major cause of diarrhea in foals. The rotavirus has been isolated from foals affected with salmonellosis which suggested the possibility that the virus may have precipitated the bacterial disease.

Pathogenesis

The rotavirus infects mature brush border villous epithelial cells in the small intestine, and to lesser extent in the large intestine. The infected cells are sloughed leading to partial villous atrophy and the atrophic villi are rapidly recovered with relatively undifferentiated crypt cells which mature over a few days and lead to healing of the lesion. The activity of the mucosal betagalactosidase (lactase) in the brush border of the villous epithelium is less than in normal animals which results in decreased utilization of lactose. This reduction in enzymes is associated with immature enterocytes on the villi during rotavirus infection. In vitro studies have suggested that lactase may be the receptor and uncoating enzyme for rotavirus which may explain the high degree of susceptibility of the newborn with high levels of lactase. The net effect of the morphological and functional changes in the intestine is malabsorption result in diarrhea, dehydration, loss of electrolytes and acidosis. The diarrhea is milk-fed calves with the experimental disease can be if the milk is withdrawn and replaced with glucose and water which is similar to transmissible gastroenteritis. The d-xylose absorption test can be used to measure the degree of malabsorption in calves infected with rotavirus.

The pathogenesis of rotaviral infection is similar in calves, lambs, pigs and foals. The lesions occur within 24 hours after infection, villous epithelial cells of the small intestine are infected and become detached and regeneration occurs within 4-6 days after the onset of the diarrhea. The intestinal villi usually returns to near normal within about 7 days after recovery from the diarrhea. However, calves and pigs may require 10-21 days to fully recover to a normal growth rate following rotavirus infection.

The effect of the interaction of other viruses or enteropathogenic E. coli is not well understood. In experimental combined E. coli and rotavirus infection in lambs the mortality is higher than when either of the two agents is administered alone. Under field conditions more than one microbiological agent is likely to be involved in the pathogenesis of diarrhea.

The pathogenesis of coronaviral enteritis in calves is similar to the rotavirus infection. The villous epithelial cells of the small and large intestines are commonly affected. The crypt epithelium is also affected which makes regeneration of villous epithelial cells much longer, which results in persistent diarrhea for several days and death from dehydration and malnutrition. The pathophysiological changes due to coronavirus-induced diarrhea in the calf have been described and are similar to the changes which occur in acute diarrheal disease in the calf caused by other enteropathogens.

The porcine coronavirus, CV777, replicates in the villous epithelial cells of both the small and large intestine and clinically resembles transmissible gastroenteritis of piglets.

A major factor in the pathogenesis of rotavirus and coronavirus infection in newborn farm animals is the amount of colostrum-derived antibody present in the intestinal lumen at the time of viral challenge. The protective effect of colostrum against rotavirus infection of the intestine is dependent upon both the volume and the antibody titers of the ingested colostrum. To be effective the colostrum must be ingested within a few hours after birth. Low levels of intestinal antibody may allow viral replication and result in clinical disease.

Clinical Findings

Calves

In experimental rotavirus infection in newborn colostrum-deprived gnotobiotic calves a profuse liquid diarrhea may occur by 10-14 hours after oral inoculation. Affected calves are mildly depressed, anorexic and may have a mild fever.

The naturally occurring disease usually occurs in calves over 4 days of age and is characterized by a sudden onset of a profuse liquid diarrhea. The feces are pale yellow, mucoid and may contain flecks of blood. Recovery usually occurs in a few days. Explosive outbreaks occur and up to 50% of calves from 5 to 14 days of age in the affected

population may develop the disease. If enterotoxigenic E. coli are present the disease may be acute; dehydration is severe and deaths may occur. Multiple mixed infection with E. coli and coronavirus are common in calves over 4 days of age and thus it may be impossible to describe a typical case of uncomplicated naturally occurring rotavirus or coronavirus-like diarrhea. There is a tendency for viral diarrhea in newborn calves to occur in explosive outbreaks, the calves are usually not toxemic, but the character of the diarrhea cannot be differentiated clinically from that caused by the other common enteric pathogens of newborn calves.

A coronaviral enteritis affecting calves from 1 to 7 days of age has been described, but there are no distinguishing clinical characteristics. The diarrhea may be persistent for several days, followed by death in spite of fluid therapy and careful realimentation with milk. The feces are voluminous, mucoid and slimy and may be dark-green or light-brown in color.

Lambs

Experimentally, newborn gnotobiotic lambs develop diarrhea 15-20 hours following oral inoculation and show dullness and mild abdominal discomfort. There are only a few documented descriptions of naturally occurring rotaviral diarrhea in newborn lambs. Affected lambs under 3 weeks of age develop a profuse diarrhea and the case fatality rate is high. It is not clear if outbreaks of uncomplicated rotaviral diarrhea occur in newborn lambs.

Pigs

Rotaviral diarrhea may occur in nursing piglets from 1 to 4 weeks of age and in pigs following weaning. The disease in nursing piglets resembles milk-scours or 3-week scours. Most of the pigs in the litter are affected with a profuse liquid to soft diarrhea with varying degrees of dehydration. Recovery usually occurs in a few days unless complicated by enterotoxigenic E. coli or unsatisfactory sanitation, overcrowding and poor management. The disease is often most severe in herds in which there is continuous farrowing with no period of vacancy for cleaning and disinfection in the farrowing barn. The disease may also occur in pigs a few days after weaning and may be a major factor in post-weaning diarrhea of piglets weaned at 3 weeks of age or earlier in the case of weaning pigs at 1-2 days of age.

Epidemic diarrhea Type II, associated with coronavirus-like particles, causes a profuse fluid diarrhea in pigs of all ages, including nursing piglets. Explosive outbreaks may occur and the morbidity may reach 100%. Mortality is usually restricted to piglets under 3 weeks of age.

Experimental rotavirus infection in newborn colostrum-deprived gnotobiotic piglets results in a profuse liquid diarrhea n 16-24 hours following inoculation. The feces are yellow and vomiting may occur. Dehydration and death may occur in 2-4 days.

Foals

Affected foals appear depressed, fail to suck and become recumbent. The temperature ranges from 39.5-41.0°C and the respiration may be rapid and shallow. A profuse, foul-smelling fluid diarrhea commences 4-12 hours after the onset of depression and affected foals become markedly dehydrated. Recovery following treatment usually occurs within 2-4 days. Death may occur within 24 hours after the onset of diarrhea.

Clinical Pathology

Fecal samples (20-30 g) should be collected from affected animals as soon after the onset of diarrhea as possible and submitted to the laboratory in a chilled state. Samples of intestinal mucosa from several sections of the small and large intestine should be submitted chilled for virus detection and possible isolation.

Because multiple mixed viral and bacterial infections are common, the request for a laboratory diagnosis must include consideration of all of the common pathogens. The viruses are much more difficult to detect than enteropathogenic E. coli. In herd outbreaks, fecal samples from several affected animals and some normal animals should be submitted. The rotavirus may be present in both normal and diarrheic animals which presents problems in interpretation and requires identification of the epidemiological factors which may have precipitated the disease in animals in which the viruses are ubiquitous.

Several laboratory tests are available for detection of rotaviruses and coronaviruses in the feces and intestinal contents and tissues. The particular test used will depend on the facilities and equipment available. Electron- microscopic examination of fecal material has been a standard diagnostic technique. It is easier to see the virus if it has been concentrated by ultracentrifugation or clumped by immune electron microscopy using specific antiserum. However, since the equipment and expertise necessary for electron microscopy are not available in many laboratories, alternative diagnostic techniques have been developed.

Several tests are based on immunofluorescence. These include immunofluorescent staining of fecal smears and of cell cultures infected with fecal preparations. The immunofluorescent staining of a fecal smear would appear to be a more convenient test for diagnostic laboratories because a diagnosis can be made in a few hours and it eliminates the need for electron microscopy. However, the immunofluorescence tests may not be as reliable as some other tests. The fluorescent antibody technique will only detect virus within epithelial cells technique will only detect virus within epithelial cells which are present in the feces for 4-6 hours after the onset of diarrhea. With electron microscopy the virus can be detected for up to 6-10 days after the onset of diarrhea. In some studies the fluorescent antibody technique detected the virus in only 20% of samples while electron microscopy detected the virus in about while electron microscopy detected the virus in about 60% of the samples. The immunodiffusion test and electron microscopy are superior to the fluorescent antibody technique. Treatment of the feces with chymotrypsin improves the detection rate.

Some of the methods for the detection of rotavirus antigens in calf feces have been compared. The enzyme linked immunosorbent assay (ELISA) is more sensitive and simple than immunoelectroosmophoresis, complement fixation, immunofluorescence on inoculated cell cultures or electron microscopy. A counterimmunoelectrophoresis test is also available and compares favorably with the ELISA test. In some studies the ELISA test and electron microscopy are considered equally reliable in detecting rotaviruses. The ELISA test utilized reagents which are both stables and non-radioactive and is ideal for handling large numbers of specimens. The test can be read with the naked eye or a simple colorimeter and there is no need for sophisticated equipment.

Solid-phase radioimmunoassay gives comparable results to electron microscopy, but requires the need for radioactive unstable reagents and expansive counting equipment.

The isolation of rotavirus from the feces of calves can be improved by treatment of the feces with trypsin which enhances the infectivity of the virus for tissue culture. Normally replication of the virus in tissue culture using conventional methods is limited.

Immunofluorescent sections of spiral colon is the diagnostic method of choice for the detection of coronavirus in calves; fecal samples are unreliable. Isolation of coronavirus in tracheal organ culture is the most sensitive in vitro culture technique. A hemadsorption-elution-hemagglutination assay test for the detection of coronavirus in feces of calves is a simple and rapid procedure. A counterimmunoelectrophoresis test is available for the detection of coronavirus in calves. Continuous cell live culture techniques for the isolation of bovine coronavirus have been described.

Several serological tests are available for the detection and measurement of rotaviral antibody in serum and lacteal secretions. The radioimmunoassay is the most sensitive test compared to the agar gel immunodiffusion, complement fixation, hemagglutination and hemagglutination inhibition test.

Pathology

The pathology of experimentally induced rotavirus and coronavirus diarrhea in colostrum-deprived and gnotobiotic calves, lambs, and piglets has been described. Grossly, the changes are unremarkable and consist of dehydration, fluid-filled intestinal tract and distension of the abomasum. The microscopic changes consists of shortening of the length of the villi and replacement of the tall columar villous epithelial cells by cuboidal and squamous cells. Segments of the small intestine may reveal villous fusion, rounded absorptive cells, villous atrophy and exposure of lamina propria. Crypt hyperplasia occurs in response to the loss of columnar epithelial cells from the villi. In coronavirus enteritis in calves there is commonly villous atrophy of both the small and large intestines and destruction of the crypt epithelial, which destruction does not occur in rotavirus enteritis. The changes are more severe in field cases of acute diarrhea in calves in which both viruses and enteropathogenic E. coli can be isolated.

Diagnosis

The cause of acute diarrhea in newborn farm animals cannot usually be determined on clinical grounds. All of the common bacterial and viral enteropathogens of calves can cause an acute profuse fluid diarrhea with progressive dehydration and death in a few days.

When outbreaks of diarrhea are encountered, a detailed examination of the possible epidemiological factors should be made and the appropriate fecal samples and tissues from affected animals submitted to the laboratory. The most reliable specimens include fecal samples obtained from animals within a few hours after the onset of diarrhea, and untreated affected animals which are submitted for necropsy and microbiological examination within a few hours after the onset of diarrhea.

The common clinical and epidemiological characteristics of the acute diarrheas of neonatal farm animals are as follows.

Calves

Enteric colibacillosis occurs primarily in calves under 4 days of age and is characterized clinically by an acute, profuse liquid diarrhea. Recovery following treatment usually occurs in 2 days. Outbreaks occur in beef and dairy calves. Rotavirus and coronavirus diarrhea usually occurs in calves over 5-10 days of age and up to 3 weeks of age. Explosive outbreaks occur characterized by an acute profuse liquid diarrhea with recovery in 2-4 days. Recovery is assisted by oral fluid therapy. Cryptosporidiosis occurs in calves from 5 to 15 days of age and is characterized by a persistent diarrhea which may last for several days. The cryptosporidia may be detected by Giemsa stain of fecal smears or by fecal flotation.

Piglets

Transmissible gastroenteritis occurs most commonly in piglets under 1 week of age and explosive outbreaks are common. There is acute profuse diarrhea and vomiting. Affected piglets may continue to nurse for several hours after the onset of the diarrhea. The case fatality rates is high in piglets under 7 days of age; older pigs commonly survive. Enteric colibacillosis usually occurs in piglets under 3 days of age. There is acute diarrhea, dehydration and rapid death. Pigs with coliform septicemia may die without obvious diarrhea and usually appear cyanotic. Entire litters may be affected and the case fatality rate may be 100%. Early treatment with antibiotics and subcutaneous fluids will result in recovery. Coccidiosis occurs in piglets from 5 to 10 days of age and is characterized by an acute diarrhea in which the feces are foul-smelling and vary in consistency from being cottage-cheese-like to liquid, and gray or yellow and frothy. The diarrhea is persistent for several days and non-responsive to antibiotics. Some pigs recover spontaneously, others die in 2-4 days. Coccidial oocysts can be detected in the feces. The morbidity rate varies from 50 to 75% and the case fatality rate from 10 to 20%. Hemorrhagic enterotoxemia due to Clostridium perfringens type C entire litters of pigs under 1 week of age, is characterized clinically by severe toxemia, dysentery and rapid death, and at necropsy there is a hemorrhagic enteritis.

Lambs

Enteric colibacillosis occurs in lambs most commonly under one week of age and is characterized by dullness, failure to suck and acute diarrhea which responds to antibiotic and fluid therapy. Coliform septicemia affects lambs under a few days of age and usually causes sudden deaths. Lamb dysentery occurs most commonly in lambs under 10 days of age and there may be sudden death or acute toxemia, tuck-up abdomen and a severe diarrhea and dysentery. At necropsy the characteristic findings are hemorrhagic enteritis.

Foals

Rotaviral diarrhea occurs in foals from 5 to 35 days of age, but most commonly in foals under 2 weeks of age. There is acute profuse watery diarrhea, failure to suck, recumbency, dehydration; recovery is common within 1 week. A mild fever is common. Less common causes of diarrhea in foals include salmonellosis, Clostridium perfringens type B and dietary diarrhea.

Treatment

The treatment of viral diarrheas in newborn farm animals is essentially the same as described for colibacillosis. There is no specific therapy for viral diarrhea, but antimicrobial agents are used orally and parenterally to treat the possible presence or occurrence of enteric and systemic bacterial infections. In the absence of complications, recovery from viral enteritis usually occurs without specific treatment in 2-5 days which parallels the replacement of the villous epithelial cells. Complete replacement and maturation probably requires several days after the cessation of diarrhea.

The withholding of milk for 24-48 hours is beneficial, but often not possible or practical when nursing beef calves or litters of pigs are involved. Milk can be withheld from hand-fed calves easily and replaced with oral fluids and electrolytes.

Oral and parenteral fluid therapy as indicated is essential. A glucose-glycine electrolyte formulation is an effective fluid therapy for pigs affected with experimental rotaviral diarrhea. The formula is: glucose 67.53%, sodium chloride 14.34%, glycine 10.3%, citric acid 0.8%, potassium citrate 0.2% and potassium dihydrogen phosphate 6.8%. A weight of 64 g of the above is dissolved in 2 liters of water to produce an isontonic solution.

When possible, affected animals, particularly calves, should be isolated from calving grounds and other newborn calves which are highly susceptible up to 3 weeks of age. When outbreaks of the disease occur in any species, the principles of good sanitation and hygiene should be emphasized to minimize spread of infection.

Control

The principles of control of viral diarrhea are similar to those described for colibacillosis. The management of pregnant animals at the time of parturition must ensure that the degree of exposure of the newborn to infectious agents is minimized. Control of population density to avoid overcrowding, and sanitation and hygiene are important.

Every effort must be made to ensure that the newborn ingests sufficient quantities of colostrum as soon after birth as possible. A unique feature of the immunology of the rotavirus infection is that the colostral antibody present in the lumen of the intestine for the first several days of life is the protective antibody. The circulating antibody which the newborn animal acquires in the first 24 hours of life is not protective against the viral diarrhea. The colostral antibodies in the lumen of the intestine of calves and lambs decrease concurrently with their decrease in the colostrum. The failure of circulating antibody to provide protection and the short duration of protection provided by colostrum makes control in calves difficult under even ideal conditions. The observation that infected calves may excrete large numbers of viral particles for weeks, suggests that control may be dependent on management of the environment of the calf and rapid development of local immunity in the intestinal tract, either by natural infection or vaccination. In the pig, in contrast, the levels of IgA in the milk increase throughout lactation and thereby provide protection until artificial weaning at 3 weeks of age when rotavirus diarrhea may occur.

In outbreaks of rotavirus or coronavirus enteritis in calves, the daily feeding of colostrum from cows of the affected herd may reduce the number of new cases. In affected herds the colostral antiviral antibody may be sufficient to prevent the disease if colostrum is fed daily for up to 30 days. If a large number of cows are calving over a short period of time the colostrum can be pooled and fed to the calves daily. Even small amounts of colostrum amounting to only a portion of the total daily intake of milk are efficacious if mixed with milk or milk replacer.

A modified live-virus rotavirus vaccine for oral administration to calves immediately after birth has been available and good results are claimed. The incidence of diarrhea in herds not vaccinated one year followed by vaccination the next year was compared by veterinarians who used the vaccine. However, the results suggest that more controlled field trials are necessary before the efficacy of the vaccine can be fully evaluated. In one study in which the double-blind technique was used to evaluated the efficacy of the rotavirus vaccine and an E. coli bacterin, there was no difference in the incidence of diarrhea in vaccinated or unvaccinated calves. In sequential field trials in which calves were vaccinated for an uninterrupted period of time, the morbidity and mortality rates were significantly lower than during the control periods before and after vaccination. In the double-blind trial, the morbidity and mortality rates in vaccinated and placebo calves were comparable. This suggests that the double-blind technique may not be satisfactory for testing the efficacy of a vaccine when vaccinated and non-vaccinated animals are allowed to co-mingle.

The use of oral rotavirus vaccine in dairy calves in endemically infected calves did not provide protection.

When modified live-virus vaccine is given orally to calves immediately after birth it is possible that the colostral antibody may neutralize the virus. This may explain the failure of the vaccine under field conditions. Most of the efficacy trials with the vaccine were done on colostrum-deprived gnotobiotic calves which were vaccinated orally at birth and experimentally challenged a few days later. It is probably illogical to vaccinate calves orally immediately after birth, particularly in herds where the disease is endemic. The colostrum of the cows will contain high levels of specific antibodies.

In utero vaccination of the fetus with a rotavirus vaccine may offer an effective method for protection. The bovine fetus from 68 to 190 days of gestation is able to respond immunologically to the rotavirus. Infection in utero with the bovine virus induces resistance to experimental disease caused by both the human and calf viruses . The calf rotavirus vaccine is ineffective for the protection of experimental rotaviral diarrhea of piglets.

Parenteral vaccination of the pregnant dam before parturition with a rotavirus vaccine may increase the level and the duration of colostral immunity. Vaccinated heifers had detectable antibody in milk 28 days after parturition, whereas in control cows no antibody was detected 4 days postpartum. There was significant lengthening of the incubation and prepatent periods in calves given a challenge infection at 7 days of age. However, the severity of the disease was similar in both groups of calves. Evidence is available which suggests that rotavirus antibody in milk can protect against a small challenge dose and maternal immunization against rotavirus may be a practical proposition. Similar results have been achieved by vaccinating ewes. Vaccination of ewes can result in an elevation of specific colostral antibody and prolong the period over which the antibody is present in the lumen of the intestines of the lambs. The antirotavirus activity is in the IgG fraction.

DISEASES CAUSED BY BACTERIA

Diseases Caused by *Escherichia Coli*

Colibacillosis occurs in all species of newborn farm animals and is a major cause of losses in this age group. Gut edema, enteric colibacillosis of feeder pigs and mastitis caused by *Escherichia coli* are also important diseases commonly caused by this organism.

Acute Undifferentiated Diarrhea of Newborn Calves

Diarrhea in newborn calves under 10 days of age is one of the most common diseases which the large animal clinician is faced with in practice. It is a major cause of economic loss in cattle herds and may assume even greater importance in the future as livestock production becomes more intensified. The effective treatment and control of neonatal diarrhea in calves have been difficult and usually empirical because the precise etiology cannot usually be determined. Field and laboratory investigations have indicated that there may not be a single etiology of calf diarrhea but rather the cause is complex and usually involves an interplay between enteropathogenic bacteria and viruses, the immunity of the calf and the effects of environment. Thus we have chosen to use the term 'acute undifferentiated diarrhea of newborn calves' to describe the acute diarrhea which occurs in newborn calves usually under 10 days of age, and sometimes older, which is characterized clinically by acute profuse watery diarrhea, progressive dehydration and acidosis and death in a few days or earlier. On a clinical basis it is not usually possible to differentiate between the common known causes of diarrhoea in newborn calves (enteropathogenic *E. coli*, rotavirus, coronavirus-like virus and *Salmonella* spp.). The common pathological lesions are dehydration, emaciation and a fluid-filled intestinal tract with no other obvious lesions.

For many years following the early work of Smith and Little in which they indicated that *E. coli* was the causative agent of calf diarrhea, there has been acceptance that it was the primary pathogen in diarrheic calves and the term colibacillosis has been in common use. We will recognize the existence of colibacillosis in calves as a disease, but wish to emphasize that diarrhea in newborn calves can be caused by many different agents influenced by several epidemiological factors (Table 47). Colibacillosis is discussed in its usually section and viral diarrhea of calves has been expanded in the light of new information. The other

causes of diarrhea in calves are found in their respective sections. This section will discuss the general picture of acute undifferentiated diarrhea of newborn calves.

Not until the categorization of *E. coli* into enteropathogenic, septicemic and, lately, enterotoxigenic strains could some questions about colibacillosis in calves be answered. It explained why *E. coli* which appeared to be the same but could be found in both diarrheic and normal animals were actually different; those in diarrheic calves possessed enteropathogenic properties while those in normal calves commonly did not. These laboratory refinements allowed the veterinary epidemiologist to study the occurrence and distribution of enteropathogenic *E. coli* in populations of both diarrheic and normal calves. Surprisingly, the enteropathogenic *E. coli* were found in only 25-30% of diarrheic calves. As an example, in one study of the occurrence and distribution of infectious agents in acute diarrhea of beef calves, one or more potentially enteropathogenic bacteria and viruses were isolated from 66% of affected calves. However, considerable laboratory effort and costs are necessary to examine all possible specimens for the common enteropathogens, and the field veterinarian must combine whatever laboratory support is available with clinical and epidemiological observations.

Concurrent with these studies on *E. coli*, the isolation of rotavirus (reovirus-like) and coronavirus-like virus from diarrheic beef calves in the United States added to the list of possible causes of calf diarrhea. Subsequent to this, these viruses and similar ones have been isolated from the human infant and many different species of newborn farm animals affected with diarrhea, including lambs, piglets and foals.

There is considerable field evidence that acute undifferentiated diarrhea of calves is associated with mixed infections. Infections with rotavirus, coronavirus, enteropathogenic *E. coli* and cryptosporidia and, complicated by hypogammaglobulinemia, are common in newborn calves with diarrhea. Multiple infections in which both enteropathogenic bacteria and viruses can be isolated from the same calf or group of calves are more common than single infection.

The rotavirus and coronavirus occur with almost equal frequency in the intestinal tracts of normal and diarrheic calves. Intestinal lesions compatible with the viral infections are found in about 70% of diarrheic

calves. Thus, these viruses are widespread in the bovine population and only under some circumstances will the infection be severe enough to cause lesions and diarrhea.

Table 47. Summary of the Relationship between the Primary Etiological Agents and the Epidemiology of Acute Undifferentiated Diarrhea of Newborn Calves

Primary Etiological Agents	Epidemiological Factors	Possible Role of Epidemiological Factor
COMMON		
Enterotoxigenic *E. coli*	Colostral immunity in calf	Low levels of serum immunoglobulins render calves highly susceptible to death from diarrhea
Rotavirus and coronavirus Dietary abnormalities	Overcrowding	Increased population density increases infection rate and high morbidity and mortality.
LESS COMMON		
Salmonella spp.	Parity of dam	Calves born from heifers may not acquire sufficient level of colostral immunoglobulins
Chlamydia sp.		
Adenovirus		
IBR	Meteorological	Changes in weather; wet, windy and cold weather commonly precedes outbreaks of diarrhea in beef calves. Higher mortality in dairy calves exposed to hot environmental temperatures. High environmental temperatures precipitate outbreaks.
BVD		
Clostridium perfringens Types B and C		
Providentia stuarti		
Cryptosporidium	Quality of diet	Heat denatured skim-milk used in milk replacers is less digestible than whole milk and precipitates diarrhea.
	Calf rearer	The concern and care provided by the calf rearer will have a direct effect on morbidity and mortality associated with diarrhea.

In one study the viruses were implicated as etiological agents in 70%, enterotoxigenic *E. coli* in 29% and *Cryptosporidia* sp. in 33% of diarrheic calves. Enterotoxic colibacillosis occurs in calves less than 4 days of age, while the viruses and cryptosporidia occur in diarrheic calves after 4 days of age. Other viruses such as parvovirus, astrovirus, calicivirus have been isolated from the feces of diarrheic calves, but their role in the etiology is yet to be defined.

To confound the issue, intensive studies have been done on the effects of colostral immunity on acute diarrhea in calves. Earlier work centered on the protective effect of colostrum against colibacillosis and recently on protection against rotavirus infection. The importance of colostral immunity is well established and while it is easy to say that calves should receive a certain amount of colostrum, the veterinarian in the field who encounters an outbreak of acute diarrhea in beef calves, for example, cannot usually determine whether in fact the calves possess protective levels of immunoglobulins.

Many interrelated epidemiological factors have been associated with a high incidence of calf diarrhea and have added to the difficulty of understanding the complexity of the disease. The effects of nutrition of the pregnant dam on the quantity and quality of colostrum and the vigor of the calf are thought to be important but there is little supporting evidence. Changes in weather and wet, windy and cold weather are thought to precipitate outbreaks of the disease in beef calves raised outdoors. Increases in population density in calf houses, and on calving grounds, resulting in a high infection rate may in part explain the high incidence in large intensified operations. Some studies have shown that the major contributing factor to dairy calf mortality is the care provided by the calf attendant. Not infrequently, however, outbreaks can occur in herds in which the management is excellent and not uncommonly an etiological diagnosis cannot be made.

Thus the disease is considered complex because one, or any combination of more than one of the specific etiological agents may be the cause of the disease; or epidemiological influences may precipitate the disease in calves which might not normally get the disease, even though they are infected with a specific enteropathogen. The term acute undifferentiated diarrhea of newborn calves is useful to encompass cases of diarrhea in calves in which the etiological diagnosis is not immediately obvious

and may not be determined even after exhaustive diagnostic work.

When a clinician is faced with an outbreak of acute diarrhea in newborn calves in which there is profuse watery diarrhea, progressive dehydration and death in a few days or earlier, the following steps are recommended:

- All affected calves should be identified, isolated and treated immediately with oral and parenteral fluid therapy as indicated.
- Antibacterials may be given orally and parenterally for the treatment of enteric and septicemic colibacillosis. When large numbers of calves are affected at one time it is not usually possible clinically or with the aid of a laboratory to determine which calf is septicemic and thus all acutely affected calves should be treated. Treatment, however, should not be continued beyond 3 days.
- Each of the commonly recognized epidemiological factors should be examined for its possible role in the particular outbreak. The common ones include quality of diet, origin of calves, overcrowding, recent changes in climate, recent stress of any kind on the herd, recent introductions into the herd, failure of calves to ingest colostrum and the calving of a disproportionately large percentage of first calf heifers which occurs in herds which are expanding their herd size.
- Fecal samples (30-50 g) should be collected from diarrheic calves at the first sign of diarrhea and from normal calves and submitted to a laboratory for the attempted isolation and characterization of enterotoxigenic *E. coli*, rotaviruses and *Salmonella* spp. Blood samples from affected and normal calves and colostral samples if available are useful for immunoglobulin and antibody studies. All moribund calves should be submitted for necropsy before they die naturally.
- Pregnant cows which are due to calve shortly should be moved to a new calving area. In a dairy herd this means a different clean calving stall, preferably in another barn not previously occupied by cattle; in beef herds it may mean moving a large number of cows to a new uncontaminated calving pasture.
- The control of the disease in future calf crops will depend on application of the principles of control which are described under colibacillosis and viral diarrhea in calves.

Acute Undifferentiated Diarrhea of Other Newborn Farm Animals

Several different pathogenic agents are now being identified in the feces and intestinal tract of diarrheic piglets, lambs, foals and kids. In principle, the situation is similar to the problem described in calves--it is difficult to know which bacteria, virus or protozoan is responsible for the diarrhea. Mixed infections are now more common than single infections. The clinician must make the most logical diagnosis based on the

clinical and epidemiological findings, the microbiological and pathological findings and the response to treatment.

Colibacillosis of Newborn Calves, Piglets, Lambs and Foals

One of the most common diseases of newborn farm animals is colibacillosis caused by pathogenic *E. coli*. There are at least two different types of the disease: enteric colibacillosis manifested primarily by varying degrees of diarrhea and septicemic colibacillosis manifested by septicemia and rapid death.

Etiology

Specific serotypes of *E. coli* are the causative agents of colibacillosis. Certain serotypes are associated with septicemia and another different series with the development of diarrhea and dilatation of an isolated loop of intestine. Both speticemic and enteric colibacillosis have been produced experimentally by the administration of relevant specific serotypes. Conversely the challenge of experimental animals with specific serotypes has been resisted by the administration of serum or colostrum containing antibodies to those specific serotypes.

Epidemiology

Colibacillosis occurs commonly wherever farm animals are maintained and is a significant cause of economic loss in raising livestock. There are many epidemiological factors which influence the disease, each one of which must be considered and evaluated when investigating the cause of an outbreak so that effective clinical management and control of the disease may be achieved. The disease is most common in animals 2-10 days of age, it may occur as early as 12-18 hours after birth and occasionally occurs in calves up to 3 weeks of age.

Morbidity and Mortality

In dairy calves raised under intensified conditions the morbidity may reach 75% but is usually about 30%. The case fatality will vary from 10 to 50%. In beef calves the morbidity will vary from 10 to 50% and the case fatality from 5 to 25% or even higher in some years. The population mortality rate in calves can vary from a low of 3% in well managed herds to a high of 60% in problem herds. Among the main causes of neonatal mortality in piglets the gastroenteropathies accounted for 2-8% of the total mortality of all piglets born in 17 herds over a 1-year period. Losses

due to stillbirths, traumatic injuries, starvation and undersize accounted for a much greater combined total loss of 13%. However, colibacillosis of piglets accounted for approximately 50% of the gastroenteropathies encountered during the pre-weaning period. Prevalence information is not readily available for foals and lambs but the disease accounts for 25% of septicemias in foals.

The prevalence of colibacillosis has increased in recent years for several possible reasons which include size of herd, shortage of qualified labor, automated livestock rearing systems and increased population density.

Immunity

Newborn farm animals are born agammaglobulinemic and must ingest colostrum and absorb colostral immunoglobulins within hours after birth to obtain protection against septicemic and enteric colibacillosis. The mortality rate from enteric colibacillosis is much higher in calves with low levels of serum immunoglobulins than in calves with adequate levels. Based on surveys, up to 25% of newborn dairy and beef calves have low levels of serum immunoglobulins because they do not receive sufficient colostrum early enough after birth which makes them very susceptible to neonatal disease especially colibacillosis. Colostral immunoglobulins are absorbed for up to 24 hours after birth in calves and up to 48 hours in piglets. However, in calves maximum efficiency of absorption occurs during the first 6-12 hours after birth and decreases rapidly from 12 to 24 hours after birth. Multiple small intakes of colostrum give significantly greater levels of serum immunoglobulins than a single, large intake and calves with continuous access to their dams are in the most favorable position. Newborn calves should ingest approximately 50 ml/kg body weight within the first 8-12 hours. Lambs with low serum immunoglobulin levels are also highly susceptible to colibacillosis.

The maximum level of serum immunoglobulins is reached in the calf at 24 hours after birth and the factors which reduce those levels below an adequate level include the factors of maternal behavior and conformation, the vigor of the calf, and environmental influences. First calf heifers do not have as much colostrum or as wide a spectrum of specific antibodies as do mature cows. Some first calf heifers do not lick and stimulate their calves to get up and suck immediately after birth as does the mature cow with an ostentatious maternal instinct. Others ignore their calves completely. The conformation of the udder and the shape of the teats may be

undesirable such that the calf cannot find the teat so easily on badly shaped udders or the teatmay be misshapen which makes it difficult for the calf to suck.

Calves which receive their first colostrum by bucket do not acquire the same high levels of serum immunoglobulins as calves which receive their first colostrum by natural sucking of the teat. In both cases the presence of the dam improves the absorption. Calves which are weak or have an edematous tongue from a prolonged difficult parturition may not be able to suck for several hours, by which time the ability to absorb colostral immunoglobulins has decreased markedly. Beef calves born outside may be subjected to several influences which affect colostral intake. They may be born during a snowstorm and suffer severe cold exposure, when born they may be dropped in a snowbank and be unable to get up even with the assistance of the dam, or in crowded calving grounds, mismothering due to mistaken identity may occur, resulting in the calf not receiving any or very little colostrum.

The disease is more common in piglets born from gilts than from sows, which suggest that immunity develops with developing age in the sow and is transferred to the piglets. Piglets which do not obtain a liberal quanitity of colostrum within a few hours after birth are very susceptible to colibacillosis. Prolonged parturition, weak piglets, slippery floors, cold draughty farrowing crates, and the condition of the sow and her colostral supply all influence the amount of colostrum ingested by the newborn piglet. Enteric colibacillosis is the major disease in piglets which are weaned from the sow immediately after birth and reared on milk replacers. A crude preparation of porcine immunoglobulin added to the milk replacer of colostrum-deprived pigs provided good protection against enteric colibacillosis when fed for the experimental period of 21 days. The susceptibility or resistance to *E. coli* diarrhea in piglets may have an inherited basis. The cell surface receptor for the K88+ antigen is inherited in a simple Mendelian way with adherence (S) dominant over non-adherence (S). Homozygous dominants (SS) and heterozygotes (SS) possess the receptor and are susceptible whereas in the homozygous recessives (SS), it is absent and the pigs are resistant. The highest incidence of diarrhea occurs in susceptible progeny born from resistant dams and sired by susceptible sires.

Effect of Meteorological Influences

While little epidemiological data are available to support the claim, many veterinarians have observed a relationship between adverse climatic conditions and colibacillosis in both calves and piglets. During inclement weather, such as a snowstorm, a common practice in beef herds is to confine the calving cows in a small area where they can be fed and watered more easily. The overcrowding is commonly followed by an outbreak of enteric colibacillosis in the calves. There is evidence that cold, wet, windy weather during the winter months and hot dry weather during the summer months has a significant effect on the incidence of dairy calf mortality.

Nutrition and Feeding Methods

Dairy calves fed milk substitutes are more prone to enteric colibacillosis than those fed cows' whole milk. Extreme heat treatment of the liquid skim-milk in the processing of dried skim-milk for use in milk substitutes for calves results in denaturation of the whey protein which interferes with digestibility of the nutrients and destruction of any lactoglobulins which are present and may have a protective effect in the young calf. Irregular feeding practices resulting in dietetic diarrhea are considered to contribute to a higher incidence of enteric colibacillosis in calves. The person feeding and caring for the calves has been an important factor influencing calf mortality due to diarrhea. While it is generally believed that general or specific nutritional deficiencies such as a lack of energy, protein or vitamin A in the maternal diet predispose to colibacillosis, particularly in calves and piglets, there is no direct evidence that nutritional deficiencies are involved. They probably are, at least in indirect ways, for example, by having an effect on the amount of colostrum available at the first milking after parturition in first calf heifers underfed during pregnancy.

Standard of Housing and Hygiene

Housing and hygienic practices are probably the most important epidemiological factors influencing the incidence of colibacillosis in calves and piglets but have received the least amount of research effort compared to other aspects, for example, control of the disease through vaccination. As the size of herds has increased and as livestock production has become more intensified, the quality of hygiene and sanitation, particularly in housed animals, assumes major importance. Where

calves are run at pasture or are individually tethered, or penned, on grass the disease is much less common.

Source of the Organism and its Ecology and Transmission

In most species the major primary source of the infection is the feces of infected animals, although the organism may be cultured from the vagina or uterus of sows whose litters become affected. In swine herds the total number of organisms on each sow was highest in the farrowing barn, decreased when the sow was returned to the breeding barn and was lowest when the sow was in the gestation barn. Calves obtain the organism from contaminated bedding and calf pails, dirty calf pens, diarrheic calves, overcrowded calving grounds, milk from cows affected with coliform mastitis and from the skin of the perineum and udder of the cow. The organism is spread within a herd through the feces of infected animals and all of the inanimate objects which can be contaminated by feces including bedding, pails, boots, tools, clothing and feed and water supplies. The organism is one of the first encountered by newborn farm animals within minutes after birth. The high population density of animals which occurs in overcrowded calving grounds in beef herds, heavily used calving pens in dairy herds and the continuous successive use of farrowing crates without a break for clean up contributes to a large dynamic population of *E. coli*. The population of bacteria in an animal barn will continue to increase as the length of time the barn is occupied by animals without depopulation, a clean-out, disinfection and a period of vacancy. In some countries where lambing must be done in buildings to avoid exposure to cold weather, the lambing sheds may become heavily contaminated within a few weeks resulting in outbreaks of septicemic and enteric colibacillosis.

Enteropathogenic *E. coli* can be isolated from only 30-60% of diarrheic calves. Heat-stable enterotoxin can be detected in only approximately 17% of calves afflicted with diarrhea presumably due to enterotoxigenic *E. coli*. The frequency of antibody titers to enterotoxin in the sera of cows in a slaughterhouse survey was 38% while in swine it was 94%, which suggests that in outbreaks of colibacillosis in calves and piglets there is an increase in the proportion of pathogenic *E. coli* detectable in the feces of diarrheic calves. Ingestion is the most likely portal of infection in calves, piglets and lambs although infection via the umbilical vessels and nasopharyngeal mucosa can occur. It has been suggested that certain serotypes of *E. coli* may enter by the latter route and lead to the

development of meningitis.

Virulence Attributes of *E. coli*

Piglets. Most enteropathogenic *E. coli* from neonatal pigs produce either K88+, 987P+, or K99+ pilus antigens which adhere to ileal villi, colonize intensively and cause profuse diarrhea when given to newborn pigs. However, there are also some enterotoxigenic strains which produce none of the three antigens. The K88+ produces heat-labile enterotoxin (LT), the 987P+ and the K99+ do not produce LT and, all three types produce heat-stable enterotoxin (STa) in infant mice. Some isolates produce neither LT or STa but produce enterotoxin in ligated intestinal loops of pigs (STb).

Calves. The major virulence attributes of the enterotoxigenic strains of *E. coli* in calves are the K99+ antigen which produces heat-stable enterotoxin (ST). The colonization in the small intestine of calves by K99+ ETEC appears to be site-specific, having a predilection for the ileum. Non-enteropathogenic strains do not adhere.

Lambs. Enterotoxigenic strains of *E. coli* can be isolated from the feces of approximately 35% of diarrheic lambs. Enterotoxigenic strain of *E. coli* have also been isolated from the blood of a small percentage of diarrheic lambs. However, the isolates have not been characterized as they have been in calves and piglets.

Pathogenesis

The major factors which are important in the understanding of colibacillosis are the immune status of the animal and the properties of the strain of *E. coli*, particularly its capacity to invade tissues and produce a septicemia, or its capacity to produce an enterotoxin which causes varying degrees of severity of diarrhea. In calves, the three common forms of the disease described several years ago were septicemic, enteric-toxemic and enteric colibacillosis. Recent work suggests that both enteric forms of the disease are caused primarily by enterotoxigenic strains of *E. coli* which are non-invasive and that the invasive strains of the organism are primarily responsible for the septicemic form of the disease.

Septicemic Colibacillosis

This occurs in all species but is most common in foals and lambs and results from the invasive strains of *E. coli* invading the tissues and systemic circulation via the intestinal lumen, nasopharyngeal mucosae and tonsillar crypts, or umbilical vessels. Calves and piglets which are

deficient in immunoglobulins are most susceptible to septicemia. The intestinal permeability to macro-molecules in the newborn piglet may predispose to the invasion of septicemia-inducing *E. coli*. Colostrum provides protection against colisepticemia in calves but does not prevent diarrhea. The clinical findings and lesions in septicemic colibacillosis are attributed to the effects of endotoxin which causes shock. The infusion of *E. coli* endotoxin into the duodenum of newborn calves reproduces the hypoglycemia and lactic acidosis which develops in calves moribund with colibacillosis. Also, colostrum-fed calves are much more resistant to endotoxin than colostrum-deprived calves. Calves, piglets and lambs which have normal levels of serum immunoglobulins are protected from septicemic strains. Animals which recover from septicemia may develop lesions due to localization in other organs. Arthritis is a common sequel in calves, foals and lambs. Meningitis is common in calves and piglets. Polyserositis due to *E. coli* has been recorded in pigs.

Enterotoxic Colibacillosis

Enterotoxigenic strains of *E. coli* possess the ability to colonize and proliferate in the upper part of the small intestine and to produce enterotoxins which causes an increase in net secretion of fluid and electrolytes from the systemic circulation into the lumen of the intestine. The adhesion of *E. coli* to the intestinal epithelial cells is mediated by bacterial pili and the mechanism of attachment to the receptors is complex. The enterotoxic form of colibacillosis occurs most commonly in calves and piglets and less commonly in foals and lambs.

The factors which allow or control the colonization, proliferation and production of enterotoxin of these strains are not well understood. An adequate level of serum immunoglobulins will protect calves from death due to diarrhea but not necessarily from diarrhea. Best protection is provided if both the serum levels and the levels in the colostrum and the milk of the first week are high. The immunoglobulins in the plasma of calves which have received sufficient colostrum are IgG, IgM (probably the more important of the two for the prevention of septicemia) and IgA. The serum IgG, concentrations of calves under 3 weeks of age, and dying from infectious disease, were much lower than in normal calves. Of the dead calves, 50% had serum IgG levels that were more than 2 standard deviations below the normal mean, and an additional 35% had concentrations greater than one standard deviation below the normal mean. In the intes-

tine, no single subclass of immunoglobulin is known to be responsible for protection against the fatal effects of diarrhea. Individually, each immunoglobulin subclass can prevent death from diarrhea even though calves may be affected with varying degrees of diarrhea. In contrast to the pig, IgA appears to be least effective. In pigs, IgA becomes the dominant immunoglobulin in sow colostrum after the first few days of lactation, and this is the immunoglobulin which is not absorbed but is retained in, and reaches a high level in, the gut and plays a major role in providing local protection against enteric colibacillosis in piglets. Porcine colostral IgA is more resistant to gastrointestinal proteolytic enzymes than IgG_2 and IgM. On the other hand, IgG is at a peak concentration in colostrum in the first day after parturition, is readily absorbed by the newborn piglet and is vital in providing protection against septicemia. Lysozyme in sows' milk may assist in the control of bacterial population in the gut of the unweaned piglet.

The production of enterotoxin by the *E. coli* results in net secretion of fluid and electrolytes from the systemic circulation into the lumen of the gut resulting in varying degrees of dehydration, electrolyte imbalance acidosis, hyperkalemia when the acidosis is severe, circulatory failure, shock and death. The response to *E. coli* enterotoxin in calves and piglets is similar to cholera enterotoxin in man and takes place through an intact mucosa. Enterotoxin stimulates mucosal adenylcyclase activity which leads to an increased cyclic AMP which in turn is thought to increase intestinal fluid secretion. The intestinal adenylcyclase system and enterotoxin-induced intestinal secretion may not be directly related in the small intestine of the pig. The fluids secreted are alkaline, and in comparison to serum, isotonic, low in protein, and high in sodium and bicarbonate ions. When the disease is confined to the intestine, it responds reasonably well to treatment in the early stages. If death occurs, it is due to acidosis, electrolyte imbalance and dehydration. Input-output balance studies of calves with neonatal diarrhea have indicated that there is no significant difference between the fluid loss of normal non-diarrheic and diarrheic dying calves because the kidney can compensate for excessive losses through the feces. However, compensation by the kidney and the maintenance of plasma volume were dependent on continued milk intake or oral fluid therapy. Severe metabolic changes may occur in calves with diarrhea. If the disease is

progressive, the acidosis becomes more severe, lactic acidosis develops because of a reduced ability to utilize lactic acid and, severe hypoglycemia may occur because of a reduced rate of conversion of lactic acid to glucose. If extensive fluids are lost, hypovolemia and shock occur. These metabolic changes may in part be due to terminal endotoxemia which may be a sequel to acute diarrheal disease.

The presence of histopathological changes such as villous atrophy and severe inflammation in the intestinal epithelium of calves and piglets infected with enterotoxigenic *E. coli* suggest that other substances such as endotoxin may be released from other strains of *E. coli* or from other enteropathogens. The enterotoxigenic *E. coli* also induce consistent pathological changes in the jejunum and ileum of colostrum-fed calves. Focal emigration of neutrophils, especially through the epithelium above aggregated lymphatic follicles, stunting of jejunal and ileal villi and adherence of bacteria to jejunal and ileal mucosae are the most consistent findings. These changes are helpful in making the diagnosis of enterotoxigenic colibacillosis in calves. While enterotoxigenic strains are considered to be non-invasive this does not preclude the possibility that invasion into the systemic circulation may occur, resulting in septicemia, or that septicemic strains may not occur together. Multiple infections rather than monoinfections are more common in calves. Rotavirus, coronavirus, and cryptosporidia may occur along with enteropathogenic *E. coli*.

In summary, septicemic colibacillosis occurs in newborn animals which are agammaglobulinemic because they have not ingested sufficient immunoglobulins, thus rendering them highly susceptible. Enteric colibacillosis on the other hand occurs in colostrum-fed animals and is caused by the colonization and proliferation of enteropathogenic *E. coli* which produce enterotoxin and cause varying degrees of diarrhea and acidosis and dehydration. While single infections occur commonly, as in piglet diarrhea, and what was previously described as enteric-toxemic colibacillosis in calves, multiple infections with enteropathogenic *E. coli* and viruses and other agents are more common.

Clinical Findings

Calves. *Septicemic colibacillosis* is most common in calves during the first 4 days of life. The illness is acute, the course varying from 24 to 96 hours. There are no diagnostic clinical signs. Affected animals

are depressed and weak, anorexia is complete, there is marked tachycardia and, although the temperature may be high initially, it falls rapidly to subnormal levels when the calf becomes weak and moribound. Diarrhea and dysentery may occur but are uncommon. If the calf survives the septicemic state, clinical evidence of post-septicemic localization may appear in about 1 week. This includes arthritis, meningitis, panophthalmitis and less commonly, pneumonia.

Enterotoxic colibacillosis is the most common form of colibacillosis in newborn calves primarily from 3 to 5 days of age. It may occur in calves as early as 1 day of age and only rarely up to 3 weeks. The clinical severity will vary dependent upon the number and kind of organisms causing the disease. The presence of a single enterotoxigenic strain of *E. coli* may cause a state of collapse usually designated as *enteric toxemia*. In this form of the disease the outstanding clinical signs include severe weakness, coma, subnormal temperature, a cold clammy skin, pale mucosae, wetness around the mouth, collapse of superficial veins, slowness and irregularity of the heart, mild convulsive movements and periodic apnea. Diarrhea is usually not evident although the abdomen may be slightly distended and succussion and auscultation may reveal fluid splashing sounds suggesting a fluid-filled intestine. The prognosis for these calves is poor and they commonly die in 2-6 hours after the onset of signs.

In the more common form of the disease in calves, the enteric form of colibacillosis, the calves are affected with a diarrhea in which the feces are profuse and watery to pasty, usually pale yellow to white in color, and occasionally streaked with blood flecks and very foul-smelling. The dry matter content of the feces is commonly below 10%. Defecation is frequent and effortless and the tail and buttocks are soiled. The temperature is usually normal in the initial stages but becomes subnormal as the disease worsens. Affected calves may or may not suck or drink depending on the degree of toxemia and weakness. The abdomen may be slightly distended in the early stages when the intestines are fluid-filled which may be detectable on succussion and auscultation. When many calves are affected within a few days in an outbreak some calves when examined early will have a distended abdomen without obvious diarrhea which usually occurs and is profuse a few hours later. Mild-to-moderately affected calves may be diarrheic for a few days and recover spontaneously with or without treatment. However, 15-20% of calves with enteric colibacillosis become

progressively worse over a period of 3-5 days, gradually become more weak, completely anorexic and progressively more obviously clinically dehydrated. Throughout the course of the diarrhea the degree of dehydration will vary from just barely detectable clinically (4-6% of body weight) up to 10-16 % of body weight. It is best assessed by 'tenting' the skin of the upper eyelid or the neck and measuring the time required for the skin-fold to return to normal. In calves with 8% dehydration, 5-10 seconds will be required for the skin-fold to return to normal; in 10-12% dehydration, up to 30 seconds. Death usually occurs in 3-5 days. Affected calves will lose 10-16% of their original body weight during the first 24-48 hours of the diarrhea. In the terminal stages there may be bradycardia with arrhythmia associated with terminal hyperkalemia. Outbreaks of the disease in beef calves may last for up to 3 weeks and in epidemics almost every calf will be affected.

Lambs. Although some cases manifest enteric signs, and chronic cases may occur, colibacillosis in lambs is almost always septicemic and peracute. Two age groups appear to be susceptible; lambs of 1-2 days of age and lambs 3-8 weeks old. Peracute cases are found dead without premonitory signs. Acute cases show meningitis manifested by a stiff gait in the early stages, followed by recumbency with hyperesthesia and tetanic convulsions. Chronic cases are usually manifested by arthritis.

Piglets. Septicemic colibacillosis is uncommon but occurs in piglets within 24-48 hours of age. Some are found dead without any premonitory signs. Usually more than one, and sometimes the entire litter, are affected. Severely affected piglets seen clinically are weak, almost comatose, appear cyanotic, and feel cold and clammy and have a subnormal temperature. Usually there is no diarrhea. The prognosis for these is unsatisfactory and most will die in spite of therapy.

Enterotoxic colibacillosis (or baby pig diarrhea) is the most common form of colibacillosis in piglets and occurs in anywhere from 12 hours of age up to several days of age with a peak incidence at 3 days of age. As with the septicemic form, usually more than one pig or the entire litter is affected. The first sign usually noticed is the fecal puddles on the floor. Affected piglets may still nurse in the early stages but gradually lose their appetite as the disease progresses. The feces vary from a pasty to watery consistency and are usually yellow to brown in color. When the diarrhea is profuse and watery, there will be no obvious staining

of the buttocks with feces but the tails of the piglets will be straight and wet. The temperature is usually normal or subnormal. The disease is progressive; diarrhea and dehydration continues, the piglets become very weak and lie in lateral recumbency and make weak paddling movements. Within several hours they appear very dehydrated and shrunken, and commonly die within 24 hours after the onset of the signs. In severe outbreaks the entire litter may be affected and die within a few hours of birth. The prognosis for these is favorable if treatment is started early before significant dehydration and acidosis occur.

Clinical Pathology

If septicemia is suspected, blood should be submitted for isolation of the organism and determination of its drug sensitivity.

The definitive etiological diagnosis of enteric colibacillosis will depend on the isolation and characterization of the *E. coli* from the intestines and the feces of affected animals. The best opportunity of making a diagnosis is when untreated representative affected animals are killed and submitted for pathological and microbiological examination. The distribution of the organism in the intestine and determination of the presence of K88+, K99+ or 987P antigens, the demonstration of enterotoxin by infant mouse test or ligated intestinal loops and the histopathological appearance of the mucose all contribute to the diagnosis. The laboratory tests used to identify enteropathogenic *E. coli* include the indirect fluorescent antibody technique anti-K99 serum agglutination test and the enzyme-linked immunosorbent assay (ELISA) test.

The routine culture of feces for *E. coli* without determining their virulence is of limited value. The determination of drug sensitivity of the *E. coli* isolated from the feces of diarrheic calves and piglets is commonly done but is of limited value without determining which isolate is enteropathogenic.

A total and differential leukocyte count may indicate the presence of a septicemia or severe intestinal infection. In some calves the plasma corticoids are increased due to the stress of infection. The packed cell volume and the total solids concentration of the blood will indicate the degree of dehydration, and the blood urea nitrogen may be increased in severe cases due to inadequate renal perfusion. The blood bicarbonate values are markedly reduced, blood pH values represent acidosis and the other serum electrolytes are variable. There is usually a decrease in

serum sodium, chloride and potassium but potassium may be elevated in severe cases of acidosis. The total plasma osmolality is decreased and fecal osmolality increased.

The determination of the level of serum immunoglobulins of diarrheic calves is considered valuable in assessing prognosis and to determine the intensivity of the therapy required for survival. However, the level of serum immunoglobulins as a measure of susceptibility or prognosis is most accurate at 24 hours after birth. After this period, it is unreliable because the serum immunoglobulins may be increased in response to septicemia, increased spuriously in dehydration, and decreased in enteric disease. A zinc sulfate turbidity test is available for an estimation of the level of serum immunoglobulins and can be used as an aid in surveillance of the colostral intake of calves. There is an increase in the excretion of fecal globulins, primarily IgG, which suggests an intraluminal leak of serum globulins.

Necropsy Findings

In septicemic colibacillosis, there may be no gross lesions and the diagnosis may depend upon the isolation of the organism from the abdominal viscera and heart blood. In less severe cases, there may be subserous and submucosal petechial hemorrhages, and a degree of enteritis and gastritis may be present. Occasionally fibrinous exudates may be present in the joints and serous cavities, and there may be omphalophlebitis, pneumonia, peritonitis and meningitis.

In enteric colibacillosis in piglets, the flaccidity of the gut is evident and although the tissues may be dehydrated the intestines are usually distended with fluid and clotted milk may be present in the stomach. The intestinal mucosae may appear normal or hyperemic; in prolonged cases there may be edema of the mesenteric lymph nodes. Villous atrophy similar to that in transmissible gastroenteritis has been described and the pathology of the experimental disease in gnotobiotic piglets has been described. Attempts should be made to culture the organism from the gut, the mesenteric lymph nodes, spleen, heart blood and cerebrospinal fluid.

In enteric colibacillosis of calves, there is dehydration, the intestinal tract is distended by yellow watery contents and gas, the abomasum is usually grossly distended with fluid and may or may not contain a milk clot. There is usually no milk clot in the abomasum of calves fed

poor quality milk replacers. The abomasal mucosae may contain numerous small hemorrhages. The villi in the jejunum and ileum are stunted and may be fused together, there is neutrophil infiltration into the intestinal mucosa and a layer of Gram-negative bacteria adhere to the mucosa. When a definitive etiological diagnosis is desirable, as in outbreaks of the disease, it is necessary to conduct the necropsy examination on diarrheic calves which are killed specifically for that purpose and the necropsy done on the fresh carcass. Postmortem autolysis of the intestinal mucosae and invasion of the tissues by intestinal microflora occurs within minutes after death, thus making evaluation of results difficult. Samples should be submitted for isolation of bacteria, demonstration and isolation of rotaviruses, and tissues for examination.

Diagnosis

The definitive etiological diagnosis of *septicemic colibacillosis* is dependent on the laboratory isolation of the causative agent, which is usually a single species of organism. The septicemias of the newborn can not be distinguished from each other clinically. The definitive etiological diagnosis of *enteric colibacillosis* in newborn calves and piglets may be difficult and often inconclusive because the significance of other organisms in the intestinal tract and feces of diarrheic animals cannot be easily determined. Table 48 lists the possible causative agents of diarrhea and septicemia in newborn farm animals. Every effort which is economically possible should be made to obtain an etiological diagnosis. This is especially important when outbreaks of diarrhea occur in a herd or where the disease appears to be endemic. The use of an interdisciplinary team will increase the success of diagnosis.

This includes making a visit to the farm or herd and making a detailed epidemiological investigation of the problem. The diagnosis depends heavily on the epidemiological findings, and the microbiological and pathological findings and sometimes on the results of treatment.

The major difficulty is to determine whether or not the diarrhea is infectious in origin and to differentiate it from dietetic diarrhea which is most common in hand-fed calves and in all newborn species which are sucking high-producing dams. In dietetic diarrhea, the feces are voluminous, pasty to gelatinous in consistency, the animal is bright and alert and is usually still sucking but some may be inappetent.

Table 48. Possible Causes of Septicemia and Acute Neonatal Diarrhea in Farm Animals

Calves	Piglets	Lambs	Foals
SEPTICEMIA			
E. coli	*E. coli*	*E. coli*	*E. coli*
Salmonella spp.	*Streptococcus spp.*	*Salmonella spp.*	*Actinobacillus equuli*
Listeria monocytogenes		*Listeria monocytogenes*	*Salmonella abortivoequina*
Pasteurella spp.		*Erysipelothrix insidiosa*	*Salm. typhimurium*
Streptococcus spp.			*Str. pyogenes*
Pneumococcus spp.			*Listeria monocytogenes*
ACUTE NEONATAL DIARRHEA			
E. coli	*E. coli*	*Cl. perfringens Type C*	*Salmonella spp.*
Salmonella spp.	*Salmonella spp.*	Rotavirus	*Cl. perfringens Type B*
Providencia stuartii	Transmissible gastroenteritis virus		Rotavirus
Clostridium perfringens Type C	*Cl. perfringens Type C*		Dietetic or disaccharidase deficiency
Chlamydia spp.	Rotavirus		
Rotavirus and coronavirus-like virus	*Eimeria spp.*		
Cryptosporidia			
Dietetic diarrhea			
Eimeria spp. (calves at least 3 weeks of age)			

Treatment

The considerations for treatment of acute neonatal diarrhea include: alteration of the diet, fluid and electrolyte replacement, antimicrobial therapy, the possible use of antiparasympathomimetics and intestinal protectants and management of outbreaks. A review of the clinical management and control of neonatal enteric infection of calves is available.

The major factor which influences survival of calves with diarrhea is the level of colostral immunity in the calf at the onset of the diarrhea. The prognosis is unfavorable if the level of immunoglobulins is low, regardless of intensive fluid and antimicrobial therapy.

Alteration of the Diet

The question of whether or not diarrheic newborn animals should be starved from milk during the period of diarrhea is still debatable. Certainly diarrheic piglets are usually treated and left free to nurse on the sow and diarrheic beef calves are commonly treated and left with the cow.

However, it is common practice to starve for 24 hours diarrheic calves which are being hand-fed milk replacer or whole milk. In one study it was shown that the offering of milk to diarrheic calves when they were willing to drink following hydration therapy in a clinic resulted in a higher but insignificant improvement in survival rate than calves which were starved from milk for 24 hours. Balance studies in diarrheic calves have shown that continued milk intake helped to maintain plasma volume which suggests that calves should continue to receive a source of oral fluid during the periods of diarrhea. Because of reduced digestibility in enteric disease, it would appear logical not to feed the animal with nutriments such as milk which must be digested but rather to provide readily absorbable substances such as oral glucose-electrolyte mixtures. These are used commonly during the period of diarrhea as a source of energy, fluids and electrolytes. Such mixtures are inexpensive, easy to use, readily available and, if used by the farmer when diarrhea is first noticed, will usually successfully treat existing dehydration and prevent further dehydration and acidosis.

Following recovery, calves should be offered reduced quantities of whole milk 3 times daily (no more than total daily intake equivalent to 8% of body weight) on the first day and increased to the normal daily allowance in the next few days. Milk should not be diluted with water as this may interfere with the clotting mechanism in the abomasum.

Fluid and Electrolyte Replacement

The dehydration, acidosis and electrolyte imbalance are corrected by the parenteral and oral use of simple or balanced electrolyte solutions. In severe dehydration and acidosis, solutions containing the bicarbonate ion are indicated. Solutions containing lactates are undesirable as they must be converted to the bicarbonate ion by the liver, which may not be functioning normally and the plasma lactates may already be increased.

An equal mixture of isotonic saline (0.85%), isotonic sodium bicarbonate (1.3%), and isotonic dextrose (5%) is a simple effective solution for parenteral use. The use of solutions containing potassium chloride is sometimes recommended on the basis that total potassium stores may be depleted in severely affected calves. This is a paradoxical situation because a serum hyperkalemia may be present when there is severe acidosis. If the acidosis and hypoglycemia are corrected with glucose and bicarbonate, the administration of potassium may be beneficial in restoring

total potassium stores. However, solutions containing potassium can be cardiotoxic particularly if renal function is not restored. For severe dehydration (8-12% of body weight) fluids should be replaced as follows: hydration therapy 100 ml/kg body weight intravenously in the first 4-6 hours and maintenance therapy 140 ml/kg body weight over the next 20 hours. The techniques for long-term intravenous administration of fluids to the calf have been described.

Maintenance therapy may be given orally if the calf is well enough to suck from a nipple bottle or drink from a a pail. Oral fluids may also be given by stomach tube, the daily dose being divided into small doses given every 2-4 hours. The compositions of oral fluids are set out below.

Formula No. 1. (Glucose-glycine-electrolyte mixture) as a powder: glucose 67-53%, sodium chloride 14-34%, glycine 10-3%, citric acid 0-81%, potassium citrate 0-21% and potassium dihydrogen phosphate 6-8%. A 64 g. amount of the above is dissolved in 2 liters of water to produce an isotonic solution. The presence of the glycine and glucose enhances the absorption of water.

This solution has a pH of 4-3 which may enhance the rennet clotting of milk. Other solutions such as Formula No. 2 with a pH of 7 or greater may decrease clotting of milk in the abomasum.

Formula No. 2. Sodium chloride 117 g., potassium choride 150 g., sodium bicarbonate 168 g., potassium phosphate 135 g. Total 570 g. For 1000 ml. of oral solution, add 5-7 g. of dry mixture, to which may also be added 50 g. of glucose.

These oral electrolyte powders can be made up for the treatment of diarrheic calves when they are first observed on the farm. Give a rate of 100-149 ml/kg body weight daily in six to eight divided doses. Several commercial oral preparations are available for the treatment of diarrheic calves. These preparations contain mixtures of glucose, electrolytes, and sources of amino acids.

In general, oral fluids and electrolytes are beneficial for calves in the early stages of diarrhea or after they have been successfully hydrated following fluid therapy. Severely dehydrated or moribund calves may not respond favourably to oral fluid therapy alone.

Calves which respond and recover usually show marked improvement from intravenous and/or oral fluid therapy within 24-36 hours. Calves which are likely to recover will respond to the hydration therapy within 6-10

hours, begin to urinate within an hour after fluid administration was begun, and maintain hydration. Calves which do not respond will not hydrate normally; they may not begin to urinate because of irreversible renal failure, their feces remain watery, they remain depressed and not strong enough to suck or drink and continued fluid therapy beyond 3 days is usually futile.

Antimicrobial and Immunoglobulin Therapy

Antibiotics, sulfonamides, and other chemotherapeutics have been used extensively for many years for the specific treatment of colibacillosis in calves. These were used because it was assumed that in calves affected with enteric colibacillosis, there was an infectious enteritis which had the potential of developing into a bacteremia or septicemia. However, there is a notable lack of well-conducted clinical trials designed to show the efficacy of antibacterials in enteric colibacillosis. Conversely, there is no information which indicated they are not useful. In one study which evaluated the efficacy of antimicrobial agents given orally and parenterally to newborn calves with acute neonatal undifferentiated diarrhea, there was a slight but insignificant improvement in survival rate when chloramphenicol was given parenterally combined with nifuraldezone orally.

One of the major difficulties confronting the veterinarian in the field is deciding if calves or piglets affected with enteric colibacillosis are bacteremic or septicemic. As a result, more animals than necessary are treated systemically to avoid deaths from septicemia. Time does not usually permit pretreatment culture of the organism and determination of the drug sensitivity, so that broad spectrum antibiotics and selected chemotherapeutics based on previously successful experience are used. Some of the commonly used ones are chloramphenicol, ampicillin and trimethoprim-sulfonamide combinations. The dosages of these drugs for maximum effectiveness for the treatment of coliform septicemia in newborn calves and piglets have not been determined but the adult dose based on body weight appears to be successful and without toxicity. Neonates may be unable to metabolize drugs as effectively as adults. The advantage of parenterally administered antibiotics in colibacillosis in calves and piglets is the enterohepatic circulation which provides a level of the drug in the intestinal lumen which may not require the oral administration of the drug.

Many oral preparations are available and used for the treatment of

enteric colibacillosis in calves and piglets. Some consist of a single drug, while others are mixtures with or without absorbent, astringents and electrolytes. They have been used on an empirical basis since controlled trials have not been conducted. Chloramphenicol, neomycin sulfate tetracyclines, sulfonamides, trimethoprim-sulfonamide mixtures, nifuraldezone and ampicillin are in common use in different mixtures too numerous to dicuss here. Any of these drugs may be used but should be discontinued after 3 successive days of treatment to avoid elimination of too many species of drug-sensitive intestinal flora and their replacement by pathogenic fungi or bacteria such as *Candida spp.*, *Proteus spp.* and *Pseudomonas spp.* If possible, particularly in outbreaks of enteric colibacillosis in calves and piglets, the causative organism should be isolated from feces or tissues and a drug sensitivity obtained even though there are some limitations on the results unless the organism is more critically characterized as discussed under clinical pathology.

In some countries, it may be illegal to use some of the antimicrobials mentioned here because of the regulations regarding their use in food-producing animals. Some are available to farmers on a prescription basis only, which makes examination of the animals and a diagnosis necessary before recommendations are made. The indiscriminate use of antibiotics in milk replacers or for treatment of newborn calves and piglets is widespread and must be viewed with concern when the problem of drug resistance transfer from animal to animal and to man are considered. Multiple antimicrobial drug resistance does occur in *E. coli* and other enterobacteriaceal when the drugs are used on a continuous basis in calves.

One of the important factors determining whether or not calves survive enteric colibacillosis is the serum immunoglobulin status of the animal before it develops the disease. Most of the literature on therapy omits this information and is therefore difficult to assess. There is ample evidence that the mortality rate will be high in diarrheic calves which are deficient in serum immunoglobulins, particularly IgG, in spite of exhaustive antimicrobial and fluid therapy. This has stimulated interest in the possible use of purified solutions of bovine gammaglobulin in diarrheic calves which are hypogammaglobulinemic. However, they must be given by the intravenous route and in large amounts, the cost of which would be prohibitive. In addition, they are unlikely to be of value once the calf is affected with diarrhea; they are protective and probably not

curative. Whole blood transfusion to severely affected calves may be used as a source of gammaglobulins but unless given in large quantitites, would not significantly elevate the serum immunoglobulin levels in deficient calves. Limited controlled trials indicate that there is no significant difference in the survival rate of diarrheic calves treated with either a blood transfusion daily for 3 days; fluid therapy given orally, subcutaneously or intravenously depending on the severity of the dehydration; or fluid therapy with antibiotic. Those calves which survived regardless of the type of therapy had high levels of immunoglobulins before they developed diarrhea. This again emphasizes the importance of the calf ingesting liberal quantities of colostrum within the first few hours after birth.

Antiparasymapthomimetics and Intestinal Protectants

Benzetimide has been used for its anticholinergic and antisecretory effect in the treatment of diarrhea in newborn farm animals. Chlorpromazine, a potent in vitro antagonist to enterotoxin in a cell system, will shorten the duration of experimentally induced and naturally occuring diarrhea in piglets infected with enterotoxigenic E. coli. Attapulgate and peptobismal are also effective in preventing fluid accumulation in ligated segments of pig intestine infected with enteropathogenic E. coli. Acetylsalicylic acid and methylprednisolone also reduce fluid accumulation. These drugs which inhibit secretion may play an inportant role in the treatment of enterotoxigenic diarrhea in newborn animals but there is only limited clinical and scientific data available to make a recommendation. However, the rational is logical. Intestinal protectants such as kaolin and pectin are in general use for diarrheic animals but likewise their beneficial value is difficult to evaluate.

Management of Outbreaks

When outbreaks of colibacillosis occur, every effort should be made to isolate affected animals from other susceptible calves and piglets. Recently born calves should be housed or pastured away from affected calves. Beef calves with the disease should be moved out of the calving grounds to an isolation pasture. All new cases should be treated immediately and death submitted to necropsy for examination.

Control

Because of the complex nature of the disease, it is unrealistic to expect total prevention, and control at an economical level should be the major goal. Effective control of colibacillosis can be accomplished by application of three principles:

1. Reduce the degree of exposure of newborn calves and piglets to the infectious agents.
2. Provide maximum non-specific resistance with adequate colostrum and optimum animal husbandry.
3. Increase the specific resistance of the newborn by vaccination of the dam or the newborn.

Reduction of the Degree of Exposure of Newborn Calves and Piglets to Pathogenic *E. coli*

Dairy calves. These comments are directed particularly at calves born inside where contamination is higher than outside. (a) Calves should be born in well-bedded box stalls which were previously cleaned out. (b) The perineum and udder of the dairy cow should be washed shortly before calving. (c) Immediately after birth the navel of the calf should be swabbed with 2% iodine. Tying off the navel at the level of the abdominal wall with cotton thread is also practiced. (d) Calves affected with diarrhea should be removed from the main calf barn if possible and treated in isolation.

Beef calves. These are usually born outside on pasture or on confined calving grounds. (a) Calving grounds should have been free of animals previous to the calving period; the grounds should be well drained, dry and scraped free of snow if possible. A top dressing of the calving grounds with straw or wood shavings will provide a comfortable calving environment. (b) In a few days following birth when the calf is nursing successfully, the cow-calf pair should be moved to a nursery pasture to avoid overcrowding in the calving grounds.

Veal calves. These calves are usually obtained from several different sources and 25-30% or higher may be deficient in serum immunoglobulin. (a) On arrival calves should be placed in their individual calf pens which were previously cleaned, disinfectd and left vacant to dry. (b) Feeding utensils are a frequent source of pathogenic *E. coli* and should be cleaned and air-dried daily. (c) Calves affected with diarrhea should be removed and isolated immediately.

Piglets. Piglets born in a total-confinement system may be exposed to a high infection rate. (a) The all in/all out system of batch farrowing, in which groups of sows farrow within a week, is recommended. This system will allow the herdsman to wean the piglets from a group of sows in a day or two and clean, disinfect and leave vacant a battery of farrowing crates for the next group of sows. This system will reduce the total

occupation time and the infection rate. The continuous farrowing system without regular breaks is not recommended. (b) Before being placed in the farrowing crate, sows should be washed with a suitable disinfectant to reduce the bacterial population of the skin.

Provision of Maximum Non-specific Resistance

This begins with the provision of optimal nutrition to the pregnant dam which will result in a vigorous newborn animal and adequate quantities of colostrum. The next most important control measure is to ensure that colostrum is ingested in liberal quantities within minutes and no later than a few hours after birth. While the optimum amount of colostrum which should be ingested by a certain time after birth is well known, the major difficulty with all species under practical conditions is to know how much colostrum a particular animal has consumed. Because modern livestock production has become so intensive, it is becoming more important for the herdsman to make every effort to ensure that sufficient colostrum is ingested by that particular species. In one study, in large dairy herds 42% of calves left with their dams for one day following birth had failed either to suck sufficient colostrum or to absorb sufficient colostral immunoglobulins. This problem can be alleviated by bottle feeding 1 liter of pooled colostrum to all calves at approximately one day after birth. The other factors which influence the ingestion of colostrum and the absorption of immunoglobulin are presented in the section on epidemiology of colibacillosis and in Chapter III on disease of the newborn.

In large herds where economics permit, a laboratory surveillance system may be used on batches of calves to determine the serum levels of immunoglobulin acquired. An accurate analysis may be done by electrophoresis or an estimation using the zinc sulfate turbidity test. Blood should be collected from calves at 24 hours of age. Samples taken a few days later may not ba a true reflection of the original serum immunoglobulin levels. The information obtained from determination of serum immunoglobulins in calves at 24 hours of age can be used to improve management practices, particularly the early ingestion of colostrum.

The administration of purified bovine gammaglobulin to calves which are deficient appears to be a logical approach but the results have been unsuccessful. Large doses (30-50 g.) of gammaglobulin given intravenously would be required to increase the level of serum gammaglobulin from 0-5 g/dl to 1-5 g/dl of serum which is considered an adequate level. The cost

would be prohibitive. The administration of gammaglobulins by any parenteral route other than the intravenous route does not result in a significant increase in serum levels of the immunoglobulin.

Dairy calves. (a) Immediately after birth, unless the calf is a vigorous sucker, colostrum should be removed from the cow and fed by nipple bottle or by stomach tube at the rate of at least 50 ml/kg body weight in the first 2 hours. (b) The calf should be left with the cow for at least 2 days. This contact will improve the absorption of immunoglobulin. (c) Following the colostral feeding period, dairy calves are usually placed in individual stalls until weaning. A recent development is the feeding of fermented colostrum to newborn calves for up to 3 weeks after birth. This provides a source of lacto-globulins in the intestinal tract and reduces the incidence of neonatal diarrhea of calves due to a wide variety of pathogens. (d) Calves should be fed regularly and preferably by the same person. One of the most important factors affecting dairy calf mortality is the concern and care provided by the calf rearer. (e) The housing and ventilation must be adequate to avoid stress.

Beef calves. (a) Beef calves should be assisted at birth, if necessary, to avoid exhaustion and weakness from a prolonged parturition. (b) Normally beef calves will make attempts to get up and suck within 20 minutes after birth but this may be delayed for up to 8 hours or longer. Beef calves which do not suck within 2 hours should be fed colostrum by nipple bottle or stomach tube. Beef calves deserted by indifferent dams need special attention. (c) Constant surveillance of the calving grounds is necessary to avoid overcrowding, to detect diarrheic calves which should be removed, to avoid mismothering, and to ensure that every calf is seen to nurse its dam. Although up to 25% of beef calves may not have sufficient serum levels of immunoglobulins, the provision of excellent management will minimize the incidence of colibacillosis. The recently developed practice of corticosteroid-induced parturition in cattle may result in a major mismothering problem if too many calves are born too quickly in a confined space. Every management effort must be used to establish the cow-calf herd, as soon as possible after birth. This will require high quality management to reduce even further the infection rate and minimize any stressors in the environment.

Piglets. (a) Every possible economical effort must be made to ensure that each newborn piglet obtains a liberal supply of colostrum within

minutes of birth. The farrowing floor must be well drained and it must be slip-proof to allow the piglets to move easily to the sow's udder. Some herdsmen provide assistance at farrowing, drying off every piglet as it is born and placing it immediately onto a teat. (b) The washing of the sow's udder immediately before farrowing with warm water and soap will reduce the bacterial population and may provide relief in cases of congested and edematous udders. (c) The piglet creep area must be dry, appropriately heated for the first week, and free from draughts.

Increasing Specific Resistance of the Newborn by Vaccinating the Pregnant Dam or the Newborn

The immunization of calves and piglets against colibacillosis by vaccination of the pregnant dam or by vaccination of the fetus or the neonate has received considerable research attention in recent years and the results appear promising. The pregnant dam is vaccinated 2-4 weeks before parturition to stimulate specific antibodies to particular strains of enteropathogenic *E. coli*, and the antibodies are then passed on to the newborn. The mechanism of protection is now considered to be the production of antibodies against the pilus antigens which are responsible for colonization of the *E. coli* in the intestine.

It must be emphasized that vaccination is an aid to good management and not replacement for inadequate management.

Calves. Vaccination of pregnant cattle with either purified *E. coli* K99 pili or whole cell preparation containing sufficient K99 antigen can reduce the incidence of enterotoxigenic colibacillosis in calves. Good protection is also possible when dams are vaccinated with a 4-strain *E. coli* whole cell bacterin containing sufficient K99 pilus antigen and the polysaccharide capsular K antigen. Colostral antibodies specific for K99 pilus antigen on polysaccharide capsular K antigen on the surface of the challenge exposure strain of enterotoxigenic *E. coli* are protective.

These favorable results suggest that vaccination may become a valuable method of control of enteric colibacillosis of calves, especially in problem herds. At the present time it appears that the autogenous bacterins will provide protection against only the homologous bacteria used in the bacterin and if heterologous strains should appear there will be no protection. In some studies in which autogenous vaccines did not provide protection against diarrhea in newborn calves, the precise cause of the diarrhea was not determined. The other problem is that calves must still

ingest colostrum in sufficient quantities soon enough to acquire the protection afforded by the vaccine.

The oral immunization of young calves using a feed-incorporated vaccine comprising heat-inactivated *E. coli* resulted in improved health attributed to a reduction in the incidence and duration of diarrhea and in the need for treatment with antibiotics. The nature of the protection is attributed to an improvement in local intestinal immune mechanisms. The technique needs further study before it can be recommended.

There has been considerable interest in the immunization of the bovine fetus in utero and the neonate after birth. In utero vaccination of bovine fetuses with an *E. coli* result in a marked increase in the number of IgA containing plasma cells in the jejunum and ileum and IgM antibody is also stimulated which provides protection against experimental challenge at birth. The use of a colisepticemic strain may provide non-serotype-specific resistance against coliform septicemia. However, this route of vaccination does not appear to be practicable at the present time.

The antibody response of newborn calves to a vaccine depends on their colostral antibody status. There is a marked failure of the colostrum-fed calf to respond to injected antigens, not because of the relatively immature lymphoid system, but rather the presence of maternal antibody to the antigens used interferes with specific antibody stimulation.

Piglets. Field experience has shown that the piglets born from gilts are more susceptible than those from mature sows, which suggests that immunity improves with parity. On a practical basis this suggests that gilts should be mixed with older sows which have been resident on the premises for some time. The length of time required for such natural immunization to occur is unknown but 1 month during late gestation seems logical.

Considerable work has shown that artificial immunity to enteric colibacillosis in piglets is feasible. Three antigen types of pili, designated K88, K99, and 987P are now implicated in colonization of the small intestine of newborn pigs by enterotoxigenic *E. coli*. The vaccination of pregnant sows with oral or parenteral vaccines containing these antigens will provide protection to newborn piglets against enterotoxigenic colibacillosis caused *E. coli*-bearing pili homologous to those in the vaccines. The parenteral vaccines are cell-free preparations of pili and the oral vaccines contain live enteropathogenic *E. coli*. The oral vaccine

is given 2 weeks before farrowing and is administered in feed daily for 3 days as 200 ml. of a broth culture containing 10^{11} *E. coli*/day.

Immunization of pregnant sows with an *E. coli* bacterin, enriched with the K88-antigen of *E. coli* results in the secretion of milk capable of preventing adhesion of K88-positive *E. coli* to the gut for at least 5 weeks after birth at which time the piglet becomes resistant to adhesion by the organism.

A combination of oral and parenteral vaccination is considered to be superior. The oral *E. coli* vaccine is administered in the feed beginning about 8 weeks after breeding and continued to parturition. The parenteral vaccine is given about 18 days before parturition and results in the production of high levels of IgM antibody for protection against both experimental and naturally occurring enterotoxigenic colibacillosis of piglets. This vaccination protocol also reduces the number of *E. coli* excreted in the feces of vaccinated sows which are major sources of enteropathogenic *E. coli*.

A simple practical and effective method of immunization of pregnant sows is by the feeding of live cultures of enterotoxigenic *E. coli* isolated from piglets affected with neonatal colibacillosis on the same farm.

The possibility of selecting and breeding from pigs that may be resistant genetically to the disease is being explored. The highest incidence of diarrhea occurs in progeny of resistant dams and susceptible dams. The homozygous dominants (SS) and the homozygous recessives (ss) are resistent.

The use of abattoir porcine serum-drived immunoglobulins as milk replacer additives for the artificial rearing of colostrum-deprived piglets in a farm environment may have a place under certain special circumstances.

Lambs. Vaccination of pregnant ewes with K99 antigen will confer colostral immunity to lambs challenged with K99-positive enteropathogenic *E. coli*. The antibodies are anti-adhesive.

CRYPTOSPORIDIOSIS

Otto M. Radostits

Cryptosporidiosis caused by the genus Cryptosporidium occurs in calves (1-4), lambs (5), foals (6) and piglets (7). The parasite has been found in the intestines of several other species of mammals and its significance is not clear. Cryptosporidia have been found along with rotavirus and coronavirus in the intestines of calves with acute neonatal diarrhea (8). It is not known if the cryptosporidia cause or only contribute to enteric disease because of the presence of other enteropathogens. The disease has occurred in a veterinary student in contact with diarrheic calves affected with cryptosporidiosis (15).

There are only a few epidemiological characteristics available. Cryptosporidia have been found in diarrheic calves as early as 5-7 days of age (2), but are more common in calves at about 15 days of age (1). This suggests that the organism is widespread and that the infection rate may be much higher than the rate of clinical disease. Outbreaks of the disease are recorded in which 60% of the calves are shedding the oocysts and 85% of the calves have diarrhea.

The protozoa have been found in diarrheic lambs from 6 to 14 days of age (5), in diarrheic foals with combined immunodeficiency (6) and in young piglets with diarrhea (7).

It would appear that cryptosporidia may be a primary cause of diarrhea in newborn farm animals or be part of a mixed multiple infection causing acute neonatal diarrhea. The presence of the cryptosporidia along with enteropathogenic E. coli may increase the mortality rate in calves affected with diarrhea (4).

The complete lifecycle of the protozoan is not known (9). The cryptosporidia appear free in the lumen of the intestine and attached to

the microvilli of the villous epithelial cells. All stages of the parasite, merozoites, trophozoites, schizonts, gametes and oocysts, can be found attached to a single villus. Varying degrees of villous atrophy occur which impairs digestion and absorption resulting in diarrhea. Oocyts are shed in the feces from calves as early as 5-12 days after birth and continue for 3-12 days (10). The shedding of oocysts in the feces usually coincides with the onset of diarrhea which occurs about 4-6 days after experimental inoculation (13).

The disease can be transmitted experimentally by oral inoculation with feces containing cryptosporidia (11). Specific pathogen-free lambs can be infected using calf feces containing cryptosporidia (11). Gnotobiotic calves and piglets can be infected with calf feces containing cryptosporidia (13) and several other species can be infected with calf feces containing the organism (12). This suggests that the cryptosporidia is not host-specific.

In the naturally occurring disease in calves there is a mild-to-moderate diarrhea which may persist intermittently for up to 2 weeks. There may be two episodes of diarrhea, the first at 15 days of age followed by recovery in 5-7 days. The second episode may occur 1-3 weeks later and last only 4 days (1).

Experimentally in calves there is a sudden onset of diarrhea 48 hours after oral inoculation. The feces contain flecks of blood and mucus plugs and the diarrhea may persist for several days until death.

The oocysts can be detected by examination of fecal smears from Giemsa stain (3) or by fecal flotation (10). The organisms can also be detected by examination of scrappings of ileal mucosa, supplemented when possible by histological examination of ileum fixed in formalin immediately after death.

At necropsy, there is villous atrophy in the ileum and large

intestines. Histologically, the cryptosporidia are associated with the microvilli of the villous epithelial cells (14). In the large intestines the lesion involve both the surface and crypt epithelium (13). Intestinal samples submitted for histological examination must be fixed in formalin immediately after death. In foals, with combined immunodeficiency, the cryptosporidia are widespread in the digestive tract (6).

Treatment has been unreliable. Sulfadimidine at 5 g/day for 3 days may be effective, but the diarrhea may recur in 7 days (2). Sulfaquinoxaline may reduce the mortality in calves affected with enteric colibacillosis and cryptosporidiosis (4). Supportive therapy for dehydration is also indicated.

There are no known specific control measures. Sanitation and hygiene and control of population density would appear to be a logical approach.

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Besnoitiosis

Otto M. Radostits

Cutaneous besnoitiosis is restricted in its distribution to southwestern Europe and African continent. Besnoitia besnoiti has been isolated from cutaneous lesions in cattle and horses and B. bennetti from burros in the United States.

Infection is thought to occur by ingestion of feed contaminated by the feces of an infected cat. Cats become infected by eating cysts in the tissues of infected intermediate hosts. The development of the disease in the cat is confined to the intestine. In cattle, infections of the teat skin may result in lesions and around the mouth in suckled calves. In cattle in the early stages there is high fever, increase in pulse and respiratory rates, and warm, painful swellings appear on the ventral aspects of the body, interfering with movement. The superficial lymph nodes are swollen, diarrhea may occur and pregnant cows may abort. Affected bulls often become sterile for long periods. Cystic stages of the besnoitia have been found in the testes of a male burro, and in the uterus and vagina of two cows. Lacrimation and an increased nasal discharge are evident and small, whitish, elevated macules may be observed on the conjunctiva and nasal mucosa. Cysts on the scleral conjunctiva are considered to be of particular diagnostic significance. The nasal discharge is serous initially but becomes mucopurulent later and may contain blood. Subsequently the skin becomes grossly thickened and the hair falls out. A severe dermatitis is present over most of the body face. The mortality rate is about 10% and the convalescence in survivors is protracted over a period of months. Necropsy lesions in cattle with the severe form of the disease are characterized by widespread vascular lesions and secondary lesions in skeletal and heart muscle, and lungs. Sarcocystis-like schizonts are present in some

lesions.

In wild goats and domesticated family goats besnoitiosis is recorded in Iran, New Zealand and Kenya. The principal lesions are on the skin and the genital tract. The cutaneous lesion is a chronic dermatitis of the legs and ventral surface of the abdomen. The hair is sparce, the skin cracked and oozing. The worst lesions are usually around the fetlock. There are also white, gritty granules subcutaneously over the hindquarters.

Horses are affected in much the same manner but the disease is less severe and the course less protracted. The disease also occurs in many species of wildlife in Australia, in impala and blue wildebeeste in Africa and caribou in Canada but on the basis of cross-infection tests the individual isolates are distinct strains.

Clinico-pathological diagnosis depends upon detection of the cysts containing a number of spindle-shaped spores in scrapings or sections of skin. Many infected animals show no clinical signs of infection, and laboratory diagnosis using a complement fixation test has been attempted unsuccessfully. Serum antibodies to Besnoitia sp. are also identifiable by an indirect immunofluorescence technique. No specific treatment is available and affected animals should be treated symptomatically for enteritis or dermatitis.

A vaccine containing Besnoitia besnoiti, grown on tissue culture, and originally isolated from blue wildebeeste, has been used to vaccinate cattle. A durable immunity to the clinical form of the disease was produced in 100% of vaccinates, but subclinical infection at a low level did occur.

COLOSTRUM FOR NEWBORN DAIRY CALVES

J.G. Linn, and D.E. Otterby
Department of Animal Science
University of Minnesota

When first born, calves have no immunity against diseases. No antibodies or immunoglobulins (Ig) are transferred across the placenta from the dam to the fetus. Thus, ingestion of colostrum is the only source of immunoglobulins (Ig) and is the method by which calves acquire immunity against diseases. This method is known as passive immunity and without it, most calves will die. Colostrum also provides a highly nutritious balanced diet for newborn calves.

COLOSTRUM

Colostrum is broadly defined as the milk-like substance secreted from the mammary gland immediately following, or in some cases just prior to, freshening. It usually includes the first six milkings following freshening and is unsaleable. However, the first milking of colostrum is the most important for calves as it is highest in immunoglobulins and nutritional value (Table 1.).

Slightly bloody or tainted first milk colostrum can be safely fed to calves, but discard extremely abnormal colostrum such as from cows with acute mastitis.

Table 1. Composition and characteristics of colostrum and normal whole milk.

Item	First milking	Second milking	Second day	Third day	Whole milk
Specific gravity (gm/ml)	1.056	1.040	1.034	1.033	1.032
Total solids (%)	23.9	17.9	14.0	13.6	12.9
Fat (%)	6.7	5.4	4.1	4.3	4.0
Nonfat solids (%)	16.7	12.2	9.6	9.5	8.8
Protein (%)	14.0	8.4	4.6	4.1	3.1
Lactose (%)	2.7	3.9	4.5	4.7	5.0
Ash (%)	1.1	1.0	.8	.8	.7
Vitamin A (ug/100 ml)	295.0	190.0	95.0	74.0	34.0
Immunoglobulins (%)	6.0	4.2	1.0	---	.09

IMMUNOGLOGULINS

The principal immunoglobulins in colostrum are IgG_1, IgG_2, IgM and IgA. Collectively, they are known as gamma globulins. The IgG_1 and IgG_2 represent about 85 percent of the immunoglobulins in colostrum. The primary function of IgG_1, IgG_2 and IgM is against systemic infections in calves, whereas, IgA appears to check the growth of bacteria and viruses in the digestive tract.

The primary source of immunoglobulins found in colostrum is blood. During the dry period, immunoglobulins are transferred from the blood to the mammary gland where they are accumulated. Cows which are not dried up, or have too short a dry period (less than 30 days) before their next lactation will not have sufficient levels of immunoglobulins in their first milk colostrum for feeding newborn calves.

IMMUNOGLOBULIN CONTENT OF COLOSTRUM

The immunoglobulin content of colostrum is affected by age and breed of cows along with successive milkings following calving. Cows in second or later lactations produce larger quantities of colostrum and have higher immunoglobulin concentrations in colostrum than cows in first lactation (Table 2). Older cows have been exposed to a wider range of diseases than young animals and therefore have produced more immunoglobulins against them. Thus, the first milking of colostrum from older cows can be fed to calves from first lactation heifers if livability problems are encountered.

Table 2. Effects of cow age on amount and immunoglobulin content of colostrum produced[a]

Lactation number	Amount	IgG_1	IgG_2	IgM	IgA
	--lb--	----------mg/ml----------			
1	53	14.5	3.8	3.1	.47
2	83	14.3	3.4	3.4	.29
3	82	18.4	3.9	3.1	.43
4	78	21.9	3.8	3.6	.48
5-8	75	18.6	3.7	3.3	.52

[a]Amount and immunoglobulin content represents the collection and composite of the first four milkings following calving. Source. Univ. of Illinois.

Guernsey cows have been found to have lower colostrum levels of IgA and IgM than other breeds. Also, the disappearance of immunoglobulins from colostrum with successive milkings following calving is greater than for other breeds. The first milking of colostrum is highest in immunoglobulin content and with each milking following calving, immunoglobulin content declines about 50 percent. In most cows, the fourth milking contains near normal milk amounts of immunoglobulins. Leaker cows can loose substantial amounts of immunoglobulins so that the first milking of colostrum is actually closer to the second or third milking in immunoglobulin content.

NUTRITIVE VALUE OF COLOSTRUM

Colostrum contains more total solids or nutrients than whole milk (table 1). Protein, primarily in the form of immunoglobulins, fat, minerals and vitamins are all higher in colostrum than whole milk. The high levels of vitamin A are particularly important to calves because liver stores are very low in newborns. Lactose or milk sugar is lower in colostrum than in whole milk. This is an advantage and a natural protective effect as high levels (above 1 lb/day) can cause young calves to scour.

FEEDING

The protection of newborn calves against diseases is dependent on the amount of immunoglobulins fed and their absorption from the digestive tract. In general, some 10 to 30 percent of all calves have low immunity levels due to a failure to absorb immunoglobulins or feeding of insufficient amounts.

Absorption from digestive tract. The highest absorption of immunoglobulins occurs immediately after birth. During the first six hours after birth, all intestinal cells have absorptive capacity, but by 24 hours most of the absorption capability is lost (Table 3). Feeding colostrum immunoglobulins after intestinal absorption has ceased is of no immunity benefit except for some local protective action against disease-causing organisms within the intestinal tract. However, bacteria present in the intestine before the first feeding of colostrum block absorption of immunoglobulins and accelerate intestinal closure to absorption. Thus, for maximum absorption of immunoglobulins, the first milking of colostrum should be fed to calves within 15 to 30 minutes of birth and before the intestinal tract becomes innoculated with bacteria.

Table 3. Effect of age at first feeding on immunoglobulin absorption.

Age at first feeding (hours)	IgG	IgM	IgA
	---Percent of calves absorbing Ig----		
12	100	100	97
16	90	97	83
20	77	70	70
24	50	47	43

Source - Univ. of Arizona

Amounts of colostrum to feed. Calves should receive six percent of their birth weight in first milk colostrum within the first six hours after birth.

This amounts to about 5.5 pounds or 2.5 quarts for a 90 pound calf. Research has shown calves fed 4.5 pounds of colostrum at birth had nearly double the blood IgG level (14.9 mg/ml) of calves fed 2.5 pounds (8.5 mg/ml) and triple calves fed 1 pound at birth (5.2 mg/ml). For protection against heavy disease challenges, calves should receive 3/4-1 pound of immunolglobulins during the first 24 hours of life. Calves should receive 10 and up to 12 percent of their birth weight in the first milking of colostrum during the first 24 hours after birth.

Methods of feeding. Milking colostrum from cows and hand feeding it to newborn calves is recommended. Calves which do nurse have a faster rate and higher amount of immunoglobulin absorption, but only 58 percent of the calves allowed to nurse consume sufficient quantities of immunoglobulins for disease protection. Thus, if calves are allowed to nurse, they should be assisted, but hand feeding of colostrum is preferred. If calves refuse to eat, colostrum should be fed using an esophageal feeder or stomach tube.

EMERGENCY COLOSTRUM SOURCES

Sometimes colostrum is not available from cows. A frozen supply of first milking colostrum from home-raised older cows should be kept for use in these situations. Frozen colostrum is preferred, but sour or fermented colostrum can be used in emergencies. Sour colostrum should be buffered with sodium bicarbonate (1 teaspoon per quart) at time of feeding to enhance absorption of immunoglobulins.

SECOND AND THIRD DAY FEEDING

Colostrum milkings should be fed to calves the second and third day of life. The immunoglobulins in this colostrum will not be absorbed but may provide some local protective action against bacteria and other organisms in the

digestive tract. Starting the second day of life, calves should be fed colostrum or milk amounts equal to 8 percent of their birth weight (about 7 pounds for a 90 pound calf). This feeding rate should be maintained until weaning.

SUMMARY

1. Colostrum is both a source of immunoglobulins for disease protection and a highly nutritious diet for calves.
2. The first milking of colostrum is highest in immunoglobulin content. Older cows will have higher colostrum immunoglobulin levels than first lactation heifers.
3. Calves should receive 1 to 2 quarts of colostrum within 30 minutes after birth. A total of 10 to 12 percent of birth weight should be fed in colostrum by 24 hours.
4. Hand feeding colostrum is recommended. Almost half of all calves allowed to nurse have insufficient immunoglobulin levels for disease protection.
5. Calves should receive colostrum the second and third day of life at a rate of 8 percent of their birth weight.

Feeding Programs for Young Dairy Calves

D. E. Otterby and J. G. Linn
Department of Animal Science
University of Minnesota

Introduction

The young calf requires a highly concentrated source of energy and nutrients in an easily digested form. Enzymes and secretions of the abomasum and intestinal tract have evolved to best digest dam's milk, but other liquid feeds have been tested and many have proven to be satisfactory for use after the first 3 days of life. The calf's digestive system changes considerably from birth to three weeks of age. Pancreatic proteases, lipase and amylase increase during this time enabling the calf to digest a wider spectrum of proteins, fats and carbohydrates than is possible at birth and for several days thereafter. The main carbohydrate-splitting enzyme, lactase, decreases with age but is abundant in early life. Thus, the feed chosen for this early feeding period must not only be concentrated in nutrients because of the calf's limited intake of dry matter, at about 1 to 1.5% of body weight, but also must fit the digestive capabilities of the calf. The main goals during liquid feeding for herd replacements are to keep the calf healthy, growing at a modest rate and to begin introduction of dry feed. Weight gains of one half to one pound per day are acceptable during the first six weeks of life. Larger gains of a pound or more a day are not necessary and in fact, would be difficult to achieve with the limited dry matter intake of calves and if achievable, would likely require a more expensive feeding program.

Requirements of the Young Calf

Recommendations for supplying nutrients to calves have been given by the National Research Council (1978) and should be used as guidelines in developing feeding programs.

Table 1. Daily Requirements of Young Calves

	Body weight	Daily gain	Crude protein	NEm	NEg	TDN	Calcium		Phosphorus		Vitamins A	Vitamins D
	lb	lb	lb	Mcal	Mcal	lb	lb	g	lb	g	-- I.U.--	
Small	55	.7	.25	.8	.4	1.2	.013	5.9	.009	4.1	1000	165
breeds	65	.8	.28	.9	.6	1.4	.280	6.4	.010	4.5	1200	200
Large	90	.9	.33	1.2	.7	1.7	.33	8.2	.011	5.0	1800	280
breeds	100	1.2	.44	1.4	1.0	2.5	.36	8.2	.013	5.9	1900	300

Adapted from Nutrient Requirements of Dairy Cattle, 1978.

The weight gain of calves during the early part of the liquid feeding phase are not likely to be great and the liquid portion of the diet alone is not likely to support large gains. Most feeding programs call for milk or milk replacer to be fed in limited amounts. Hence, if calves are to make gains similar to those in Table 1, they must begin consuming dry feeds, i.e. calf starters, early. Limited or modest liquid feeding is designed to promote early consumption of starter and yet maintain satisfactory growth and health. For replacement heifers, starter should be introduced on day 4 of life to reduce cost of the feeding program and to initiate rumen development.

Liquid Feeds

Choice of liquid feed depends on availability, cost, convenience and preference. Each of the following can be used satisfactorily provided good management practices are followed.

Whole Milk. Whole milk is and has been the standard in feeding practice and in experimentation for judging and comparing other feeds. Although whole milk varies in composition among cows, it is readily accepted and easily digested by the calf. The nutrient content corresponds closely to or exceeds the need of the calf (see Table 2). Nutrients that may be limiting are some of the trace minerals such as cobalt, iron, copper, manganese and zinc. Quality of protein and energy availability are excellent.

Recent recommendations suggest that whole milk be fed at about 8% of birth weight (Table 3). This amounts to feeding milk solids at 1% of body weight. Amount offered is not increased throughout the period of liquid feeding.

Table 2. Nutrient contents of whole milk and National Research council recommended nutrient content of milk replacers.

Item	Whole milk	NRC recommendations for milk replacers
Crude protein, %	26.9	22
NEm, Mcal/lb	1.64	1.09
NEg, Mcal/lb	.91	.70
TDN, %	130	95
Fat, %	28	10
Calcium, %	.89	.7
Phosphorus, %	.72	.5
Potassium, %	1.16	.8
Magnesium, %	.08	.07
Sodium, %	.34	.1
Chlorine, %	.92	.5
Cobalt, ppm	.005	.1
Iron, ppm	<10	100
Copper, ppm	.8	10
Zinc, ppm	23	40
Manganese, ppm	--	40
Selenium, ppm	--	.1
Vitamin A, I.U./lb	6800	1720

Adapted from Nutrient Requirements of Dairy Cattle, 1978.

Table 3. Suggested rates for feeding whole milk to replacement calves.

Body weight lb	Whole milk lb	Solids content lb
50-60	5	.6
61-70	5.5	.7
71-80	6.5	.8
81-90	7.0	.9
91-100	8.0	1.0
101-110	9.0	1.1

Waste or Discard Milk. Waste milk can be used as a source of liquid feed for young calves. Waste milk is defined as unmarketable milk from cows that were

treated for mastitis, metritis or other health problems. This milk can be fed fresh in the same manner as whole milk. Extremely abnormal milk (bloody or watery) should not be fed. Excess waste milk can be fermented or preserved chemically. Fermentation of milk from three to six milkings post antibiotic treatment will proceed as normal milk or colostrum. Milk from the first milking after treatment does not ferment as well. But studies indicate that a mixture of all unmarketable milk post treatment usually ferments well. Some reports have suggested high mortality in calves fed mastitic milk and a higher incidence of mastitis in first-parity heifers fed mastitic milk as calves. A recent report showed that calves fed pasteurized milk containing an added culture of live Staphylococcus aureus did not show evidence of the organism in body tissues 10 to 14 days after exposure. Also, lactating first calf heifers fed Staphylococcus aureus in pasteurized milk as calves did not have an increased incidence of infection as compared to controls. Mastitic milk should not be fed to calves under two days of age when the intestine is permeable to large protein molecules. Incidence of diarrhea appears to be no more prevalent in calves fed mastitic milk than in calves fed whole milk. It is recommended that calves fed mastitic milk be housed separately to prevent introduction of organisms into the teat canal through suckling. Also, calves fed milk containing antibiotics should not be marketed for meat unless a suitable withdrawal period is used before slaughter. Although mastitic milk may be slightly higher in solids than whole milk recommended feeding rates are similar.

Surplus Colostrum. The use of excess colostrum as the liquid diet beyond 3 days of age was advocated years ago, but little was used. Many thought that colostrum was not a suitable feed for calves older than a few days of age. Research work in the 1960's and early 1970's stimulated dairy producers to include surplus colostrum in feeding programs and dispelled many of the notions

that colostrum was not nutritious or would cause severe digestive problems. It has been estimated that enough surplus colostrum is produced in most dairy herds to feed heifers born in the herd from birth to weaning at 28 days of age. Suitable storage for the surplus must be provided. Methods for storage include refrigeration, freezing, souring and chemical preservation.

Refrigeration. Colostrum can be stored for about 7 days in the refrigerator and not change appreciably in composition or acidity. Length of life will depend on sanitary conditions at milking and how quickly the material was cooled after milking. Some dairy producers have used small bulk tanks for cooling and storing colostrum. This works well in larger herds where volume of colostrum is relatively large

Freezing. This is an excellent method of preserving colostrum for an indefinite period. Nutrient content is preserved with little loss. Milk cartons or plastic 1/2 or 1 gallon jugs make good storage containers. Disadvantages are thawing time and the need for freezer space to store the excess colostrum which can be considerable. Thawing at ambient temperatures will require several hours so frozen colostrum should be removed from storage early to allow sufficient time for thawing.

Souring or fermenting. Colostrum can be stored at ambient temperatures and allowed to ferment. The product will become acid and can be kept for 3-4 weeks. Several research trials and practical experience by dairy producers indicate that this is an acceptable method of storing surplus colostrum. Calves have performed satisfactorily when fed soured colostrum. Some have suggested less scouring problems when soured colostrum is fed, but this is not a universal observation.

The following guidelines should be followed for successful souring of colostrum: A covered container should be used to store the fermenting colostrum. A container with a plastic liner is easier to clean before each reuse. Furthermore, a plastic liner prevents the soured milk (which becomes quite acidic) from corroding a metal container and causing excessive intake of zinc or other minerals. Some producers innoculate the fresh batch with 3 to 5 tablespoons of fermented colostrum from a previous batch to help start proper fermentation. Fermented colostrum should not be held for long periods before feeding, especially in summer. Research has shown that over time this product can become very acid and putrid with long storage. The fermenting colostrum should be stirred daily, during storage and prior to feeding. Stirring helps prevent scum from forming and reduces size of large curds. Fermented colostrum should be stored away from direct sunlight. Extremely bloody colostrum should not be fermented. Feeding the fermenting colostrum can begin on the 4th day after birth (after feeding the fresh undiluted colostrum directly from the cow the first 3 days). By doing this, the colostrum hasn't yet completed its fermenting process and won't be as acid tasting to the calf. This helps teach the calf to consume the soured product.

If two or more batches of soured colostrum have been collected for at least a week and have fully fermented, they may be mixed together to save space and shorten the time spent mixing.

Colostrum produced during a 3-5 day period may be mixed together. Otherwise, a new batch should be started for colostrum produced after this period. If the colostrum is diluted, warm water can be used to increase acceptability.

Chemically preserved colostrum. Acids may be used to reduce pH of colostrum and preserve it for extended periods. Colostrum preservatives prevent or decrease microbial fermentation. Adding acid to the colostrum has the same

effect as acid produced by microbes--an environment too acidic for further growth of most microorganisms. Propionic, acetic and formic acid all have been successfully used. Propionic acid that is used for preserving high moisture corn can be used for treating colostrum. Rate of addition is about .7% propionic acid by weight.

If acetic or formic are used, the rates are less because these are stronger acids than propionic. Acceptability of acid-treated or soured colstrum is sometimes a problem. Addition of sodium bicarbonate (.6% of weight of colostrum or .8 ounce per gallon) just prior to feeding can reduce the acidity problem.

Feeding. Colostrum is a good source of nutrients. Total solids of mixed colostrum usually range from 14 to 18%. Fat and protein will be somewhat higher than normal milk, but lactose will be lower. Therefore to feed the same quantity of solids as whole milk only about 75% of the amount needs to be fed (Table 4). Thus, calves that would receive 8 pounds of whole milk will require only 6 pounds of mixed colostrum. A convenient method of feeding is to mix 3 parts of colostrum with 1 of water. If solids are quite high a 2.1 dilution can be used. If most of the colostrum is from the 5th and 6th milkings postpartum, no dilution is required.

Table 4. Feeding and dilution rates for colostrum[1]

Body weight	Colostrum	Water
lb	lb	lb
50-60	3.6	1.2
61-70	4.2	1.4
71-80	4.8	1.6
81-90	5.4	1.8
91-100	6.0	2.0
101-110	6.6	2.2

[1]Solids content is assumed to be 16 percent.

Milk Replacers. The first true milk replacers were developed over 30 years ago. Initially they were formulated from dried skim milk, dried buttermilk, dried

whey and animal fat. As costs of milk products increased, non-milk sources of protein were included. When large amounts of non-milk proteins were part of the replacer, quality suffered and performance of calves was less than desirable. Through research by universities and private organizations, much improvement has been made in milk replacers. The best sources of protein for milk replacers are milk products processed under controlled conditions to avoid heat damage. All-milk products (protein) are commercially available. They are generally more expensive than replacers that contain a portion of the protein from other sources. Certain of these non-milk proteins are satisfactory and include chemically modified soy, soy isolates and soy protein concentrates. Soy flours have not given good performance and often have resulted in digestive disturbances. Anti-nutritional factors in soy flours are antigens and trypsin inhibitor. Trypsin inhibitor activity can be reduced by heat and antigens by extraction and/or other modification. Performance of calves fed milk replacers with modified soy products has been satisfactory, though not equal to whole milk. Other products such as alfalfa, blood, fish, meat solubles, pea, rapeseed and others have been investigated. Meat solubles, fish protein concentrate, soy flour, distillers, dried solubles, brewers dried yeast, oat flour and wheat flour have been inferior sources of protein.

Carbohydrates used in milk replacers are generally supplied from whey products to provide lactose. Glucose is a satisfactory carbohydrate, but is not often used in replacers. Sucrose and starch are not good sources of carbohydrate for young preruminant calves. Crude fiber contents of milk replacers should be low, and those replacers containing larger amounts usually are high in cereal grains and should not be used for young calves.

Good quality animal or hydrogenated vegetable fats are incorporated into most replacers at the rate 10 to 20%. These products are highly acceptable as energy sources.

Fortification of milk replacers with fat and water soluble vitamins, minerals and low concentrations of antibiotics is common practice. The National Research Council recommends 2.6 ppm niacin, 13 ppm pantothenic acid, 6.5 ppm riboflavin, 6.5 ppm pyridoxine, 6.5 ppm thiamine, .5 ppm folic acid, .1 ppm biotin, .07 ppm B_{12} and .26% choline per unit of replacer. The National Research Council suggests 20-40 ppm antibiotics, such as chlortetracycline and oxytetracycline, in milk replacers for promoting growth and concentrations of 50-100 ppm for prevention of reduction of diarrhea. Many veterinary practioneers prefer no antibiotics in milk replacers, but these are not as available as the other.

Feeding rates and mixing suggestions for milk replacers are suggested by individual manufacturers but reconstituting to the approximate solids content of whole milk is the general practice. A possible reconstitution schedule is in Table 5.

Table 5. Reconstitution schedule for milk replacers.

Body weight	Replacer	Water
lb	lb	lb
50-60	.6	4
61-70	.7	5
71-80	.8	5.5
81-90	.9	6
91-100	1.0	7
101-110	1.1	8

Milk replacer can be fed warm or cold, but use of warm water in mixing will facilitate mixing and may improve acceptability of the replacer by calves.

Method of Feeding Liquid Feeds

Feeding milk or milk replacer by open pail is a common practice although nipple feeding by pail or bottle is chosen by many calf raisers. No real advantage in calf health or performance has been shown for either method. Feeding

large numbers of calves by either of these methods is slow and tedious and many large producers have been interested in automated feeding. Mobile units with refrigerated storage of feed that automatically dispense feed at given intervals have been used where large numbers of calves are fed. Stationary automatic dispensers also have been used. Many large operators dispense milk by hose from mobile thermos tanks into individual pails rather than use automated systems. Others have organized feeding with equipment for mixing and transporting liquid-feed in nipple bottles or pails. Time for clean-up of equipment also has been reduced through organization and provision of good facilities. Cleaning and sanitizing calf feeding equipment in manner similar to that used for milking equipment is recommended.

Frequency of Feeding

Most calf raisers feed twice daily. Half the amounts given in the feeding schedules (Tables 3 and 4) can be offered at each feeding. Feeding twice daily assures at least two observations per day of the calf and probably is more satisfying to the calf. Once-a-day feeding of liquid has been shown to be an acceptable management practice. A review of several experiments indicated good performance of calves fed milk, colostrum, or milk replacer once daily. Quality of milk replacer, amount, and concentration of replacer require careful attention, but labor requirements for feeding can be reduced as much as 40%. Reduction of liquid feeding to 6 times per week also has been reported. Although this procedure resulted in satisfactory gains and health, it has not been adopted as yet by many calf growers. If once-a-day feeding is practiced, calves should be observed at least once in addition to feeding time for general health and well-being.

Feeding Calves Housed in Hutches

Feeding programs for calves in hutches can be similar to those used for

calves in nurseries during most of the year. During extremely cold weather feeding rates should be increased because energy requirements of the calves will be greater. Although exact information is not available, increasing the amount fed by factors of 1.25 to 1.5 and offering the feed three times daily has helped provide the nutrients needed by these calves. Young calves that appear to be extremely cold and are doing poorly should be placed in warmer quarters.

Weaning

Abrupt weaning of calves at an early age is an acceptable practice. Research has shown that calves may be weaned successfully at three weeks of age, but most producers wean between 4-8 weeks. Weaning later than 8 weeks could lead to fat calves. If weaned at 21 days, calves may suffer from slightly depressed growth rates. However, by 12 weeks of age, early-weaned calves and later-weaned calves (6 weeks) are of similar weights. Weaning according to starter intake (1-1½ lb/day) is a good practice, but abrupt weaning usually stimulates dry feed consumption. Starter intake can be encouraged by placing the dry feed in the pail immediately after the liquid has been consumed. In general, early weaning can reduce feed and labor costs and good results can be obtained with weaning at 21 to 35 days of age. Poor doing calves or calves eating less than 1 lb of starter per day should be fed liquid until performance improves and dry feed is consumed in satisfactory amounts.

Calf Starters

Calf starters are concentrated sources of nutrients usually formulated from air dry grains and supplements.. They must be relatively high in protein and energy and must be readily acceptable to the calf. Recommended nutrient content is in Table 6. Commercial calf starters are available and, in general, are of excellent quality. Starters can be put together by the producer and these will achieve satisfactory growth in the calves. Plant protein sources are generally

used for calf starters. Soybean meal is commonly used although sunflower, linseed, cottonseed, rapeseed, peanut, meat and fish meals have given satisfactory results in research studies. Nonprotein nitrogen has been used successfully in some studies, but usually is not recommended for calves under three months of age.

Shelled corn, corn and cob meal, and oats are all good grains to include in calf starters. they will provide adequate energy and are readily acceptable to the calf. Dried whey or other dry milk products can be included if price permits. Molasses can be added up to 5-10 percent for energy, increased palatability and for reducing dust.

Crude fiber content of calf starters probably should be 5% or more. Oats, wheat bran, corn and cob meal or small amounts of good quality chopped alfalfa will supply enough fiber. Extremely low crude fiber does not support gains as well as moderate fiber. The exact percentage has not been ascertained and probably depends on other factors such as physical form of fiber. Fiber below 5 to 6% probably will not produce best growth, and some bloating may occur. Simple mixtures with 13.9% crude fiber have supported growth as well as complex mixtures containing 6% crude fiber.

Starters must be supplemented with adequate vitamins A, D and E and minerals because this feed will be the main source of nutrients after weaning. Supplemental calcium and phosphorus that are readily available, salt and trace minerals are necessary ingredients.

Antibiotics, buffers and flavors are sometimes added to starters. If antibiotics are included, instructions on their use should be followed carefully. Minnesota research did not show any benefit in performance of calves when sodium bicarbonate was added at the rate of two percent of the feed. Flavoring agents

Table 6. Recommended nutrient content of calf starters[1].

Crude protein	16.0
Net Energy m, Mcal/lb	.86
Net energy g, Mcal/lb	.54
TDN, %	80
Fat, %	2
Calcium, %	.60
Phosphorus, %	.42
Magnesium, %	.07
Potassium, %	.80
Salt, %	.25
Sulfur, %	.21
Iron, ppm	100
Cobalt, ppm	.10
Copper, ppm	10
Manganese, ppm	40
Zinc, ppm	40
Lodine, ppm	.25
Selenium, ppm	.10
Vitamin A, I.U./lb	1000
Vitamin D, I.U./lb	140

[1]Adapted from 1978 NRC.

can be used to enhance feed intake, but not all are successful. Molasses is good for this purpose.

Physical form of starters is of extreme importance. Grains that are coarsely ground or rolled are preferable. Whole grains are acceptable if other ingredients can be mixed in effectively. Liquid molasses can help to achieve uniform distribution of ingredients in the mixture. Finely ground starters are mealy and will not be accepted as well as more coarsely prepared feeds. Pellets are not accepted as well as coarsely ground mash. Mashes generally have a higher proportion of larger particles, suggesting that 50% of the particles should be larger than 1190 um (16 mesh).

Some examples of good calf starters are in Table 7. Excellent starters are available commercially.

Table 7. Examples of some good calf starters.

Ingredient	A	B	C	D	E
Corn, cracked or coarsely ground, lb	50	50	39	55	50
Oats, rolled or crushed, lb	34	29	--	12	20
Barley, rolled or coarsely ground, lb	--	--	38	--	--
Wheat bran, lb	--	--	10	12	8
Soybean meal, lb	14	14	10	13	14
Molasses, liquid, lb	--	5	--	5	5
Dicalcium phosphate, lb	1	1	.5	.5	.5
Ground limestone, lb	1	1	1.5	1.5	1.5
Trace mineral salt, lb	1	1	1	1	1
Vitamin A (I.U.)	200,000	200,000	200,000	200,000	200,000
Vitamin D (I.U.)	50,000	50,000	50,000	50,000	50,000

High moisture grains can be fed to young calves, but must be supplemented with adequate protein, minerals and vitamins. Also, uneaten grain must be removed and replaced daily.

Feeding starter

Starter should be offered to the replacement heifer on day 4 of life. This is especially important when limited liquid feeding such as described here in is practiced. Many calves will nibble at starter very early and the sooner dry feed consumption occurs, the better. Calves can be encouraged to begin eating starter by placing a small amount in the pail immediately after feeding liquid. Most calves should be eating starter well by 30 days of age and consumption should increase markedly after weaning. By three months of age, most large breed calves should be consuming 4-5 lb/day. It is not necessary that they consume more than this to achieve good growth and health.

Forage Feeding

Many calf raisers prefer to include forage in the diet at an early age. While this is commendable, the presence of absence of forage in the feeding program during early life makes little difference provided a good starter is available. Young calves under 8 weeks of age will eat very little hay.

Minnesota research has shown that calves fed starter only (Table 8) after weaning did about as well as those fed starter and forage.

Table 8. Starter and hay intakes of young calves.

Age	Feed intake		
	Starter only	Starter + Alfalfa hay	
	lb/day	lb/day	
2-4 wk	.70	.54	.02
4-6 wk	2.00	1.42	.06
6-8 wk	4.4	3.7	.18
8-10 wk	5.68	5.32	.31
10-12 wk	6.79	6.70	.47

Weight gains for the 10-week period were about equal, 115 lb for those receiving starter only and 110 lb for those fed starter and hay. The data suggest that forage should be introduced by 8-12 weeks of age, but prior to that time other management considerations can determine whether or not forage is included. Good quality legume or grass hay or haylage and corn silage are satisfactory forages. If ensiled feeds are used, moldy, poor quality feed should be avoided and feed managers should be cleaned daily.

Water

Calves fed limited liquid should receive supplemental water early in life. This is important for calves fed milk once daily and is especially critical during warm weather. Purdue studies showed that calves offered milk replacer (10% of body weight) plus starter and supplemental water during the liquid feeding period (birth to 4 weeks) tended consume more starter and performed better after weaning. Water can be offered free-choice on day 4 and thereafter at feeding time.

Summary

The feeding program during early life of the calf must be carefully planned

and managed if the calf is to thrive and grow. The feeding program can be summarized as follows:

Day 1	Dam's colostrum
Day 2	Dam's colostrum
Day 3	Dam's colostrum
Day 4	Liquid feed of choice, introduce starter and water
Day 5 to weaning	Continue feeding program
Weaning to 12 weeks	Starter (up to 5 lb daily), introduce forage

If quality feeds are used and fed in satisfactory amounts under good conditions of management in the program described, replacement calves that are growthy, vigorous and healthy should result.

FEEDING DAIRY HEIFERS FROM 4 TO 24 MONTHS

J. G. Linn and D. E. Otterby
Department of Animal Science
University of Minnesota

The major goal in feeding dairy heifers is to produce an efficiently, but adequately, grown animal for calving at 24 months of age with full lactation potential. This requires heifers to be of desirable size and weight for breeding at 15 months of age. The nutritional plane of heifers both before and after breeding has a significant effect on their growth and development and whether or not this goal is achieved. High planes of nutrition excellerate heifer growth, but tend to fatten rather than develop skeletal growth. Also, heifers on high planes of nutrition are usually bred before 15 months to calve earlier than 24 months. Holstein heifers calving before 23 months of age lose 300 to 400 pounds of milk (200 pounds for Jerseys) from the total first lactation yield for each month they calve before 23 months of age. On the other hand, low planes of nutrition usually delay the calving of heifers past 24 months. No increases in milk production result from extending the heifer growing period past 2 years of age but costs of keeping nonproducing animals are increased. This paper will review some of the nutritional factors affecting the growth, reproduction and subsequent milk production of replacement dairy heifers.

GROWTH RATES

The National Research Council weight and age guidelines for growing heifers are listed in table 1. Large breed heifers are suggested to weigh 756 pounds and small breed heifers 479 pounds at breeding ages of 15 months. Most Holstein and Brown Swiss heifers will reach 750 pounds by 15 months, however, 500 to 550 pounds is a more preferable weight at breeding age for Jersey and Guernsey heifers and 600 to 650 pounds for Ayrshire and Milking Shorthorn heifers.

Table 1. NRC and Canadian weight guidelines for growing heifers.

	NRC[a]		Canadian[b]		
Age	Large breeds	Small breeds	Holstein Brown Swiss	Guernsey Ayrshire	Jersey
(months)	- - - - - - - - - - - - - - - - lb - - - - - - - - - - - - - - - -				
3	194	126	249	178	110
6	336	214	390	309	275
9	476	302	530	439	400
12	616	391	670	569	510
15	756	479	810	699	605
18	897	567	950	830	670
21	1037	655	1090	960	760
24	1177	744	1230	1090	856

[a]National Research Council - Nutrient Requirements of Dairy Cattle (1978).

[b]Ontario Ministry of Agriculture (Hoard's Dairyman, Sept. 25, 1981).

Recent information from Canada indicates height along with weight should be considered in assessing growth of heifers. The recommended height and weight of Holstein and Brown Swiss heifers at different ages is in Figure 1 and the weight recommendations for all breeds compared to NRC guidelines are in table 1. The rates of gain for large breed heifers are similar (1.6 vs. 1.54 pounds per day) for the NRC and Canadian guidelines. The major difference between the two guidelines for large breeds is Canadian heifers start off and remain about 50 pounds heavier than NRC heifers throughout the heifer rearing program. The two guidelines differ significantly on weights of small breed heifers. The Canadian recommendations are for much heavier weights at all ages and a faster growth rate (1.1 to 1.4 pounds per day) than the NRC guidelines (1.0 pounds per day).

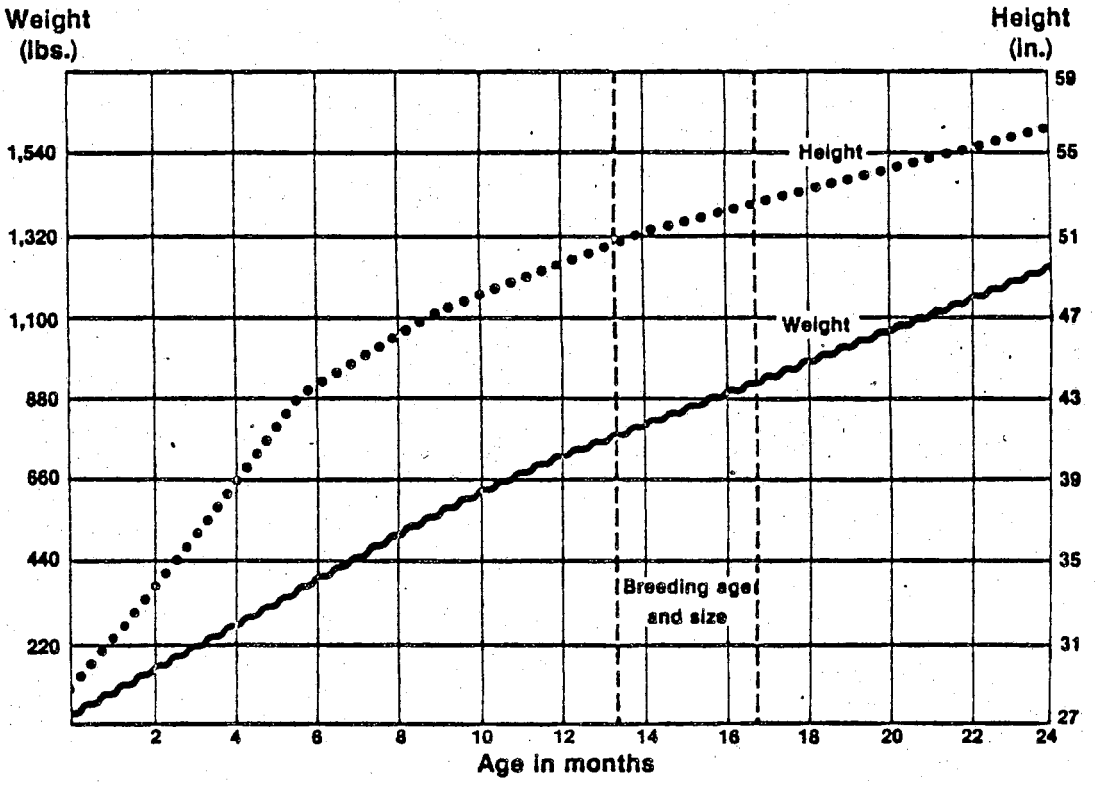

Figure 1. Canadian height and weight chart for Holstein and Brown Swiss heifers.

NUTRIENT REQUIREMENTS

The recommended nutrient content of rations for growing heifers is presented in table 2. Meeting these nutrient needs of heifers is not difficult, however, consideration must be given to dry matter intake and actual quantities of certain nutrients consumed. Growing heifers require different amounts of crude protein, energy, calcium and phosphorus at different body weights and different rates of gain. Table 3 lists the amounts of crude protein, energy, calcium and phosphorus required at different body weights and recommended gain to achieve breeding at 15 months and freshening at 24 months of age.

Table 2. Recommended nutrient content of rations for growing heifers[a].

Nutrient	Concentration in feed DM	Mineral	Concentration in feed DM
Crude protein	12%	Calcium	.40%
Net energy		Phosphorus	.26%
maintenance	.57 Mcal/lb	Magnesium	.16%
gain	.27 Mcal/lb	Potassium	.80%
TDN	60%	Salt	.25%
Crude Fiber	15%	Sulfur	.16%
Acid detergent		Iron	50 ppm
fiber	19%	Cobalt	.10 ppm
Vitamins		Copper	10 ppm
A	1,000 IU/lb	Maganese	40 ppm
D	140 IU/lb	Zinc	40 ppm
		Iodine	.25 ppm
		Selenium	.10 ppm

[a]National Research Council - Nutrient Requirements of Dairy Cattle.

Table 3. Dairy nutrient requirements of growing heifers[a].

Body weight (lb)	Daily gain (lb)	Crude protein (lb)	TDN (lb)	Calcium (grams)	Phosphorus (grams)
Large Breeds					
200	1.6	.85	4.3	17.2	9.1
300	1.6	1.04	5.7	18.6	11.3
400	1.6	1.30	7.1	20.4	13.6
500	1.6	1.47	8.4	22.7	16.3
600	1.6	1.61	9.6	23.6	17.2
700	1.6	1.74	10.5	24.5	18.1
800	1.6	1.85	11.5	25.8	19.0
900	1.6	1.92	12.3	26.3	20.4
1000	1.4	1.97	12.6	27.2	20.9
1100	1.2	1.99	12.8	27.2	20.9
Small Breeds					
100	.8	.36	2.2	9.1	5.9
200	1.2	.75	3.9	15.4	8.2
300	1.2	.96	5.2	16.8	10.4
400	1.2	1.22	6.6	19.5	13.1
500	1.2	1.41	7.8	21.8	15.4
600	1.2	1.56	8.9	22.7	16.3
700	1.0	1.67	9.5	23.1	16.8
800	.6	1.58	9.1	21.8	16.3
900	.4	1.51	8.9	20.4	15.9

[a]National Research Council - Nutrient Requirements of Dairy Cattle.

FOUR MONTHS TO BREEDING

Nutrition

The goals during this period are to have heifers: 1) gain an average of 1.6 pounds per day for large breed heifers and 1.3 pounds per day for small breed heifers; 2) weigh 40 percent of their mature weight at 12 months of age and 3) bred at 15 months of age. Nutrition is a major determinant in attaining these goals.

The nutritional factors which affect the growth and/or reproduction in heifers are: 1) the amount and quality of feed or energy supplied; 2) protein, 3) mineral and mineral balances and 4) vitamins A, D and E. Any serious nutrient deficiency can adversely affect growth and/or reporduction. Thus, the nutrient requirements in tables 2 and 3 must always be met with diets that are balanced and adequate in essential nutrients.

Heifers from 4 months to breeding age generally cannot consume enough nutrients from forages alone to support good growth rates. As a general guideline, 3 to 4 pounds of grain dry matter per day along with ad-libitum forage should be fed during this period. A 3 to 4 month old heifer will consume about 5 to 6 pounds of dry matter per day, of which 75 percent should be grain. As heifers grow, their total dry matter intakes will increase. Forage intakes should be allowed to increase while grain amounts are held relatively constant and thus, grain will become a smaller proportion of the total dry matter consumed as heifers approach breeding age. Table 4 provides a guideline for dry matter intakes and the proportion of grain needed in rations fed to heifers of different ages. However, the actual amount of grain fed and the supplemental nutrient content of the grain mix needed to balance the ration should be based on the quality of forage being fed.

Table 4. Dry matter intake and ration composition guidelines for promoting good growth rates in large breed dairy heifers.

Age	Dry Matter intake	Ration composition (DM basis)		
		Forage:Grain	TDN	Crude protein
(Months)	(lb)		(%)	(%)
3	5- 6	25:75	78	16
6	8-10	50:50 (65:35)[a]	71	13
9	11-13	60:40 (80:20)[a]	68	12
12	15-17	75:25 (100:0)[a]	60	12
15	17-19	85:15 (100:0)[a]	60	12
18	20-22	100:0	60	12
21	21-23	100:0	60	12
24	21-23	90:10	60	12

[a]Forage to grain ratio when over 50% of the forage DM is from corn silage.

Mammary gland development

The key period in mammary gland development is between 3 and 9 months of age. During this period, mammary tissue is growing 3.5 times faster than body tissue. Heifers fed high concentrate rations from calfhood to breeding age develop less mammary secretory tissue than heifers raised at normal, recommended growth rates. The exact effect overconditioning has on mammary gland development is unknown but not solely caused by fat infiltration into the secretory tissue. Fattening of heifers prior to puberty appears to have an inhibitory effect on mammary secretory tissue development and/or change the endocrine stimulation of ductular growth. Overconditioning of heifers after 15 months of age does not affect mammary secretory tissue.

First estrus

First estrus in heifers is dependent upon a combination of size and weight but primary weight. A general guideline is heifers will show their first estrus at 40 percent of their mature weight which should be about 12 months of age. Heifers fed high planes of nutrition will show estrus at an earlier age than heifers grown at recommended rates, but underfeeding of heifers will delay estrus. Underfed or very slow growing heifers may ovulate but estrus signs often are suppressed. Heifers in good condition and gaining weight at breeding time generally show more definite signs of estrus and have improved conception rates over heifers in poor condition and/or losing weight. Over-conditioned or fat heifers have been reported to require more services per conception than heifers of normal size and weight.

BREEDING TO 2 MONTHS BEFORE CALVING

Heifers should be growing well at the time of conception. After conception and during gestation, the goal is to feed a balanced ration for a steady but slow growth rate. A return to a more rapid growth rate during the last third of gestation will be desired for reasons discussed later. If there is a period when less than recommended growth rates are tollerable and will have the least effect on overall heifer performance, it is during the middle trimester of gestation. However, heifers should not be so severly restricted in nutrient intakes that weight loss occurs.

Good to high quality forage is probably all the feed bred heifers will require. However, adequate amounts of all essential nutrients (table 2) are needed and rations should be balanced to see which nutrients are deficient and need supplementation. Corn stalks, very poor hay or mature, dry pastures will not provide adequate nutrients for growth. All or nearly all corn silage rations will need protein supplementation and probably should be limit fed to avoid fattening heifers.

FORAGE PROGRAMS

Heifers fed rations sufficient in nutrients to support good growth rates and conception are not affected by source of forage in the ration. Heifers fed to 18 months of age on all corn silage, corn silage and hay silage or corn silage and hay had similar dry matter intakes, growth rates, ages at first breeding and conception rates. The feeding of an all silage (40% corn silage-60% grass silage) or hay-silage (40% hay-20% grass silage-40% corn silage) ration to

heifers from 57 to 574 days did not affect age of breeding, services per conception or health of heifers. Heifers fed the all silage ration gained slightly faster (1.71 vs. 1.65 pounds per day) and were slightly heavier at 574 days (1016 vs. 983 pounds) than heifers fed the hay-silage ration.

Pasture can be used for heifers over 400 pounds and can be the only feed for bred heifers. Some grain should be fed to prebreeding age heifers on pasture and as pastures mature and dry out, grain or additional forages will need to be fed to older heifers on pasture. The quantity and quality of pastures must be monitored closely and additional feeds offered when needed, if pastured heifers are to gain at recommended rates.

TWO MONTHS BEFORE CALVING TO CALVING

How heifers are fed during this period can affect milk production during first lactation. Heifers should move from a slow steady growth rate during the first and second trimester of gestation into a rapid growing phase the last half of the third trimester. Rapidly growing heifers at calving time, but needing additional growth during the first lactation, were found to be more persistent milkers than full sized heifers at calving. Also, heifers slightly undersized at calving (80% of normal) will reach full milk production potential and normal size if fed sufficient nutrients for both growth and milk production during the lactation.

The exact amount of grain to feed before calving will depend on forage quality, size and condition of the heifer. A thumb rule would be to feed grain at 1 percent of body weight starting about 6 weeks before calving. Be sure rations are balanced in protein, minerals and vitamins. Excess salt intakes can contribute to udder edema and should be avoided the last 2 weeks before calving.

Well grown heifers will have a minimum of problems at calving but ease of calving can be affected by plane of nutrition in two ways: 1) an effect on calf size and 2) an effect on fatness of the dam. At equal body weights, fat, overconditioned heifers are almost always younger and consequently will have less skeletal growth than leaner normally grown heifers. Thus, fat heifers have higher incidences of dystocia because of small pelvic openings and usually a larger than normal sized calf at birth. Underfed or poorly grown heifers also will require more assistance at calving and have a higher death rate at calving than normal sized heifers.

SUMMARY

Heifers should be grown at a slow but steady growth rate. Extremes in overfeeding and underfeeding will adversely affect the health, calving and milk production potential of heifers. Daily gains averaging 1.6 pounds per day for large breeds and 1.2 pounds per day for small breeds from birth to 24 months of age are recommended. Two periods when faster growth rates may be desirable are at time of breeding and starting 6 weeks before calving. Heifers should weigh about 40 percent of their mature weight at puberty and about 80 percent of mature weight at calving. Heifers needing some additional growth during the first lactation will reach full milk production potential, be more persistent milkers and will grow when fed sufficient quantities of nutrients for both growth and milk production.

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