PRECONDITIONING SEMINAR

Sponsored by

OKLAHOMA STATE UNIVERSITY

and

ELANCO PRODUCTS COMPANY

INTRODUCTION

In September 1967, about 200 animal and veterinary scientists met at Oklahoma State University to discuss the problems and scientific basis for developing a "preconditioning program". This group of scientists and industry leaders assembled on their own initiative to share their research findings and ideas because of the widely felt need to reduce the huge dollar losses which occur today in newly arrived feedlot cattle.

Part of the planning for the program was done by the staff at Oklahoma State University. Many others played key roles in the development of the seminar. Among these are Dr. George Crenshaw, University of California at Davis; Dr. John Herrick, Iowa State University; Dr. H. E. Geyer with the Federal Extension Service in Washington; Dr. Don Williams, a veterinarian at Ada, Oklahoma; and Dr. Bob Schock from American Cynamid. Oklahoma State University staff active in the planning were, Dr. L. S. Pope and Dr. W. F. Taggart, in Animal Science; Dr. Wiley Wolf, Dr. Everett Besch and Dr. E. W. Jones from the College of Veterinary Medicine; and Dr. Mike Howell from the Entomology Department.

Oklahoma State University and Elanco Products Company, A Division of Eli Lilly and Company, have assembled into this proceedings the transcripts of the formal presentations made at the seminar. It is hoped that the ideas in this proceedings will serve as a guide in the formulation of even more research and educational programs aimed at improving the health and animal husbandry practices which are so essential to efficient beef production.

Donald R. Gill
Extension Animal Nutritionist
Oklahoma State University
Program Chairman

INDEX

	Page
Preconditioning Feeder Cattle *Dr. John Herrick*	4
Cost of Conditioning Calves After Shipment *Earl Harriss*	9
Why do Feeder Calves Fail to Perform When They Reach the Feedlot? (part I) *Dr. George L. Crenshaw*	10
Why do Feeder Calves Fail to Perform When They Reach the Feedlot? (part II) *Dr. R. A. Ivie*	12
A Review of Viral Diseases of Feedlot Cattle *Dr. D. G. McKercher*	13
Bacterial Diseases *Dr. J. R. Collier*	16
Relationship of BVD to Feedlot Performance *Dr. William M. Lynch*	21
Relationship of IBR to Feedlot Performance *Dr. Richard C. Searl*	22
PI-3, How it Relates to Feedlot Performance *Dr. J. H. Carroll*	24
The Effects of Internal and External Parasites on Feedlot Performance *Dr. D. E. Howell*	27
Experimental Use of a New Worming Agent in Arkansas Cattle *Dr. Dan E. Goodwin*	29
The Effects of Internal Parasites on Feedlot Performance *Dr. Everett Besch*	31
Fundamental Concepts Relative to Internal Parasites and the Conditioning of Calves for the Feedlot *Dr. Norman F. Baker*	34
A Brief Resume of Bovine Anaplasmosis and Its Relation to Feedlot Cattle *Dr. E. W. Jones*	37
Effect of Starvation and Refeeding Upon Rumen Function *Dr. R. L. Baldwin*	40
Feeder Cattle on High Grain Rations *Dr. R. W. Dougherty*	43
Nutrition of Ruminants as Affected by Stress *Dr. D. C. Church*	46
The Energy and Protein Requirements of Starting Calves *Dr. W. N. Garrett*	52
Effect of Vitamin and Mineral Status Upon Feedlot Performance of Weanling Calves *Dr. Allen D. Tillman*	54
The Role of Cations in Neuromuscular Functions *Dr. Eric I. Williams*	57
Management of Calves and Adapting the Calf to Its Future Environment *Dr. Donald R. Gill*	59
Preconditioning of Feeder Cattle Prior to Interstate Shipment *Dr. Richard F. Bristol*	62
Immunization Programs—(Pre-weaning) *Dr. James J. Sheldon*	66
Immunizations Programs—(Post-weaning) *Dr. Robert Dickson*	68
Feeding Calves Prior to Shipment *Jack Algeo*	69
Identification of Cattle *Dr. Don Williams*	73
Factors Affecting Shrink in Feeder Cattle *Dr. H. L. Self*	75
Summary of Problems and Research Opportunities for Those in Animal Science Research *Dr. O. Burr Ross*	82
Summary of Problems and Research Opportunities for the Veterinary Research Institutions *Dr. Nelson B. King*	85
The Opportunities for Field Research and Educational Programs in Extension *Dr. Dixon Hubbard*	88
Problems and Research Opportunities for the Pharmaceutical and Biological Industry *Dr. N. H. Casselberry*	93

PRECONDITIONING FEEDER CATTLE

Dr. John B. Herrick
Extension Veterinarian
Iowa State University

Cattle feeders throughout the nation are aware of the problems of moving cattle from the point of origin or where they are raised to the feedlot where they are then forwarded to market. The variation in sizes, ages, management, and disease exposure, plus the variation in systems of marketing add to the complexity of recommending specific procedures for handling all feeder cattle. The loss from shrink, feed utilization, and efficiency plus a 1 to 2 percent death loss due to prevailing management practices and disease manifestations has been estimated to amount to approximately $10 to $20 per animal. Thus, it can be readily seen that the movement of cattle per year into feedlots throughout the United States produces a gigantic loss to cattlemen.

The reputation of the feeder calf producer is made or broken in the feedlot. Many large feedlots have developed elaborate record keeping systems, and in many cases they are composed of electronic data processing systems, to help analyze performance of each lot of cattle. As these feedlots accumulate data, they become better able to avoid buying poor-doing cattle. The data accumulated at these feedlots are now pinpointing some previously unrecognized problems.

No one specific program will forestall all these losses yet a program aimed at removing as many of the voids in the proper handling of cattle is in order. The following program is aimed at the producers of feeder cattle where many of the preventive aspects of these losses can be implemented. Perhaps the greatest motivating force for the implementation of these programs can come from the buyer himself. "Buyers' resistance" can in actuality bring these practices into being faster than any other motivation.

In many of the feedlots new cattle seldom recover their purchase weight in three weeks. This is in spite of the best nutrition that can be provided. Receiving pens in many feed yards can best be compared to the emergency room of a large hospital. In other words, the best feedlot health program is often implemented too late. The progressive feedlot has reached a road block towards improving its health programs. To bypass this road block a few feedlots have bought their own cow-calf units to get control of the health program early enough to reduce the feedlot losses due to morbidity and death.

It is realized that a large gap exists between the problem and its ultimate solution; however, there is enough known that if only a few practices were put into use, feedlot morbidity and death losses could be reduced considerably.

A program aimed at the prevention of these problems is entitled "Preconditioning." Recommended procedures are outlined.

1. Pre-weaning prior to shipment: The fear and anxiety resulting from the calf being taken from the cow, the physical irritation of bawling, the change of water and feed, and the fatigue by "fence running" adds stress that predisposes the calf to shrink and disease. Facilities should be so provided by the producer of the calf that it can be taken from its mother at least three weeks prior to shipment. It is realized that many producers do not have such facilities. In view of the overall value of such, they are encouraged to immediately make plans for them. Weaning puts a good deal of stress on the calf. He may even become sick following weaning, but his chances of coming through this stress period is better in the environment in which he was raised than if exposed to disease organisms in markets and other areas.
2. Preconditioning prior to shipment—calves and yearlings:
   a. Feeders to be shipped should be confined and put under observation for at least three weeks. If at all possible, they should be started on hay and grain and be accustomed to feed bunds and watering facilities. (See attached leaflet PM 401.) The type and level of rations that have been fed to the cattle prior to shipping are very important from the standpoint of shrink and getting the cattle started on feed.

      Animals that have been on a liberal amount of feed then undergo a 48 hour or more starvation period in transit or exhibit over a 5 to 6 percent shrink from payweight are apt to undergo digestive disturbances and become "poor doers" if not started out correctly in the feedlot. This is based on the lactic acid build up in the paunch if a high carbohydrate diet is fed immediately followed by acidosis and dehydration.

   b. Following the stress period of weaning, parasite determination and treatment should be implemented. Not all cattle need worming—just those with worms!

Grub control is a must and is a responsibility of the feeder calf producer. Due to the difference in the heel fly season from one section of the country to the other and the complexity of treating for grubs off season in the feedlot, feeder cattle should be treated for grubs at the point of origin at the correct time of the season and certified as such. This gives the buyer of feeder calves grub control and also louse control. Buyer resistance at the feeder end could really implement this action.

c. Fecal examinations should be conducted. If treatment is necessary for the control of internal parasites, such treatment should be instituted at the point of production of the calves or yearlings.

d. Blackleg-Malignant Edema Immunization: For maximum protection, Blackleg-Malignant Edema immunization should be administered after the calf is three months old. If vaccinated prior to the three months-of-age bracket, revaccination should be administered at weaning time.

e. The viral diseases, according to present-day knowledge, that are the greatest concern to the cattle producers are Shipping Fever Complex (Para-Influenza 3), Infectious Bovine Rhinotracheitis, and Bovine Viral Diarrhea. In many stock cow herds, IBR is a problem within the cow herd and vaccination of the cows prior to breeding is recommended. Calves or yearlings should be vaccinated for these diseases at least two weeks prior to shipment or exposure. Veterinary counsel is suggested for the timing and procedure of vaccination of animals for these viral diseases because of their complexity.

f. Veterinary certification of immunization and parasite control should accompany the cattle and give the purchaser assurance that such procedures have been followed. Vaccines and serial numbers of each should be designated on such certification. (See attached Preconditioning Certificate.)

g. Cattle identification: National cattle identification is sorely needed within the cattle industry. Until such program is enacted, the producer of feeder cattle should make every effort to identify his cattle individually and have such identifications designated on the preconditioning certificates.

Practices such as vaccinating sick cattle, use of bacterins in lieu of vaccines and faulty administration in the handling of vaccines will alter the effects of such programs. The success in the use of a vaccine depends not only on the efficacy of the product, but also the state of health of the animal at the time of the vaccination.

All efforts should then be made to see that the cattle are loaded and handled with great care and with a minimum of excitement. Cattle moving directly from site of origin to feedlot suffer the least stress. Long shipments should abide by rest, feeding and watering regulations. Regulations governing rest and feed and water for animals in transit should be directed toward those shipped by truck as well as those by rail. There apparently is a direct correlation between the time cattle are in transit and the disease resistance they possess.

Will Preconditioning Pay?

Naturally, all the answers are not completely worked out. No single procedure will fit all ranches or shipping situations. Considerable research needs to be conducted; research which can only be started on the ranches where calves are produced and completed in the feedlot where they are finished. Extensive studies are now under way to evaluate various procedures.

The following costs are realistic and will be verified by accumulated data within the next calendar year. It would appear that cattle feeders are now recognizing the value of preconditioning. More field research is needed to conclusively point out the value and the limitations of such a program. Basically, it is sound because it is preventive medicine.

ESTIMATED PRECONDITIONING COSTS

Feed costs		15-20c a day
Yardage		5c a day x 30 days
Interest on animal		3c a day
	TOTAL	$7 - $8.50
Grub treatment		
Vaccinations		
Worming		$3 - $6
Surgery		
Electrolyte - optional		
	TOTAL	$10 - $14.50

Gain During Preconditioning Period (30 days)

20 to 60 pounds
Average 40 pounds
Cost 20 to 30 cents
Average 25 cents

25c x 40 lbs. = $10.00

In many cases a profit is shown for preconditioning period. A product to gain feedlot reputation.

Handling and Feeding New Feeder Cattle

Prepared by Nelson Gay and William G. Zmolek, extension livestock specialists. Iowa State University of Science and Technology Cooperative Extension Service, Ames, Iowa, October, 1967, Pm-401.

The more than 2 million feeder cattle shipped into Iowa every year come in a variety of age, weight, size and color. They're shipped from 20 to 2,000 miles. Some come directly from the producer; others may go through a half dozen "hands." But *all* feeder cattle are subject to setbacks from two causes:

1. *Stress* caused by handling and shipment. Stress lowers disease resistance and feed utilization. Any management practice that reduces the intensity of stress will benefit cattle.
2. *Shrink* is inevitably linked with handling and shipment of cattle. Feeders shipped to Iowa State University outlying experimental farms have lost 4 to 13 percent from pay weight, and only about one-half of this shrink was of gut fill. The rest was tissue loss of moisture, minerals, energy and proteins. To regain shrink and maintain the health of the animal, these moisture and nutrient losses should be restored as soon as possible after arrival in the home yards. (Limited research indicates that feeder shrink is *directly* related to the amount of handling and transportation time from ranch to feedlot.)

Management for Newly Arrived Feeders

Many feeder cattle, especially calves, are not used to eating from a bunk or drinking from an automatic waterer. Corn silage and grain may be strange and unfamiliar to them. It's impossible to recommend one best management system for all feeder cattle. However, there are two essentials in handling new feeder cattle:

1. Rest. *Do not handle* cattle on arrival. Allow them access to water and hay for 12 hours. Water should be in tanks and allowed to trickle. Hay should be medium or higher quality.
2. *Feed some concentrates.* Offer about ½ percent of body weight daily. Mixing the concentrate with hay or silage will prevent overeating of concentrates by individual animals. It's difficult to founder cattle on corn silage or hay. In fact, small feeders under 400 pounds have difficulty getting enough energy from such sources to make satisfactory gains.

The ration should contain supplemental vitamins, minerals (including trace), and protein. Supplements containing urea should not be used initially. They are not as palatable as vegetable protein supplements, and cattle need time to become accustomed to urea. Many commercial feed companies produce "stress feeds" with high levels of vitamins and minerals. These may be useful in starting newly-arrived feeder cattle.

Nutrient Content of Starter Rations

Energy: The rations should contain readily available energy. Sources may be grain, supplements, complete commercial feed. Expect a daily intake of air dry feed equivalent to 2 to 3 percent of body weight. A 450-pound calf might consume 2.5 to 3 pounds of grain and supplement and 12 to 15 pounds of silage or 8 pounds of hay daily.

Vitamins: Vitamin A is the most important. A level of 40,000 to 50,000 IU per head per day is desirable for 5 to 7 days. This should be supplied as synthetic vitamin A. Many feeders offer supplementary vitamins D and E which has little research basis but may provide some insurance. If the cattle originated in a drouth area or are in extremely thin condition, injectable A, D, and E might be helpful.

Minerals: A supply of all essential minerals is desirable. However, it is not wise to force feed minerals indiscriminately or to offer free-choice a complex mineral mixture to which molasses and flavoring agents have been added.

Rations for Starting New Feeders

Several starting rations for new feeder cattle are shown in table 1. These rations can be fed for 1 week after which the grain portion can be increased.

TABLE 1. RATIONS FOR STARTING NEW FEEDERS.

Fleshy, heavy creep-fed calves, 450 to 550 pounds			
	1	2	3
Corn silage	15-20	—	—
Cracked shelled corn	4-6	4-6	—
Ground ear corn	—	—	4-6
Haylage	—	8-12	—
Mixed hay, chopped	—	—	4-6
Supplement 2	2.0	1.0	1.0
Oats	—	—	—
Light non-creep-fed calves, 300 to 400 pounds			
	1	2	3
Corn silage	12-15	—	—
Cracked shelled corn	2	1	—
Ground ear corn	—	—	2-3
Oats	—	1	—
Haylage	—	6-8	—
Mixed hay, chopped	—	—	3-4
Supplement	2.0	1.0	1.0

Bunk space should be adequate, and if small calves are involved, be certain they can reach the bunks. To insure freshness and acceptability, feed more than once a day. Stale or rained-on silage is not eaten as readily as good smelling, fresh silage. Clean, fresh water is also important.

Ration A listed in table 2 has been successfully self-fed to yearling steers immediately on arrival. As much as 24 to 26 pounds per head per day have been consumed with no ill effects. Each ration in the series listed in table 2 is designed to be self-fed 5 to 7 days before working up to ration E or F.

Conditioner Supplements

If desired, "conditioner" supplements may be used for the first 2 to 3 weeks. Conditioner supplements differ from regular commercial supplements in that they are more palatable, contain higher levels of vitamin A (40,000 to 50,000 IU per pound), and a high level of antibiotics (300-350 mg, per pound) to help combat low-level diseases. Since antibiotics have not been proven to be a sure cure for all low-level diseases in new feeder cattle, the use of high-level antibiotics should be the decision of each individual cattle feeder. These supplements also contain higher than normal amounts of potassium, magnesium, and chlorides. These elements are lost along with moisture during shipping and are essential for restoring normal water balance.

Tables 3 and 4 present two examples of conditioner supplements. Commercial supplements of similar analysis would serve just as well.

TABLE 2. RATION SERIES FOR SELF-FEEDING.

Ingredient	A	B	C	D	E	F
			Pounds			
Corn grain	250	520	800	1020	1240	1400
Cobs	1200	1000	830	640	460	325
Molasses	150	100	80	80	80	80
Dehydrated alfalfa	100	100	50	50	50	50
Supplement 1	300	280	240	210	150	125
Dicalcium phosphate	—	—	—	—	10	10
Salt	10	10	10	10	10	10
Calculated Analysis						
Crude protein	10.5	10.7	10.4	10.6	10	10
TDN %	53	58	62	65	68	71
Ca %	.48	.44	.36	.32	.38	.35
P %	.28	.30	.31	.32	.40	.41
Vitamin A, I.U./lb.	4920	3290	3640	3240	2690	2440
NE m. megcal/lb.	.60	.66	.72	.76	.80	.82
NE p. megcal/lb.	.38	.38	.42	.44	.46	.47

Upon arrival cattle may be immediately started on ration A. Cattle should be fed each ration 5 to 7 days depending on how they appear to be going on feed. Fresh feed should be in front of the animals at all times. If molasses is not priced competitively with corn on an energy basis, it could be omitted from rations C, D, E and F. Ration F should make for slightly faster gains and shorter time to reach the choice grade, but will be slightly more expensive than ration E, which is adequate for finishing. Yearling cattle in medium flesh should reach the choice grade in 140 to 160 days on these rations.

TABLE 3. CONDITIONER SUPPLEMENT FORMULATION NO. 1 FOR SELF-FED RATIONS.

Ingredient	lbs.	Protein lbs.	Ca. lbs.	Phos. lbs.	Vitamin A	K	Mg	Nacl
SBOM	1260	567	4.0	8.4	—	23.8	3.4	—
Dehy	200	32	3.0	.5	20M	3.2	.6	—
Molasses	100	—	.8	—	—	2.4	.3	—
Dicalcium	100	—	26.0	20.0	—	—	—	—
$CaCO_3$	50	—	19	—	—	—	—	—
KCl	60	—	—	—	—	31.0	—	—
$MgCO_3$ (MgO)	40(20)	—	—	—	—	—	11.0	—
NaCl	20	—	—	—	—	—	—	20
Vitamins	10	—	—	—	40M	—	—	—
Trace minerals	10	—	—	—	—	—	—	—
Urea	150	422	—	—	—	—	—	—
*Antibiotics+								
Total	2,000	1021	52.8	28.9	60M	60.4	15.3	20
Percent	—	51	2.6	1.4	30,000IU	3.0	.76	1.0

Feeding Directions:
*If desired, antibiotics (tetracyclines) can be included at a rate such that each animal will receive 350 mg/hd/day. Some evidence indicates a combination of 350 mg tetracycline plus 350 mg sulfamethazine may be useful.

TABLE 4. CONDITIONER SUPPLEMENT FORMULATION NO. 2.

	lbs.	Protein lbs.	Ca. lbs	Phos. lbs.	Vitamin A	K	Mg	Nacl
SBOM	1410	635	4.5	9.4	—	27.8	3.8	—
Dehy	200	32	3.0	.5	20M	3.2	.6	—
Molasses	100	—	.8	—	—	2.4	.3	—
Dicalcium	100	—	26.0	20.0	—	—	—	—
$CaCO_3$	50	—	19.0	—	—	—	—	—
KCl	60	—	—	—	—	31.0	—	—
$MgCO_3$ (MgO)	40(20)	—	—	—	—	—	11.0	—
NaCl	20	—	—	—	—	—	—	20
Vitamins	10	—	—	—	80M	—	—	—
Trace minerals	10	—	—	—	—	—	—	—
*Antibiotics+								
Total	2,000	667	53.3	29.9	100M	64.4	15.7	20
Percent	—	33	2.6	1.5	50,000	3.2	.78	1.0

Feeding Directions:
*See footnote on Supplement 1.

THE IOWA BEEF AREA

Preconditioning Certificate

Certificate No._____ Date_____

The animals identified by_____ brand,_____ breed,_____

age,_____ number of steers,_____ number of heifers_____

total number have been preconditioned as follows:

	DATE	BRAND	SERIAL NUMBER
CASTRATED			
DEHORNED			
BLACK LEG			
MALIGNANT EDEMA			
SF4 (P13) VACCINE			
BVD VACCINE			
IBR VACCINE			
LEPTO VACCINE			
GRUB TREATED			
WORM TREATMENT			
OTHER			

Ration during preconditioning period:_____

SELLER _____

ADDRESS _____

BUYER _____

ADDRESS _____

CERTIFIED BY _____

COST OF CONDITIONING CALVES AFTER SHIPMENT

Earl Harriss
Harriss Feed Yard
Brawley, California

In trying to arrive at a true cost of recovering purchase weight I have compiled the results on several lots of calves we fed last year that are typical of our operation.

These calves were purchased during September and October Sales in East Texas, assembled and shipped about 1,200 miles to my feed yard in Imperial Valley, California. They were No. 1 and No. 2 cross-breds, a total of 3,069 head averaged 377 pounds purchase weight. They were held in the feed yard an average of 19 days then put on alfalfa pastures. While in the feed yard they were branded, vaccinated, horns tipped, bull calves castrated, and sick calves doctored. During this period the feed cost was 17.83¢ per head per day. The cost of processing, including medicine and vaccine was 11.27¢ per head per day or a total cost of 29.19¢ per head per day. For the 19 day period this amounted to $5.54 per head. The death loss was 1.15% or an additional $1.21 per head, making a total cost of $6.75 or 1.79¢ per pound on purchase weight. When the calves were weighed out of the feed yard to pasture they averaged 371 pounds or 6 pounds less than purchase weight.

In the case of another lot of 1,849 head of calves kept in the feed lot on a growing ration, the actual cost of medicine and processing was $5,095.05 or an average of $2.76 per head. Since these calves were grown in the feed lot a check weight was not taken to determine feed cost to recover purchase weight. In this particular lot the death loss was 1.46% through the growing period of 97 days.

All of the above figures include the actual cost of medicine used, plus 50¢ per head for branding, vaccinating, dehorning, etc., and an additional 50¢ per head for castrating bull calves. These are normal charges in our area.

You would also be interested in knowing that the death loss percentages do not include those calves that were determined to be chronic pneumonia cases or had not made a satisfactory recovery and were sold before putting the calves on feed. In the lots of calves included in this report these sales amounted to 3/10ths of 1 percent.

Using these figures then it would appear that a realistic cost of recovering purchase weight and getting calves started on feed would be somewhere between one and three-fourths and two cents per pound.

WHY DO FEEDER CALVES FAIL TO PERFORM WHEN THEY REACH THE FEEDLOT?

George L. Crenshaw, D.V.M.
Extension Animal Health Specialist
University of California, Davis

When we observe what has occurred, it is readily recognizable that in the past decade or two the cattle industry, particularly the feeding industry, has changed considerably. Historically, there were large numbers of farmers in the middlewest and lesser numbers in other sections of the country who marketed their grain by feeding cattle. At the same time, ranchers were selling grass-fat animals, so there were several types of cattle operations and classes of cattle marketed.

Today most of our cattle are fed in feedlots, and these feedlots are ever-increasing in size and capacity. The cattle fed are younger since it is no longer so economical for either the producer or feeder to handle old, growthy steers. Also, we have a sophisticated consumer who demands a well-finished carcass which is not too fat.

When the small feeder was the only one involved in finishing cattle, some of the same problems that are prevalent today were occurring at that time. It is questionable that as many problems occurred, however, because older calves were fed which had, in many instances, been maintained since weaning on the home ranch or grown in an environment, particularly pasture, which resembled their original one.

Because of these practices, calves were given a better chance to mature sufficiently in a more favorable environment; and, if a disease outbreak did occur, many were not as severely affected. Also, not so many groups of cattle from a variety of sources were mixed together. Furthermore, because the size of feedlots was smaller, cattle from different sources were not as apt to be fed in adjacent pens.

Currently many of these factors have changed. As mentioned previously, we are sending cattle to the feedlots at an earlier age and in order to do this, we are weaning both heavier and lighter calves at younger ages. In this era of rapid and readily available transportation cattle may be shipped shortly after weaning, frequently to an auction, in search of the highest dollar. Since there are a great number of salesyards, we may find our calves passing through several of them before reaching the ultimate owner. The trend is away from the commission yard with fat cattle which used to be the rule; but, feeder cattle have found their way in increasing numbers into salesyards; consequently, the following factors may be considered as responsible for some of the conditions that currently exist:

1. Weaning of calves at an earlier age which have either (a) insufficient immunity to many diseases, or (b) inability to develop immunity due either to youth or to the presence of maternal antibodies.
2. More severe weaning stress due to weaning at an age when calves are still dependent upon their dam.
3. Shipment of calves with a shorter stay under the home ranch environment.
4. Increased exposure to disease
   a. In trucks
   b. At salesyards
   c. At rest stops
   d. In feedlots

When we consider these factors, plus associated stressing factors, it is amazing that we do not have more problems. Some of the more important stressing factors are:

1. Weaning
2. Shipping
3. Handling and sorting
4. Surgical
5. Nutritional
6. Vaccination
7. Environmental
   a. Confinement
   b. Climatic changes

Our approach towards solving many of the problems we are faced with in feedlot cattle has been at best poor. Rather than attempting to alter the existing system, we have been content to institute stopgap measures by depending on our armamentarium of chemotherapeutants and antibiotics.

Cattle have been bred primarily for improved efficiency and rate of gain. Disease resistance breeding programs so far have been confined to range cattle. Unlike poultry,

we have not had sufficient research programs to develop strains of cattle with predictable responses when they are placed under a specific set of circumstances.

Parasite problems have been of increasing importance basically because of increased concentrations of cattle both on the range and in the feedlot and to a dwindling supply of competent labor. On the range, for example, helminths and lice occur; in feedlots coccidiosis due to poor sanitation and anaplasmosis because of improper surgical and vaccination disinfection techniques may be problems.

Labor and handling problems on the range have resulted in fewer calves being dehorned and castrated. Surgical shock is greater for older animals, and if this current range practice is to increase—and it probably will since non-castrates gain faster and more efficiently—we must develop different methods of handling this situation. Comparatively, little or no research has been conducted on the use of endocrine products for nonsurgical castration or to overcome some of the effects of testicular hormones in bull calves.

Labor insufficiency has also meant that poor or no immunization programs are conducted on ranches. Consequently, the feedlot operator is required to conduct these programs which often consist of too little too late. For example, frequently sub-conlical parainfluenza and BVD infections may be occurring in calves prior to weaning. Thus, when weaning plus other stressing factors are imposed on calves, we find these diseases manifesting themselves as part of the shipping fever syndrome. IBR is not usually a problem of range calves, but vaccination for this disease may stress calves and contribute to other disease syndromes.

While we are on the subject of immunization, the response of sick animals to vaccination should be considered. Although we are aware that immunization is best effected on healthy animals, in many instances sick animals are vaccinated, which subsequently manifest clinical signs of the disease for which they have presumably been vaccinated.

Although we encounter a number of producers who demand top dollar for a product which they have often handled apathetically, they are not entirely responsible for performance failures. After cattle arrive at feedlots, many factors may be involved which influence performance.

In feedlots we observe a desire to do everything to a calf at one time, including vaccinating against a multiplicity of diseases, grub treatment, dehorning, castrating, worming, branding, and treatment for sickness.

Frequently, sick calves may be undertreated, occasionally overtreated, and often in either case improperly treated because of an inaccurate diagnosis of the disease and improper evaluation of the problem. Many of us have lost sight of the fact that groups of cattle consist of individuals each of which has idiosyncrasies. Instead, we rely on group treatment, often through the feed and/or water; do not treat symptomatically; and fail to recognize factors essential to recovery from illness. For example, the sicker animals may not consume an adequate amount of medication in feed or water. These animals should be identified, treated, placed in a hospital pen, and appropriate records kept of their disposition.

Lack of foresight has often been demonstrated in the design of receiving, hospital, and convalescent pens, and often accessibility to these pens is unsatisfactory. In some instances the same crews may be used for both shipping and receiving cattle. With the major emphasis being on marketing, sick animals may be treated erratically. These factors only serve to emphasize that many feedlots were designed only for feeding cattle and have expanded without sufficient planning.

There is also a lack of appreciation of the influence that concentration of animals has on the incidence of infectious disease. It is known that as organisms pass through animals their pathogenicity may increase, yet we place new cattle in recently-vacated pens or adjacent to other cattle which may be sick.

In this era of scientific achievement we are deficient in knowledge of the nutritional and medical requirements for newly received and sick animals. For example, we are still measuring nutritional results by weight gain and feed efficiency evaluation, both of which are slow and non-specific methods, while medically we are still unaware of the electrolyte requirements of most dehydrated calves. We have used little imagination in our approaches to these problems.

There is anxiety by some feeders to try new but unproved methods or products on too large a scale; i.e., use of *Hemophilus sp.* bacterin in control of meningo-encephalitis when there is no scientific evidence to substantiate its efficacy. Along with this we have extreme reliance on drugs such as corticosteroids with insufficient knowledge of, or respect for, contraindications.

Fundamentally, these factors as presented are due to poor rapport between the cow/calf man, the feeder, the animal husbandman, the nutritionist, the economist, and the veterinarian. When better communication and understanding are developed between these groups, considerable progress will result in solving many of the problems facing the cattle industry today.

WHY DO FEEDER CALVES FAIL TO PERFORM WHEN THEY REACH THE FEEDLOT? (PART II)

R. A. Ivie, D.V.M.
Follett, Texas

In the previous papers that have been presented this morning each speaker has emphasized the importance of stress on those calves that have arrived at the ranch or feed lot. It would seem logical therefore, that, if we can reduce the stress factor, the calves would start gaining quicker, and the incidence of sickness and death loss would be reduced.

The factors that we are talking about that cause stress are:

1. Gathering and weaning the calves.
2. Shipping of the calves.
3. Feeding and watering during this entire period of time.
4. Working the calves, such as branding, castration, dehorning, vaccination, and control of external and internal parasites.

1. Many of the calves shipped into the Texas panhandle area have been taken away from the mother cow and shipped to market the same day. The order buyers purchase these calves in small groups or even one at a time. They are then placed in a pen together with little thought to feed and water. The animals are so excited at this stage they possibly will not eat or drink very much any way. This may cover a period of two or three days to a week.
2. When the order buyer fills his order, the calves are loaded on trucks and transported to their destination. There may be a period of twelve hours to seventy-two hours, before the calves are unloaded. Calves are shipped from Florida, Alabama, Georgia, Mississippi, Louisiana, and east Texas panhandle.
3. At this stage of the game (the destination point) the calves are hungry, thirsty, and utterly exhausted. They are offered feed and water. Too often the man receiving the cattle has made little preparation for them. He has not cleaned out his watering tank at least once in past ten years. He has too little area for feeding and has given little thought as to the best feed or to the amount of feed.
4. The rancher or feed lot operator is in a terrific hurry to get these calves out on pasture or started on feed, so, he immediately starts to work his calves, castrating, dehorning, branding, vaccinating, with every conceivable product on the market.

The ideal situation would be to be able to buy preconditioned calves; however, at the present time this is not possible on a large scale. The next best thing to do is to provide proper management and close supervision of calves after they arrive at final destination.

1. Purchase your calves as close to their origin as possible.
2. Purchase from order buyers that will show an interest and keep these stress factors to a bare minimum.
3. Keep a close check of the truckers so that the calves are delivered in the least possible time.
4. When the calves are unloaded, have clean water available. Know ahead of time what and how you are going to feed through the stress period and provide adequate feeder space.

Now is when the work really starts. In our practice we recommend a water medication from the time the calves are unloaded until they are turned out or placed on full feed. We have a ration that we use to provide a balanced ration without upsetting the digestive tract. Antibiotics can be added to this feed if it is thought to be necessary.

We suggest the calves be worked after a twenty-four hour rest period. This usually includes castration, dehorning, branding, vaccination, and controlling internal parasites. The cattle should be confined in an area so that any animals that appear sick can be removed without exciting the sick animals or the entire group. The calves should be observed closely each day. Any animal that appears droopy should be pulled out and have his temperature taken. If he is running temperature, record it and treat the calf, keeping him separated from the rest of the herd. If he is not running temperature, return him to the herd.

The stress period usually lasts for ten to fourteen days after the calves have arrived at the final destination. This does not mean to say that you will not have calves to get sick after this period. We have seen calves have pneumonia six weeks after being turned out to pasture.

With the above outlined program we have been able to regain orginal shipping weights within a two week period and in some instances in seven days. We have been able to reduce the morbidity from 60% to 100% to zero to 30%. This, we feel, is quite an accomplishment.

A REVIEW OF VIRAL DISEASES OF FEEDLOT CATTLE

D. G. McKercher, D.V.M.
School of Veterinary Medicine
University of California
Davis, California

Thank you, Mr. Chairman. Members of the Conference. I will not refer to the clinical aspects of the viral infections which I am about to discuss because I feel that all of you are quite familiar with them. Rather, I will dwell on the ecology, and epidemiology of these diseases and the problems associated with their prevention and control.

During the past 10 to 15 years a number of viruses has been isolated from cattle, particularly from the respiratory tract. The disease-producing significance of the more recently isolated ones, namely the adeno, the rhino and the reoviruses, has not been established. However, since these viruses infect other species, it is quite possible that they produce naturally-occurring disease in cattle also. However continued research will be required in order to clarify the situation.

There are three well known bovine viruses, two of which, namely, the virus of infectious bovine rhinotracheitis (IBR) and bovine viral diarrhea (BVD) virus which are the causes of severe and economically important diseases, particularly of feedlot cattle. The third one referred to above is the parainfluenza-3 (PI-3) virus. While the first two viruses mentioned produce severe clinical disease without the intervention of secondary bacterial infection, the disease produced by the PI-3 virus is quite mild. However, when complicated by secondary bacterial invasion it is, in the opinion of most investigators, manifested by so-called "shipping fever".

The Ecology and Epidemiology of Respiratory Viral Infections of Cattle

1) *Infectious Bovine Rhinotracheitis:* Environmental factors play an extremely important role in respiratory infections by providing not only stressful conditions, as has been mentioned this morning, but also by providing ecologic situations which might alter or extend the pathogenic potential of the virus. Of the three viruses mentioned, the IBR virus is the only one which gives indications of having undergone changes in pathogenic behavior as a result of ecologic factors. It is possible that other of the bovine viruses might inadvertently be induced to cause new manifestations of disease as has the IBR virus. The postulated ecologic developments which contributed to, or resulted in this situation in the case of the latter virus are briefly as follows.

It is now known that the IBR virus is the cause of "Bläschenausschlag" or, as the disease is known in this country, infectious pustular vulvovaginitis (IPV). This is a venereally transmitted infection which has occurred in Central Europe for over a century. Husbandry practices relevant to small, isolated dairy herds indigeneous to these areas, and particularly the use of one bull for all or most herds in the village, maintained the venereal infection to the exclusion of the respiratory disease which this virus caused subsequently on this continent.

Since the virus can persist in the prepucial tissues of recovered bulls, and possibly in the genital tract of the female, it undoubtedly was introduced into this country in carrier cattle. This must have occurred prior to 1930 since an embargo was placed that year on the importation of cattle from Europe. The virus established itself in small herds in the eastern parts of the United States and, because of the similarity in the type of dairy operation in these areas and in Central Europe, the venereal infection was maintained. In fact it is now known, in retrospect, that the first infection caused by this virus in the United States was IPV and not IBR. Eventually the virus gained access to cattle in feedlots. It was unable to produce venereal infection in these animals since such cattle are essentially reproductively inactive. Apparently, however, it established itself in the respiratory passages of these animals.

In situations where large numbers of cattle are maintained in close physical contact, respiratory viruses can pass very rapidly from animal to animal and in the process, build up a high degree of pathogenicity for the system of the body which they infect. It was through such a process that the severe respiratory syndrome which became known as infectious bovine rhinotracheitis is believed to have evolved.

Later, it was found that the virus was the cause of conjunctivitis in cattle. It also acquired the ability to invade the blood stream and, consequently the fetus, with the production of abortion. The blood borne virus also invaded the brain tissue causing a meningoencephalitis. In this slide I have attempted to show the ecologic developments which resulted in this one virus acquiring the unique ability to produce five distinctive clinical syndromes.

In situations where the concentration of cattle is not great, as in cow-calf and range cattle operations, the opportunity for spread of the virus is minimized so that we rarely find IBR under such conditions. Generally speaking, it is not a problem until cattle are brought together in large numbers as in feedlots. Under these conditions relatively few carrier animals can trigger a large scale outbreak.

Encephalitis, IPV and abortion due to the IBR virus are of little significance in feedlot animals although conjunctivitis can be a minor problem. Whether the IBR virus causes keratoconjunctivitis has not been established.

2) *Bovine Viral Diarrhea:* BVD is a much more complex disease than IBR and is much less well understood. Clinically it resembles rinderpest; however, any relationship between it and the latter has been excluded by serologic and immunologic testing. Apparently BVD originated in the eastern or midwestern United States sometime during the early 1940's. Since that time it has been recognized in essentially all parts of the world with the possible exception of South America. As far as is known the BVD virus does not produce any clinical syndrome other than BVD.

Little is known about the epidemiology of BVD. The disease is contagious, being transmissible via the respiratory passages. Some cattle shed the virus in the urine for long periods of time following clinical recovery, thus providing a very effective mechanism for natural spread of the disease.

3) *Parainfluenza-3:* Ecologically there is little known about the PI-3 virus. It is closely related to the PE-3 virus of man, hence it is interesting to speculate that originally it might have been a human pathogen which became adapted to cattle. This is in contrast to the swine influenza virus which, in taking the reverse route, played an important etiologic role in the 1918-19 influenza pandemic in man.

Parainfluenza-3 virus infection is contagious and it occurs widely, as revealed by the high incidence of antibody to the virus in cattle. However, since the incidence of antibody is much greater than that of shipping fever, with which this virus is believed by most to be associated, it is apparent that the great majority of cases of PI-3 infection are subclinical in nature. It is possible that an immunity to the virus is short lived so that animals repeatedly become reinfected as in the case of the common cold in man.

Prevention and Control

1) *Infectious Bovine Rhinotracheitis:* Vaccination against infections caused by the IBR virus is widely practiced, (over 26 million doses of the vaccine were produced in 1966) and it has been uniquely successful. Colostral antibody to the virus disappears about two weeks after birth so that calves can be vaccinated at an early age. The vaccine gives a good immune response and antibody levels in the range of 1:2 or 1:4 are sufficient to protect against the natural disease. Moreover, the immunity is durable, persisting for years. It is possible, however, that it is maintained through periodic field re-exposure of vaccinated and convalescent cattle to the virus.

2) *Bovine Viral Diarrhea:* Vaccination against BVD has been somewhat less successful than against IBR although it has reduced considerably the losses caused by this infection. However, postvaccinal reactions occur to some extent and are of considerable concern to both the manufacturer and the user of the vaccine.

A number of reasons has been advanced to account for these undesirable effects. Some claim that the vaccine virus is not sufficiently attenuated and that when given under adverse environmental conditions, which frequently exist in the field, the combined effects of environmental and physiologic stress lead to complications. It is felt also by some that when the BVD virus is combined with a second virus, the synergistic effect of the two results in clinical disease in highly susceptible animals. Still another claim is that the vaccine virus is sometimes contaminated with virulent field BVD virus from the fetuses used in the preparation of the cell cultures for the production of the vaccine virus. Finally, postvaccinal reactions have been attributed also to immunologic tolerance. It is postulated that cattle which became infected prenatally from infected dams are incapable of responding immunologically to the virus, whether it be the modified vaccine virus or virulent field virus. Such animals eventually succumb to the infection, ostensibly through their inability to produce protective antibody against the virus. However, thus far none of these theories has been substantiated.

In studies of a recent postvaccinal outbreak of BVD, it was found that some of the animals in the herd were in the incubative stages of either BVD or PI-3 infection. It was believed that the stress of vaccination precipitated these infections, which otherwise might have remained subclinical, into acute clinical disease. It is also possible that the appearance of the clinical phase of these infections coincided closely with the time of vaccination. In either situation indications would be that the vaccine was at fault.

The maternal antibody level to the BVD virus persists for as long as six months following birth but it apparently does not afford protection during this entire period. This is based on the fact that by the time the passive immunity

normally should have worn off, a high percentage of cattle are immune, presumably as a consequence of natural infection acquired after the colostral antibody dropped below a protective level. BVD antibody levels which are inadequate to protect against the natural infection interfere, unfortunately, with the vaccine virus, rendering vaccination ineffective. For this reason it is advisable to delay vaccination until the animals are six months or more of age.

3) *Parainfluenza-3:* A vaccine against shipping fever consisting of inactivated PI-3 virus and a strain of *Pasteurella multocida,* has recently been marketed. Two doses are recommended for maximal effects. A second vaccine which is either available, or will be shortly, contains the modified IBR and PI-3 viruses. While it is still too early to assess the effectiveness of these products in either controlling or reducing the incidence of shipping fever, it is encouraging that an effort is being made towards this end.

The difficulties in immunizing against this important infection are formidable. The fact that repeat infection may occur in the same animal indicates that the immunity is poor. This is due to some extent to the antigenic nature of the etiologic agents involved but particularly to the nature of the infectious process itself. Viruses produce a more durable immunity than do bacteria, with the strongest response to either occurring in the case of generalized infections. In a localized infection of mixed etiology, such as shipping fever, in which the viral infection is mild, the resultant immunologic response is weak and hence soon disappears. Because of the weak immunologic response given to the bacterial antigen, in this case the *Pasteurella* organism, the animal soon becomes susceptible once again. Thus, the problem posed in attempting to immunize against shipping fever is not one of protecting only against a virus which, at most, causes a very mild infection, and hence a weak, transitory immunity, but it involves also protecting against one or more bacterial agents which are relatively poor antigens.

Discussion

In considering the efficacy of vaccination, the existence of stress and the role it plays in any vaccination program cannot be ignored.

Stress is one of the great detriments to good health in all species of animal. It increases susceptibility to disease as was so dramatically demonstrated by Pasteur who succeeded in infecting chickens with the anthrax organism after lowering their general body resistance by forcing them to stand in iced water. Stress may also precipitate latent or mild infection into acute disease.

The importance of stress as it relates to the feedlot industry is recognized and improvements designed for its alleviation have been made in the transportation of cattle. These include frequent rest and watering stops and protection against inclement environmental conditions. Many laymen and probably even some veterinarians fail to realize that vaccination is also a great stress producer. When physiologic stress from vaccination is augmented by environmental stress factors, highly unfavorable results may ensue. Despite this situation, essentially all vaccinating of beef cattle is done after these animals enter the feedlot. This is an extremely dangerous practice. However, since vaccination was not done earlier, the feedlot operator has no choice but to take the risk. Nevertheless, the folly of this practice is reflected in the high incidence of post-vaccinal reactions which occur in feedlot cattle.

The ideal time to vaccinate is at an earlier age when environmental stress factors are at minimal levels and when vaccination can be carried out over a period of time. Under these conditions, post-vaccinal reactions are minimal. In addition, cattle would arrive at the feedlot fully protected.

The concept of preconditioning *per se* is a sound one. Whether the necessary cooperation can be obtained from all segments of the beef cattle industry involved, and whether the coincidental problems which are almost certain to arise can be resolved is another consideration. Nevertheless, this approach to the reduction of disease among feedlot cattle has much to recommend it, and it should be given a fair trial.

BACTERIAL DISEASES

J. R. Collier, D.V.M., Ph.D.
Colorado State University
Fort Collins, Colorado

I'm pleased to attend this meeting in which there is representation of so many disciplinary and specialty areas pertinent to the theme of conditioning cattle for the feedlot. Certainly the talents of geneticists, nutritionists, veterinary scientists and practitioners, the pharmaceutical and biologics manufacturer, the cattle producer, feedlot operator, and others should be brought to bear on this theme. It is evident that each of these areas can and must make significant contributions, if workable plans are to be developed and applied. It is equally evident no one specialty group has all of the answers. Teamwork among these groups is essential and effective communication is the key. In some respects this needed communication and cutting across the boundary lines of specialties is analagous to the ecumenical movement in the church.

There are many bacterial diseases or infections of beef cattle that are of concern both to the farmer or rancher and the feedlot operator. It is my interpretation that, for the purposes of this conference, attention should be directed toward and limited to those bacterial diseases or infections which are (1) currently considered most significant from the standpoint of preconditioning cattle and (2) for which prophylactic immunization of proven value is available or (in the case of shipping fever) perhaps is being developed. In this light blackleg, malignant edema, anthrax, shipping fever, leptospirosis, and perhaps bacillary hemoglobinuria should be considered. For one or both of these reasons, the bacteria associated with calf diphtheria, footrot, and the "sleeper" syndrome will not be considered here.

Fortunately, effective prophylactic immunizing products are available for protecting cattle against blackleg, malignant edema, and bacillary hemoglobinuria, anthrax, and leptospirosis. These diseases are well known and, in may opinion, need no review for the purposes of this conference. In a preconditioning program for cattle which are produced and will remain within the continental United States there will likely be some variation between (and perhaps, within) geographic regions as to requirement for immunization against each of the diseases just mentioned. For example, there appears to be a common need in all geographic regions for routine vaccination against blackleg and malignant edema. Certainly cattle that are produced in or that will be shipped into an area where anthrax is endemic must be vaccinated against this disease. The need for vaccination against leptospirosis would be subject to similar considerations. Cattle originating in areas in which bacillary hemoglobinuria is endemic should be vaccinated against this disease before shipment to a feedlot. "Go anywhere" cattle presumably would require numbers of known effective immunizations. Here cost must be justified.

Shipping fever (SF) is a disease of the utmost concern in conditioning cattle for the feedlot. In view of its importance and in view of the controversy that has existed relative to the etiology of SF, detailed consideration of this disease is indicated. SF most commonly affects weaned calves that have been shipped but also may affect yearling cattle and calves that have not been weaned or shipped. Clinically, varying degrees of depressed attitude (as evidenced by a lowered head with one or both ears dropped), serous ocular discharge, mucopurulent nasal exudate, febrile reaction, anorexia, dyspnea and cough may be observed. At necropsy lesions are most prominent in the respiratory tract and include inflammation of the mucosa of the airways (with or without fibrinous casts in the head sinuses) and a fibrinous pleuritis and pneumonia with hemorrhage into the alveoli and a marked thickening of the interlobular septa. Varying amounts of the anterior and ventral portions of the lungs are affected.

Sick cattle in which the pneumonia is not advanced generally respond favorably to appropriate doses of selected antibiotics or sulfanomides or combinations of these drugs. In general, attempts at prophylactic immunization of cattle against SF using pasteurella bacterins have failed, as have the use of tranquilizers, pasteurella antiserum, and preventive medication.

Considerable information relevant to the nature and control of this disease has been developed through research within the past 15 years. Unfortunately, much of this information has not become generally known or applied.

In general the information gained through investigation of epizootics of SF and by experimental production of this disease has confirmed the significant role of *Pasteurella multocida* and *Past. hemolytica* in its etiology, and provides a rational basis for formulating a working hypothesis concerning the pathogenesis of SF. Studies of the antigenic (serologic) structure of these *Pasteurellae*

provide a valid basis for selection of the correct immunogenic strains to be included in bacterins. Studies pertaining to the disease known as epizootic pasteurellosis (hemorrhagic septicemia) of cattle and work buffalo of Asia, Africa, and eastern Europe provide guidelines for the production of more effective bacterins for use in protecting cattle against the bacterial component of SF.

Field investigations have revealed that virus (Myxovirus parainfluenza-3), bacteria *(Past. multocida* and/or *Past. hemolytica)* and physiologic stress(es) are associated with natural epizootics of SF. Experimentally, clinical signs and lesions resembling those seen in field cases of SF have been induced in cattle subjected to combinations of MP-3, *Pasteurella spp.*, and stress, but not in cattle given these agents singly or in other combinations. Virus alone induced no observable effect or only a mild to moderate transient pneumonitis. Fluid culture of *Pasteurella spp.* given intratracheally induced little effect while inhaled aerosol induced febrile reaction and certain other signs of respiratory disease. From this information one can logically conclude that virus, bacteria, and stress are essential factors in the etiology of SF.

Now Let's consider a working hypothesis as to the role of the MP-3 virus and these Pasteurellae in SF. Delineation of the exact or total contribution of the MP-3 virus or these Pasteurellae to the pathogenesis of SF has not been achieved or at least not recognized. This delineation of cause and effect is pertinent to understanding the pathogenesis of SF and, more importantly, to provide a rational basis for a prophylactic immunization program against SF.

In my opinion, the evidence accumulated to date strongly supports the hypothesis that the Pasteurellae are the principal or perhaps sole cause of the advanced clinical signs, terminal lesions, and death loss associated with SF and that the MP-3 virus simply predisposes cattle to this severe and sometimes fatal pasteurellosis but does not elicit the advanced clinical signs, or lesions, or death loss. If this is the case, then the virus infection in SF can logically be considered as an increment of physiologic stress, albeit infectious in nature.

Let us examine evidence in support of this hypothesis. For the past 25 years chief reliance for the control of SF has been placed in early diagnosis and prompt treatment of affected cattle using sulfonamids, or antibiotics, or both. It is reasonable to assume that many cattle thus medicated would have recovered without the benefit of this drug therapy. It is equally reasonable to assume that in many other cattle a severely debilitating disease and perhaps death was averted by therapy.

If this is the case, the benefits of therapy must have been derived from control of infectious agents susceptible to antibiotics or sulfonamids. The Pasteurellae are the only antibiotic-sensitive microorganisms that have been shown to be significant in the etiology of SF. In view of the foregoing, it is evident that the benefits of antibiotic and sulfonamid therapy of cattle affected with SF was due to control of these Pasteurellae.

Furthermore, in cattle medicated with these drugs the MP-3 virus was free to exert its utmost effect without competition from bacteria. This suppression of bacteria in vivo has an effect analagous to that achieved in vitro in tissue cultures which are inoculated with virus with antibiotics routinely added to suppress bacteria. In considering the requirements for growth of MP-3 virus and the Pasteurellae within the bovine respiratory tract it is apparent that the virus has the advantage in the early stage of SF and that the advantage belongs to the bacteria during the later stages. At the outset of infection there are, in the linings of the airways, large populations of healthy epithelial cells for the virus to attack and little competition from bacteria, in other words, an excellent substrate for viral growth.

In contrast, the Pasteurellae likely have difficulty in establishing infection on intact epithelial membranes but enjoy an increasingly favorable growth medium once host epithelial cellular debris and serous fluid have been elicited by virus. The Pasteurellae then can grow in prodigious numbers and exert pathogenic effects perhaps through the action of endotoxin. Studies of the hemodynamic and skin toxicity properties of the endotoxin of *Past. hemolytica* indicated that this toxin has a potency comparable to that of the endotoxin of *Salmonella spp.* and certain strains of *Escherichia coli.* The growth of large populations of *Past. spp.* in the lungs of cattle affected with SF and the attendant release of endotoxin could account for hemorrhage as well as the massive outpouring of fibrin and inflammatory cells seen in SF. If so, this occurrence would be analagous to pneumococcal pneumonia of man in which toxin released by the pneumococci promotes extension of the pneumonic lesions. Incidentally, it is recognized that initiation of antibiotic therapy in advanced cases of pneumococcal pneumonia may hasten death of the patient due to increased death rate of the bacterial cells and proportional increase in the release of toxin.

Doubt that *P. multocida* or *P. hemolytica* are pathogenic for cattle has stemmed from the fact that these bacteria can be isolated from the nasopharynx of some healthy cattle. In my opinion, this situation is analagous to the carriage by some healthy humans of pneumococci or meningococci in their nasopharynx. These human carriers do not have pneumococcal pneumonia or bacterial meningitis, yet occasional people do have pneumococcal pneumonia and occasional epidemics of bacterial

meningitis do occur in youthful people recently weaned from their home environment, collected from various geographical areas, "shipped" to college or to boot camp, where they are placed in close contact with each other in classrooms and dormitories which constitute excellent chambers for aerosol inoculation. Few physicians doubt the pathogenicity of either the pneumococcus or the meingococcus, but there are some veterinarians who still doubt the pathogenicity of these *Pasteurellae* for cattle.

It is apparent that carrier cattle constitute the source of Pasteurellae for infecting other cattle. Dissemination or transmission of these bacteria is assumed to be by contact. In outbreaks of SF in unweaned range calves in early fall there is invariably a history of the cattle being gathered with the attendant close contact and a certain amount of excitement which may be stressful. There is opportunity for nose to nose contact with exchange of nasal secretions among calves and perhaps opportunity to inhale infective aerosols created by coughing of carrier cattle irritated by dust or excitement. In this way both the virus and bacterial components of SF can be disseminated.

In explosive outbreaks of SF it seems likely that these Pasteurellae invade deeply into the lungs as droplet particles expelled by the coughing of a sick calf. The terminal bronchioles and even the alveoli may, in this way, be directly invaded. There is precedent for this belief in work done many years ago in attempts to experimentally induce bovine contagious pleuropneumonia. Healthy cattle inoculated intratracheally with virulent pleural exudate did not develop pleuropneumonia. Finally typical pleuropneumonia was induced by stanchioning two cattle, one healthy animal and one with pleuropneumonia, facing each other with their heads several feet apart. A linen tube 2 or 3 feet in diameter was secured to the head of each animal so that the healthy animal was forced to breathe the aerosolized material produced by coughing of the sick animal.

It is well known that pasteurella infection may spread rapidly among cattle confined in railroad cars or trucks in transit and that fewer cases of SF occur in newly-arrived cattle that are dispersed out of doors, even in bitter winter weather, as compared to those which the owner pampers by confining them in a nice warm barn which insures close contact and quiet air for the dispersion of infective aerosols.

The rapid onset of lower respiratory tract infection of cattle that inhaled aerosolized *Pasteurella hemolytica* live culture has been demonstrated experimentally. Four cattle averaging 320 lb. in weight and maintained in isolation were inoculated with an aerosol of *P. hemoly-*

Table 1. Clinical Data on Calves Inoculated with *P. hemolytica* 30 Days after Inoculation with IBR Virus

Calf number	Rectal temperatures in hours postinoculation											Clinical signs of disease (in days) and outcome ▼	
	0	3	7	13	24	36	48	60	72	84	96		
1.	1.4*	2.0	5.2	6.6	5.6	6.2	5.6	5.9	4.3	4.0	3.9	depression anorexia cough dyspnea prostration (died at 96 hours)	3.5 3.5 3.0 3.0 1.0
2.	1.8	2.2	2.9	3.9	5.8	5.2	2.5	3.2	2.0	2.1	1.6	depression anorexia cough (recovered health)	1.5 1.5 1.5
3.	2.0	2.4	3.6	7.0	7.0	5.6	6.1	5.8	6.6	6.4	5.7**	depression anorexia a. marked b. partial cough dyspnea (chronic pneumonia)	4.0 4.0 3.0 7.0 2.0
4.	2.6	2.2	3.8	5.7	5.9	5.4	3.6	4.7	2.8	3.0	2.8	depression anorexia (cough (recovered health)	2.0 2.5 2.0

* 1.4=101.4F,etc.
** calf No. 3 was febrile for an additional 4 days.

tica. One ml. per calf of a 24 hr. tryptose broth culture of *P. hemolytica of Bibersteins type 1 was aerosolized into* a plastic nose bag for inhalation. These cattle had been inoculated 30 days previously with IBR virus intratracheally and had gone through the relatively mild respiratory disease that this virus elicits in cattle on a low energy diet, but they had returned to clinical normalcy and were clinically normal for more than two weeks prior to inoculation with *P. hemolytica.* All of these calves developed high febrile reactions and clinical signs including anorexia, depressed attitude, cough, and increased respiratory rate.

Two of these calves returned to clinical normalcy within 72 hours postinoculation. The clinical signs and febrile reaction persisted for 8 days in a third calf which went into chronic pneumonia. The remaining calf died at almost exactly 96 hours postinoculation (Table 1). At necropsy gross lesions involving approximately 80 per cent of the lung tissue and pleura were indistinguishable from those seen in field cases of SF.

Finally, as a result of serologic typing of the *Pasteurellae* associated with SF and as a result of the development of an effective bacterin for prophylaxis of the septicemic pasteurellosis seen in Asia there is hope that an effective bacterin can be developed to protect cattle against the SF *Pasteurellae* and some guidelines as to how to go about producing it.

The strains of *Past. multocida* associated with SF of cattle fall into Carters type A. The strains of *Past. multocida* causing the Asiatic hemorrhagic septicemia of cattle and work buffalo are all members of Carters serologic type B. These two types do *not* induce cross protective immunity. Carter reported that the *Past. bubalaseptica* strain which was derived from an American Bison that died of hemorrhagic septicemia many years ago was in fact the same serotype as that encountered in Asia today (Carters type B). It is ironic that the "buffalo" strain was for many years used to produce bacterin and hemorrhagic septicemic antiserum to protect cattle against SF. In doing this a futile attempt was made to immunize against Type A using a bacterin prepared from Type B. No doubt the rationale for the use of this buffalo strain in the production of biologics stemmed from the belief that "any pasteurella that killed a buffalo would surely immunize a cow". In an AVMA Journal editorial of some years ago the question, "what and where is hemorrhagic septicemia"?, was raised. Perhaps the answer is that it disappeared with the bison and the more primitive cattle that were prevalent on the great plains at that time. The strains of *Past. hemolytica* associated with SF have also been serotyped.

In recent years many experiments have been aimed at immunizing cattle against SF. The infectious agents associated with SF (MP-3 virus, *P. multocida* and *P. hemolytica,* and occasionally IBR virus) have been used as immunizing antigens. In general the results have been confusing; on challenge with homologous kinds of microorganisms the level of immunity was good, but the same immunizing agents failed to induce protective levels of immunity under field conditions.

In view of this confusion of rationale for use of a bacterin to immunize cattle against the Pasteurellae in SF is not clear. If virus infection is necessary to predispose cattle to serious (perhaps fatal) pasteurellosis, then effective immunization against the prevalent respiratory virus(es) should not only control these viruses but preclude pasteurellosis. In that case there would be no need to attempt immunization against the pasteurellae. If, on the other hand, no reliable vaccine can be developed against even one prevalent respiratory virus, or it proves to be impractical to immunize cattle against all of the respiratory viruses that may predispose them to pasteurellosis, or if it turns out that primary pasteurellosis of the respiratory tract may occur in cattle that are sufficiently stressed (whether the stress be all noninfectious in nature or in part due to infections in other organ systems), then attempts to prophylactically immunize cattle against these Pasteurellae is clearly indicated.

The results of drug therapy in SF indicate that avoidance of serious or fatal pasteurellosis may depend on avoidance of the build-up of large populations of Pasteurellae in the respiratory tract and particularly in the lungs. Presumably, then, specific immunity should be sought against the bacterial cell to avoid establishment of infection. However, it may also be possible and important to increase the nonspecific resistance of young cattle to endotoxin of Pasteurellae by immunogenic procedures.

Experience gained in the development of improved bacterins against fowl cholera in the U.S.A. and against epizootic pasteurellosis (also known as hemorrhagic septicemia) of buffalo and cattle in other countries provides guidelines or at least points of departure for preparation of more effective bacterins for aid in the control of SF. In original work and in subsequent reviews the following principles have been emphasized: (1) incorporate the significant serotype(s) of Pasteurellae in the bacterin, (2) insure that bacterial cells produced for bacterin are in the virulent phase (rather than dissociated into avirulent forms), (3) get an adequate quantity of cells (2 mg. dry wt. in case of epizootic pasteurellosis bacterin) per dose, (4) use an oil adjuvant.

One can hope that an improved pasteurella vaccine (bacterin) will be developed which will protect with a single dose. In view of the record of Gram-negative bacteria as immunizing agents, it is more realistic to

expect that two or more doses will be required. The final dose should be given at least two weeks before shipment of cattle.

Summary:

1. With the exception of the pasteurellosis component of SF, time-proven biologics are available for selective use in the conditioning program for prophylaxis of the bacterial diseases of major concern in cattle entering the feedlot.
2. It has been established beyond reasonable doubt that certain serotypes of *Past. multocida* and *Past. hemolytica* are significant in the etiology of SF. Prophylactic measures to avoid or minimize this pasteurellosis perhaps can be achieved by immunizing cattle against all viruses that may predispose them to pasteurella infection, or perhaps by use of improved pasteurella bacterins, or perhaps by the use of a combination of these products.

RELATIONSHIP OF BVD TO FEEDLOT PERFORMANCE

William M. Lynch, D.V.M.
The Dow Chemical Company
Dallas, Texas

Thank you, Mr. Chairman.

At the present time most of the cattle being vaccinated with bovine virus diarrhea (BVD) vaccine are vaccinated on or after entrance into the feedlots. This is the least desirable time for vaccination from the standpoint of prevention because at this stage of the cycle these animals very likely have been exposed to several virus and/or bacterial diseases.

In an effort to gather information, economic benefits of vaccination, The Dow Chemical Company has bought and owned cattle for feed-out in custom feed lots. Using these animals we have run several controlled vaccination experiments.

During 1965 we purchased 863 head of cattle from Mississippi, Texas, and Oklahoma. Of these 510 were vaccinated at the feed lot with Mucovax-2® (Pitman Moore brand of Bovine Rhinotracheitis—Bovine Virus Diarrhea Vaccine) while 353 head were used as controls and were given only Rhivax® (Pitman-Moore brand of Bovine Virus Diarrhea Vaccine) at the destination feed lots. In this manner BVD was the variable.

In 1966 we used 2396 head again with roughly one half serving as controls. Experimental design was the same as for 1965.

During 1967 approximately 2800 head of cattle were used in similar experiments.

Of the 3610 vaccinates for the three years only three head developed signs of "Iowa" mucosal disease (MD)— a malignant form of virus diarrhea. Of the 2847 head of controls one case of Iowa MD developed. When one considers the exposure of the cattle this is an extremely small percentage of MD.

In 1967 one group (600 head) of which 300 head were given Mucovax-2® plus a simultaneous (40 cc) dose of Pitman-Moore's Bio. 591 (Antibacterial Serum Bovine), the death loss was halved for this group compared to the controls receiving only Rhivax. Average daily gain, feed conversion, and total cost of gain favored the 300 head receiving Mucovax-2 and Bio. 591.

In October, 1966 we purchased 580 head of cattle at Lufkin, Texas. One half were given 20 cc Bio. 591 and Mucovax-2 prior to loading for transport to the feedlot while the other half were given Mucovax-2 alone. In November, one of the principals died of MD.

In the 1966 experiments all groups of principals turned in a superior economic performance. For the average of the four vaccinated groups (1300 head of principals) the average added economic benefits was $3.28 per head over the performance turned in by the controls which were vaccinated only for infectious bovine rhinotracheitis (IBR).

Our 1967 trials are being closed out now and data is available on only two lots. These trials were aimed at a type of backgrounding program.

Cattle were purchased at auction markets until 600 head had been accumulated. By random selection the 600 were divided into groups of 300 head each. The control group was sent direct to the destination feedlot where the normal feedlot vaccination and handling program was carried out. The principal groups were sent to a feedlot near the collection point for processing. Principals were held in the feedlot near collection point for thirty to forty days and then forwarded to the same destination feedlot as their control mates for finishing.

Principals were vaccinated with Mucovax-2, blackleg-malignant edema bacterin, implanted with diethylstilbesterol, poured with Ruelene 25E® insecticide, castrated, dehorned, and branded.

Again the BVD vaccinated animals turned in a superior performance. In one group sent from Alabama to Nebraska, the death loss in the controls nearly reached the disaster stage with nearly three times the death loss of the principals—30 vs. 11 head. Cost of medication for principals was $272.00 vs. $1270.00 for the controls.

One thing learned was that backgrounding for so short a period is not economic when cost of two shipping costs are added in. Furthermore since calves had grown we had to consign several head from the backgrounding lot so cattle wouldn't be too crowded in trailers sent to destination feedlot.

Their short summary of three years' work does show the economic benefit of vaccination for BVD even upon entrance to the feedlot.

I feel that even more benefit would accrue if calves were vaccinated on their ranch of origin as a part of a specification preconditioning program.

Since virus vaccines are designed for prevention, they are best given prior to exposure.

RELATIONSHIP OF IBR TO FEEDLOT PERFORMANCE

Dr. Richard C. Searl
Fort Dodge Laboratories
Fort Dodge, Iowa

Thank you Mr. Chairman. Gentlemen. IBR is most commonly recognized in our feedlots as an acute upper respiratory infection which typically results in a high morbidity with a low mortality figure. The lesions as we now well know are those of a catarrhal to fibrino-necrotic type and occur in the nose, sinuses, larynx, pharynx and the trachea. Pneumonia may develop but it is a secondary complication. Alimentary tract, symptoms and lesions are not part of the syndrome observed in the field. Outbreaks of BVD may be confused with IBR as are some outbreaks of so-called shipping fever complex, PI-3, Pasteurellosis and this complicated syndrome.

It is somewhat disconcerting to know that experimentally intravenous innoculations of IBR virus on occasion may produce digestive disturbances, whether this often or ever occurs in the field in uncomplicated IBR infection I do not know. This certainly is a commonplace virus as indicated by all the serological surveys which have been conducted. It has become increasingly commonplace to see IBR infection in newly arrived feedlot cattle. And probably more commonly it is superimposed upon slowly resolving cases of shipping fever. In this instance severe fibrino-necrotic lesions are noted and usually a chronic suppurative pneumonia. Diagnostic procedures utilized at this time incriminate only IBR virus as producing these lesions with the aid of secondary bacteria. In actuality these severe pneumonic lesions are a continuation of pathologic processes already in progess. This may be confirmed as indicated by the location, the age of the lesions, etc. These pneumonic areas do not develop as a result of either virus activity or inhaled tracheal exudate. The mortality in these outbreaks may be excessive.

Indications are that both physical and biological stresses are important in initiating IBR clinical infection and are equally important in influencing the severity of the outbreaks. Under adverse conditions simultaneous exposure to two or more virus diseases or bacterial complications greatly increases the severity of the disease and may be accompanied in some instances by immunologic failure. It has been suggested by some that animals may harbour the IBR virus in an apparently inactive form for long periods of time. Here an animal showing recrudescense of viral activity does offer an explanation for some of the outbreak sources observed. The forms of IBR, we have observed in the feedlot, particularly in the midwest, and I assume this is so otherwise, in the order of their frequency of observation are the upper respiratory form, the ocular form or conjunctivitis and although I have not observed them, encephalitic cases are reported I think these are rather rare and infrequent at this time The clinical variations and the reasons for such outbreak variations are not clearly understood at this time Some feel that they may be due to the primary site o viral infection. The spread of IBR through the herd may be either rapid or it may be slow. Vaccination is certainly indicated before exposure and if not practiced, i indicated during an outbreak.

To acquaint you with some problems associated with immunization that I have observed, I would like to repor on one case where I was working with a local practitioner I think this case report represents one of the problem that we are faced with, in the feedlot preconditioning program, which includes the period 30 days prior t movement and thirty days after movement or arrival a the lot. We had a group of 115 steer calves, weight 400 t 450 pounds, consisting of 81 herefords and 34 angu calves. These were good quality calves. They wer shipped from Kansas City, Missouri to a feedlot in th Fort Dodge, Iowa area on the first of April 1967. The were obtained from the yards and a previous history o these calves was not obtainable. However, according t the practitioners, they were healthy upon arrival an on the third of April which was two days after the left Kansas City, they were vaccinated for IBR with single antigen product upon arrival at the feedlot. Thes animals required no treatment for the ensuing tw months, but on 6/6/67, and this is approximately tw months after their arrival, and after their vaccination they became sick.

Only the herefords were observed to be sick. The mo bidity was approximately 20% (25 animals)—all sick an mals were herefords. Temperatures ranged from 104 t 107.3. There was dyspnea, depression, conjunctiviti open mouth breathing and tenacious long nasal exudat but there was no scouring or pneumonic sounds, or lesions or other visible symptoms. Practitioners reporte the use of antibiotics tended to reduce this temperatur within twenty-four hours. Two of these animals had die one on the 10th and one on the 11th of June. I was calle in on consultation on these on the 11th. I would concu with the previous history provided by the veterinaria as being correct with the added observation that the were in addition to these 115 head, a group of health

IBR vaccinated cattle in an adjacent lot separated by only a fence but housed under the same barn roof. The autopsy of the one animal posted revealed rhinitis, pharyngitis, laryngitis, tracheitis, and in spite of vaccination, a presumptive diagnosis of IBR was made. Acute and convalescent serum samples were obtained from this group. In all 5 representative animals were bled, 1 healthy hereford, 2 sick herefords and 2 healthy angus calves. Results of serum neutralization studies were as follows: All 5 animals were negative on both samples to B.V.D. One sick hereford had an ascending titre to I.B.R. and a positive but non-ascending titre to P.I.$_3$. The other sick hereford had an ascending titre to I.B.R. and an ascending titre to P.I.$_3$. The healthy hereford steer showed a positive but non-ascending titre to I.B.R. indicating a past immunizing contact with I.B.R. antigen or infection. This healthy hereford calf had an ascending P.I.$_3$ titre. The two angus calves both showed a titre to I.B.R. which did not ascend, one of those showed an increase in P.I.$_3$ titre and the other remained negative to P.I.$_3$.

The conclusions that I have drawn of this case, and they may be incorrect, and my explanation of the results are as follows: The I.B.R. vaccine used was a viable antigenic product. It was properly given and did elicit an immune response. Some of the hereford group had passive antibody protection at the time of vaccination and this fact possibly accompanied by the stress of movement did not permit the development of an immune response. As a result, some were immunized and some were not. The serological findings are compatible with this assumption. It also confirms the the P.I.^{3} virus was present in the herd but in the abasnce of adequate stress bacterial complications etc., no clinical syndrome was produced. This further points out to me, or suggests to me, that passive antibody may interfere with the response to viral antigens. This does not appear compatible with the previous recommendation to vaccinate at 2 weeks of age. We cannot wait until there is a 100% opportune time. You will have to vaccinate and get a good percentage response from those involved, such as did here, and we were protecting ninety-plus out of 115 in spite of a few who did not respond, or you have to wait for a time when they are all susceptible to infection.

In a preconditioning program as far as I.B.R. vaccination goes, calves certainly should be injected 2 weeks prior to movement, and we'd hope they would all be immunized. We should make no guarantees, and if situations and indications would warrant after arrival at feedlots, or after on feed, they may and should be re-vaccinated if conditions so indicate. Thank you.

PI-3, HOW IT RELATES TO FEEDLOT PERFORMANCE

Dr. L. H. Carroll
Elanco Products Company
Garland, Texas

My remarks this morning will relate primarily to my company's experience with a vaccine we call BAR-3®, which is a killed product containing the PI-3 virus and two species of Pasteurella, multocida, and hemolytica.

I would like to review briefly what we know about the PI-3 virus. Its role as a pathogen was demonstrated by Dawson and Darbyshire using colostrum deprived calves. They were able to demonstrate clinical symptoms and a temperature response, as well as microscopic lesions of pneumonia in experimentally innoculated calves. They also recovered the virus from lungs with pneumonia with no complicating bacteria present. Abinanti, 1961, et al, and Woods, 1964, et al, were able to produce respiratory signs with PI-3 in experimentally exposed calves. Hamdy, et al, (1963) observed a febrile response and a leucopenia in calves that were given virus alone. They also observed some pneumonic lesions in these calves. Gale and King duplicated this work. However, the clinical symptoms described here were not the severe respiratory distress referred to as "Shipping Fever." Where this complex has been experimentally reproduced, it has required the combination of the PI-3 virus and one or more Pasteurella species.

Serological surveys have established that PI-3 is prevalent in the cattle population. The demonstration of an increase in titer of four fold or greater in all groups of feedlot calves studied during a three-year period by Hoerlein, et al, indicates widespread infection of feeder calves with PI-3 during shipment. Surveys of market cattle bled at slaughter showed at least 70% were carrying antibodies to PI-3. A survey of mature cows in Nebraska reported that 86.2% had significant antibody titers. The incidence of PI-3 in New York state was 48% in a similar survey.

The role of PI-3 in the Shipping Fever Complex can perhaps best be described by quoting several published papers on the subject: Hoerlein and Marsh (1959) state —"the etiological factors in shipping fever are apparently a complex of several elements. The hypothesis of a virus, perhaps latent, being activated by the stress of shipment to make a favorable environment for secondary bacteria finds some support in these experiments.

Hetrick (1962) from his observations felt that PI-3 acted as a trigger mechanism for the Pasteurella species. "Due to the widespread occurrence of both PI-3 and Pasteurella multocida in cattle with respiratory illness, it is suggested that the combined effect of these two agents may be responsible for a portion of the naturally occurring bov respiratory disease. Presumably, the virus first multipl and alters the respiratory mucosa in some manner, af which P. multocida multiplies and is responsible for t severity of the syndrome."

Numerous workers have been unable to produce t clinical symptom referred to in the feedlots as "Shippi Fever" with the PI-3 virus alone. But most agree th Shipping Fever as we know it is the result of field co ditions superimposed on a PI-3 infection. These conc tions interplay with each other and include such factc as the host animals resistance, external stress factors su as climate and crowding, the bacterial flora in the respir tory tract, and others.

These remarks have been an attempt to provide son perspective on how the PI-3 virus affects the feedl animal as a disease producing organism. As previous mentioned, my experience with the PI-3 virus has bee limited to field evaluations of our BAR-3 vaccine.

TABLE No. 1 summarizes one of our first trials. The cattle were obtained from a ranch near Marfa, Texa Serologic studies of blood samples from these catt were negative on HI for PI-3 antibody prior to the fir vaccination. Also, 20 nasal swabs taken prior to vaccin tion were negative in tissue culture. One-half the heife and steers were vaccinated on the ranch twice, thre weeks prior to shipment and at shipment. The anima were then shipped to our research facilities at Greer field, Indiana, with a stopover at a public stockyard enroute. Exposure did occur enroute since the control developed an HI titer shortly after arrival.

TABLE NO. 1

GREENFIELD FIELD TRIAL NO. 1

HEIFERS	Vaccinated*	Non-Vaccinated
Number	60	60
Number Requiring Treatment**	4	22
Percent Requiring Treatment	6.6	36.6
Number Died	2	1
PI-3 Isolates	3	23
STEERS		
Number	60	60
Number Requiring Treatment**	7	4
Percent Requiring Treatment	11	6.6
Number Died	0	1
PI-3 Isolates	8.3	13.3

*Three weeks prior to shipment
**Treatment based on temperature of 105° F. for two days

Serum neutralization tests were also performed, and there appeared to be a direct correlation in the development of SN antibodies and HI antibodies. Most of the viral isolates made in tissue culture from nasal swabs proved to be PI-3.

Pasteurella multocida was isolated consistently on arrival and again six weeks later. Pasteurella hemolytica was not a common isolant in this trial. Several strains of bovine mycoplasma were also isolated.

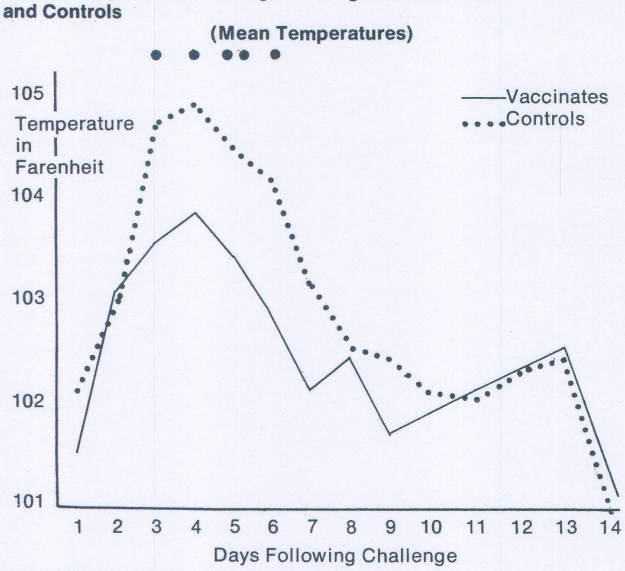
From this trial we can conclude:

1. That a killed PI-3 vaccine can provide protection when used in susceptible calves and when a PI-3 outbreak occurs relatively uncomplicated.
2. That some correlation exists between the development of HI and SN antibodies.
3. That vaccination and the corresponding antibody increase tends to eliminate the carrier state of the PI-3 virus from the nasal passages.

Vaccinated and unvaccinated controls were experimentally exposed by aerosol to a virulent culture of the PI-3 virus and *Pasteurella sp.* (Table No. 2). After experimental challenge, temperatures were higher and persisted longer in the unvaccinated controls. The differences were significant at the 0.01 and 0.05 probability levels between the third and sixth day post challenge. The rate of virus shedding was also higher in the unvaccinated calves.

TABLE NO. 2

GRAPH I. Pyremia Following Challenge in Vaccinates and Controls



•Significant differences between groups
*Probability level = 0.01
**Probability level = 0.05

In another group of calves an evaluation was made using the incidence and severity of lung lesions as a measure of the effect of the vaccine (Table No. 3). The number of lungs showing lesions and the severity of the lesions were reduced in vaccinated calves following challenge. Post mortem lesions were categorized as follows:

Normal—no lesions or only small focal areas.

Moderate—involvement of one to two lobes with consolidation.

Severe—Several lobes involved with consolidation, absessation, fibrin, edema, and emphysema.

TABLE NO. 3

GROSS SIGNS OF PNEUMONIA BETWEEN VACCINATES AND CONTROLS

Lungs	Vaccinates	Controls
Normal	13/17	5/14
Pneumonia	4/17	9/14
	(1/17 severe)	(4/14 severe)

Sweat at Nebraska has reported that calves infected with PI-3 virus fail to gain weight as well as noninfected calves. Woods, et al, reported that weight gains in calves vaccinated with inactivated vaccines were greater than unvaccinated controls. Our efforts to replicate this work are still in progress. We have limited data on six trials and complete information on two other groups. In order to acquire complete data on weight regain with feed efficiency, we have to own the cattle ourselves and feed them in our own facilities. It was discovered about halfway through the second trial that we had a pen effect in one of the four pens which will require more replications to produce statistically significant data.

In four groups of cattle we have in-weights and a subsequent weight measurement some 30 to 40 days later. With these measurements the average percent shrink is some one to two percent less in the vaccinated cattle.

We are continuing the study with complete periodic weigh periods and feed efficiency data to market. We feel we must determine the predictability of any weight advantage carrying through from starting the cattle on feed to market. From our data so far, it would appear that on the average, BAR-3 vaccinated cattle have a better accumulative average daily gain and require less feed per pound of gain than non-vaccinates. Certainly, the severity of an outbreak of respiratory disease and any mortality would influence these figures. To date our measurements have been made in very healthy cattle, not necessarily by design, I might add.

As for the vaccine control of disease outbreaks in the field, we now have completed some 17 trials involving approximately 9000 head of cattle with BAR-3. Our information to date can be summarized as follows:

1. Vaccination after arrival in the feedlot is usually unsuccessful in cattle obtained through auction channels. Blood samples taken at the feedlots in all of our trials consistently indicate exposure has already occurred. Exceptions to this would be cattle taken from one ranch directly to a feedlot.

2. BAR-3 has consistently produced a very high HI antibody increase following two injections.

3. The shipping fever syndrome as it is observed in the western feedlots has a many and varied probable etiology. In one trial where the calves were vaccinated on the ranch, a severe respiratory outbreak occurred and persisted when the cattle were moved to the feedlot. We were unable to isolate either PI-3 or Pasteurella from the animals in the sick pen.

4. The number of required treatments and the number of animals requiring retreatment are not always an accurate measure of vaccine effect. Individual interpretation as to a specific diagnosis makes this difficult As an example, BAR-3 is of little value in an outbreal of IBR.

5. A specific diagnosis is rarely determined in most feed lot respiratory problems. We know that IBR, BVD and PI-3 can initiate respiratory problems. Curren research indicates that the adenoviruses, Rhinoviruses Reoviruses, the Psittacoid group, and possibly the Mycoplasma group can potentially be involved in the Bovine Respiratory Complex.

6. At the present time a program of vaccination of calves on the ranch prior to sale for IBR, BVD, PI-3, and the *Pasteurella sp.* offers the soundest approach to control of the respiratory complex. I want to add we strongly feel that more economic evaluations will be necessary before the cattle industry accepts such a practice as routine.

In summary, research work has proven that the PI-3 virus is a significant disease-producing factor in the Bovine Respiratory Complex. I feel that at this time we have yet to conclusively document the full extent of its economic importance.

Thank you.

THE EFFECTS OF INTERNAL AND EXTERNAL PARASITES ON FEEDLOT PERFORMANCE

Dr. D. E. Howell
Entomology Department
Oklahoma State University

The arthropod factors in conditioning cattle for the feed lot are several, but they're not clear cut. The previous speakers have indicated that this is not a well worked out area—the same thing would apply here. First let's consider cattle grubs. The cattle grubs are essentially found throughout our entire cattle industry. Granted, there are two species involved, the northern and the southern heel fly or cattle grubs and for an extended period of time we have been trying to determine how much damage these particular insects are doing when they get into the animal. Many states have weight gain trials, many states have tried to demonstrate a difference in the grade as they leave the feed lot. The results aren't conclusive. This State has used some seven thousand animals in tests designed to evaluate the losses caused by cattle grubs.

In most cases the animals were treated before entering the feedlot with one of the systemic insecticides which provide almost complete protection against grubs. The tests were initiated from soon after the last heelfly eggs had hatched in April until late October as only the southern cattle grub was present.

Weights were obtained monthly during the period of the experiment and the gains of the treated animals were compared with those of similar untreated animals kept in the same lots.

Data accumulated here over a 12 year period indicate a gain of approximately 10 pounds per animal may be expected but these data are variable and at times treated groups showed less weight gain than the untreated checks. In general, the difference in weight gain between the treated and check groups is greater when the nutritional level is less desirable than under very favorable conditions. This was noted both in the pasture and the feed lot.

These data suggest that grub control usually pays. This seems more apparent when the test animals are slaughtered during the period the grubs normally appear in the back and the additional losses not measured by weight differential can be easily seen. Carcass trimming may be extensive when grubs are numerous and grade loss may occur.

The advantages of early treatment are apparent in the slaughter house. While grubs can be killed from time of penetration of the skin until shortly before they drop out to pupate, those killed after they have encepted in the back often remain in the cyst and reduce the carcass value. In addition early killing prevents the damage caused while the grubs are migrating through the body.

Time of treatment is important and may vary with locality, weather, parasite load and time of slaughter. The gains derived from early grub treatment should be compared with additional benefits due to control of other parasites such as hornflies, ticks, lice and mosquitoes. Early treatment for grubs in June may provide 2-4 weeks control of hornfiies, mosquitoes, and summer ticks, *Amblyomma americanum,* while in August populations of these parasites would be so low that control measures would be of little value. In late September of many years spray application of a systemic could be expected to control grubs, hornflies, mosquitoes, and greatly reduce winter infestations of lice and winter ticks. Another important factor is the time interval required between application of the pesticide and slaughter which may vary from 14-60 days depending on the systemic used.

Late application of systematic insecticides, after October, may result in some toxic reactions and should be avoided. In more southern areas the last application should be made by October 15, even though better louse and tick control would result from late October or November treatment.

Studies in Kansas indicated that an infestation of grubs provides considerable protection against an infestation the next year. Early treatment to control small grubs may result in much less protection against the next season's grubs than would have occurred if the grubs had been killed when they were ready for pupation.

The effect of movement of calves from one region to another on the cycle of the grub in the host is often asked. Cooperative work involving the experiment stations at Lethbridge, Alberta, Canada and Stillwater, Oklahoma indicated that grubs in cattle which were shipped from northern Oklahoma to Alberta Canada in late spring after the eggs had hatched and the larvae entered the

body of the calves, matured at approximately the same time as grubs from local cattle which had been exposed to very different external conditions. The Oklahoma *Hypoderma lineatum* grubs emerged over a longer period than the Canada grubs while the *H. bovis* grubs pupated very close to the same time. Adult females flies obtained from grubs of Oklahoma cattle sent to Canada laid eggs on Canadian cattle which produced larvae which pupated at almost the same time as grubs coming from eggs laid by flies from local Canadian cattle.

The effects of treating cattle for lice before going into the feedlot are not easily evaluated. Uniform populations normally are hard to find and it is difficult to obtain statistically valid data.

In general heavy populations of lice result in greatly reduced weight gains when compared with louse free animals and under such circumstances louse control pays big dividends. The economics of louse treatment of range animals depends to a considerable degree on the cost of bringing the animals to the treating areas and the ability of the animals to keep their louse populations at a low level. Insecticide treatment to control minimal louse populations just before the cattle enter the feedlot usually are worthwhile as louse populations tend to increase when the cattle are more crowded if carrier animals are present. Early treatment often makes later treatments unnecessary and the loss of 0.1—0.2 pound per animal per day that may be caused by heavy louse populations is avoided. If the very small number of animals which are very susceptible to louse attacks can be removed from the feedlot, a full season's control with one treatment in the fall is far more likely to occur.

The importance of the small filarid parasite *Stephanofilaria stilesi* which uses the hornfly as an intermediate host is not well understood. Recent surveys indicate that a high percentage of the animals show typical lesions which cause some irritation but the importance of these pests and the value of control is not clear. This parasite can be controlled by controlling its intermediate host, the hornfly.

Reports occur frequently of irritation and weight loss due to infestations with follicle mites *Demodex sp*. As with *Stephanofilaria* the importance of this pest is not clear nor are adequate control measures available.

Previous experience with temporary parasites such as stableflies, horseflies, or hornflies may reduce the annoyance caused by these pests when they are attacked at a later date. Animals which have been protected from stablefly or horsefly attack during the spring and early summer react much more to attacks in late summer than animals which have not been protected earlier. This may create a minor problem for the feedlot operator.

EXPERIMENTAL USE OF A NEW WORMING AGENT IN ARKANSAS CATTLE

Dan E. Goodwin, D. V. M.

Overgrazing and underfeeding encourage worm population explosions on many Arkansas farms. Successful concontrol programs are based on a recognition of those two very important factors. Control aids include properly administered worming agents such as copper sulphate, nicotine sulphate, tetrachloroethylene, lead arsenate and phenothiazine. To determine the effectiveness of thiabendazole*, a recently introduced worming agent, the following trials were conducted at Winrock Farms.

TRIAL NO. 1

On October 8, 1964, at Carlisle, Arkansas, a group of 66 Santa Gertrudis bulls in fair condition, varying in age from 7 to 13 months, were weighed individually after which they were paired with the animal having the nearest weight. A coin toss determined which animal of the pair would be alloted to the treatment group. The remaining animal was assigned to the control group. Identified fecal samples were collected from about half of each group as they passed through a work chute.

The experimental animals were drenched with thiabendazole at the rate of 3 grams per 100 lbs. body weight. The other animals served as untreated controls. The experimental animals subsequently received on December 11, 1964 and February 8, 1965, thiabendazole via feed at the rate of 3 grams/100 lbs. body weight.

Both groups were maintained together in the same dormant pasture (Lespedeza, Bahai grass, Dallisgrass) and received a wintering ration composed of 3 lbs. 41% cottonseed meal and about 50 lbs. sorghum silage. The trial was terminated on March 25, 1965 at which time individual weights were obtained. Fecal samples were again collected from the previously sampled animals.

RESULTS:

Ten of the younger, smaller animals were unable to compete with the older animals at the feed trough and were removed from the pasture before the experiment was concluded. (4 of these were from the treatment group and 6 were from the control group.) One other animal (control group) was removed from the experiment because of an incorrectly recorded initial weight. Data on the remaining 29 animals of the treatment group and the 26 animals of the control group follows:

*Thibenzole (R) Merck & Co., Inc.

TABLE NO. 1

	Treatment	Control
Average weight on 10-8-64	559.65 lbs.	571.10 lbs.
Average weight on 3-25-65	676.55 lbs.	630.00 lbs.
Average gain (168 days)	116.90 lbs.	58.90 lbs.
Average daily gain	.695 lbs.	.350 lbs.
Average number worm eggs per gram feces 10-8-64	659	896
Average number worm eggs per gram feces 3-25-65	217	390

Five of the control animals lost weight during the trial. All of the experimental animals gained weight. At the conclusion of the trial the animals that had received thiabendazole appeared to be thriftier and had more bloom than their control mates.

TRIAL NO. 2

On October 8, 1964, at Carlisle, Arkansas, 29 Hereford bulls in good condition, varying in age from 10-20 months, were weighed and alloted to treatment and control groups in the same manner described for Trial No. 1. Fecal samples were randomly collected from 15 of the 29 bulls to establish the degree of infection. As the samples were unidentified the same egg per gram count was assigned to each group. Thiabendazole was administered by drench at a dosage rate of 3 grams per 100 lbs. body weight to 15 experimental animals. The remaining 14 animals served as untreated controls. The bulls received a high energy grain ration in preparation for sale. Both groups were maintained together in a small trap. The experiment was terminated January 4, 1965, at which time final weights were recorded. Fecal samples were not obtained at the conclusion of the trial.

RESULTS:

TABLE NO. 2

	Treatment	Control
Average weight 10-8-64	802.33 lbs.	794.64 lbs.
Average weight 1-4-65	920.43 lbs.	875.07 lbs.
Average gain (88 days)	118.10 lbs.	80.43 lbs.
Average daily gain	1.342 lbs.	.914 lbs.
Average number worm eggs/gram feces 10-8-64	347 lbs.	347 lbs.

There was no obvious difference in the appearance of the two groups at the time the trial commenced or at its termination.

TRIAL NO. 3

On October 9, 1964, at Morrilton, Arkansas, 23 recently weaned 8 month old Santa Gertrudis heifers in good condition were weighed and alloted to groups in the same manner described for Trial No. 1. The treatment group was drenched on 10-9-64 with thiabendazole at a rate of 3 grams per 100 lbs. body weight and received via feed the same dose on January 8, 1965. Fecal samples were collected from half of each group as they passed through a work chute.

Both groups were maintained together in a dormant coastal bermuda grass pasture and received a ration composed of a gain test grain ration and sorghum silage. The trial was terminated on March 25, 1965 at which time final weights were recorded and fecal samples again collected from the previously sampled animals.

RESULTS:

Table No. 3

	Treatment	Control
Average weight 10-9-64	470.00 lbs.	464.54 lbs.
Average weight 3-25-65	693.00 lbs.	690.36 lbs.
Average gain	223.00 lbs.	225.82 lbs.
Average daily gain	1.23 lbs.	1.25 lbs.
Average number worm eggs/gram feces 10-9-64	850 lbs.	357 lbs.
Average number worm eggs/gram feces 3-25-65	119 lbs.	140 lbs.

The heifers were all in excellent condition at the end of the trial and no apparent differences in bloom were observed.

DISCUSSION:

The Santa Gertrudis and Hereford bulls that were located in the Eastern Arkansas Delta environment where internal parasites are generally considered to be more threatening, responded well to the drug regardless of ration or initial condition, whereas the Santa Gertrudis heifers in Western Arkansas, in good initial condition, receiving a good ration, derived no apparent benefit from thiabendazole.

It is generally thought that high energy grain ration possess some anti-parasite activity and that animals receiving adequate amounts of such rations will not suffer from parasitism to the same degree that underfed animals do. This was presumably confirmed in the Santa Gertrudis heifer trial where the treated well-fed group failed to outperform the untreated well-fed group. This philosophy is violated however when one considers the superior response of the well-fed Hereford bulls receiving thiabendazole. The Hereford bulls originated in Oklahome range country where parasites are not so bothersome—it may be that they had less resistance or immunity to internal parasites and were more vigorously attacked, the treatment group therefore deriving more benefit from medication than the comparable Santa Gertrudis heifer treatment group receiving the grain ration and perhaps possessing some natural resistance.

It is interesting to note that in the case of the Hereford bulls the response to treatment was not readily perceivable and without recorded weights would probably have gone unnoticed. This illustrates the insiduous nature of internal parasites.

Three hundred worm eggs per gram of feces is generally considered to reflect significant parasitism. The cattle groups in all of these trials then apparently harbored significant worm burdens.

In Trials 1 & 3 where both pre-treatment and post-treatment fecal egg counts were made, the treatment groups had a greater reduction in fecal egg count. The control group also enjoyed a reduction in egg count and in this regard it needs to be recognized that the control animals probably benefitted from a reduction in egg shedding by the experimental animals and that the experimental animals were being constantly reinfected by the larvae hatching from the eggs shed by their more heavily parasitized control mates.

In no instance did the animals refuse to eat thiabendazole pellets when poured on top of their regular ration. Ease of administration with apparent uniform intake avoided costly handling procedures.

(Grateful recognition is given Dr. Robert Kilgore, Fayetteville, Arkansas, Field Veterinarian, Merck & Co., Inc., whose contribution of knowledge and time materially aided the successful conduct of these trials.)

THE EFFECTS OF INTERNAL PARASITES ON FEEDLOT PERFORMANCE

Dr. Everett D. Besch
Department of Parasitology and Public Health
College of Veterinary Medicine
Oklahoma State University

Although this panel had to operate at long distance, there was an attempt to coordinate our efforts and present discussions that would be of interest to you. I will not talk about worms, but will discuss coccidia, small protozoan parasites that attack the lining of the small intestine of domesticated and wild animals. I am sure that much of what is said will be familiar to you, but it might be well to be repeated. Also, I will attempt to bring you up to date with some of the ideas and concepts concerning coccidiosis that have been developed recently.

Coccidia, microscopic protozan parasites, are single celled animals and are parasitic in both vertbrate and invertbrate hosts. They develop within cells of the host's body especially in the lining cells of the digestive tract. The species that infect the digestive tract of ruminant animals are known to occur in specific locations in the small intestines, cecum, colon or rectum. Some species invade the mucosal cells or lining cells of the intestinal tract proper, others may locate in the cells of the mucosal crypts while still others develop in submucosal areas of the intestine. Coccidia that have completed development pass from the destroyed cell into the contents of the intestine and then are passed from the host animal in the feces. These oocysts complete their development outside of the host. The infective stages (those stages that will invade host cells when infested) develop in oocysts deposited on pastures or on the ground in lots. Cattle are exposed to infection through ingestion of contaminated herbage or water.

Too many times we encounter the notion that all animals have parasites or spontaneously acquire parasites and must be routinely treated at the age of six weeks or eight weeks. Like all other animals, parasites begat parasites. Host animals must be exposed to infective stages usually in contaminated food and water in order for the nematode or protozoan parasites to gain entrance and develop within their bodies. The extent of pathology or damage that occurs from infection depends on the number of parasites in the infective dose, on the part of the digestive tract involved and on the extent of cellular destruction.

During the development process for coccidia, the host cells are invaded and usually are destroyed as are some of the cells that surround the invaded cell. Not only are the lining cells destroyed but also the underlying or submucosal areas of the intestines. This developmental pattern of the parasites involves an intra-host multiplication process.

The form of the parasite that is deposited in the fecal material in an unsporulated oocyst. This stage develops from sexual reproduction of the coccidia and when in the external environment undergoes development or sporulation. During this process spores or infective stages develop which when ingested by the cow will leave their protective shell and invade an intestinal cell. From one sporulated oocyst containing eight spores, after cellular multiplication in the host, there could be over a million unsporulated oocysts deposited in the fecal material. The details of this developmental pattern are familiar to many of you and are too complicated to discuss further. It is important to stress that intra-host or celluar multiplication process is an important aspect of the epidemiological definition of coccidiosis.

Infection of the host invokes the development of resistant which is not complete and which usually causes the infected animal to be a carrier. A carrier animal is infected but does not show signs of infection. Consequently, the carrier state is the most important factor in the ecology of the coccidiosis. Cattle that are carriers constantly shed oocysts and are the source of pasture contamination. We have found the coccidia in the majority of the bovine animals from Oklahoma that were examined by fecal examination. There are eleven species known to occur in cattle, three of which are pathogenic and of importance in the South Central Plains. An animal infected with coccidiosis, but with no apparent symptoms is said to have coccidiasis, a nonclinical condition. Coccidiosis is the term used to refer to a clinical infection or one that can be identified by symptoms. The former condition, coccidiasis, is very common in our cattle population while the latter condition, coccidiosis, occurs in a small percentage of the animals maintained on pasture or in a lot. Some of you may not agree with this statement if you have experienced an outbreak of coccidiosis in a particular group of animals. The incidence of clinical disease in the cattle population taken as a group is low and infrequently observed while the incidence of nonclinical disease, although difficult to define, is very high. This insiduous form of the disease is the

most important (as each animal is a carrier) has caused untold losses to the cattle industry and will continue to plague us until certain fundamental problems of the disease condition are defined. I do not believe there has been a recent estimation of the overall cost of this disease to the cattle industry.

It has been stated that the carrier animal is the source of pasture contamination and that the infective stages develop outside the host animal. Cattle on pasture are exposed gradually to a small number of these infective stages while grazing. The cow's body responds to these parasites and a resistance against infections begins to develop and will reach a level that is protective against clinical disease if this type of exposure is repeated frequently enough. However, exposure of the host involves a subsequent multiplication of the parasite within the host and which in turn results in a gradual increase in the number of oocysts that are deposited on the pasture area. As the number of infective stages increase on the pasture so does the number of chances for exposure to these stages per animal increase. Also, the number of infective stages that may be ingested at any one time increases. If this cycle continues long enough, more and more animals become infected, pasture contamination will increase to the extent that a brief exposure to the susceptible individual will result in clinical disease. This process of infection was illustrated in the line drawing patterned after the discussion by Boughton. As illustrated the most important animal in the infection process was the carrier or multiplier animal. It is evident that the calf that does not participate in earlier part of the pasture-host sequence does not develop a resistance. When this animal is exposed to a high level of pasture contamination, he may develop clinical disease and may even surcumb to the disease.

You may be wondering what this has to do with our subject of pre-conditioning of feedlot animals. The animal with a previous experience with those species of coccidia that are pathogenic will have a better chance of responding to the contamination associated with the feed lot. Young animals may have an increased susceptibility to disease due to the stress of weaning, to the stress of transportation, and to the stress of the lot conditons. These are the animals that break with clinical coccidiosis in a relatively short period of time after being put in the lot. Another situation can be identified. An animal can develop a resistance to the infection potential of his own environment, but may not have a protective resistance when he is exposed to greater numbers of infective stages that build up in a lot soil or to a contamination caused by different species of coccidia. The pre-exposure or the exposure of the animal prior to entering into the lot, may be of importance in respect to the animal's response once he comes into the highly contaminated premises of a feedlot condition. The ecology of the relationship that we see here, the intra- and inter-host-relationships are very complex. The contributing factors that may have an influence on this ecology need to be defined.

When we speak of resistance to a metazoan or protozoan parasite we refer to a relative state of protection. We use the terms resistance and immunity rather liberally. Immunity in disease is practically unheard of, but there is a few diseases to which a true immunity may develop. Immunity refers to a total absence of disease. Since this state is difficult to achieve, the term resistance is more acceptable to define the host's response to a given pathogen. Resistance to parasitism in a relative phenomenon, a state that is influenced or attenuated by a variety of factors. Most of these factors are associated with stress conditions. Diet can be a stress condition, weaning can be a stress condition, production, lactation, pregnancy, climatic or seasonal changes can be stress conditions. These can change the host response to a parasite; these can change the resistance of the host to a given parasite. The degree of change that may occur with the development of a stress condition or a combination of stress factors is difficult to assess. We have no means or mechanism or technique for assessing this change, consequently, the animal, who may be resistant to a pasture contamination situation, coming into a log may be exposed to a single or a variety of stress conditions that may actually decrease his resistance to the point of susceptibility. This animal when esposed to the infective stages of the pathogenic strain of coccidia could very really, and very likely break with coccidiosis, a clinical case of coccidiosis. The stability of this resistance to coccidiosis is not to well defined. Some of the work that has been done in chickens implies the level of resistance to coccidiosis is dependent upon the boostering effect of repeated exposures to sporulated oocysts. This has been interperted to mean that a chicken receiving sufficient numbers of infective stages to produce resistance but not disease can be caused to become susceptible if removed from the contaminated environment and placed in a clean environment. In other words, the loss of the boostering effect caused a decrease in the level of resistance to coccidiosis. We need to determine whether this situation applies to cattle. If it does it would be possible to produce a susceptible animal under feedlot conditions. Definition of the stability of resistance to coccidiosis in cattle may help to explain the clinical cases that occur in animals about to complete the feeding program.

The importance of the five to seven species of coccidia that occur in cattle other than those that have been classed as pathogens has to be determined. We need to

know what effect these species have on the pathogenticity or potentiation of the pathogenticity of the pathogenic species. We need to define some of the relationships that may occur between coccidia and helminth parasites. There is some work being done on this and the results that are being obtained are very interesting. Stomach worms or intestinal worms of cattle may potentiate the pathogenicity of the coccidial species. Much of what we know or hope to know about coccidiosis in cattle has resulted from study of experimental infections. During the course of these infections the size of the infective dose was defined, the resulting pathology was described and the response of the animal to challenge was elicited. Information was obtained from animals exposed to sporulated oocyst in a liquid inoculum and given by drench. Recently it has been shown that the manner by which the animal is exposed to these infective stages will influence the actual response that occurs. It has been determined that one of the best ways to produce coccidiosis in animals is to feed the sporulated oocysts in dry feed. Infections can be produced more consistently and with a lower number of sporulated oocysts in the inoculum. These results should be applicable to a lot where the dry feed would be available to the animal and contamination of the dry feed also occurs. As a result of these findings, we need to re-evaluate the whole problem of bovine coccidiosis in the United States.

FUNDAMENTAL CONCEPTS RELATIVE TO INTERNAL PARASITES AND THE CONDITIONING OF CALVES FOR THE FEEDLOT

Norman F. Baker, D.V.M.
School of Veterinary Medicine
University of California, Davis

As you are all aware, the term "internal parasites" includes many organisms, all of which are infectious but most of which are not contagious. These are not characteristics unique to pathogenic organisms considered as parasites, i. e. protozoa, helminths, and arthropods, but also are characteristic of many bacterial, viral, and fungal organisms. Further, the basic events which may lead to epidemic disease of parasite origin are identical to those of other disease agents. These basic events are (1) introduction of the causal organism into a susceptible population, (2) introduction of the causal organism into a susceptible population, (2) introduction of a susceptible population into an already infected (endemic) environment, (3) an increase in the infecting inoculaton of the organism in an already infected population, and (4) reduction of resistance of a population in which the causal organism is present (endemic). It is true that many specific biological characteristics of helminths (worms) differ markedly from those of individual viruses, but certainly by no greater degree than do those of certain bacteria. It is largely due to the failure to realize that worm "parasites" viral "parasites", bacterial "parasites" etc. all have these previously mentioned basic events in common that has set internal parasites and their control aside from other agents of disease.

Time does not allow discussion of the entire spectrum of internal parasites and as a result this discussion will be primarily concerned with nematodes (roundworms) of the gastrointestinal tract. The objective will be to present fundamental biological characteristics of the parasite, and the parasites relationship with the host, insofar as these may affect performance and or morbidity in the feedlot.

Fundamental to all such discussions of parasites is an understanding of the life cycle. For the most part those roundworms of the gastrointestinal tract which are the most important from the economical viewpoint, have similar life cycles. These life cycles are direct, and with few exceptions (*Strongyloides papillosus* and *Bunostonum phlebotomum*) passively enter the host via the mouth. The two exceptions may actively penetrate the skin. The life cycle is typically described as follows. The adult female and male located in the lumen, or on the mucosa, of the gastrointestinal tract mate and the female deposits fertile eggs. These eggs which are approximately 1/250 inch in length, pass out of the animal with the intestinal excrement. Within the egg, a first stage larva develops which hatches and actively feeds. Shortly, a moult takes place and with shedding of the old "skin" a second stage larva which also actively feeds is produced. A second moult takes place and the third stage, which is the infective larva results. This larva retains the "skin" of the second stage larva as a sheath around it and consequently does not feed although it may be quite active. The total time required to reach the infective stage is approximately seven days under ideal conditions. No further development occurs until the larva is ingested with food or water by the appropriate host. Subsequent to ingestion the sheath is cast off and the larva penetrates the mucosa of the gastrointestinal tract. Within the body there is growth followed by a third and fourth moult resulting in the early fifth stage, young adult worm. It is either as the fourth or fifth stage that the individual worm returns to the lumen and continues its development to maturity. The period from infection to passage of eggs is usually considered to approximate three weeks.

This is a generalized cycle and does not include many important biological properties of the group and or individual species of worms. For example, the entire development of *Nematodirus spp.*, a trichostrongyle of considerable importance in some areas, from germ cell to infective larva may take place within the egg. In some species the embryo within the egg may withstand many months of dessication, whereas the hatched first and second stage are soon killed by dessication. The infective larva is somewhat intermediate with respect to dessication. Further, there is considerable evidence, and speculation, to indicate that there are at least three, and possibly four routes of development which may be taken during larval development in the host. First, the larva may return to the lumen immediately after the third moult (first within the host). Second, the larva may return to the lumen immediately after the fourth moult (second within the host). Third, the larva may remain within the mucosa until it is a mature worm. Fourth, the larva may reach the early fourth stage and become arrested in it development. In this instance the larva may remain within the mucosa for several months before further develop

ment occurs. The practical importance of such varied development is apparent when it is realized that for the most part anthelmintics are only active against those individuals within the lumen.

It should further be noted that these parasites do not multiply within the host and thus for every individual parasite in the host, at least one, usually three or more, infective larvae must have been ingested.

With the foregoing background it will now be well to view the biological relationship in the form of a four stage infection cycle, figure 1.

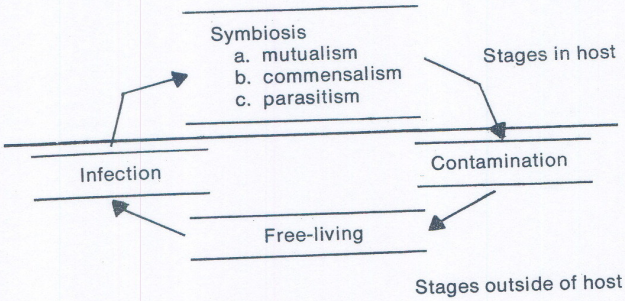


Figure 1. Infection cycle of gastrointestinal roundworms.

The four stages of the infection cycle consist of: (1) symbiosis, (2) contamination, (3) free-living, and (4) infection. This infection cycle is a dynamic one and the rate at which it "turns" will be determined by the rate-limiting stage. For example, if the host is immune to infection, there will be few worms present in the symbiotic stage and thus little contamination. In this instance the symbiotic stage would be rate limiting. If there was a high level of contamination, large number of eggs deposited per unit of area, but the climatic conditions were such that no development to the infective stage occurred the free-living stage would be rate limiting.

In figure 1, it is noted that within the symbiotic stage there are three divisions, namely mutualism, commensalism, and parasitism. These three types of animal associations may be defined as follows. Mutualism is that association wherein both species of animals in the symbiotic relationship are benefited. Commensalism is that state in which one animal species (helminth) is benefited while the other (cattle) is neither harmed or benefited. Parasitism is that association wherein one species (helminth) may be harmed or benefited, but wherein the other (cattle) is definitely harmed. In all biological associations there is change and one must realize that within the symbiotic relationship the association between species of parasites and their hosts may at different times assume any of the three forms. The objective of the veterinarian, and the livestock producer, is to recognize which form the association is in and predict which direction change is likely to take. This is in fact to say that the objective of all parasite control is directed at maintaining the association in a mutualistic or commensalistic form.

In order to discuss all aspects of the foregoing in relationship to parasites and their hosts would require a sizable volume. However, it should be noted that we are primarily concerned with animals entering feedlot. In this respect there are many of the foregoing fundamentals which offer immediate limits insofar as the infection cycle and consequently epidemic disease is concerned.

Under feedlot conditions there is little chance for free-living development of larvae and further even if there were, there would be little chance for infection via contamination of food or water. In addition, where fecal material is routinely removed the contamination stage is also greatly reduced. In essence then we can conclude that with rare exception, and for practical purposes, the infection cycle is inoperative under feedlot conditions. Further, as pertains to the basic events which may result in epidemic disease, we find that in only one instance, namely a reduction of resistance of a population in which the organism is already present, might an epidemic occur as the result of events within the feedlot. If then gastrointestinal parasitism is to be a problem in the feedlot, it can be concluded that the symbiotic relationship was one of parasitism, prior to movement into feedlot or changed from a mutualistic or commensalistic association to one of parasitism after entering feedlot. Let us then examine what can be done to diagnose, avoid, and control these latter two possibilities, particularly with reference to conditioning of calves for feedlot. In order to do this we first should differentiate two types of disease which have recently been described for *Ostertagia ostertagi*, the middle stomach worm of cattle. Type I disease is that resulting from a gradual accumulation of adult worms more or less fitting the pattern given in the previously discussed "typical" life cycle. In this instance, the onset of the disease in a herd is insidious with a small percentage of animals showing physical signs. As time progresses, and no change in management intercedes, increasing numbers show signs and the signs become increasingly apparent in individuals. This form of disease would be most common in young animals and it is during this "slow" transition from a commensalistic state to the parasitic state of symbiosis that the term subclinical parasitism has been so often mistakenly applied. Type II disease results from a build up of the previously mentioned arrested larvae with relatively few adult worms being present. While the larvae remain in the arrested state, the symbiotic association is usually commensalistic or

mutualistic. What factors are responsible for the arrestment of these larvae are not completely elucidated but may be endocrinological and immunological in origin. In any case, at some later date the "arresting factors" are removed and the accumulated larvae resume their development resulting in an explosive epidemic of disease. While this classification of disease is still restricted to the middle stomach worm there is much evidence to suggest that it applies to many parasitic roundworms.

From the conditioning standpoint, one may usually diagnose Type I disease by physical observation, history and the use of quantitative fecal examination. Physical observation should take particular note of such items as state of hair coat, food intake, constipation or diarrhea, alertness, and uniformity of condition of animals of common origin. The history should give particular attention to the origin and potential rate of the infection cycle from which the animals came. The cyclic rate will always be higher on irrigated pasture with high animal concentration than on dry rangelands with low animal concentration. Fecal examination should be conducted on a representative number of individual animals. If the animals are of common history and origin 15 to 20 individual samples will usually suffice. If the animals have been put together from several sources proportionately more samples will be needed. In Type I disease where physical signs are more or less questionable, parasite egg counts will vary widely from less than 50 eggs per gram to more than 500 eggs per gram. There need not be any correlation between the fecal condition (formed or diarrheal) and the high and low parasite egg counts. Where this condition is diagnosed most of the newer anthelmintics will give satisfactory results. Where the anmials have recently been removed from an environment where a high infection rate was occurring, a second treatment may be necessary. Such treatment should be administered during the conditioning period or shortly after the animals are placed in feedlot if maximal economic benefit is to be obtained.

The type II syndrome presents a much more difficult diagnostic problem and presently the only means of predicting its occurrence must be based on history and oriin. This is the case since these animals may show no physical evidence of the condition and all parasite egg counts may be low at the time they enter feedlot. In Scotland and Great Britain, where it was described, the accumulation of arrested larvae occurred after September. Of considerable concern relative to this type of disease is the evidence indicating that the presence of relatively small numbers of adult parasites may in some way be partially responsible for maintaining the larvae in the arrested state. Removal of these adults by "shotgun" use of anthelmintics may result in an explosive onset of the disease some two or three weeks after treatment. Obviously, we still have much to learn relative to this problem.

In the great majority of instances where the type I condition exists in a state of commensalism or mutualism and the animals are to be placed in feedlot, either on growing rations or fattening rations, no benefits will accrue from the use of anthelmintics.

In summation, we can say that internal parasites do play a role in the conditioning of calves for feedlot. An understanding of the biological relationship of parasite and host is essential if they are to be dealt with in the most economical manner. Internal parasites are fundamentally the same as other infectious noncontagious disease agents insofar as the occurrence of epidemic disease is concerned, and the feedlot is unique in offering conditions which allow interruption and control of the infection cycle.

A BRIEF RESUME OF BOVINE ANAPLASMOSIS AND ITS RELATION TO FEEDLOT CATTLE

E. W. Jones, D. V.M.
Veterinary Medicine & Surgery
Oklahoma State University

Anaplasmosis is an infectious anemia which was first recognized at the turn of this century. Despite this, it remains both an intriguing and troublesome disease. The infectious agent has not been isolated (Hruska, 1965), its development within the body has not been demonstrated (Brock, 1962; Jones and Brock, 1966), and its exact nature has not been sufficiently well defined to permit definite classification (Brock, 1962; Hruska, 1965; Jones and Brock, 1966). Resistance to this disease, unlike that of many diseases, diminishes with age (Jones and Brock, 1966; Jones et al, 1968) and, subsequent to infection, a state of premunition or latent infection exists (Brock, 1958; Jones and Brock, 1966; Jones et al, 1968). This persistant infection, which may be lifelong, provides protection from subsequent infection (Christensen, 1963; Henning, 1956; Mott, 1957; Ristic, 1960). The mechanism of resistance related both to age and to latent infection is undetermined (Jones and Brock, 1966; Jones et al, 1968).

Anaplasmosis has been reported from more than 40 states. However, the areas in which the incidence is highest are the Pacific Coast, the intermountain states, the lower Mississippi Valley, the Gulf Coast and the Atlantic Coast south of New Jersey (Christensen, 1963; Ristic, 1960).

During patent disease, anaplasma bodies are observed within the red blood cells (RBC). Once these cells are parasitized they are removed rapidly from the circulation, probably by the reticuloendothelial system (RES) (Baker et al, 1961; Jones and Brock, 1966). Removal of the infected RBC from the circulation may be facilitated by an autoimmune mechanism (Schroeder and Ristic, 1965). Despite incrimination of the RES in infected RBC clearance, the ultimate fate of the anaplasma organism and the manner by which other RBC are infected is unknown (Jones and Brock, 1966; Williams, 1966). The degree of the parasitemia varies markedly and is directly related to the age of the animal (Jones et al, 1968). More than 50% of the RBC may be invaded and subsequently destroyed in mature and aged cattle (Jones et al, 1968). Infected RBC appear to be destroyed rapidly, for the increasing parasitemia and developing anemia parallel each other closely, and in aged animals in particular, as many as 50 to 75% of the circulating RBC may be destroyed during a 5 to 10 day period (Jones et al, 1968). After the percentage of infected RBC has reached a peak, this declines often as rapidly as it increased. The duration of the parasitemia usually persists for 1 to 3 weeks (Jones et al, 1968). Longer periods have been observed in aged cattle, less resistant animals (Jones et al, 1968), and surprisingly, in some calves less than 2 to 3 months of age (Jones and Brock, 1966). In fact, in aged cattle, recrudescing parasitemia similar to that which is characteristic of splenectomized cattle, is observed (Jones et al, 1968).

Development of severe anemia causes increased RBC production which seems directly related to the intensity of the anemia and is usually maximal between the second and third week of patent disease (Jones et al, 1968). Evidence of accelerated RBC production is frequently inapparent in animals dying from acute anaplasmosis (Brock, 1958; Brock et al, 1959; Jones and Brock, 1966). If anaplasmosis induces more than 60 to 70% loss of the RBC, there is evidence of anoxic tissue damage (hepatic, renal, and myocardial) (Brock et al, 1959; Jones and Brock, 1966). The onset of convalescence is indicated by drinking, by return of the appetite and, in the blood, by evidence of increased RBC production. Convalescence usually requires 1 to 2 months, is short in calves and young adults and prolonged in aged animals and in calves infected shortly after birth (Jones and Brock, 1966; Jones et al, 1968).

Clinical findings in this disease result from the loss of the RBC and consequent anoxic tissue damage (Jones and Brock, 1966). Surprisingly, evidence of RBC loss is rarely apparent clinically until 40% or more of the RBC have been destroyed (Jones and Brock, 1966). Failure to recognize clinical anemia until 40 to 50% of the red cells are lost is probably responsible for the belief that anaplasmosis presents no problem in the young animal (Jones and Brock, 1966). Based upon clinical findings related to the degree of RBC loss, anaplasmosis can be classified as subclinical, mild, acute and severe. In subclinical anaplasmosis less than 40 to 50% of the red cells are destroyed; in mild clinical disease $>50\%$ of the red cells are destroyed and there are but transient clinical findings of 2 or 3 days duration; in acute clinical disease 60 to 70% of the red cells are destroyed and there is obvious pallor, loss of appetite and weakness; and in severe anaplasmosis $>70\%$ of the red cells are destroyed

and there is a marked pallor, weakness, even recumbancy, evidence of cardiac embarrassment and often death (Jones and Brock, 1966). In the feedlot, it is obvious that with the age group involved, the type of disease syndrome commonly encountered will be that which is either clinically inapparent or mild in nature. Acute and severe anaplasmosis are nevertheless occasionally observed in young adult cattle (Jones et al, 1968). At present there is little or no available information concerning the influence of anaplasmosis upon feedlot performance (Jones and Brock, 1966).

The diagnosis of anaplasmosis is facilitated by consideration of the geographic location, the season of the year, the age of the cattle in question as well as by clinical, laboratory and necropsy findings. Since the disease is transmitted predominantly by insect vectors it occurs more frequently during summer and fall. Accidental mechanical transmission, such as by surgical instruments or hypodermic needles, may however occur, in which case anaplasmosis occurs during any season (Jones and Brock, 1966). The incidence of clinical anaplasmosis is directly related to age; cases are therefore more usually observed in cattle 1 to 3 years of age or older and mortality is higher in mature and aged animals (Jones and Brock, 1966; Jones et al, 1968). Possible subclinical disease of the young should not be overlooked and can only be detected by examination of the blood. Common complaints by the owner of infected animals include fatalities, weakness, depression, loss of condition or production and occasionally pallor and jaundice (Jones and Brock, 1966). The most significant clinical findings are fever, pallor, weakness, constipation, normal colored urine and watery blood (Jones and Brock, 1966). Microscopic examination of the blood permits confirmation of the diagnosis and is essential for adequate prognosis. Evidence of an immune response can be detected by the complement fixation test and capillary agglutination test (Jones and Brock, 1966 and Jones et al, 1968).

Control of anaplasmosis necessitates consideration of the reservoir of infection, the mode of transmission, natural and acquired resistance and prophylactic therapy. The predominant reservoir of infection is the carrier or recovered animal (Jones and Brock, 1966). Such a reservoir assumes major significance to the individual assembling groups of animals from diverse locations, when the mixing of uninfected and carrier cattle easily results. In some regions, wild remunants may also serve as reservoirs of infection (Christensen, 1963). Since *A. marginale* survives for extended periods in certain ticks, these arthropods, likewise, act as a source of infection (Christensen, 1963; Henning, 1956; Howell, 1957; Piercy, 1956). Carrier cattle, of course, can be identified serologically by the complement fixation and capillary agglutination tests (Jones and Brock, 1966). Isolation of such animals is an effective means of control where transmission is by biting flies (Jones and Brock, 1966). Where ticks transmit the disease and wildlife reservoirs exist, control by isolation is less effective since pastures may be or may become contamination with vector ticks capable of infecting either deer or cattle (Christensen, 1963; Ristic, 1960). Once clinical disease occurs within a group of animals, a much more potent reservoir of infection exists; the blood of such an animal being highly infectious (Jones and Brock 1966).

Should exposure or potential exposure to anaplasmosis occur, prophylaxis can be achieved by the use of tetracyclines (0.3 to 0.5 mg. per lb. of body weight per day) in a feed or pasture supplement (Brock, 1959; Franklin et al 1962). As little as 0.1 mg. per lb. body weight as a feed supplement has been observed to prevent acute disease in infected cattle (Brock, 1959). Acute disease may, however, occur at the termination of therapy due to prolongation of the incubation period. It would seem therefore that a dose of tetracycline somewhere between 0.1 to 0.5 mg. per lb. body weight per day is adequate to prevent clinical disease in the feedlot. Alternatively 1 to 2 mg tetracycline per lb. body weight by intramuscular injection at 4 week intervals minimizes clinical disease and prevents death loss (Brock, 1964; Miller, 1962).

Despite predominant transmission by arthropod vectors the prophylaxis of anaplasmosis by vector control has met with but limited success (Roberts, 1962). Intensive control of biting insects by synergized pyrethrins may be beneficial (Anon., 1964). In addition to these control procedures, resistance comparable to that of the young bovine can be induced by the subcutaneous injection of a killed anaplasma antigen (vaccine) (Brock et al, 1965). The use of 2 doses, 6 or more weeks apart, is required. Development of maximal resistance requires approximately 9 months from the time of initial vaccination (Brock et al 1965).

In summary, the feedlot owner and his veterinarian will usually encounter subclinical or mild anaplasmosis. A syndrome, which although obscure, nevertheless produces significant red blood cell loss and may well impair performance in the feedlot. The owner should consider the localities from which animals have been shipped the hazard of mixing uninfected and carrier cattle, and therefore the need for prophylaxis.

REFERENCES

1. Anon.: Report of the Committee on Anaplasmosis. Proc. 68th U.S. Livestock San. A. (Oct., 1964): 112-114.
2. Baker, N. F., Osebold, J. W. and Christenson, J. F.: Erythrocyte Survival in Experimental Anaplasmosis. Am. J. Vet. Res., 22, (May, 1961): 590-596
3. Brock, W. E.: A Study of the Pathogenesis of the Anemia in Acute Anaplasmosis. Ph.D. Thesis, University of Oklahoma, May, 1958: 1-93.

4. Brock, W. E.: Anaplasmosis Control and Treatment. Oklahoma Vet. (Sept., 1959): 8-11.

5. Brock, W. E.: Recent Research on the Characteristics of the Etiologic Agent of Anaplasmosis. Proc. 4th Nat. Anaplasmosis Conf. (April, 1962): 11-13.

6. Brock, W. E., Stillwater, Okla.: Unpublished data, 1964.

7. Brock, W. E., Kliewer, I. O., and Pearson, C. C.: A Vaccine for Anaplasmosis. J.A.V.M.A., 147, (Nov., 1965): 948-951.

8. Brock, W. E., Pearson, C. C., Kliewer, I. O. and Jones, E. W.: The Relation of Treatment to Hematological Changes in Anaplasmosis. Proc. 63rd U.S. Livestock San. A., (Oct., 1959): 61-67.

9. Christensen, J. F.: Anaplasmosis. In *Diseases of Cattle.* Edited by Walter J Gibbons, 2nd ed. American Veterinary Publications, Inc., Santa Barbara, Calif., (1963): 655-665.

10. Franklin, T. E., Heck, F. C., and Huff, J. W.: A Review of the Treatment of Anaplasmosis. Proc. 4th Nat. Anaplasmosis Conf. (April, 1962): 50-53.

11. Henning, M. W.: Animal Diseases in South Africa. 3rd ed. Central News Agency Ltd., Johannesburg, South Africa (1956): 562-585.

12. Howell, D. E.: Transmission of Anaplasmosis by Arthropods. Proc. 3rd Nat. Res. Conf.: Anaplasmosis in Cattle (June, 1957): 14-16.

13. Hruska, J. C.: A Study of the Cryogenic Preservation and in Vitro Cultivation of Anaplasma Marginale. Ph.D. Thesis, Oklahoma State University, August, 1965.

14. Jones, E. W. and Brock, W. E.: Bovine Anaplasmosis: Its Diagnosis, Treatment, and Control J.A.V.M.A., 149, (Dec., 1966): 1624-1633.

15. Jones, E. W., Kliewer, I. O., Norman, B. B., and Brock, W. E.: Anaplasma Marginale Infection in Young and Aged Cattle. Am. J. Vet. Res., 29, (March, 1968).

16. Miller, J. G.: Protective Measures Against Anaplasmosis in Jamaica for Imported Animals. Proc. 4th Nat. Anaplasmosis Conf. (April, 1962): 49-50.

17. Mott, L. O.: The Nature of Anaplasmosis. Proc. 3rd Nat. Res. Conf.: Anaplasmosis in Cattle (June, 1957): 1-9.

18. Piercy, P. L.: Transmission of Anaplasmosis. Ann. New York Acad. Sci., 64, (July, 1956): 40-48.

19. Ristic, M.: Anaplasmosis. Advances Vet. Sci., 6, (1960): 111-192.

20. Roberts, R. H.: Control of Anaplasmosis Through Insect Control, Washington County, Mississippi, 1961. Proc. 4th Nat. Anaplasmosis Conf. (April, 1962): 85-86.

21. Schroeder, W. F. and Ristic, M.: Anaplasmosis. XVII. The Relation of Autoimmune Processes to Anemia. Am. J. Vet. Res., 26, (March, 1965): 239-245.

22. Williams, E. I.: Blood Transfusions in Anaplasmosis. M. S. Thesis, Oklahoma State University, July, 1966.

EFFECT OF STARVATION AND REFEEDING UPON RUMEN FUNCTION

Dr. R. L. Baldwin
Animal Husbandry Department
University of California, Davis

Ruminal changes that occur as a result of starvation of feeder calves prior to and during shipping, and the subsequent disruptions of rumen function that occur during refeeding, are cause for considerable concern. The most logical solution to the problem is to avoid it by not starving calves prior to shipping and by keeping shipping periods short enough such that severe ruminal changes do not occur. However, the feeder does not seem able to control these factors involved in sale and transport and, hence, will continue to be faced with the problem of refeeding starved calves for some time to come. Selection of the best refeeding program for a given lot of cattle is dependent upon knowledge of the condition of the animals and prior treatment, and the decision can only be made after the cattle arrive at the feedlot. This presentation is not, therefore, directed at suggesting a general refeeding program applicable to all types of cattle. Rather it describes the problem in the hope that the background presented will be helpful to feeders in deciding how to refeed specific lots of cattle.

Effects of Starvation

The rumen is a big fermentation vat in which many different types of bacteria and protozoa live together in a dynamic balance similar to the balance which exists in nature. Under normal conditions, the bacteria and protozoa break down feedstuffs to form products which the animal can use, thus providing a service to the animal. The animal, in turn, provides conditions in its rumen which assure microbial survival and the maintenance of an appropriate balance among various bacterial and protozoan species. One of the most important contributions the animal makes to the maintenance of a proper microbial balance is the periodic consumption of food, with the resultant release of nutrients necessary for microbial growth and survival. Many types of rumen microorganisms are very sensitive to the severe depletion of nutrients that occurs in the rumen during long periods of starvation. When the starvation period extends to 48 hours and the supply of nutrients in the rumen is essentially zero, many types of bacteria and protozoa die.

One of the most dramatic things first noted upon microscopic examination of rumen contents from animals starved for 48 hours or so, is the almost complete lack protozoa. Normally, at low magnification, one can fi 25-30 protozoa per microscopic field. Many times doze of fields can be examined without finding any protozoa rumen contents from starved animals, although son times 3-4 protozoa per field may be observed.

Based upon specific measurement of DNA - a measu ment directly related to bacterial numbers - we ha found that the concentration of bacteria per milliliter rumen contents is reduced to 25-50% of normal after st vation. Couple this observation with the fact that the v ume of rumen contents is reduced to less than one-h normal after 48 hours starvation, and it is obvious that number of bacteria in the rumen is reduced to 10-25% normal by starvation. This dramatic reduction in nu bers is significant in itself; however, a more import observation is that the surviving bacteria represent o a few of the many types of bacteria ordinarily prese In other words, the microbial death loss is different Reasonably large numbers of small cocci, including *Str tococcus bovis* remain after starvation, while very f large cocci and pleomorphic rods are observed. Th observations are important to a later discussion of lac acid production and utilization, because S. *bovis* i lactic-acid producer while the primary lactic-acid utiliz of the rumen, *Peptostreptococcus elsdenii* and the sele monads, are large cocci and rods, respectively. The sp chetes which are so obvious in normal rumen fluid and noted above, the protozoa, are almost entirely absent a starvation.

In addition to examining microscopically, the change the rumen microflora that occur during starvation, generally use quantitative criteria to assess changes fermentative activity and capacity. The effects of star tion upon two measures of fermentative capacity are p sented in table 1. It can be seen that, after 48 hours s vation, the capacity of the rumen microorganisms to ment added sugar is reduced to about 10% of normal. production in the presence of nonlimiting amounts energy nutrients (substrates) is similarly decreased a 48 hours. The apparent increase in fermentative capa after 24 hours starvation is a reflection of the fact t water passes from the rumen more quickly than do bacteria, resulting in higher bacterial numbers per liliter of rumen contents.

Gas production in the absence of added substrate is used as an index of actual microbial activity in the rumen. Fermentative activity is reduced to zero by starvation for 48 hours. This reflects the total depletion of nutrients in the rumen, which, in turn causes the dramatic decreases in protozoa and bacteria in the rumen, as discussed above.

The effects of starvation on the rumen are summarized in table 2.

Refeeding

The goals of any refeeding regime selected for starved animals are:

1. To provide maximum energy input to restore health and disease resistance.

2. To provide for rapid restoration of rumen function and rapid adjustment to growing or fattening ration.

3. To prevent rumen dysfunction.

Various refeeding techniques are employed in attempts to satisfy these objectives. One is to refeed a low-energy ration, such as a low-quality roughage. This technique generally satisfies the third objective, in that rumen dysfunctions are rarely encountered. However, such diets do not provided energy for restoration of animal health and disease resistance, and do not provide for rapid adjustment to growing and fattening rations. Other refeeding techniques involve the use of high-energy rations that are comprised of high-quality hay and concentrates in various combinations. These methods provide energy such that animals regain their strength and condition quickly and, if nothing goes wrong, they also provide for rapid restoration of rumen function and easy adjustment to growing and fattening rations. The problem is that sometimes something goes wrong and leads to rumen dysfunction. This problem can be directly attributed to the effects of starvation upon the rumen microflora. When we refeed a high-energy ration to an animal with an unbalanced rumen microbial population, it is reasonably easy to unbalance the rumen population further, with dire results, as discussed below.

The effects of refeeding hay and concentrate rations to animals starved for 48 hours are compared in table 3. Two criteria of adjustment are presented. One of these, glucose utilization, is an index of fermentative capacity, as discussed previously. Fermentative capacity was slowly restored in the animals refed hay until, after 50 hours of refeeding, it reached prestarvation levels. This does not mean that everything was normal again, but it does indicate development in the right direction. When the concentrate ration was refed, fermentative activity appeared to increase in a desirable fashion for the first 2 hours. Thereafter it decreased drastically, such that after 26 hours a number of animals were in severe trouble, and immediate steps had to be taken with several animals in order to save them. Hence, the 50-hour value, which indicates partial restoration of fermentative capacity in animals refed concentrate, reflects the results in animals that were not as severely affected. The most probable cause of the obvious rumen dysfunction observed in animals refed concentrate was the rapid buildup of lactic acid in the rumens of these cattle (table 3). Lactic acid is a strong acid and caused the p^H of the rumen contents to drop to about 4.5. At this p^H many rumen bacteria are killed, causing the microbial population to become more unbalanced. Under these conditions, lactic acid is absorbed by the animal more quickly than it can be utilized. This results in a buildup of lactic acid in the blood, and acidosis. Acidosis causes dehydration, loss of appetite, metabolic disorders and, if allowed to proceed unchecked, death. What causes this buildup of lactic acid in the rumen? The sudden availability of readily usable carbohydrates to a rumen microbial population unbalanced by starvation results in a rapid growth of microorganisms (*S. bovis* and others) which form D- and L-lactic acid (D and L represent two types of lactic acid which are formed and used at different rates, as indicated in table 4). These bacteria form lactic acid at such rapid rates that neither the other rumen microorganisms nor the animal can use or degrade the lactic acid fast enough (table 4). As a result, lactic acid builds up, the rumen microbial population becomes further unbalanced due to low p^H, fermentation stops, the animal experiences acidosis, and goes off feed. It is very difficult to bring these animals back to normal. It is often necessary to change diets or to transfuse affected animals with rumen fluids from normal animals in order to save them.

The data in table 5 indicate the relative effectiveness of three diets in reestablishing the rumen microbial population after 48 hours starvation. Both the high-roughage diet and the 55%-concentrate diet produced ruminal microflora with good fermentation characteristics, and total rumen microbial numbers were in the normal range (table 5). Of course, calves refed the roughage diet must still go through an adaptive period when switched to higher energy rations, while the animals fed the 55% concentrate ration are already fairly well adapted. When animals were refed the high-roughage diet with 300 mg of antibiotic per day, the development of the ruminal flora was retarded.

Table 1. Effect of Starvation on Fermentation

Period of starvation (hours)	Glucose utilization[1]/‡	Gas production	
		With substrate[1]/	Without substrate[2]/
2	100[3]/	100	100
24	115	140	70
48	10	15	0

1/ Fermentative capacity.

2/ Fermentative activity.

3/ Data expressed as percent of 2-hour values.

Table 2. Ruminal Changes During Starvation

1. Fermentative activity and capacity decrease to 10-15% of normal.
2. Rumen protozoa dramatically decreased often to essentially zero.
3. Rumen bacteria numbers decrease to 10-25% of normal.
4. Balance between microbial species disrupted by differential death loss.

Table 3. Effects of Refeeding Technique on Fermentation[1]/

Time after refeeding (hours)	GLUCOSE UTILIZATION[2]/		Lactic acid[3]/	
	Hay	Conc.	Hay	Conc.
0	10	5	0	0
2	80	55	10	25
26	60	10	15	142
50	100	70	12	55

1/Refed after 48 hours starvation.

2/ Values expressed relative to prestarvation capacity.

3/ umoles/ml.

Table 5. Effect of Refeeding Technique on Fermentation[1]/

Diet	Gas production		pH	DNA
	With substrate	Without substrate		
High roughage	65.0	21.0	6.7	0.32
55% concentrate	73.0	28.0	6.1	0.34
High roughage with antibiotic	43.0	3.8	6.9	0.24

1/ Refed 1 week after 48-hr starvation.

Table 4. Lactate Production and Utilization

Form of lactic acid	Production[1]/	Utilization	
	Rumen	Rumen	Animal
L	1090[2]/	46	450
D	410	34	35
DL	1500	52	—

1/ Maximum reported rate.

2 All values reported as umoles/hour for 150-kg steer.

FEEDER CATTLE ON HIGH GRAIN RATIONS

Dr. R. W. Dougherty
USDA-Agricultural Research Service
National Animal Disease Laboratory
Ames, Iowa

Mr. Chairman. There has been a lot said here about stress. I have undergone a lot of stress since leaving Des Moines. As some of you know, the state of Iowa is a very conservative state. When I got on the Braniff Airlines and saw some of those hostesses with their new uniforms—I don't know whether it is psychedelic or what it is—but, even if you are over forty years old, it has quite a traumatic effect. Last night at the Mixer, Dr. Gill told me they had sent a plane over to Tulsa for some of us and it did not land because the landing gear wouldn't come down. He hasn't told me yet whether this thing has come down or not. Then, to add to the trauma, Dr. Baldwin has covered my subject very ably and very thoroughly, so I don't know just what I will do from now on.

Another traumatic experience occurred when I talked with Dr. Harry Geyer at the AVMA meeting in Dallas. Dr. Geyer, who was a classmate of mine (if any of you want to know anything about his student days, I'll be very glad to get in touch with you), asked me if I would appear on this program, and I agreed to do so. He said it was just going to be a little informal group, talking back and forth. Then when I got the first program I noticed I wasn't on it and I thought, "Well, I have made it again!"; however, the next program had me on it. So if you have the first program, you can leave now; if you have the second one, you can put up with me. I haven't prepared anything formally because I thought it was going to be more of a discussion group and, as I said before, the previous speaker has given you much of the material that I had planned on giving you; but he has done it much more ably than I could.

My interest in this field dates back to 1948 (this is historical now). I was in a graduate exam the other day and found this graduate student had no conception of anything occurring beyond 1955—this was the "Dark Ages." However, I will risk going back a little into the "Dark Ages". Shortly after going to Cornell in 1948 Hurricane Hazel, or one of those violent ladies, swept through upstate New York and, as some of you know, in that older country there are many old apple orchards or apple trees in the pastures. The hurricane brought down apples in great quantities—these were little wormy apples, but the cattle ate them and enjoyed them and became toxic. I saw eighteen dairy cattle in one field staggering around in a depressed state. This was enough to create some interest in this condition. At that time, some of the clinicians believed that eating apples was related to apple cider, hard cider, alcohol, which was related to the staggering gait of the cattle. I think anyone acquainted with fermentation knows that distillery experts have quite a lot of trouble maintaining the correct bacterial population that will make ethanol efficiently. Certainly the mixed population in the rumen didn't seem quite logical to us as alcohol fermenters, so Dr. Cello, who is now in California and who was working with me, ran some of the material for ethanol, or alcohol, of any kind and found only traces of reducing substances.

Recently Dr. Allison, who is in our group, published a paper entitled "Ethanol Accumulation in the Rumen After Overfeeding with Readily Fermentable Carbohydrates". I want to read one little paragraph from this paper. I have great respect for Dr. Allison's work and for him too.

"Considering the acute nature of the overfeeding indigestion syndrome, and the absence of or low level of ethanol in the blood, it seems likely that ethanol did not contribute significantly to the symptoms of overfeeding observed in our experiments."

Notice, he uses the word "symptom", which I like—I'm a reactionary—and because some people want to call it signs, I still want to call it symptoms. Dr. Allison may be influenced some by my mal-thinking. There is a little ethanol formed, but apparently under these conditions we certainly wouldn't expect the alcohol accumulation to have much, if anything, to do with the symptoms of overfeeding.

In using the term overfeeding, I am talking about grain engorgement. We have produced this condition many times experimentally by putting cracked corn, wheat, rye grain—any type of grain—in the rumen through a fistula. I tried to force it down the esophagus by various methods, but gave that up a long time ago.

Readily fermentable carbohydrates are the important thing. The end products accumulate much more rapidly than they can be utilized by the bacteria or by the animal itself. Some of the very fine early work was done by Turner and Hodgets of Australia. Dr. Turner is one of the most able men I have ever met. I had a little experience working with him when I spent some time in Australia in 1956. This was a brief sojourn during my stay in New Zealand. I happened to have a brother in the foreign service in Australia who arranged a trip to Australia for

me. I landed at Melbourne while the Olympic Games were going on. I said to the fellow who met me at the airport, "Wouldn't it be a shame if I happened to be here and wouldn't be able to see just one little bit of the Olympic Games." He said, "Well, that is too bad, but the fellow you are going to see tomorrow morning, Dr. Turner, doesn't even know the games are on." I did get to see about a half a day of the 1956 Olympics.

Turner and Hodgets did some beautiful work. The early work was reported in the C.S.I.R.O. publications, which I knew nothing about at the time we started our work in 1949. I did spend a little time with Dr. Turner in his laboratory, and I even got to do a rumen experiment or two with him. The predominantly gram-negative microbial population shifts to the gram-positive type. Lactic acid accumulates rapidly. At that time Dr. Turner believed that the symptoms depended upon lactic acid formation and the accumulation of large amounts of lactic acid in the blood. This leads to changes in the alkali reserve and a lowering of rumen and blood p^H. Towards the end of his career (he retired several years ago) I think he was beginning to feel that the acidosis theory was not quite enough. I am not saying that the lactic acid picture is not important—it certainly is. But, in my mind, it doesn't quite answer the whole problem because you cannot reproduce it by administering lactic acid. At least I don't think the exact symptoms or signs have been reproduced by administering large amounts of D-lactic acid.

There is one other thing I would like to stress—you can see I am very well prepared. Dr. Baldwin has gone over the accumulation of lactic acid, the acidosis and the p^H. The p^H of rumen ingesta in our work has dropped from its normal 6.6 to 6.8, to about p^H of 4 or occasionally we could get it slightly below 4. Death occurs in some animals in 12 to 24 hours with practically no gross lesions. In the ones that go longer, you can see a rumenitis developing and some sluffing of the rumen epithelium. In the ones dying acutely, there is very little gross pathology or even histopathology.

Dr. Allison gave me these slides showing that the ruminal microflora is mainly gram-negative under normal conditions. These slides show a transition to a predominately gram-positive population after overfeeding a sheep with grain. More gram-positive organisms were seen 28 hours after feeding and during the next two hours there was a very marked increase in the proportion of gram-positive organisms and this change was coincident with a rapid drop in ruminal p^H. We have overfed cattle but have found that sheep are a lot less expensive because the mortality rate is pretty high. Dr. Bond and Dr. Ryan, graduate students of mine at Cornell, have worked on this problem. Dr. Dunlop, who is now at Saskatoon, used grain engorgement for his thesis problem. These theses are all available.

More recently, Drs. Mullenax, Keeler and Allison, in our own laboratory, took a rather unusual approach. They did some work where they took the predominently gram-negative population and using ultrasonic equipment destroyed the bacteria and isolated some of the endotoxins. They were able to produce almost the same symptoms or signs with endotoxin administration as are obtained when the animal is overfed with grain. This does not mean that the endotoxins have anything to do with the disease, but it is quite interesting. They hypothesized that as the gram-negative population decreased they broke up and endotoxins were liberated very rapidly. These would not be present in such large quantities under ordinary conditions of growth and death of the micro-organisms.

In this (this is a very old slide, about 1950), Drs. Hungate and Bryant (these are two pretty prominent names in the microbiology field) worked with us at Cornell for a year. At that time we were using wheat largely as the overfeeding grain. One day Dr. Hungate suggested that if we would grind the wheat real fine we would make it more available for the bacteria. We had it pulverized in a very fine hammer mill. The results were disappointing. The very finely comminuted material was probably going down into the lower tract too rapidly for the lactic acid fermentation to occur. This is one thing that we learned the hard way. We use the fistula method for putting the grain in the rumen—I like this picture because this young man was a student at the Veterinary School at Cornell at that time and worked for me odd hours, and apparently he was the very sensitive type—you can tell by the expression on his face. Just to show you what a wonderful influence I had on him, he became a cardiovascular physiologist and he is now a very prominent one. This apparently cured him of working with the digestive tract.

I am presenting these statements semi-factitiously, but not entirely. I know the lactic acid story is sufficient for some people. I would like to say, as Mark Twain once said, "Difference of opinion is what makes horse races." Dr. Baldwin and I have a slight difference of opinion here, but it does not mean that mine is right. A number of years ago, Dr. Akin asked me to write a little review to renew the interest in bloat, and at the end of it I put a couple of paragraphs from the poem about the blind man and the elephant.

"And so these men of Indostan
  Disputed loud and long
Each in his own opinion
  Though each was partly right,
And all were in the wrong."

"So oft in theologic wars,
The disputants, I ween,
Wail on in utter ignorance
Of what each mean,
And prate about an elephant,
Not one of them has seen!"

The moral:

That is just the way I felt and I have felt that way ever since about problems with which I have worked.

Normal ingesta will depress the dog's blood pressure but not as rapidly as the ingesta obtained from a sick animal. We used about 200 dogs in this work. With the isolated guinea pig ileum we got a depression of activity and lengthening of the gut. Of course, this is not a histamine reaction—histamine would shorten the gut. If we injected rumen fluid from a normal animal intravenously into a sheep, we could partially depress rumen activity, but if we used the ingesta from a sick animal we could depress it more.

I don't know what I have missed. I am sure I have missed a good many things, but I have one or two statements to make in winding this up. Dr. Crenshaw made a statement this morning saying that we need to know more about ruminant physiology. I certainly endorse this heartily. I have been in this business for thirty-some years; the progress has been disappointing. But when I started with this work in 1936 you could count the people working full time in this field on the fingers of one hand and have a few fingers left over. I don't know how many of you remember that—I do. In 1964 we had a symposium at Ames and there were 630 present, some from foreign countries, but at that it represented only a small proportion of the total number of people interested in this type of work. Research workers are increasing in numbers, and if the grants can hold up we may do better than the blind men with the elephant.

We have an environmental facility at Ames in the National Animal Disease Laboratory which has been extremely disappointing in its mechanical operation. We have re-engineered it and we know how much it will cost to rebuild it into an effective unit. All we need now is a special appropriation from Congress. I believe that we can answer many questions better with this facility. I heard shipping fever discussed this morning. More progress will be made with a well controlled environmental facility where you can control wind velocity, light, temperature, humidity and altitude. On the overfeeding problem we are just now starting some experiments again; I hope to get back into it.

I would like to make one statement, and this is probably the only statement I will make that is worth remembering. In grain engorgement it is a matter of intake. In the beginning I was so enamoured with my veterinary training, which has some advantages and probably some disadvantages, that I was looking for an immunity or some type of resistance. I would like to tell a little anecdote on myself. We have a small acreage and about 35 Suffolk ewes. A couple of years ago my little daughter, who likes to feed the lambs with a bottle, had a couple of lambs on this regime. She enjoyed this so much that the lambs were getting pretty good size and were still on the bottle. They were also getting liberal amounts of grain. Finally I said this bottle business has got to stop—I can't afford this, not even on my salary, which leaves something to be desired. We put one of them down with the other lambs that were creeping, but still nursing. In about 15 hours this animal was dead and the rumen was simply packed with grain, a mixture of corn and oats. I thought I would repeat this experiment; it's pretty hard when it comes out of your own pocketbook, but it is a little more impressive than when you do it on grant money. I put the other one in the creep and the same thing happened. I decided that this probably was a good experiment, but quite expensive. These lambs were used to grain—quite a bit of grain in their diet. When they went into the creep situation with the rest of them, all they had to do was stand there and eat grain; the bottle had been removed and there were no distractions or interruptions—in other words, total intake of grain was responsible for the symptoms and death. I do not think that these animals had developed any resistance; I think it is mostly a shift in the microflora situation. I think, Dr. Baldwin, that there is a lot to do yet to increase our understanding of this shift from gram-negative to gram-positive microflora in animals placed on a high grain ration. I have heard some of the stories, but I doubt if we know the whole story. Intake and appetite are very important in the overeating syndrome. I think this is one place where we have to do much more investigational work. Thank you.

NUTRITION OF RUMINANTS AS AFFECTED BY STRESS

Dr. D. C. Church
Department of Animal Science
Oregon State University

The well-fed calf (or lamb) is generally thought to be less subject to infectious disease and metabolic disturbances than one receiving an inadequate nutrient supply. With present-day production methods, transportation of cattle over long distances, and the economic need for rapid and efficient production, the importance of maintaining the young animal in an adequate nutritional state is apparent. Death of animals and loss of production developing from stresses applied during transportation and adaptation to a new environment in the feedlot can be, and probably frequently is, the difference between profit and loss to many feeding operations.

Nutrition in Stressing Situations

The nutrition and feeding of ruminants during periods of stress has been investigated extensively for only a relatively few situations. In the case of short-term stressing situations, data of a quantitative nature are much less readily available and are primarily restricted to such situations as energy expenditure during exposure to different temperatures, water restriction, starvation, and as related to the prevention of such maladies as grass tetany, acidosis, ketosis, and bloat.

Thermal Stress: The influence of thermal stress—either hot or cold—is pertinent to this discussion, since it may be a factor in transportation or during adaptation to a new situation in the feedlot. A brief review of relatively recent papers on both sheep and cattle follows.

It is well known that a change in environmental temperature will alter the energy requirements of an animal, however, the extent to which requirements will be modified is dependant upon several factors. One of the primary factors is the amount of heat loss as influenced by body covering, coat color, and amount of adipose tissue. Balxter *et al.* (1959) demonstrated very clearly that the length of fleece of sheep was inversely related to the heat production required to maintain body temperature; or to put it another way, the critical temperature (that temperature at which nutrients must be oxidized to maintain body temperature) was higher in animals with closely clipped fleeces. Presumably the same situation would apply to cattle, although to a lesser extent since hair length varies less than wool length. Cattle do have some ability to increase effective length of hair by erecting the hairs and thus providing more insulation.

The effect of wetting the fleece on heat loss has bee studied by Alexander (1958). When the heat productio of dry lambs was measured in still air, they produce 2,000 kcal./m.²/24 hr. When the fleece was wet and th lambs exposed to a wind of 10 m.p.h., an additiona 1,812 kcal./m.²/24 hr. of energy was required to main tain body temperature in lambs with fine, short coat and 998 kcal./m.²/24 hr. in lambs with coarse, long coat Other data of this sort are lacking, but this one pape illustrates the tremendous heat loss that can result whe the body is exposed to wind with the result that insula ing properties of the fleece were reduced.

The amount of adipose tissue (fat) carried by the anim also affects heat loss. Lambourne and Reardon (196 obtained a partial answer on this aspect by using adu wethers of "equal size but a varying degree of fatness When these sheep were kept in metabolism pens indoor liveweights of 25, 32, and 47 kg. could be maintained o a daily intake of approximately 200, 300, and 420 g. digestible organic matter. When the metabolism pe were placed outdoors in winter, the maintenance requir ments rose, the rise appearing to be greatest for the th sheep.

A second major factor affecting the critical temperatu is the level of feeding. Blaxter *et al.* (1959) and Blaxt and Waiman (1961) have demonstrated with sheep ar cattle, respectively, that an increased level of feedi resulted in a reduction of their critical temperature. the case of two steers at the maintenance level of feedin the critical temperature ranged from 5.7° to 6.8° C. T values during fasting were 17.8° and 18.4° C. The d ference between the two being due primarily to the pr duction of more heat from rumen fermentation and oth exergonic (heat producing) reactions occurring in t tissues of steers fed at the maintenance level. When t steers were fed to gain about 1 pound per day, the critic temperature was further reduced to −1° C. An increasi plane of nutrition will also result in a reduction in he tolerance when temperatures are on the high side (Rog son, 1960; Yeates, 1956; Soderquist and Knox, 196 ultimately resulting in a reduced feed intake and a red tion in performance.

Effect of thermal stress on metabolism or nutrient quirements: In an interesting study with goats Applem and Delouche (1958) observed that a decrease in

vironmental temperature from 20° to 0° C. resulted in an increase in time spent consuming roughage; a slight decrease in water consumption, drinking frequency and time spent drinking; a considerable increase in remastication rate; and an increase in aggressiveness, time spent standing and in movement. Temperatures increased from 20° to 40° C. resulted in a decrease in time spent drinking; a decrease in remastication rate, aggressiveness, time spent standing and in movement. Temperatures between 35° and 40° where believed to be the point at which the heat was causing stress. In warm weather Rainey *et al.* (1967) reported that low roughage-fed cows produced significantly more fat-corrected-milk than moderately high roughage-fed cows, although the rations did not result in any difference in milk composition. Rea and Ross (1961) carried out lamb feeding trials in the summer and winter. Lambs fed in the summer gained more when fed rations containing 60% concentrate as compared to rations with 40% concentrate, whereas there was no difference in lambs fed during the winter. When lambs were maintained in metabolism studies at 40° or 80° F., differences in digestibility for the two rations in favor of the 60% concentrate ration were greater at 80° F. than at 40° F. Another paper along this line is that of Soderquist and Knox (1967) who studied energy retention at temperatures of 0°, 23°, or 35° C. with lambs subjected to these temperatures for 50 days. The high temperature resulted in a reduced feed intake, increased water consumption, a marked reduction in N retention and a negative energy balance. Animals exposed to the high temperature produced four times as much methane as those in the cold, and twice that produced at the intermediate temperature. The increased methane production may be a reflection of less acidic conditions in the rumen, since methane production is reduced as the rumen pH drops.

The author is not aware of published data adequately showing changing preferences for roughage vs. concentrates as the temperature increases. It would be interesting to determine if voluntary consumption of roughage would decrease with rising temperatures. The data of Appleman and DeLouche (1958) indicates a decline in roughage consumption as the temperature went up, based on eating time. They stated that grain (1 lb. fed/day) was rapidly consumed up to a temperature of 35°. A decline in roughage consumption might be anticipated in view of the known facts that roughage gives rise to greater relative quantities of acetic acid during rumen fermentation than do concentrates, and in view of the fact that acetic acid tends to result in a higher heat increment when metabolized in the tissues at intakes above maintenance. Weldy *et al.* (1962) studied the volatile fatty acid production of Holstein or Hereford cows kept in environmental chambers at 70° and 90° F. as compared to pairmates housed under ambient conditions (40-78° F.). Total VFA production was not lower in the chamber groups, however, acetic acid concentration was less in both breeds. Gengler *et al.* (1967) also demonstrated that increasing ambient temperature from 18° to 35° C. or the application of a heated coil in the rumen resulted in reduced VFA production. In the case of dairy cows studied under tropical conditions, Tsai *et al.* (1967) reported that feeding a low-fiber ration (12.5%) resulted in an increase in the production of fat-corrected milk and in concentration of ruminal propionic acid; decreased milk fat percentage, rectal temperature, pulse rate, respiration rate and percent ruminal acetic acid as compared to feeding a ration with 18.5% fiber. Cock *et al.* (1966) reported that urea-containing rations resulted in a lower heat increment, and in the case of rations having 5-20% crude protein, that the highest heat increments were from the rations with 15 and 10% protein.

Graham and Serle (1966) reported that pregnant ewes had higher levels of plasma urea and creatinine at 35° than at 20° C., but differences in urinary excretion were not apparent; and Christian and Williams (1960) reported data that indicated that Mg requirements of sheep exposed to inclement weather were higher than those for protected animals. Kondos and McClymont (1966) reported that cold stress (4° C. for 18 hrs.) increased the toxicity of CC_{14} in newly shorn sheep in medium condition. Unshorn sheep were less susceptible. Sheep in poor condition were most susceptible. Heat stress (40° C. and 55-60% relative humidity for 12 hrs.) rendered the drug less toxic, probably due to increased elimination of CC_{14} immediately after administration.

There have been several reports related to the effect of solar radiation stress on cattle. Rainey *et al.* (1967) found that cows protected by shade had lower body temperatures and respiration rates than unprotected cows, but there was no effect when low and moderate levels of roughage were fed. Williams and others (1960) reported that solar radiation did not increase body temperatures when the temperature was above 90° F., although the respiration rate was increased. Perhaps this is explained by the data of Murray (1966) who reported that evaporation from the skin of cattle under field conditions was almost twice that observed when similar temperatures were imposed under laboratory conditions. This was believed to be due to the direction action of solar radiation in stimulating activity of sweat glands. Hair color would appear to be an important factor since Riemerschmid and Elder (1945) reported that white hair reflected almost twice as much heat as black hair. Perhaps black-haired animals sweat more to counteract this absorp-

tion; if not, it would seem that they should be less tolerant of hot climates.

With respect to other environmental variables, Shrode and co-workers (1960) investigated the effect of air temperature, wind velocity, solar radiation, and vapor pressure on cattle. From a practical point of view, they concluded that air temperature was by far the most important variable of those studied. Riggs (1966) has recently reported data obtained on steer calves fattened in six locations differing in altitude (976 m. difference), in rainfall (889 mm. difference), relative humidity (16% difference), mean temperature (9° C. difference) and with variations in wind movement. Differences in rectal temperature, pulse rate and respiration rates were observed. Atmospheric temperature was significantly correlated with respiration rate and with body temperature. Wind movement appeared to have a greater effect at the warmer locations, however, gain of cattle at the three lower, wetter and warmer locations was almost identical to that of the three higher, drier and cooler locations. The same was true for feed and water intake and feed efficiency. Carcass characteristics were almost identical at all locations. He concluded that the difference in stress did not cause any difference in productive performance and one would, therefore, conclude that no differences occurred in nutrient utilization.

Thompson *et al.* (1963) found that thyroxine secretion and turnover rates and PBI values decreased under hot conditions (75-95° F.) in Holstein heifers. Blood plasma levels of 17-hydroxycorticocosterone increased and daily body gain and feed consumption decreased (1.8 to 1.1 lb./day and 15.7 and 14.7 lb./day, respectively) as compared to cool temperatures (38-65° F.). Heat production, respiration rate, and rectal temperature rose following initial exposure to heat, then declined on continued exposure, apparently as a result of compensatory adjustments brought about by altered thyroid and adrenal cortical function. Yousef and Johnson (1966a) injected thyroxine into Holstein cows kept at 18 or 32° C. and found an increased "resting or standing" metabolism, pulse rate and lactation. The latent period was 2-3 days and 1 to 2 days at 18 and 32°, respectively. Data suggested that thyroxine metabolism decreases progressively under hot conditions. The high temperature decreased pulse rate, oxygen consumption, milk production and feed intake and increased rectal temperature and respiration rate, indicating a serious disturbance in the cow's thermal equilibrium. These authors (1966b) also studied the effect of injecting growth hormone. As with thyroxine, the injection of GH at 18 and 32° C. increased resting metabolism and pulse rate. GH increased thyroid activity and decreased the respiratory quotient. Results suggest that the calorigenic action of GH may be due to a synergetic reaction with thyroxine or to the increase in fa metabolism (indicated by a decreased RQ).

Conclusions on effect of thermal and other environmenta stresses on nutrient metabolism: Available data, althoug limited in many respects, indicates that a thin, poorly fed animal is apt to be more severely affected by col environmental temperatures, particularly if its body i wet and exposed to a wind. Such an animal also ha fewer reserves to call on, consequently, it cannot surviv conditions that would affect a well-fed animal very littl In the case of well-fed animals, hot temperatures wi result in reduced production and heat stress which doe not appear to be markedly increased by radiation, win or humidity. A limited amount of data indicate that th heat stress might be alleviated somewhat by reducin the amount of fiber in the ration and by substituting ure for native protein. Heat stress tends to reduce feed intak as well as energy and nitrogen retention and gain c milk production. There may be a minor effect on volatil acid production in the rumen. The requirement of M may be greater during adverse (cold, wet) weathe Limited data indicate that CC_{14}, used for treatment o fukes, is more toxic in a cold environment, and data o hormones such as thyroxine, indicate that administratio of such compounds would tend to accentuate heat stres

Conclusion

Restricted feed intake: Restricted intake of feed durin transportation and subsequent adaptation to a new en vironment is probably one of the more important stresse imposed on feeder cattle. Consequently, it is pertinent t look briefly at some of the research in this area sinc data on nutrient metabolism may be useful in determi ing what nutrients are needed for repletion followin feed restriction. It is assumed, of course, that energy the most limiting factor in these situations.

In the case of ruminants, fortunately, food reservoirs i the reticulo-rumen should tend to delay the onset starvation as compared to monogastric species; howeve it is also possible that recovery may be delayed whi rumen organisms are returning to normal.

Hyden (1961) reports that salivery secretion is reduce in animals deprived of food, thus this would tend to co serve those minerals secreted in saliva. With respect rumen microorganisms, Warner (1962) found that tl numbers decline. The protozoa (*Entodenia spp.*) disa peared as did *Oscillospira* bacteria in sheep starved days, and that it took 2-3 days for the numbers of bacter to return to normal after refeeding was begun. Nes (1961) also noted that protozoa disappeared and th rumen motility was less frequent and vigorous. Meis *et al.* (1958) found that starvation resulted in an increa

in rumen pH and that the ability of rumen fluid to digest cellulose decreased greatly when a steer was starved for 3 days; 3-4 days were required for cellulolytic activity to return to normal after feeding was resumed. Juhasz (1962) also reported that rumen samples of starved cows and sheep increased in pH and ammonia concentration, and Robertson and Thin (1953) observed that it required 6 days for rumen VFA concentrations to return to prefasting levels.

In the case of calves starved 4 days, Blaxter and Wood (1951) noted a constant fall in metabolism. Energy and nitrogen balance were negative and the urinary excretion was increased markedly for urea, creatine and uric-acid. Sulfur excretion also increased, although Cl, K, Na and C excretion decreased and there was no evidence of acidosis or ketonuria during starvation; a slight ketosis during realimentation was a constant symptom, however. In a longer term study, Meyer and others (1962) concluded that supplemental protein was most helpful in relieving the stress of partial starvation in sheep fed poor quality roughage.

Weeth *et al.* (1959) found that snowbound, pregnant ewes developed visible symptoms of ketosis and a pronounced eosinopenia. Recovery was prompt in most ewes when emergency feed was supplied, however, some ewes required force feeding with soluble carbohydrate before they would eat. Most ewes would not eat a ration of 15% tallow and 85% grass hay following starvation, although this feed was consumed when offered before fasting. Procos (1962) found no effect of fasting on total ketone levels in wethers although there were some differences in individual ketones. In starved lactating cows, Robertson *et al.* (1960) reported that Ca dropped until milk fever developed. Blood Mg dropped and took some time to return to normal after refeeding while P was unchanged. Blood glucose increased from 35-40 mg.% prefast to 60-70 in non-milk fever cows and went up to 160 mg.% with milk fever. Lactic acid increased as did pyruvic acid and total ketones (from 1.2 to 56 mg.%). Saba *et al.* (1966) reported that fasting of ewes for 6 days caused plasma ketones and non-esterified fatty acids to increase. Blood glucose decreased along with insulin activities, but blood was not affected. Williams and Christian (1959) found that decreasing food intake from 1000 to 300g. for dry, nonpregnant ewes caused no significant change in blood sugar, ketones, animo acid nitrogen or nonprotein nitrogen, P, Ca, Mg, or hemoglobin. Jensen *et al.* (1954) found that five days of starvation produced only slight changes in concentration of major inorganic ions in the plasma, although there was a marked reduction in urinary excretion of cations and carbonate and an increased excretion of phosphate. The acid-base balance of the blood was not distorted by starvation or thermal stress, although ketones in plasma and urine increased upon starvation. In pregnant ewes, starvation tends to result in an increased level of liver fat (Ferguson, 1954; Wright, 1955).

In the case of young calves (0-19 days of age) deprived of both milk and water, Dalton (1966) observed that the urine became progressively more concentrated, although there was no significant increase in mean osmolality of blood plasma.

Johnson and Yousef (1966) found that fasting for 8 hrs. by dairy cows caused a significant increase in the disappearance rate of I^{131}-labeled throyoxine, but fasting for 24 hrs. did not result in differences from normally-fed cows.

Hight and Barton (1965) found that partial starvation reduced the content of muscle protein and fat in the leg, loin and rib of sheep carcasses. Prolonged under-nutrition reduced the weight of the heart. Suzuki (1965) reported that the percentage of muscle declined in starved animals, with the loss being greater in muscles of the better cuts. Starvation reduced diameter of muscle fibers and the glycogen content of the muscle.

Conclusions regarding restricted feed intake on Nutrient Metabolism: This very brief review of literature on this subject clearly indicates that a total lack of feed for a few days results in a rapid loss of normal celluloytic activity by rumen microorganisms and, presumably, the loss of other normal functions as well. Protozoa rapidly disappear from the reticulo-rumen, and rumen motility becomes less frequent and less vigorous. Rumen pH tends to increase as does the ammonia concentration.

Limited data indicate a rapid fall in metabolism of the young animal when fasting. Energy and nitrogen balance are negative, and urinary excretion is increased for those N-containing compounds found in cellular protein. Acidosis and ketosis do not seem to be pronounced problems in animals other than pregnant or lactating females. Blood glucose and tissue glycogne may be expected to decline rapidly. In the case of minerals, limited data indicate that tissue levels of Mg, and possibly P, may decline appreciably.

Following feed restriction or starvation, data would indicate supplementation with a readily consumed source of energy (high fat or high starch sources not recommended), a liberal allowance of protein, and of Mg and P.

Effect of Water Restriction.

Factors affecting water requirements: As Winchester and Morris (1956) clearly point out, water intake is a function of dry matter consumption and ambient tempera-

ture, with other environmental and physiological factors having lesser effects. Data reported by Riggs *et al.* (1953) and McIlvain (1953) indicate that salt added to protein supplements may increase water consumption from 22-100% over no salt addition, and Kelly *et al.* (1955) found that salt added to a ration increased water intake 40-60%. Ritzman and Benedict (1924) found that steers on high protein allowances consumed 26% more water than similar animals on low protein rations.

Relative humidity has a negligible effect on water consumption at temperatures below 75° F. (Ragsdale *et al.*, 1953) and wind up to 9 m.p.h. did not influence water intake in dairy cattle (Brody *et al.*, 1954). Ittner and others (1951) found that providing cooled water (65° vs. 88° F.) to feedlot cattle resulted in a reduced intake of water and a greater daily gain in Hereford cattle when the maximum mean temperature was 100.5° F. At colder temperatures, Bailey *et al.* (1962) found that a reduction in environmental temperature from 15° to −12° C. caused a reduced water intake in sheep from about 1600 to 800 ml./day. At an environmental temperature of −12° C. the temperature of the drinking water (0 to 30° C.) did not influence the amount of water consumed. Williams (1959) studied the water intake in feedlot steers when temperatures ranged from −35° to 45° F. He found that water intake was significantly correlated with air temperature on the same day with feed intake on the previous day. When ambient temperatures are moderate, cattle tend to do most of their drinking in the forenoon and late afternoon and evening. At 90° F., the periods during which no water is consumed tend to be shortened and animals tend to drink every 2 hrs. or more often (Ragsdale *et al.*, 1950, 1951).

Effect of Water Restriction: The first noticeable effect of moderate water restriction is a reduced feed intake (Balch *et al.*, 1953; French, 1956; Bianca *et al.*, 1965). Balch and others demonstrated that restricting the water intake of dairy cows to 65% of normal resulted in a reduced feed intake accompanied by a marked reduction in water excreted via urine and feces. Surprisingly, there was no effect on nitrogen balance or digestibility of dry matter. Water levels in the reticulo-rumen were maintained at about the same levels as before restriction, presumably by greater saliva production or by recycling water through the rumen wall. Ruckenbush *et al.* (1967) found a significant correlation between water and hay intake over 24 hrs., and between water intake in 24 hrs. and rumination time in the next 24 hrs. It was interesting to note that sheep dosed with 41. of water still drank 0.51. of supplementary water, although water intake the next day was reduced. When sheep were deprived of water for 5 days there was no significant change in feeding behavior until the 4th and 5th day when intake decreased 80 and 90% respectively. Rumination time also decreased. French (1956) studied the effect of infrequent watering on consumption and digestibility by Zebu cattle. He found that digestibility was not markedly lower when water was available only once in 48 hrs. as compared to *ad libitum* availability, but restriction to drinking every 72 hrs. reduced digestibility. With moderate restriction, feed intake was reduced and there was a higher digestibility of fiber. Weeth and Lesperance (1965) have studied intermittent watering in Hereford cattle which were watered ad libitum, once daily, and on alternate days with tap and 0.5% NaCl water during the summer. They concluded that once daily watering was adequate, under conditions imposed even with water of high quality. When the saline water was given, water and feed consumption were reduced by less frequent watering as was urinary output. Urinary N was greatly increased. An example of adaptation to restricted water intake has been reported by Taneja (1965). In this report it was found that 169 sheep could be maintained on the water ration required for 100 sheep if watering was done every third day.

In cases of total water deprivation, Bianca *et al* (1965) found that steers could maintain normal body temperature when the ambient temperature was 15° C., but at an ambient temperature of 40° C., rectal temperatures rose due apparently to a reduced rate of evaporation from the skin surface (Bianca, 1966). By the end of four days the cattle had lost about 12% of bodyweight which was regained in the first day of rehydration (Bianca, *et al.*, 1965) The primary means of water conservation was a reduction in fecal water which was associated with reduced feed intake and fecal output. Smaller reductions in evaporative and urinary water loss took place. Blood chemistry indicated hemoconcentration and did not return to normal for at least two days after water was made available. Weeth *et al.* (1967) found that total water deprivation resulted in a decrease in feed consumption amounting to 50% of each preceeding day's consumption (4 days which returned to normal after 4 days of access to water Urine volume was reduced 72%, fecal weight by 91%, and water composition of the feces by 16%. Plasma volume was decreased by 28% by the 4th day of water deprivation Macfarlane and others (1961) found that control of body temperature in sheep failed when 31% of bodyweight was lost by the end of 10 days without water. During rapid dehydration more Na was excreted than K.

Conclusions regarding water need and restrictions: Data indicate that water intake is primarily influenced by environmental temperature and feed consumption (or energy produced). Other environmental factors appear to have little influence. From a nutritional point of view, feeding "high" levels of salt or protein result in the need for more water to excrete salt or protein metabolites in the urine

Restriction of water will restrict feed intake. Further restriction, particularly in a hot environment, will severely reduce performance. As the temperature goes up, ruminants drink more frequently if water is readily available. Warming water in a cold atmosphere does not appear to greatly increase intake. In a hot environment, cooling water may be beneficial; although it may not be economical to do so. It water is restricted, the addition or presence of excessive amounts of salt puts added stress on the animal.

Effect of stress on Vitamin A Utilization: There are a number of factors that affect the utilization of vitamin A which has been clearly shown to be borderline or deficient in many feedlot situations. For example, it was demonstrated by Church *et al.* (1956) that a low plane of nutrition resulted in less depletion in lactating cows than a moderate plane of nutrition. Subsequent work by Erwin *et al.* (1963) indicated that depletion of vitamin A reserves in feedlot steers was accelerated by increasing levels of protein and energy and that the addition of an antioxidant reduced the depletion. In the case of deficient levels of protein, Anderson *et al.* (1962) reported that sheep fed rations with 5.9% crude protein stored only about one-half as much of an intraruminally administered dose of vitamin A acetate as did sheep receiving a ration with 10.4% crude protein. However, a recent report by Hayes *et al.* (1967) indicated that the half-life of tritium-labeled vitamin A was the same in steers fed either a 6 or 12% crude protein ration; consequently, some uncertainty exists on this subject. Feeding rations with high levels of urea apparently has no detrimental effect on vitamin A metabolism (Durdle *et al.*, 1962).

With respect to various stressing factors, Braun (1945) demonstrated some time ago that such factors as parturition, abortion or acute infections such as localized abcesses and mastitis or operations such as dehorning and castration will result in a rapid decline in plasma vitamin A, and, presumably, in reduced body reserves in the liver and other tissues. This is, undoubtedly, one explanation for many reports that certain situations were complicated with vitamin A deficiency. In the case of parturition, blood levels take about 2-3 weeks to return to pre-parturition levels. The effect of parturition may be partly a withdrawal of vitamin A from the blood and transfer to milk and colostrum. However, Kendall *et al.* (1954) demonstrated that treatment of cows with progesterone tended to increase blood vitamin A prior to and following parturition as compared to untreated controls. Jordan *et al.* (1963) has demonstrated that treatment of beef cattle with triiodothyronine (a thyroid hormone) resulted in increased blood and liver levels of supplemented steers and increased blood levels of unsupplemented animals. McGillivray (1961) reported that injections of adrenocorticotrophin or cortisone (adrenal hormones) resulted in elevated blood vitamin A levels and more rapid depletion of liver vitamin A in rats. Whether this same mechanism occurs in ruminants is unknown, but it seems plausible. The author is not familiar with data showing the effect of diethylstilbestrol on blood or liver vitamin A in ruminants, but perhaps this is one of the explanations for the apparently higher requirements for vitamin A noted in recent years in commercial feedlots.

One other factor regarding vitamin A should be mentioned. Page *et al.* (1959) reported that heat stress and solar radiation resulted in more rapid depletion of liver vitamin A than did low ambient temperatures or animals protected by shade.

Conclusions regarding vitamin A: Data indicate that a high plane of nutrition or a high energy ration tend to deplete vitamin A reserves (and thus increase the requirement). Deficient protein may reduce utilization and apparently an excess also does. Parturition, infections, and operations may be expected to result in reduced reserves. Hormone treatment and the stress of heat and/or solar radiation apparently also increase the rate of vitamin A utilization.

Summary

The best means of preventing production losses resulting from various stresses and during adaptation to a new environment can probably be largely achieved by control of parasite problems, immunization against specific maladies, adequate management of feeder stock enroute and after arriving at the feedyard and by the development and proper application of what we might call preventative nutrition. The ruminant animal, being what it is, preventative nutrition might best be applied by achieving a greater degree of continuity in providing similar feedstuffs to animals transported to new locations and placed in drastically different situations, than they have been accustomed to. Adaptation of rumen microorganisms to new feedstuffs requires time. In the meantime, the animal may be inadequately nourished because (1) it refuses to consume an adequate amount of feed or water, or (2) feedstuffs consumed may be utilized poorly, or (3) end-products produced in the rumen, as in the case of lactates, may be very detrimental to the animal. Consequently, a greater degree of continuity in source of feed very likely would help to prevent a variety of ills. The frequent references in trade publications that "backgrounding" of weaner calves or feeder stock is beneficial tends to bear this contention out. Something as simple as feeding alfalfa for a couple of weeks prior to shipping, during extended shipments, and following arrival at the feedyard might be adequate for this purpose.

THE ENERGY AND PROTEIN REQUIREMENTS OF STARTING CALVES

Dr. W. N. Garrett
Animal Husbandry Department
University of California, Davis

I. Major factors influencing energy requirements

A. Maintenance

1. Body weight (W)

a. Energy requirements for maintenance are proportional to metabolic body size (W 0.75 lb.).

b. Estimates of requirements:

Digestible energy, kcal./day, 76W 0.75 lb.

Metabolizable energy kcal./day 62W 0.75 lb.

Net energy (California system) kcal./day 43W 0.75 lb.

2. Activity

a. Estimates of the maintenance requirements made under practical conditions (as those above) will automatically include an allowance for normal activity, i.e. drylot feeding or grazing good pastures.

b. Walking on the level maintenance requirement is increased by about 1% for each 100 lb. of body weight for each mile traveled.

c. Walking uphill requires an additional increase of about 2.5% for each 1000 ft. of ascent.

3. Some estimates of the maintenance requirement for energy are shown in table 1.

B. Production—growth and fattening

1. Energy required for growth and fattening depend to a large extent on two factors:

a. Chemical composition of tissue deposited, i.e. the amount of fat, protein, water and mineral matter—more precisely, the energy content of the gain. Estimates vary according to methods used, breed, sex and hormone treatment, but are generally in the range of 1000-1500 kcal./lb. of gain for calves 150-450 lb.

b. The efficiency of utilization of the feed energy. This is variable depending to a large extent on the source of energy, i.e. roughage concentrate and how the efficiency is es timated probably also on the compositio of the gained tissue.

2. Some estimates of the energy required fc production are shown in table 1.

C Environment

1. The critical temperature (environmental ten perature at which feed or stored energy is use to maintain body temperature) depends upc the feeding level, source of energy and th condition of hair coat as well as the temper ture, humidity and air velocity.

a. For general conditions the following a probably close estimates of the critic temperatures of beef calves:

Fasting—	65°F
Maintenance—	40°F
1.0 lb./day gain—	25°F
2.0 lb./day gain—	10°F

2. It seems apparent that the energy requirem of calves will sometimes be increased beca of low environmental temperatures even un fed conditions. Calves being held at main nance or under fasting will have stress due cold at above freezing temperatures. Beca of the relationship between feeding level a critical temperature it is not possible to sim (in table 1) indicate the increased energy ne due to low temperatures.

II. Major factors influencing the protein requiremen calves

A Maintenance

1. Body weight (W)

a. There is a relationship between basal tabolism and endogenous urinary nitr excretion (a measure of protein catabo under fasting conditions). For light ca the estimates are usually between 0.08 0.1 gm. of nitrogen per unit of meta

body size W 0.75 lb. This is equivalent to 0.5—0.63 gm. of protein. This figure must be adjusted upward for the biological value and the true digestibility of the protein as well as the metabolic fecal loss of nitrogen (related to food intake and to the feedstuff being consumed). Using appropriate estimates, the final figure which closely estimates the maintenance crude protein requirement (lb./head/day) for calves is 0.0055W 0.75 lb.

2. Activity

   a. There is no protein requirement for exercise if total caloric intake is adequate.

B. Production

1. The amount of protein required for growth is largely dependent upon the rate and composition of the gain.

   a. The gain of fast-growing calves will ordinarily contain 16-18% protein or 0.18 lb. of protein/lb. of gain. Adjusting this figure for the average biological value and the average true digestibility of feed protein and for losses of protein associated with the amount of feed consumed, the estimated crude protein allowance for calves is 0.55 lb./lb. of weight gain.

C. General

1. It must be remembered that calves require amounts of protein, not a certain percentage of their diet. Thus a 400-lb. calf requiring 1.45 lb. of total protein and eating 11 lb. of feed should receive a diet containing 13% protein. However, this same calf eating an all-concentrate diet consuming only 9.5 lb. of feed would require a 15% level of protein in his diet.

2. Suggested protein allowances are given in table 1. Because of the assumptions made in developing this table, the protein requirements listed for weight gain are somewhat on the liberal side. This will be especially true for rations containing large amounts of concentrate.

Table 1. Suggested energy and protein allowances for maintenance and growth of calves[1]

Daily gain		Body weight, lb.						
		200	250	300	350	400	450	500
lb.		For maintenance alone						
0	CP[2]	0.29	0.35	0.40	0.44	0.49	0.54	0.58
	DE[3]	4.04	4.77	5.48	6.15	6.80	7.43	8.03
	NE_m[4]	2.29	2.70	3.10	3.48	3.85	4.20	4.55
		For gain alone—must be added to maintenance requirement (CP and DE) or used in conjunction (NE_m) to obtain total requirement						
0.5	CP	0.28	0.28	0.28	0.28	0.28	0.28	0.28
	DE	1.17	1.38	1.59	1.78	1.97	2.15	2.33
	NE_p	0.36	0.43	0.49	0.55	0.61	0.67	0.72
1.0	CP	0.55	0.55	0.55	0.55	0.55	0.55	0.55
	DE	2.35	2.77	3.18	3.57	3.94	4.31	4.66
	NE_p	0.74	0.88	1.01	1.13	1.25	1.37	1.48
1.5	CP	0.83	0.83	0.83	0.83	0.83	0.83	0.83
	DE	3.52	4.15	4.77	5.35	5.91	6.46	6.99
	NE_p	1.15	1.36	1.56	1.75	1.93	2.11	2.28
1.75	CP	0.96	0.96	0.96	0.96	0.96	0.96	0.96
	DE	4.10	4.84	5.56	6.24	6.90	7.54	8.15
	NE_p	1.36	1.60	1.84	2.07	2.29	2.49	2.70
2.0	CP	1.10	1.10	1.10	1.10	1.10	1.10	1.10
	DE	4.70	5.54	6.36	7.14	7.88	8.62	9.32
	NE_p	1.57	1.86	2.13	2.39	2.64	2.89	3.12
2.25	CP	1.24	1.24	1.24	1.24	1.24	1.24	1.24
	DE	5.28	6.23	7.15	8.03	8.87	9.69	10.49
	NE_p	1.79	2.12	2.43	2.73	3.01	3.29	3.74
2.5	CP	1.38	1.38	1.38	1.38	1.38	1.38	1.38
	DE	5.86	6.92	7.95	8.92	9.86	10.77	11.65
	NE_p	2.02	2.38	2.73	3.07	3.39	3.70	4.01

[1] To be used with the values listed in table 2.
[2] Crude protein, lb./head/day.
[3] Digestible energy, megacalories/head/day (for TDN divide by 2).
[4] Net energy, megacalories/head/day (NE_m is for maintenance, NEp is for production).

Table 2. Energy content of feeds*

	Crude protein	For maintenance (NE_m)	For Weight gain in addition to maintenance (NE_p)	For maintenance and weight gain (DE)
	%	megcal. per 100 lb.		
Dry roughages (90% dry matter)				
Alfalfa hay, 24% fiber	17.5	51	24	105
Alfalfa hay, 29% fiber	14.6	46	16	100
Alfalfa hay, 34% fiber	13.7	42	8	96
Alfalfa meal, dehydrated	20.2	56	32	110
Barley hay	7.3	50	21	98
Barley straw	3.7	41	6	82
Bermuda grass hay	7.1	43	12	92
Cottonseed hulls	3.9	42	9	88
Oat hay	8.2	47	16	96
Prairie hay, good quality	6.5	47	16	96
Sudan grass hay, immature	8.8	48	19	98
Silages (30% dry matter)				
Alfalfa, wilted	5.3	18	9	34
Corn, dent	2.3	22	13	42
Sorghum, sweet or dual purpose	2.3	20	9	33
Concentrates (90% dry matter)				
Barley, 46-48 lb.	9.0	87	58	158
Beet pulp, molasses, dried	8.4	83	55	145
Citrus pulp, dried	6.2	81	54	154
Corn, dent, No. 2	8.7	92	60	162
Corn and cob meal	7.4	81	54	150
Cottonseed, whole	23.1	82	49	155
Cottonseed meal, expeller	41.0	75	50	146
Cootonseed meal, solvent	41.0	64	41	133
Fat (98% dry matter)	—	203	127	380
Hegari grain	9.0	87	58	161
Hominy feed, 5% fat	10.6	96	67	168
Linseed meal, expeller	37.5	78	52	151
Linseed meal, solvent	36.6	73	49	150
Milo grain, Southwest	9.0	87	58	160
Molasses (cane, 71% dry matter)	3.0	65	41	110
Oats	9.0	75	51	144
Potatoes, dried	9.7	76	51	144
Rice bran	14.3	68	45	136
Screenings, grain, high quality	12.8	63	40	125
Soybean meal	44.0	84	56	156
Wheat bran	16.4	69	46	133
Wheat mixed feed (mill run)	15.8	78	44	140
Whey, dried	12.8	84	55	156

*To be used with requirements as listed in table 1.

EFFECT OF VITAMIN AND MINERAL STATUS UPON FEEDLOT PERFORMANCE OF WEANLING CALVES

Dr. Allen D. Tillman
Animal Science Department
Oklahoma State University

The approach to the subject will be made by addressing my attention to the question of vitamin and mineral requirements of cattle and then to ask my colleague, Dr. Eric Williams, to discuss the "Role of Cations in Neuromuscular Function."

The nutrition of ruminants is complex because of the symbiotic relationship between the microorganism in the gastrointestinal tract and the host animal. While it is true that the microorganism simplify ration formulation of these animals because of synthesis of certain nutrients, there is destruction of some elements in the rumen, the fermentation process is wasteful of dietary energy, and is the cause of many disorders peculiar to the ruminant. In this review some of these factors will be considered.

Vitamin A

One of the most important nutrients for maintaining animal health is vitamin A. The ruminant obtains vitamin A from dietary carotenes or as supplemental vitamin A. In feeding cattle, the nutritionist must realize that the carotenes of corn silage, alfalfa hay and other carotene sources must be converted to vitamin A before these are of value to the animal. There are many factors affecting the conversion of the carotenes to vitamin A. Some of these are as follows:

1. *Plant species:* The carotene of some plant species like alfalfa are very poorly converted.
2. *Processing conditions:* Any condition which is conducive to oxidation of the formation of stereoisomers results in reduced conversion.
3. *Storage losses:* Storage losses are variable and can be great. Regardless of storage conditions, there is some loss of potency upon storage under practical conditions.
4. *Level of Intake:* The conversion of the carotenes to vitamin A is inversely related to intake.

These observations indicate that the carotenes are unreliable sources of vitamin A. It is recommended that some preformed vitamin A be added to the diets of fattening cattle regardless of roughage source.

Also there are many factors affecting vitamin A utiliza tion and the subsequent requirements of cattle: Some o these are as follows:

1. *Isomeric form.* The cis isomers, which can resul from storage and other changes, are much les active than the all-trans form.
2. *Absorption.* Vitamin A has to be absorbed before i can be utilized. Absorption in cattle is affected b dietary level, composition of diet, level of fat, leve of vitamin E or an antioxidant, emulsifiers, antibio tic level, and protein level.
3. *Stress Factors.* Any stress factor involving reticu loendothelial system prevents deposition of th vitamin A ester in the liver and increases the dietar requirements for this vitamin. The following stres factors will increase the requirements of cattle fo vitamin A: Infections by bacteria, virus, fungus toxic factors, regardless of source; parasitic infesta tions; high environmental temperature; and stresse associated with high production. Vaccination an stresses associated with it cause decreased status

Vitamin A requirements of animals are increased as w expect and obtain greater production from all animal production stresses. The recent tendency for many peopl to associate the presence of dietary nitrates with in creased cattle requirements for this vitamin does no seem to be justified, especially, if trace mineral level are adequate. The recent associations of vitamin A de ficiency symptoms with the feeding of corn silage to cattl might be explained by a deficiency of iodine in th silage; it has been shown that reduced thyroid activit affects vitamin A status of animals fed carotenes a source of vitamin A.

The author recommends that supplemental vitamin A as an ester of the vitamin, be added to *all* feedlot ration

Vitamin D

Feedlot cattle in the Southwest are exposed to dire sunlight thus it is doubtful if supplemental vitamin D needed in this area. In other areas, the situation is di ferent.

Vitamin E

Recent work by the Iowa group indicates that vitamin E gave increased performance in cattle fed all-concentrate diets. Selenium might also be efficacious in such diets. Because of lack of knowledge concerning the specific function of vitamin E or selenium, or both, this whole area is confused. If the vitamin E level of the diet is low, response to added vitamin E might be expected. What diets are low in this vitamin, is a difficult question. The relationship of antioxidants, vitamin E and selenium at the cellular level also is not known at this time. For this reason, supplemental vitamin E is usually recommended for cattle rations.

In view of the recent results from Cornell with fowls, which indicates that high levels of vitamin E might increase the requirements for selenium, one must ask about selenium. Selenium is an elusive element and many factors affect its level in soil and plants. As it is a toxic element at higher concentrations, our attention has been directed toward this aspect of its metabolism. Research in this important field must be increased. There are distinct possibilities that this element should be added to cattle fattening rations even though it is unlawful to add it to feeds being sold interstate.

Vitamin K

Conditions would have to be quite abnormal to show need for dietary vitamin K in cattle.

B-Complex vitamins

Under normal conditions, regardless of type of ration, cattle can synthesize sufficient levels of the "B" vitamins to meet their needs. My laboratory has done much research in this area and has found that low ruminal pH results in lowered "B" vitamin synthesis. These results indicate that during periods of stress, especially when cattle are brought onto concentrates too quickly, that "B" vitamin injections or if added in the feed could possibly be beneficial. Our work would indicate that once the stress condition was removed and ruminal pH returned to normal that further "B" vitamin supplementation was not necessary. Cattle, which are "off feed" and sick could very well respond to "B" vitamin supplementation or injections.

MINERALS

It is doubtful if the mineral content of the ration of feedlot cattle is a major factor in the short time stress phenomena noted in feedlot cattle. However, the dietary level of minerals is important in determining the over all efficiency of the cattle. The following represents (table 1) levels of minerals, which will meet the requirement of feedlot cattle.

These levels are estimates and should be treated as such. The author is concerned about the present tendency to use standard mineral premixes for use everywhere without regard to the composition of the ration. It is his contention that the mineral imbalance plays an important part as regards mineral adequacy in nutrition. Adding known minerals to rations of unknown composition without regard to mineral balance is dangerous. To illustrate this point, I present Figure 1.

Table I.
ESTIMATE OF OPTIMUM MINERAL LEVELS FOR CATTLE

CALCIUM	0.4%	COPPER	10PPM
PHOSPHORUS	0.3%	IRON	100PPM
MAGNESIUM	0.1%	MANGANESE	30PPM
POTASSIUM	0.50%	ZINC	60PPM
SODIUM	0.2%	COBALT	0.1—.5PPM
CHLORINE	0.2%	IODINE	.2—.4PPM
SULFUR	0.15%	MOLYBDENUM	1.0PPM
		SELENIUM	0.1PPM

Phosphorus: (a) Ca, Mg, Mn, Zn, Fe, Al and Be interfere with the absorption of P, as well as the opposite being true, due to the formation of insoluble phosphates. (b) Low Cuhigh Mo intakes increase the loss of body P (copper is required for phospholipid synthesis).

Calcium: (a) High Ca levels in the diet reduce the absorption of Mn, Zn and F. (b) An excess of either Ca or P interferes with the absorption of the other due to the formation of insoluble tricalcium phosphate. (c) Large intakes of either Ca or Mg increases the urinary excretion of the other; but, both Ca and P prevent the absorption of excess Mg. (d) SO_4 increases the excretion of Ca.

Copper: (a) Cu is required for the proper metabolism of Fe. (b) Cd and Ag increase the severity of Cu deficiency. (c) High dietary Zn reduces liver stores of Fe and Cu while low Zn favors excess storage of Fe and Cu. Excess Cu causes low storage of Zn. (d) Mo limits Cu storage in the presence of adequate sulfate. Sheep with high liver Cu have low Mo levels and Cu toxicity may develop with low Mo intakes.

Sulfur: (a) Sulfate—S limits Cu and Ca storage and protects against Se toxicity. (b) High Zn increases fecal S. (c) SO_4 decreases liver Mo.

Molybdenum: (a) Tungstate (W) increases urinary excretion of Mo.

Cobalt: (a) Cobalt increases the urinary excretion of I. (b) Fe accumulates during Co deficiency (Co needed for Fe metabolism).

Iodine: (a) As and F have goitrogenic activity.

Fluroine: (a) Ca and especially Al salts protect against F toxicity.

Selenium: (a) SO_4 and As reduce Se toxicity.

Sodium—Potassium: (a) Deficiency symptoms of either of these elements are aggravated by an excess of the other. The high K content of forage may explain the high salt requirement of Herbivera.

Manganese: (a) High Mn interferes with Fe utilization. (b) High Mn lowers serum Mg.

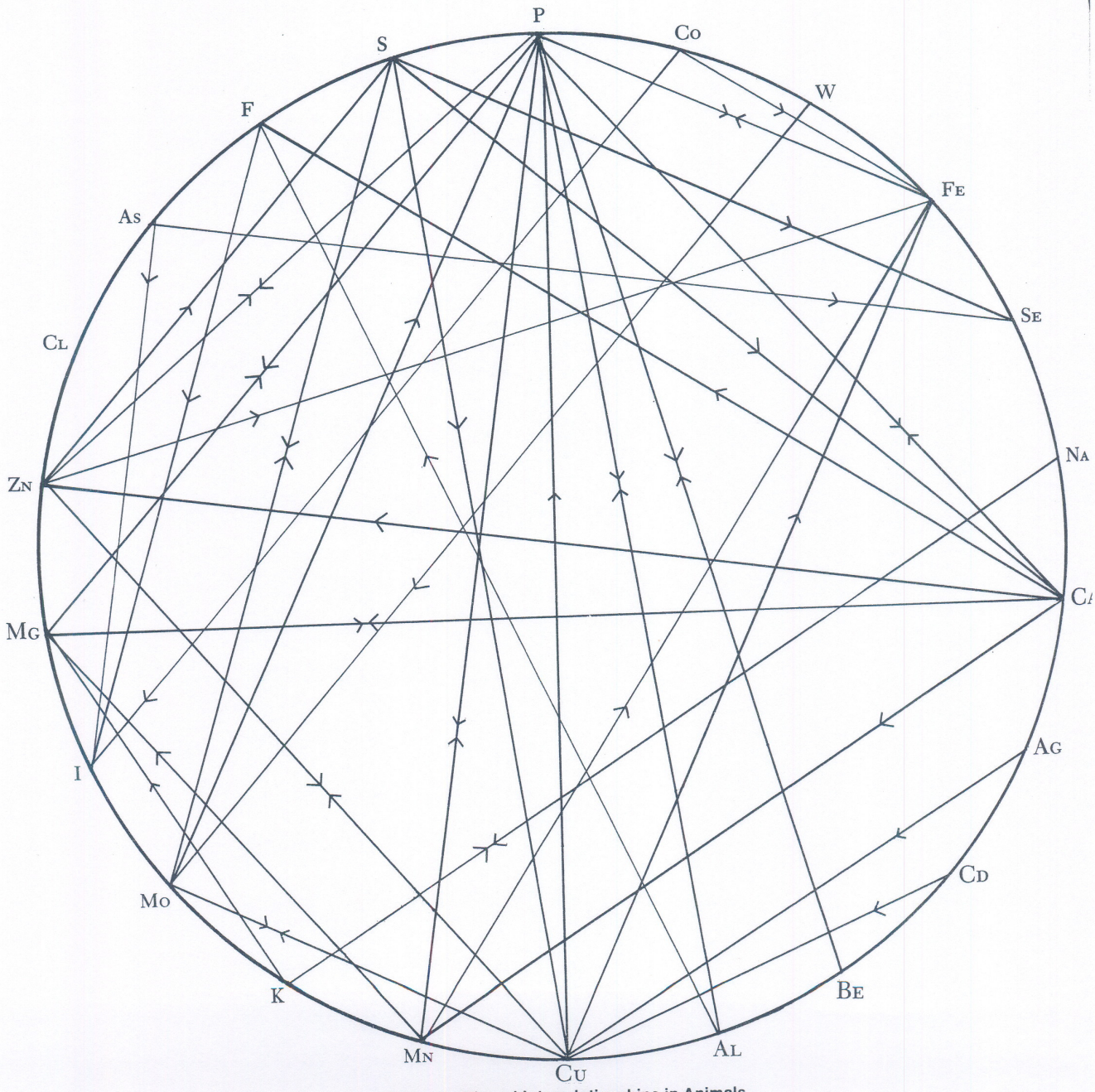


Figure 1. Mineral Interrelationships in Animals

THE ROLE OF CATIONS IN NEUROMUSCULAR FUNCTIONS

Dr. Eric I. Williams, F.R.C.V.S., M.S.
Department of Veterinary Medicine & Surgery
Oklahoma State University

Summary

The concentrations of Ca^{++}, Mg^{++}, K^+ & Na^+ in the extracellular (ECF) and intracellular fluids (ICF) influence neuromuscular mechanisms. This paper deals with the role of these cations on the various components of the neuromuscular unit viz:—(a) *the resting nerve cell membrane* which is readily permeable to K^+, but only slightly permeable to Na^+, (b) The initiation of a *nerve impulse* (action potential)—stimulation of a normal resting nerve cell membrane above threshold level results in an increased permeability to Na^+, depolarization and a nerve cell to Na^+ & K^+. Magnesium ions do not influence the resting nerve cell permeability or the initiation of a nerve impulse. Changes in Ca^{++} in the ECF affects the permeability of the nerve cell to Na^+ & K^+. Magnesium ions do not influence the resting nerve cell permeability or the initiation of a nerve impulse. (c) *neuromuscular junction*—the release of acetylcholine (ACh) necessitates a nerve impulse and depends on the concentrations of Ca^{++} & Mg^{++} in the ECF. These cations exert opposite effects on ACh release and thus on the transmission of activity from nerve to muscle. (d) *muscle contractions* which are produced by action potentials, following the release of ACh, on the surface membranes of skeletal muscles. The resulting electric currents cause the relase of Ca^{++} in the muscle fiber, which activate a reaction between the contractile muscle proteins, actin and myosin. A high concentration of Mg^{++} within the muscle cell will interfere with this reaction. Reference is made to the danger of excessive calcium therapy for the "downer" cow. The "alert downer" cow probably does not need potassium therapy. The significance of Ca^{++} & Mg^{++} in wheat pasture poisoning is discussed.

Reference—Additional information on this presentation is available in:

Breazile, James E. and Williams, Eric I., "The Role of Cations in Neuromuscular Functions." Scientific Proceedings of the 104th Annual Meeting, American Veterinary Medical Association, July 9-13, 1967, Dallas, Texas.

EFFECT OF CATIONIC IMBALANCE ON NEUROMUSCULAR FUNCTION

Cation		Resting cell membrane	Nerve impulse	Neuromusc. junction	Muscle contraction	Overall effect
K^+	Hyper K^+	Incr. excitability followed by decreased excit. if maintained (due to cathodal block)	Same as previous column	Increased release of ACh	Increased, later decreased	Twitching later paresis
	Hypo K^+	Decreased excitability	Decreased excit.	Decreased release of ACh	Decreased	Muscle weakness (depressed "downer")
*Na^+	Hyper Na^+	Increased excit. (resting membrane potential less than normal)	Increased excit.	Increased release of ACh per nerve impulse	Increased	Increased excit. without spontaneous tetany
	Hypo Na^+	Decreased excit. (resting membrane potential increased -hyperpolarized)	Decreased excit.	Decreased release of ACh per nerve impulse	Decreased	Paresis
*Sodium ion imbalance alone is not likely to occur as a specific clinical entity due to efficient homeostatic mechanisms.						
*Ca^{++}	Hyper Ca^{++}	Incr. excit. due to effect on K^+ permeability; later decreased excit. due to cathodal block	Decreased excit.	Incr. release of ACh	Incr. (stronger contr.)	Paralysis due to tetany later paresis

Cation		Resting cell membrane	Nerve impulse	Neuromusc. junction	Muscle contraction	Overall effect
	Hypo Ca^{++}	Incr. excit. due to effect on Na^{+} & K^{+} perm. later decrease	Incr. excit., later decreased	Decr. release of ACh	Weak contr.	Paresis
*This condition may occur due to excessive Ca therapy of a 'downer' cow.						
Mg^{++}	Hyper Mg^{++}	Nil	Nil	Decreased ACh release	Decreased (interference with Ca-actin-myosin interaction)	Paresis
	Hypo Mg^{++}	Nil	Nil	Increased ACh release	Increased	Tetany followed by paresis

Note: The concentration of Mg^{++} in the extracellular fluid has no significant effect on resting cell membrane excitability and the production of a nerve impulse.

MANAGEMENT OF CALVES AND ADAPTING THE CALF TO ITS FUTURE ENVIRONMENT

Dr. Donald R. Gill
Extension Nutritionist
Oklahoma State University

The method in which calves are handled on the ranch is probably the key link in the preconditioning program. The practices used will not only affect the calves' feedlot performance, but also the profitability or loss for this operation to the rancher.

The most important management practice in the conditioning program is that of injecting a period of time on the ranch, between when the calves are weaned and when they leave the ranch. The reason for this is simple, in that the stress of weaning in itself is enough of a health hazard to the weanling calf, that exposure to disease and additional stress are to be avoided. The just weaned calf with a sore throat from bawling is a poor risk when mixed with other cattle from other origins in the close confinement of a sales ring, truck or feedlot.

Basically the whole preconditioning program must be designed to reduce the risk or probability of disease both infectious and noninfectious.

The reduction of stress is the key to the success of the whole program. If the stresses to which cattle are subjected are not reduced it is unlikely that any drug or vaccine is going to help eliminate the major problems which lead to so much economic loss to the cattle industry today.

Only part of the total problem which causes feeder cattle to fail to perform or even die, can be attributed to infectious disease. Many are confused about this because the weakened calf after his ability to resist disease is reduced to almost nothing, eventually succumbs to some infectious organism.

Data collected by Dr. Richard Bristol at Iowa State University clearly points out that the respiratory disease complex while accounting for a major part of the death and morbidity in just weaned and shipped feeder cattle, is seen at much lower frequency in calves weaned 30 days or so before shipping. In many cases the incidence is reduced by more than 20 times.

Holding calves on the ranch for a period of time after weaning accomplishes many things which can greatly increase the animal's ability to do well in the feedlot.

1. It allows a calf to recover from the stress of weaning under much more favorable conditions than are available elsewhere.
2. It provides time for the calves to build up an immunity following vaccination with viral and bacterial agents. This is important as it has been demonstrated that the stressed animal may not develop a good immunity even when properly vaccinated with an otherwise effective agent.
3. It provides time for the rancher to accustom the calves to troughs or bunks. Calves should come to the feedlot prepared to eat from bunks, and to drink from water troughs. Not all cattle taken from the producing ranches can do this.
4. It provides an opportunity for the rancher to look over his calves. If his reputation means anything to him he can possibly remove some of the poor doing calves, and sick calves and either treat them or dispose of them as nonconditioned calves.
5. Feeding—a calf should be exposed to dry feed, preferably out of a bunk or trough, before it leaves the ranch. The period of time between weaning and shipping will be ideal. Feedlots prefer non-creep-fed calves. In general creep-feeding may be used when grass or hay are short, but should not be used in conjunction with good grass or hay. The fleshy calf is not in demand by feeders who keep good records unless they can be bought with a good discount.

   Any supplemental feeding program should be designed to obtain bred-in growth potential without producing unwanted flesh and fat. Following weaning, concentrate feeds may be used at a rate of up to one pound per 100 pounds of body weight per day. However, the use of good quality roughage or forage may produce a more saleable product. In many cases gains may not be any better with concentrate feeding if the calves are on good forage or hay.
6. Management—all calves should be castrated, dehorned and branded while they are small. No calf should be shipped within 30 days following any of these procedures, and then only if they have fully recovered.

The economic affects of the weaning before shipping are being studied at a number of universtities and ranches. To-

day it is impossible to tell a rancher the effects of early weaning on his pocketbook. However, some trends are emerging which indicate that holding calves on the ranch may increase net returns to the ranch, ignoring any premium for a better conditioned calf.

Data from Oklahoma State University herds over a number of years indicated that the gross return to the ranch is going to be dependent on the length of time following weaning before the cattle are shipped.

Table 1 shows a summary of weights taken on a group made up of both Hereford and Angus calves in 1961. These calves were separated into light and heavy calves based on the dams' previous history. These calves were weighed off the cows with no shrink. These weights are comparable to what a rancher might see if he has a scale on the ranch close to the working area.

Table 1

O.S.U. DATA 1961

Straight breed Hereford and Angus	
Calves weaned	10-5-61
Maintained on grass	
Average weight	(no shrink)
Average age—225 days	
Heavy	493 pounds
Light	448 pounds
Into feedlot—14 days following weaning	10-19-61
	(overnight stand)
Average weight	
Heavy	488 pounds − 5 pounds
Light	451 pounds + 3 pounds
14 day feedlot check weight	(525 pounds)
Heavy—ADG from weaning	1.17 pounds
Heavy—ADG from weigh in	2.73 pounds
Light	(487 pounds)
Light—ADG from weaning	1.39 pounds
Light—ADG from weigh in	2.57 pounds

The calves were moved to the feedlot 14 days later and given an overnight stand without feed or water before weighing. This is usually equivalent to a four percent pencil shrink. At 14 days post weaning this group of calves, after an overnight stand, showed no weight gain compared to the unshrunk weaning weight. However, since they weighed the same after being subjected to a shrink you may consider that they gained about four percent during this two week period. After these cattle had been on feed 14 days they were check weighed and the data are presented based on feedlot in weight and on weaning weight.

In Table 2 data on a group of calves weaned in 1963 are presented. These calves were held 35 days following weaning at an average age of 200 days. These calves like the others were maintained on pasture with little or no supplemental feed. Weigh conditions in all tables are similar to those described in the first table. These calves indicated a weight gain of 1.69 pounds per day from weaning to entering the feedlot. These are very good gains considering that the weaning weight was unshrunk and probably about four percent high compared to their shrink in weight at the feedlot.

Table 2

O.S.U. DATA 80 HEAD 1963

Weaning date	10-2-63
Average age	200 days
Average weight	457 pounds (no shrink)
In feedlot—35 days following weaning	11-6-63
Average in weight	516 pounds (overnight stand
15 day check weight	562 pounds (no shrink)
Gain	
Weaning to in	1.69 pounds
Weaning to check	2.10 pounds
Initial to check	3.05 pounds

The data for 1964 and 1965 are presented in Tables 3 and 4 respectively.

The data in these tables suggest that the longer the period of time following weaning until feedlot check-in, or in a ranchers case, sale; the better will be the weight gain following weaning.

Tests are in progress at the present time at Oklahoma State University to provide a better understanding of weight patterns following weaning. These data will be available later. It appears that a calf will lose weight immediately following weaning and will continue to do so for a number of days. The number of days will be variable, depending on various factors. Some of the important factors are: the milking ability of the cow, the feed available both before and after weaning for the calf, and the degree of stress to which the calf is subjected following weaning.

Many ranchers, hesitant to hold calves after weaning, are being surprised to find that it is not hard to market enough additional weight to easily pay for holding the calves. Just weaned calves shrink more in transit between the ranch and the sale.

Table 3

1964 O.S.U. DATA

Weaning date	10-8-64
Average age	200 days
Average weight	490 pounds (no shrink)
In feedlot—28 days following weaning	11-5-64
Average weight	523 pounds (overnight stan
ADG	1.2 pounds
20 day feedlot check	(590 pounds)
ADG from weaning	2.03 pounds
ADG from weigh in	3.29 pounds

Table 4

O.S.U. DATA

1965 Crossbred Heifers
Angus Sires Hereford Mothers

Weaning date	9-29-65
Average age	219 days
Average weight—not shrunk	393 pounds
These cattle were weaned and carried on grass until November 15th.	(44 days later)
On November 15th	437 pounds
These cattle averaged*	

*After overnight stand without feed or water.

Actual gain under these conditions was about one pound per day.

Optimum conditioning on the calves can be obtained when the calves are held for about 30 days on the ranch. The timing will vary with the ranch and conditions. This type of contitioning will provide the calf time to recover from the stresses of weaning, and provide time to develop a good immunity to disease when a well planned vaccination program is carried out in conjunction with this phase of preconditioning.

The rancher should remember that weaning about 30 days before shipping is just a part of what can be an excellent program of preconditioning calves for the feedlot. If a rancher wants to sell truely conditioned calves his program starts with the bull and the cow and ends with the delivery of the prepared product to the feedlot. The program is no better than the weakest link. The rancher who has no control of the movement of his calves from his ranch to the feedlot could see all his efforts erased by a couple days of mismanagement of the calves between his ranch and the feedlot.

PRECONDITIONING OF FEEDER CATTLE PRIOR TO INTERSTATE SHIPMENT

Richard F. Bristol, D.V.M., M.S.
Veterinary Clinic Sciences
Iowa State University

Introduction

The findings and recommendations upon which this report is based are the results of a preliminary survey conducted by the Department of Clinical Sciences of the College of Veterinary Medicine at Iowa State University. The survey was undertaken to determine if any correlation might exist between certain management practices of the rancher-source of feeder calves, the practices of the feeder-seller, and the disease problems encountered by the feeder-seller.

Three geographical areas were surveyed for sources of feeder calves. These source areas were the counties surrounding Choteau, Montana; Ainsworth, Nebraska; and Mitchell, South Dakota.

All cattle that were traced to their final destination were shipped to the state of Iowa for their final feeding period.

The number of farms or ranches involved in the survey was 63, averaging 171.9 calves sold per farm. The extremes in numbers of calves marketed for feeding purposes per farm or ranch varied from 50 to 864 animals per year. The total numbers of animals involved in this initial work were 10,832. The total numbers of feeder calves traced to their ultimate destination were 2,656 calves.

The questions asked of the rancher source of the feeder calves were as follows:

1. Number of calves sold
2. Sex
3. Age at date of sale
4. Method of sale
   a. private negotiation or contract
   b. public auction
5. Method of shipment
   a. rail
   b. truck
6. Surgical procedures performed by owner-source
   a. castration
      1. those castrated at least 30 days prior to sale
      2. those castrated less than 30 days prior to sale
   b. spay or other procedures
      1. those spayed at least 30 days prior to the sale
      2. those spayed less than 30 days prior to sale
7. Vaccination procedures employed prior to shipment
   a. type of vaccines administered
   b. method of administration
   c. date of administration
      1. at least 30 days prior to selling and shipment
      2. less than 30 days prior to selling and shipment
8. Antibiotics administered prior to shipment
   a. oral administration
   b. parenteral administration
   c. type used
   d. date administered
9. Did any co-mingling with other animals take place at the time of sale?
10. Were pesticides administered?
    a. type
    b. when administered
11. When were animals weaned?

The feeder seller was querried in these areas:

1. Date of arrival of the calves
2. Condition of the calves upon arrival
   a. Were any animals visibly ill?
   b. Were veterinary services required?
   c. Veterinarian's diagnosis.
3. Feeding and handling practices at the time of arriva
   a. types of feed available to the calves
   b. availability of water
   c. availability of shelter
4. Medical practices upon arrival
   a. vaccines given
      1. type

2. how administered
3. date administered

b. other chemo-therapeutic agents employed
1. antibiotics
2. sulfonamides
3. route of administration

c. pesticides used
1. type
2. date of administration
3. method of administration

d. Were veterinarians employed in the above procedures?
1. how employed

5. Disease problems after arrival
a. type of problem
b. date the animals were first affected
c. steps taken to eliminate the condition or conditions
1. Professional assistance
2. chemo-therapeutic agents employed
3. method of administration
d. morbidity
e. mortality

6. Additional comments of the owner in regard to health and general well being of the animals upon final sale

RESULTS

It is not within the scope of this paper to discuss or report on all of the findings in this survey. This preliminary report will confine itself to certain pre-selling practices of the rancher-seller, shipment of the animals and the method of selling.

1. Method of sale (see chart 1)
2. Vaccination procedures (see chart 2)
3. Weaning procedures (see chart 3)
4. Methods of shipment (see chart 4)
5. Surgical procedures (see chart 5)

Slightly over one-fifth of the animals involved in the survey were traced to their eventual destination. It was found that animals sold by private contract were much easier to trace. The owners were more cooperative in answering questions relative to the animals and more detailed information could be obtained. Of the animals sold at auction, only 710 animals of the total number could be traced; this represented only eight percent of the animals surveyed.

It is from animals sold on private contract that the correlations have been obtained. In evaluating the data obtained, the feeder-sellers estimation on record of the number of cases of respiratory infection that were treated in a particular lot during the first 30 days after arrival on the premises are compared with the management practices of the rancher-seller.

CHART 1
Method of sale

Number of Calves	Auction	Per-Cent	traced	Per-cent	private sale	per-cent	traced	per-cent*
10,832	8,882	82	710	8	1,950	18	1,198	38

*Percent figures are given in the nearest whole number.

CHART 2
Vaccination procedures

Number of calves	Vaccinated	When administered (At least 30 days prior to sale)	When administered (Less than 30 days prior to sale)
10,760*	Clostridium Chauvei Septicus	8,470	784
	Pasturella	1,340	4,623
	IBR		468
	BVD	2,142	378
	Leptospira pomona		1,423

*One owner did not answer this question.

CHART 3
Weaning procedures

Number of animals	(Weaned less than 30 days prior to sale)	(Weaned more than 30 days prior to sale)
7,264*	2,647	4,617

*Some ranchers did not answer this question.

CHART 4
Method of shipment

Number of animals	Method of shipment: Truck	Method of shipment: Rail	Number of hours in transit: Less than 24 hours	Number of hours in transit: More than 24 hours	Number of hours in transit: More than 36 hours
9,628*	8,276	1,352	1,847	412	397

*Some ranchers did not answer this question.

CHART 5
Surgical procedures

Number of Calves	Castrated	Surgery not performed	Surgery performed: At least 30 days prior to sale	Surgery performed: within 30 days of sale
10,832	6,427	4,279	6,076	571

CHART 6
The time in transit compared to respiratory involvement

Animals traced from rancher-seller to feeder-seller time in transit when compared to rate of respiratory disease.

Total number of animals:	2,656	Percent*
Cases of respiratory involvement:	231	10
In those less than 24 hours in transit:	37	16
In those over 24 hours in transit:	64	27
In those over 36 hours in transit:	130	56

*Percent expressed to the nearest whole number.

CHART 7
Incidence of respiratory involvement when compared with castration practices

No. of calves	No. Castrated	Castrated: (At least 30 days prior to sale)	Castrated: (Within 30 days of sale)	Uncastrated
10,832	6,427	6,076	571	4,279
No. of calves traced 2,656	2,227	2,106	121	429
No. of respiratory condition cases treated	231	192	27	12
Percent treated*	10	8	21	3

*Percent figures are given to the nearest whole number

CHART 8
Weaning practices in comparison to respiratory involvement

No. of calves	Total no. of cases 231	percent
(Weaned 30 days prior to sale)	12	.5
(Weaned within 30 days of sale)	219	99.5

SUMMARY AND DISCUSSION

The weaknesses of a survey of this type are quite obvious. The information received from the rancher-seller and the feeder-seller are dependent on few, if any, written records and the memory of the individual owner. By and large, the records of professional people in regard to exact numbers of animals treated, the types of medication involved, and the response to treatment was woefully unavailable. By the same token, definitive diagnosis of animals affected with an illness during the first 30 days after arrival was extremely difficult to establish again due to inadequate records and lapses of memory. Thus, the term "respiratory conditions" is used. It has been established that respiratory involvement can exist as a secondary condition to other disease states.

Nonetheless, the information that has been received is of some value in establishing a trend of infection rate.

The numbers involved in this survey should be expanded so that these trends can be established and the habits of interstate movement of animals recorded.

It was noted early in the survey that it was extremely difficult to trace the movement of animals sold at public auction. In some cases (11) the animals were traced through three states and then lost. One group of 97 calves was followed through four states, six professional dealers of cattle and finally lost in the Omaha, Nebraska, stockyards after having been grouped with cattle from Cherry County, Nebraska. The cattle were originally from Mitchell, South Dakota. The records of the public auction are almost impossible to examine or to trace.

One of the most startling incidents of the entire survey concerned a group of 53 calves sold at public auction at Ainsworth, Nebraska. These animals were traced through four professional dealers, two public auctions, one public stockyard, and a private sale. The time elapsed between the date of the original sale and their final destination was eight days!

This group of calves had a 100 percent incidence of respiratory conditition and a total death loss of 48! It would seem that the time of weaning calves is a factor in the incidence of recognizable respiratory infection within thirty days of arrival upon the farm. The effects of vaccination are difficult to determine other than to state that in the small number of animals traced, the use of the Viral Vaccines at the time of shipment when correlated with the incidence of respiratory infection show a distinct rise in incidence.

The use of the auction barn as a method of sale and acquisition of animals tend to show an increase in the incidence of respiratory infection.

There is a radical increase in correlating the castration practices with the incidence of respiratory infection. However, this is based on a small numerical sampling.

A correlation between the time in transit and the incidence of respiratory disease is well defined and the numerical sampling is adequate. It would seem that as the time in transit increases there is a corresponding rise in the incidence of respiratory infection.

RECOMMENDATIONS

Although this survey is in its preliminary stages and much information is still to be obtained, it would seem that certain practices of disposing of feeder calves and the management of the time of disposal markedly affect the incidence of respiratory infection. Further, the interstate movement of these animals, although presumably under the influence of Federal regulations and therefore recorded, are almost impossible to trace. The wide range in the movement of these cattle and the diseases that could be and are disseminated from one area to another would call for stricter record keeping on the part of veterinarians, public health officials, and owners.

A. Steps that can be taken at the regulatory level

1. Animals that are subject to interstate movement be marked as to the state of origin
2. Stricter enforcement of Federal regulations regarding the movement of such animals
3. A requirement that recognized professional dealers have legible and permanent records of the animals sold and/or purchased by them for resale. That these records be made available to any prospective purchaser.

B. Steps that can be taken at the rancher-seller level to alleviate conditions where a correlation exists between a management practice and the incidence of respiratory disease.

1. An educational program should be instituted to inform the rancher-seller of detrimental practices.
   a. Use of vaccines at least 30 days prior to sale
   b. Weaning at least 30 days prior to sale
   c. Castration at least 30 days prior to sale
   d. Treatment for grubs at point of origin
2. An educational program should be undertaken to inform the feeder-seller of the pitfalls in obtaining animals
   a. short transit time
   b. yard-weary cattle
   c. the increase in respiratory involvement that correlated the surgical, vaccination, and management procedures that occur at time of shipment.

IMMUNIZATION PROGRAMS—(PRE-WEANING)

James J. Sheldon, D.V.M.
Department of Animal Pathology
University of Arizona

We're talking about pre-weaning immunization programs. This can't be an instantaneous thing. This is basically the approach that we have to take in the Arizona area, at least, in developing a preconditioning program, and especially tying this into the range operation and preweaning immunization. We have heard many good suggestions aimed at delivering the healthiest possible calf to the feedlot. I think Dr. Bristol emphasized many of the areas that get completely away from immunization but are equally, if not more, important. We were also re-acquainted with specific and unique problems involved in protecting this calf against the most common and most serious pathogens responsible for major economic loss to the feeder. We heard about some of the physiological factors and nutritional factors that come into this same overall goal. I believe it should be obvious to all of us at this point; that to overlook any of these areas in establishing a sound preconditioning program certainly puts us in a position of falling short of this goal. It was also emphasized that this entire program is based on education on the feeder level and at the cow-calf level in the beef industry. With proper education of both of these groups, I think we can alter marketing and shipping procedures through demand from influential groups that are responsible for much of this shipment and marketing methodology. If we are to achieve this goal of delivering a healthy calf to the feedlot, I think we have to consider two major factors. First, we can't get to first base with this program at any time, if we don't look at the entire program from the time the calf is dropped and again look ahead at the possible ultimate geographic distribution and destination of these calves, and the specific problems that they are likely to encounter at their final destination. I think this is essential for a sound program. Along with this, any planned health program must be geared to protect the cow-calf operator and his herd, and yet be planned to allow for maximum protection for that calf at the time it reaches the feedlot and throughout the feeding period. The second major area that can't be overlooked is the necessity for this three to four week minimum holding period after weaning if we are to be successful. I think Don Gill emphasized this very well.

We can talk about a lot of pre-weaning programs but if we wean calves and put them on the truck and take them to the feedlot, we're still asking for trouble. Now the animal newly weaned certainly does experience a psychological upset, and this term has been used with a little apprehension, but I believe it's a good term. If we put ourself in this calf's position we can recognize that there is a little psychology here. The holding period does allow for the completion of a health and nutritional conditioning program that enables that calf to go to a strange environment and withstand the many factors of stress, disease and exposure; as well as prepare the calf to adapt to sound feedlot rations. Pre-weaning immunizations must be aimed at the protection of the animal on a program that fits the need of the cow-calf operation, and secondly, fits initial procedures in the entire preconditioning schedule. Much of this is governed by the type of immunization that we are aiming for at the end of that three to four week holding period—not at the time of weaning. There is a need for maximum protection during the feeding period, but a good start can be made during the pre-weaning period. This is why we are obligated to look at the entire program from the time the calf is dropped through the destination of the animal.

The first step is to review the protection needs for the ranch and that area, from conversations I've had at this meeting, it's obvious to me that variation exists, based on geographical areas of the country and prevalent disease problems in various range areas. Careful consideration here must also be given to labor and handling procedures, as well as cow-calf health during the pre weaning period.

Many things that have been discussed relate to the use and abuse of vaccines applied during the pre-weaning and, as Dr. Bristol pointed out, the post-shipment period In our area, it's not unusual for a cowboy to put hi lepto vaccine and his clostridial bacterins in a saddl bag with one needle, one syringe, ride all day in th Arizona sunshine, and he expects that these product are going to do the job. I think the re-education is a essential to the cow-calf man, and must begin at thi level. Re-education is necessary for management pro cedures on the range as well. I have a tremendous amour of respect for the cowboys in the Arizona area, as far a their ability to handle cattle and gentle these calves, bu I think it's time that we gave up the manure-turpentin poultice, for swelling after castration. This is all pa of a re-education program, and it's somebody's job to d this. The typical Arizona rancher sees his calves onc from the time that they're dropped until they're weane

From this, can we develop a pre-weaning immunization schedule where we can convince him that collecting his calves one additional time prior to weaning can be beneficial? The good ranchers certainly will go along with this one additional collection period.

If the rancher collects his calves twice, we can schedule the following procedures and immunizations: IBR, leptospirosis, blackleg, malignant edema, administeration of Vitamin A, castration; and branding can be carried out during the first collection. You all recall Dr. McKercher's comments regarding IBR protection possibilities after the second week of life. If this is done before the animal is four months of age, re-vaccination for the clostridial organisims and leptospirosis plus the re-administration of vitamin A is indicated; plus the first PI-3 vaccine, if the two dose series is used, can be carried out with a second collection of calves six to eight weeks prior to weaning. Dehorning can also be performed at this time where necessary.

We heard from Dr. Marsh, from Illinois, and he wants some indication of what's been done. This brings up another thing—and that is records. It's not a good idea to write the vaccination procedure on a feed tag or scrap of paper. This is all part of our education and re-education job; to make this rancher record conscious, so that we have some idea of when these biologics were administered and the need for re-administeration. The whole problem of re-education of cowboys and people that are handling biologics is a big job. I think it's one, that if we're going to be successful with a preconditioning program, certainly can't be overlooked. This has been very well emphasized already.

My final comment here is that any program of preconditioning should not be sold to the feeder or the rancher on the basis of a panacea. It represents a specific step forward in all areas of the country, as far as herd health is concerned and control of major diseases responsible for economic loss, but I think we're still going to lose a few along the way. To sell this on the basis of a cure-all has already shaken up some of the ranchers in the area that I am familiar with.

IMMUNIZATION PROGRAMS—(POST-WEANING)

Robert Dickson, D.V.M.
Bakersfield, California

Immunization programs have got to be based on the evidence of the animal need in his new environment. The outline of diseases of the new environment were covered admirably by Dr. McKercher and Dr. Collier. Our consideration supposedly today is whether or not you can immunize against these entities and when should it be done and how. The basic use of antigens in the past has oftentimes been a crutch on which we lean, and I'm afraid that we could add antibiotics into that picture and some of the other things. Oftentimes when the management changes overcome the problem we continue the crutch long after its indication has passed. Attempts to establish immunity to the pasteurella group of organisms has been going on for years and years. Most of you in the room, I'm sure, have had some contact with the theoretical promises of immunization and the heartbreak of field "fizzle-ization"; you can spell it with a "ph" if you want to, to make it technical, but this "fizzle-ization" seems to happen with autogenous bacterins and commercial bacterins as well. Repeats of these expensive and time consuming trials continue, and if the proper criteria are met as outlined, certainly some forward steps can be made; if, in fact, the organism that you're dealing with can be used as an antigen; or if it's a good immunilogical agent. Some of them aren't. The use of viral antigens is really more recent in the background of trying to prevent shipping fever. I'm told, by the popular press and unfortunately also by promotional agents of some companies, that these antigens are showing great promise. In fact, one is led to believe that the results will be incredible. From some of the published literature, and some of the lack of published literature, and from some personal experience with the newer products; I think the work just hasn't been done to prove them incredible. I heartily hope that we aren't headed for some "fizzle-izations". Man's and management's limitations on developing products and on all our products may in themselves be the best preventative procedures. In fact, if the limitations are complied with, perhaps we can do without the antigens in many cases.

Post-weaning immunization, or pre-conditioning immunization should include the following goals: 1. Immunization against repeated sales yard transfer and residence. 2. Immunization against transportation failure and mis-handling. 3. Immunization against starvation and death of rumen micro-organisms. (To me, this is one of the greatest areas for research at this moment.) 4. Immunization against immediate and drastic changes of feed and intake. 5. Immunization against dust and heat, and I guess cold, snow and rain. 6. Immunization against nutritional imbalances and deficiencies. 7. Immunization against improper vaccination. Added together they mean immunization against poor management.

Changes in management can start with educational programs and programs such as this, the sharing of information, and programs in which I'm happy and want to take a part. Research must continue, and this kind of sharing of ideas can generate more interest—I'm all for it. Pre-conditioning, as has been said, is not the entire answer; everyone realized it, but it will certainly bring into focus some of our needs in management changes.

In conclusion, and in discussing immunity of shipping fever the point must be made again; that, although causitive agents are isolated and even if immunological agents are developed that work under proper conditions, proper management of cattle is the best prophylactic agent against shipping fever.

FEEDING CALVES PRIOR TO SHIPMENT

Jack Algeo, Consulting Nutritionist
Santa Ynez, Research Farm
Santa Ynez, California

Many advances have been made in ruminant research in recent years, especially in selection for economic rather than show ring factors, and in nutrition. However, the area of stress and nutrition appears to be regressing rather than going ahead. From 1954 to 1959, when I was resident nutritionist at Sinton and Brown Co., the death loss was held to about 0.25% on yearlings and 1.0 to 2.0% on long-shipped calves, on about 50,000 head per year.

Today we seldom see figures under 0.5% on yearlings and 1.5 to 3.0% on calves. Therefore, with major efforts in management, feeding and veterinary medicine, we seem to be "advancing backwards into the future."

I shall try to set out some principles which I hope will be helpful and present some data on preconditioning, rather than list specific rations, since feeds and rations *per se* must vary greatly over the country.

Programs now in use in backgrounding feedlot cattle indicate many commodities can be used, provided we can be sure it is a paying proposition. For example, we see calves from the following programs coming into our yards:

Alfalfa — pasture, green chop, pellets, cubes and haylage

Ensilage — corn, sorghum, by-product wastes

Combinations of the above with grain, mill feeds, cottonseed hulls, etc.

Our problem areas seem to be in defining accurately the proper amounts of nutrients and drugs to feed, and in management. For example, what are the optimums (not just requirements) for protein, NPN, energy and vitamins under stress and shipment? How long should calves be fed prior to going to the feedyard and to what weights?

I do not feel that high protein levels, such as 14 to 20% are helpful. It is not possible, as shown by Mitchell (1959) and Holt *et al.* (1962) to build protein stores in animals for future use in the same way that energy can be stored. Thus, it is probably fallacious to feed weaned calves 20% protein diets in an attempt to build a store to hold them over shipment. In fact, when experimental animals are fed normal vs. high protein levels, and are then subjected to depletion, weight losses are about equal. In one test with rats, 79% of those on standard protein levels survived 169 days of semistarvation, whereas only 39% of those on high protein diets survived for that length of time. However, weight losses were equal.

Also, there may be detrimental effects from high protein during periods of stress. The Purdue workers (Beeson, personal communication, and Wilson *et al.*, 1966) feel that calves should not be fed high protein legumes on arrival but, preferentially, should be fed low protein grass hays. This is probably a good recommendation for preconditioning as well as receiving. From our experience, we like some (25%) coarsely chopped legume with oat, Sudan, or other grass hay and molasses for filling calves. Afterwards, they can be transitioned to high alfalfa or other programs. We have at times used slightly unbalanced, but very palatable rations for 3 to 5 days just to fill calves after long shipping. One subject which needs investigating is the allowable NPN level in rations for stressed calves. We have made use of 10% of the total protein in the form of NPN with no problems; yet others say none should be used.

The effect of nutrition on disease has been shown in studies with species other than ruminants. For example, Hill (Cornell Nutrition Conference, 1961) demonstrated an increased susceptibility to *Salmonella* infection by increased protein levels in chicks. He also showed a protein and vitamin interaction was involved and that susceptibility was reduced by increasing vitamins. The chicks in question were challenged with *Salmonella* after being held on various levels of protein and vitamins in the diet. Those with the highest protein levels had least resistance to the infection.

Studies of this type in calves are vitally needed. Salmonellosis has been a serious problem in calves in many of our feed yards. In the past, many newly received calves have been started on rather high protein levels, due to the high quality alfalfa in some areas. By so doing, are we predisposing the animals to the infection? Recently, we have used Sudan or grain hay, plus molasses and oral antibiotics in starting calves. The incidence of Salmonellosis appears to be lower; however, the proof is yet to come, since it is in the fall shipping period that the greatest losses usually occur. We intend to pursue this interrelationship this fall.

Another nutrient factor to be considered is energy. In our observations we have noted that when the NE_p of growing rations is above 35 to 38 megcal/cwt. the cattle become too fleshy and economic results are not as good in the finishing period. Thus, it does not seem wise to precondition calves on even moderately high levels of grain

(6 lbs./head/day would be considered moderate). For our purposes, we much prefer calves without high or moderate grain backgrounds. The data which you will see later tend to confirm this position. It is fallacious to believe that by getting calves onto full or nearly full feed prior to shipment, they can be loaded on trucks, hauled long distances and then put right back on feed. Even ("Superbugs") are not likely to remedy the poor results of such a program.

Due to the fact that Western area feeders deal with volume movements of cattle from all sources and must, of necessity, use "put together" cattle rather than strings of fresh calves from the same genetic and environmental background, our efforts must be directed toward lowering losses in animals from this source.

It is highly possible that strings of feeder cattle for small yards may be purchased directly from ranches and profitably preconditioned. However, for the rancher to do this, he would have to be located in an area of relatively cheap feeds. We know instances of excellent results with calves fed 90 days on the ranch. These calves were not "over fed" energy and were held long enough for the rancher to make some money on their gains for his efforts. This is not a philanthropy and the feeder will have to pay for the extra costs which will inevitably occur in short (30 days) preconditioning periods if the rancher has to purchase feed and medical supplies, as well as pay for more labor. We would estimate preconditioning in the short run to cost $8-$12/head. Chart 1 illustrates the effect of short term preconditioning compared to a relatively long term preconditioning. Again, we would like to emphasize that someone must pay for the short period program of preconditioning.

One obvious disadvantage we have noted is that in order to cheapen his gain cost, the rancher may add grain at a rate which will give him a high initial spurt of compensatory growth in the calves directly off range. When this occurs, the feeder who subsequently buys the calves loses any possibility of obtaining the compensatory growth gains and will experience higher feedlot gain costs. It has been our experience that calves from conditioning yards start and perform well in the feedyard unless, as indicated above, the conditioning operator increases the energy, usually grain, too much, or carries the cattle to the heavier weights. In one instance, calves backgrounded on alfalfa green chop with 3.5 pounds of grain per head per day, gave excellent results during the later feeding period. However, the backgrounding operator wanted more efficient gains and the grain was increased to 10 pounds per head per day without our knowledge. The result in the fattening period was an increased feed and conversion ratio of about 1 full point, or 3c per pound on the final gain cost.

Another item we should discuss is that of hormone implantation in growing or preconditioning programs. Our results do not appear to be appreciably changed by the administration of 12 or 15 mg of DES in a 90 to 120 day growing regime. However, higher levels, or reimplanatation at 60 to 80 days will reduce the response to hormones in the fattening phase. We would like to point out in this regard that a straight short term preconditioning program *should not* include hormone implantation of any type. I say this for two reasons: (1) The feeder may have preference for a different product or program than the rancher has in mind. (2) In our experience a hormone challenge in the fact of a nutrient deficient period such as weaning and long shipment can be detrimental. In one experiment by Gassner and Algeo (unpublished data) a few years ago, calves were randomly split into two groups. One group was put on poor quality, low protein dry range and the other on excellent Gramma grass pasture. The calves were further sub-grouped into 0 and 24 mg DES implanted groups. On the excellent pasture, a rather typical DES response of 0.25 pounds per head per day gain over the control was noted. However, the reverse was true on the deficient pasture and the implanted calves lost 0.3 pounds per head per day compared with their controls.

Regarding preconditioning of so-called "put together" calves, I have some data which are rather damaging to our preconditioned ideas on the subject. These data were generated by Dow Chemical Company in cooperation with two California feeding companies. Our group at Santa Ynez was not involved directly in the studies and it is through the generosity of the Dow Company, McCabe Cattle Company and the Noble Ranch that we are able to present these data.

In these studies, mixed and crossbred calves were purchased at Austin, Texas and divided at random into direct shipped and preconditioned groups. The calves for preconditioning were sent to Lake Jackson, Texas for a period of 37 to 41 days. They were fed rations similar to those fed the direct shipped calves on feed in California during the same period. Although feed commodities at Lake Jackson differed from those available in California, the nutrient content of the rations, including protein and mineral constituents as well as energy, were calculated to be the same.

Table 1 shows the design of the investigation. Note that the average total length of time on feed, including preconditioning for the preconditioned replicates, was 199. days. Thus, there was no real difference in the time required to get the cattle to market condition

The data shown in Table 2 are limited to the first period for each treatment. The direct shipped controls con

verted slightly better than the preconditioned calves but gained less per day. However, as indicated in Table 1, there was no real difference in total time between the groups.

Table 3 shows that although the preconditioned groups had appreciably less loss through death and consignment ("chronics") than the control, the cost of feed was slightly higher and the freight costs were considerably higher. The freight differential involved in shipping the calves for preconditioning to the East and back again Westward, accounts for most of the higher overall costs in the preconditioned groups. Had the preconditioning been accomplished at some point west of Austin, there might have been an advantage in favor of the calves on the preconditioning program.

Table 4 merely illustrates the design of the second trial, which was similar to the first one.

As can be seen by the data in Table 5, the overall feed conversion of the preconditioned calves was again poorer than that of the direct shipped calves.

In the second trial (Table 6), as in the first one, the cumulative costs of gain were lower for the direct shipped controls than for the preconditioned calves. This was despite a 44% increase in loss from death and consignment in the control groups.

Table 7 contains pooled data from both trials.

Although it is probably on the fringes of the area of preconditioning, I would like to present some data on performance in the feedlot of cattle from varying backgrounds. These data cover 28,605 animals received a year ago this fall in Arizona and California feedlots. These animals were shipped in the winter and spring of 1966-67. While these data do not represent a controlled experiment, they do rather emphatically point out that the greatest problem is in auction cattle and that well backgrounded calves from alfalfa, silage or wheat pasture have the lowest mortality.

Table 8 shows the effect of background on death loss and medicinal costs.

LITERATURE CITED

Beeson, W. M. 1967. Personal communication.

Gassner, F. X. and J. W. Algeo. 1957. Unpublished data.

Hill, C. H. 1961. Cornell Nutrition Conference.

Holt, L. E., Jr., E. Hale, Jr., and C. N. Kadju. 1962. Journal of the American Medical Assn.

Mitchell, H. H. 1959. Protein and Amino Acid Nutrition. Academic Press, New York, New York.

Wilson, L. L., K. G. MacDonald and H. H. Mayo. 1966. Annual Indiana Cattle Feeders' Day.

Chart 1. Economic Aspects of Preconditioning Calves on the Ranch.

	Preconditioning Period 30 days	90 days
1. Avg. investment/weaned calf	$80.00	$80.00
2. Rancher's cost to produce/lb. calf	22.85¢	22.85¢
3. Avg. weaning wt./calf, lbs.[a]	350	350
4. Growing ration cost/ton (40% concentrate-60% roughage + overhead)	46.00	46.00
5. Avg. daily consumption, lbs.	9.90	10.97
6. Avg. daily feed cost/calf	22.77¢	25.22¢
7. Medical cost/calf	1.50	1.50
8. Death cost/calf (@ 1%)	0.80	0.80
9. Avg. daily gain, lbs.	1.0	1.6
10. Interest/calf (6%/annum)	39.45¢	1.18
11. Total cost/calf (including init. investment)	89.53	106.18
12. Final sales wt./calf, lbs.	380	494
13. Final cost/lb. calf to rancher	23.56¢	21.49¢
14. Rancher's necessary premium per lb. of calf sold to break even	0.71¢	-0-

[a] Weaning weights reflect no weaning stress shrink, since daily gains were calculated to allow for gain back to original weaning weight during the period succeeding weaning.

TRIAL I

TABLE 1. Preconditioning of Calves, Design

	Control - direct shipment				Preconditioned				
	Rep 1	Rep 2	Rep 3	$\bar{x}$	Rep 4	Rep 5	Rep 6	$\bar{x}$	
No. head	100	100	99		98	96	98		
Days fed, avg.	200	200	189	196.3	166	161	156	161.0	199.3[a]
Init. wt., avg.	387	377	396		467	437	455		
preconditioning period, days	0	0	0		37	37	41		

[a] Average time including preconditioning

TRIAL I

Table 2. Effect of Preconditioning on ADG, Feed Consumption and Feed Conversion

Item	Control - direct shipment				Preconditioned			
	Rep 1	Rep 2	Rep 3		Rep 4	Rep 5	Rep 6	$\bar{x}$
First Period								
No. days	76	75	69	73.3	74	71	66	70.3
ADG	2.09	1.77	1.79	1.88	2.39	2.08	2.14	2.20
Feed consumption, lbs.	14.1	12.7	12.4	13.07	15.4	15.7	15.2	15.43
Feed conversion ratio : 1	6.71	7.13	6.89	6.91	6.41	7.51	7.07	6.99
Cumulative								
ADG	2.57	2.44	2.53	2.51	2.74	2.65	2.67	2.69
Feed consumption, lbs.	16.9	16.0	16.9	16.60	18.3	18.6	17.9	18.26
Feed conversion ratio : 1	6.56	6.54	6.65	6.58	6.66	6.99	6.67	6.77

TRIAL I

Table 3. Effect of Death Loss, Consigned Animals and Economics.

Item	Control - direct shipment				Preconditioned			
	Rep 1	Rep 2	Rep 3	$\bar{x}$	Rep 4	Rep 5	Rep 6	$\bar{x}$
Death loss	0	2	2	1.33	1	1	0	0.68
No. consigned	3	3	6	4.00	0	2	0	0.68
Dead + consigned	3	5	8	5.33	1	3	0	1.32
Dead + consigned, %				5.35				1.37
Costs: Freight, avg.	8.99	8.75	9.08	8.94	12.37	11.53	12.03	11.98
Total cost/*lb.* gain, ¢	23.81	24.04	24.43	24.09	24.79	25.57	25.20	25.18
Feeding cost/lb. gain, ¢	19.94	19.89	20.30	20.59	20.21	21.27	20.37	20.61

TRIAL II

Table 4. Preconditioning of Calves, Design.

Item	Control - direct shipment			Preconditioned				
	Rep 1 Fdlt	Rep 2 Fdlt	$\bar{x}$	Rep 3 Fdlt	Rep 3 Fdlt	Rep 4 Fdlt	Rep 4 Fdlt	$\bar{x}$ Pre + Fdlt
No. head	100	100		100		100		
Days fed, avg.	227	235	231	194	226	187	222	224
Init. wt., avg.	390	383	386.5	383		390		386.5
Precondition period, days	0	0	0	32		35		33.5

TRIAL II

Table 5. Effect of Preconditioning on ADG, Feed Consumption and Feed Conversion.

Item	Control - Direct Shipment			Preconditioned Rep 3		Rep 4			
	Fdlt	Fdlt	$\bar{x}$	Fdlt	Pre + Fdlt	Fdlt	Pre + Fdlt	$\bar{x}$ Fdlt	$\bar{x}$ Fdlt + Pre.
ADG	2.26	1.99	2.12	2.56	2.20	2.59	2.18	2.57	2.19
Feed consump., lbs.	15.46	14.89	15.17	17.77	17.05	17.68	16.93	17.72	16.99
Feed conver. ratio:1	6.84	7.49	7.16	6.93	7.75	6.82	7.77	6.87	7.76

TRIAL II

Table 6. Effect of Preconditioning on Death Loss Consigned Animals and Economics

	Control - Direct Shipment			Preconditioned Rep 3		Rep 4			
	Fdlt	Fdlt	$\bar{x}$	Fdlt	Pre + Fdlt	Fdlt	Pre + Fdlt	$\bar{x}$ Fdlt	$\bar{x}$ Pre + Fdlt
Death loss	1	10	5.50	2		5		3.50	
No. head consigned	1	1	1.00	1		1		1.00	
Dead + consigned	2	11	6.50	3		6		4.50	
Dead + consigned, %			6.50					4.50	
Costs Cost/lb. gain, ¢	21.60	23.31	22.45	20.96	23.50	20.86	23.77	20.91	23.63

Table 7. Pooled Data, Trials I and II, Feedlot Effects.

Item	Control 1	2	3	4	5	Σ	%	$\bar{x}$
No. head	100	100	99	100	100	499	—	—
Death loss	0	2	2	1	10	15	3.01	
Consigned	3	3	6	1	1	14	2.80	
Dead + Consigned	3	5	8	2	11	29	5.81	
ADG	2.57	2.44	2.53	2.26	1.99	—	—	2.36
Feed conversion ratio:1	6.56	6.54	6.65	6.84	7.49	—	—	6.82
Total cost/lb. gain, ¢	23.81	24.04	24.43	21.60	23.31	—	—	23.44
	Preconditioned							
No. head	98	96	98	100	100	492	—	—
Death loss	1	1	0	2	5	9	1.83	
Consigned	0	2	0	1	1	4	0.81	
Dead + Consigned	1	3	0	3	6	13	2.64	
ADG	2.74	2.65	2.67	2.20	2.18	—	—	2.49
Feed conversion ratio:1	6.66	6.99	6.67	7.75	7.77	—	—	717
Total cost/lb. gain, ¢	24.79	25.57	25.20	23.50	23.77	—	—	24.57

Table 8. A Comparison of Prior Background Effects on Death Loss in Cattle Shipped to Selected California and Arizona Feedyards.

Background	NO. Lots	No. Head	Percent Death Loss	Medicine Cost/Head	Purchase Weight
Good range	6	11,334	2.11	1.96	527
Dry range	4	8,968	1.11	1.26	614
Alfalfa pellets & cubes	2	374	0.80	1.46	649
Silage & grain	4	3,139	0.28	1.61	607
Wheat pasture	1	1,612	0.68	1.62	566
Auction	3	3,178	3.30	2.17	487
	E = 28,605		$\bar{x}$ = 1.68		

IDENTIFICATION OF CATTLE

Don Williams, D.V.M.
Ada, Oklahoma

The problem of identification is with everybody, it's not necessarily a problem of the cattle industry. Look at the millions and millions of dollars which are spent on television trying to identify their product in the mind of the consumer. It's a problem with government; most of us have been in the service at some time and had an identification number tacked on us. We all have it in social security. Identification is quite a challenge with cattle. I don't know of anything that's been investigated longer and we still have any more problems than in cattle identification. Going back in history, you would have to go back to the early days and cover the branding, when brands were placed up one side and down the other. For a long time, this practice declined this, attempting to prevent damage to hides. Now, with decrease in hide prices and an increase in individual identification we see a wide distribution of brands on the animals. A list of identification methods would also have to include ear notches and "dewlapping." In identification of cattle, one can see that there has been thousands of dollars spent in trying to find a better method. They have been identified from one ear to the other and from top to bottom. They go from ankle bands above the hoof to brands all across the topside of the animal. They go forward from the tail, where there is a plastic double tag with bolts that screw around the tail. Now coming on the market is a plastic tag which will fit in the nose like a nose ring. We have a breeder in our area who has ear tags in both ears, neck chains, number brands on the side, and tattoos; and yet none of the numbers coincide. That pretty well covers the cow from both ends.

To be more specific, we're talking this morning about the identification of pre-conditioned cattle. I will confine my remarks to a program developed by the American Association of Bovine Practitioners to identify these cattle which have been immunized and pre-conditioned, under certain requirements, for shipment. We feel that this is needed if the feed lot owner is able to put confidence in these cattle received and to know that these animals have been treated according to certain specifications, at a point a thousand miles and several sale yards away.

We would like to say that to correctly identify an animal for pre-conditioning, that first, the identification should be such that it is recognized specifically as an identification for preconditioning. Second, there should be some way to identify this animal as to origin, both for trace back in disease problems and secondly for those animals which show superior feedlot performance. Third, this animal must be identified individually, so that it goes through all these various sale barns, is regrouped, split and re-split, it can be traced back to its origin. Fourth, we think that they should be identified as to treatment. As Dr. Marsh emphasized there has to be some way to know what happened to this animal at the point of origin.

The American Association of Bovine Practitioner's program will be known as *Certified-Pre-Conditioned for Health* and be identified by metal tags in such a manner that it will have a meaning on a nationwide basis. The tags will be a green chartreuse tag similar to what is used on the Brucellosis program at the present time. In addition there will be attached a plastic coated tag that is called a bangle. It is felt that this will allow the animals to complete all their trips through the sale barns and get to the point of origin with their identification still intact. We realize that it is possible for some of the bangles to be lost, however, this can be overcome by identifying the serial numbers on the green metal tag with others in the shipment, and comparing treatment on that basis (some of the bangles from the original shipment should still be present). The metal ear tag will have the same type serial number as is presently used in the government tags which are used for Brucellosis and Tuberculosis eradication. The first two digits will indicate the state of origin, in Oklahoma that happens to be 73, then there will be three letters, the first one which shall always be a "P". This has been agreed upon by the United States Department of Agriculture. (In calfhood vaccinations they use the letter V here). The letter P- is for pre-conditioning. The other two letters will be used serially which with the four numeral digits following will allow about six million seven hundred and fifty thousand cattle to be identified from one state. The first such tag in Oklahoma would be 73PAA0001. It is anticipated, and all work done to the present time has indicated, that these numbers will be filed in the office of the State Veterinarian in the state of origin so that at any time the owner of the cattle can be traced through state of origin. On the back of the tag will be the letters AABP, for American Association of Bovine Practitioners; and CPH-Certified Pre-Conditioned for Health.

We will have three sets of Bangles which hang from the metal tag. The white tag with the black lettering requires the immunization, castration, de-horning and some feeding—or at least be sure that the animals are

aware of feed bunks. However, these bangles will carry the notation that this animal has not been pre-weaned. The tag with red lettering is the same program with the exception that this animal will have been pre-weaned at least two weeks. Now, this variance points up the problem of what type of a program shall be designed. An attempt has been made to vary these three programs enough that the dollar value will indicate to the calf producer which program is to his benefit. If it becomes evident that the feed lot owner does not want to pay for weaning, we anticipate that more of the calves will be sold without weaning.

The blue tag will be the deluxe program. These cattle will have been immunized not only against blackleg, malignant edema, leptospirosis, IBR and PI3, but these cattle will also be immunized against bovine virus diarrhea and pasteurella. They will be grub treated and wormed if necessary. On the back of each of these bangles is a place for the date, the treatment and the remarks. This will allow the feeder to get much more information on the cattle he receives.

Again, we feel that placing this tag on an animal in this method is a means of fulfilling all of the requirements of (1) specifically identifying this animal as to a pre-conditioning program, (2) identifying him as to origin, (3) identifying him individually. These requirements will be met by a fairly permanent identification, the metal ear tag. We say fairly permanent, because to our knowledge there is no permanent identification that can be easily read.

The fourth requirement, identification, as to treatment, will be accomplished by the use of the bangle. The bangle will be used almost as an individual health certifica and will probably last as long as is needed.

In closing, I would like to call your attention to ser titers of some Oklahoma calves weaned from a clo herd and maintained on the premise. Approximately of these range calves, which have never been throug sale barn or been on a truck, had titers for IBR, 55% BVD, and 47% to PI3. Thus as much as we would like push all the problems and all the troubles to the feed l and to the method of handling after they leave our cc calf operations, we're convinced that we furnish the s to the problems of the feed lots, in our shipment of rar cattle. Until we are able to get some method of immur ing these cattle and handling them to prevent this str feedlots will continue to have their problems. This h has transferred ownership and a pre-immunization p gram was instigated and for a period of four years, ab seven hundred and fifty of these calves have been shipp annually to the midwest and the results have been v very good. Most of the time, they have been able to vac nate two weeks prior to weaning with IBR, Malign Edema, Blackleg, & Leptospirosis. Since the developm of a bovine virus diarrhea vaccine this has been includ (Under range conditions, no problems with the use this vaccine have been encountered, as has been repor in the feed lot at times of stress.) Again, the results w excellent.

May I repeat? Our shipping fever is due to three thing stress, a viral trigger and pasteurella; and if we can ta aim on all three, we'll make some headway. Thank y

FACTORS AFFECTING SHRINK IN FEEDER CATTLE

Dr. H. L. Self
Animal Science Department
Iowa State University

We at Iowa State have a lot at stake in the cattle feeding business, so it is natural that we also have had an interest in shrink for many years. Our active pursuit of this elusive character, which we refer to as shrink began in 1961 when we first attempted to establish some benchmarks as to exactly what is happening to cattle being transferred from wherever they happen to be, which might be a pasture or an auction barn, or some other facility to the feed yard.

Our Chairman, Dr. Pope, mentioned the Outlying Experimental Farms, we have sixteen such farms, but only seven of them have livestock. We use several hundred steers in the various research programs at these farms during the year. It gives us an opportunity to follow through to the carcass on a good many of these animals, that we would likewise not be able to do if handled by private feeders.

The origin of our research into certain aspects of shrink began as many other research projects do. First there is a problem, second there is recognition of the problem, third there is someone with an interest in pursuing the problem and fourth, resources are available in sufficient quantity to permit the problem to be studied.

Bill Zmolek, Extension Beef Cattle Specialist at Iowa State, and I visited about the problem of shrink in beef cattle and discussed the type of information needed. It was decided to devote enough time and personnel to make at least a minimal contribution toward answering some of the questions. Gene Summers, an Extension Staff member at that time, worked on this project and then later, Dr. Nelson Gay came on the staff and has worked with us on the project.

Many of the asked but unanswered questions were undoubtedly derived from what you might call "old wives tales". For example, it has been said that it is better to transport cattle in gasoline powered trucks, than in diesel powered trucks; better or worse to transport by rail; better to go on an interstate than on a primary highway; better to move at night than it is in the daytime, or vice versa; or this factor or another factor, or a weather front moving through or something else will have a bearing on the amount of shrink and the amount of fill, and various related factors. As we think through the mass of viewpoints expressed on these various subjects, it soon becomes evident that we have had very little factual information upon which to base recommendations. We have felt, and here I speak primarily for myself, that we must first establish our present position, a benchmark, if you please, by collecting data on routine shipments terminating under our supervision. This permitted greater uniformity of post-shipment treatment and reduced the variation in length of recovery periods and other factors under observation.

In some of our shrink studies we were not able to control all factors as well as we would have liked to, but in general we have attempted to keep under control most of those factors which we had an opportunity to control. On those we could not control, we made note of the circumstances. In the first three years, the cattle were purchased by order buyers at any location and under any conditions that the order buyer might use to complete the order. This might be in an auction barn, it might be that part of the cattle were bought from a ranch and the required numbers were filled out from an auction barn, or from some other source. We did not know the origin in most cases, we did not know whether or not pencil shrink was involved, but we did obtain the payweight, and we had some semblance of the conditions under which the pay weights were obtained. From the point of purchase the cattle were transported to the research farm as arranged by the order buyer, as is the usual custom. We had no role in selecting the transfer agent. In general the cattle were weighed on trucks on local elevator scales as they arrived at the farm. Subsequent weighing of the empty truck gave an estimate of the on-farm arrival weight before feed and water, the cattle were usually weighed individually also after they were unloaded and prior to receiving feed or water. Most of the shipments went to the Allee Experimental Farm in northwest Iowa, near Storm Lake for use in a feed lot management research program. In all instances, the cattle were unloaded off the truck and given access to a complete ration in self-feeders. They received feed in no other form or manner from the time they were unloaded as feeder cattle until they went to slaughter. The first ration offered was 65% ground corncobs with the rest being grain and supplement. After a period of 5 to 7 days, the amount of cobs was reduced to 55%, and at weekly intervals it was reduced 10 percentage points to 45, and 35, and finally to 25% ground corncobs by the end of the first 30 days. They remained on this 25% throughout the remainder of the test, except for a few lots in which the cob portion was dropped to 17%.

I have pinpointed procedure up to this point without dealing specifically with shrink and its significance to the cattle industry. For example, the significance of shrink to the Iowa Cattle feeder who is historically a relatively small operator is fairly easy to demonstrate. Please keep in mind that I am referring to the relatively small feeder in the western cornbelt. Someone mentioned yesterday that the average number of cattle per feedlot in Iowa is 64 head. I don't know whether that's correct or not, but I know we have a lot of people who feed only one or two truck loads of cattle each year and are classified as feeders. Others may feed only the calves they raised.

TABLE I

SIGNIFICANCE OF SHRINK TO THE CATTLE FEEDER

Buy
800 lb. YEARLING FEEDER STEER — 9% Shrink . . . 72 lbs.

Sell
1088 lb. SLAUGHTER STEER . . . . . 4.5% Shrink . . . 49 lbs.

TOTAL SHRINK — 121 lbs.

Assume
AVERAGE DAILY GAIN OF 3.0 lbs.
DURING 120 FEEDING PERIOD

$$\frac{\text{121 lbs. lost to shrink}}{\text{3.0 lbs. A. D. G.}} = \text{40 DAYS TO REGAIN SHRINK}$$

But to get on with the demonstration of the role of shrink to a cattle feeder we can use the example shown in Table I. Assume you buy an eight hundred pound yearling steer and he shrinks nine percent during shipment, for a total shrink of 72 pounds. Let us assume this animal does reasonably well in the feed lot and gains an average of three pounds. During transit to market, he shrinks 4½% or another 49 pounds. Adding the 49 pounds to the 72 pounds gives a total of one hundred and twenty-one pounds of shrink that this feed lot manager has suffered. Divide the average daily gain of three pounds into the one hundred and twenty-one pounds lost as shrink. You will find that it takes forty days of the hundred and twenty day feeding period just to regain the shrink. In other words, a third of the time that the cattle were owned they were being fed to regain shrink. I readily admit that this example may have been dramatized just a bit but not very much. The nine percent shrink is not excessive, based on our studies nor is the 4½% shrink too far off. We must recognize that when feed-lot cattle stand overnight without feed or water before they are weighed there will be less shrink. Quite often these are the circumstances under which shrink data are collected. The amount of shrink will be low and the dressing percentage will be high. Such data are then quoted with a great deal of pride due to the lack of understanding of the problem. We should remember that as long as a feed lot operator has steers in his lot, drinking his water, and eating his feed, it's costing him money. Consequently, when that steer walks away from the bunk, and/or the water trough his weight at that instant is the weight we should use as the base weight for all of our data, including shrink and dressing percentage. In the studies reported here, we have used not only the live weight at the farm, but also the hot carcass weight at the packing plant. This eliminates error due to the wide variations among the various packing plants in the amount of adjustment (pencil shrink) used to obtain cold carcass weight.

With these background conditions in mind, let us now look at the data on shrink. The first group of cattle were obtained in the fall of 1961, from near Valentine, Nebraska. They were transported a little over three hundred miles from that point to the Allee Experimental Farm. The weighing conditions are reported to us by the order buyer was that they were to be driven five miles without feed or water to the weighing pens. The pertinent data are shown in Table II.

TABLE II

DATA ON SHRINK AND PURCHASE PRICE FOR YEARLING STEERS (Fall 1961)

DATE	ITEM	PER HEAD	PER CWT	
10-14	114 STEERS	—	—	
	WEIGHT BOUGHT	731	—	
	SHRINK (EST.)	93	12.7	
	WEIGHT DELIVERED	638	—	
	STEER COST	$175.44	$24.00	
	OTHER COST	$ 6.12	$ .84	$24.84
	DELIVERED COSTS	$181.56 (EST.)	$28.46[a]	
11-13	WEIGHT	734	—	
	FEED/DAY	20.5	2.8	
	FEED COST	$9.55	$ 9.90[b]	
	CURRENT COST	$191.11	$26.40	

[a] COST PER HUNDRED WEIGHT DELIVERED
[b] FEED COST PER HUNDRED WEIGHT GAINED FROM DELIVERY WEIGHT

On October 14, we paid for an average of 731 pounds. They arrived at the Allee Farm that same day weighing 638 pounds for a total loss of 93 pounds per steer (12.7%) from pay weight to arrival weight at the farm. May I draw your attention to one or two other items in this table. First, I want to point out that this is the format for the next several slides. The next item to which I invite your attention is the purchase price of $24.00 per hundred pounds. Freight, commission and so forth amounted to

an additional 84c per hundred weight. This would be the price that a feed lot operator would say he paid for these cattle. Or he might say that they were laid in for $24.84. In reality 638 pounds were delivered for a total of $181.56 per steer. This meant that on a per hundred weight delivered basis these cattle actually cost him $28.46 per hundred, which is $3.64 more than we would ordinarily hear quoted by the average Iowa feeder. These cattle were weighed 30 days after delivery. At that time, they average 734 pounds, which is three pounds heavier than the purchase weight thirty days earlier. During that first 30 day period they consumed an average of $9.55 of feed per head, which brought the total cash outlay per head to $191.11. The so called purchase price was $24.84, but in reality, after owning the steers for 30 days, we still had $26.04 per hundred in those steers. I must agree with you that we got shafted on this group of cattle, and a wise person would have changed to another order buyer, but we didn't because they had asked for another chance. The next spring this same organization purchased 120 steers for us. The data are shown in Table III.

TABLE III
DATA ON SHRINK AND PURCHASE PRICE FOR YEARLING STEERS (Spring 1962)

DATE	ITEM	PER HEAD	PER CWT.
10-14	120 STEERS	—	—
	WEIGHT BOUGHT	702	—
	SHRINK (EST.)	28	4.0
	WEIGHT DELIVERED	674	—
	STEER COST	$177.54	$25.30
	OTHER COST	9.02	1.28 ($26.58)
	DELIVERED	$186.56	$27.68[a]
4-23	WEIGHT	702	—
	FEED/DAY	14.9	2.16
	FEED COST	.82	.03[b]
	CURRENT COST	$187.38	$26.69

[a] COST PER HUNDRED WEIGHT DELIVERED.
[b] FEED COST PER HUNDRED WEIGHT GAINED FROM DELIVERY WEIGHT.

On April 19, they arrived weighing 674. We had paid $25.30 on an average weight of 702 pounds. This amounted to a shrink of only four percent. These cattle were purchased off of wheat pasture near Salina, Kansas, and I think I mentioned that the others were North Central Nebraska cattle, or Sand Hills cattle. These Kansas cattle had been put together the previous fall and grazed over winter on wheat pasture. This second group arrived with a purchase cost of $25.30. Other costs amounted to $1.28 per cwt for a total of $26.58. However, you will note that the actual cost for pounds delivered was $27.68. Four days later, on April 23rd we had only $26.69 in those cattle, which is not far from the original cost plus trucking. During that initial four day period the cost of the feed required to get these steers back to their pay weight and to their original cost per hundred pounds averaged only 82c per head.

The data in Tables II and III illustrate the two extremes observed in our studies the past several years. The first group (Table II) had 12.7% shrink and the second group (Table III) had only 4% shrink.

Table IV summarizes the first three years of observations on shrink in cattle bought under the conditions as described for Table II and Table III.

TABLE IV

SUMMARY OF DATA ON SHRINK AND PURCHASE PRICE FOR YEARLINGS AND CALVES (3 YEAR PERIOD)

	Average Per Head	Per Cwt.
970 Yearlings[1]		
Weight bought	676	0
Shrink	65	9.62
Weight delivered	611	0
Animal Cost	$149.13	$22.06
Other costs (feed, truck.)	8.22	1.22 ($23.28)[a]
Total Costs	$157.35	$25.75[b]
335 Calves[2]		
Weight bought	506	0
Shrink	48	9.49
Weight delivered	458	0
Animal costs	$103.19	$20.39
Other costs (feed, truck.)	7.03	1.39 ($21.78)
Total Costs	$110.22	$24.07[b]

[1] Yearlings were purchased from the fall of 1961 through the fall of 1964.
[2] Calves were purchased in the fall of 1963 and spring and fall of 1964.
[a] Total cost per hundred pounds based on purchase weight.
[b] Total cost per hundred pounds based on delivered weight.

Both calves and yearlings were involved. The yearlings were purchased in the fall of '61 through the fall of '64. The calves were cheaper than the yearling because there's a difference in the relative price of feeder cattle in those different seasons. The group of 970 yearlings was made up of 9 shipments. The 335 calves involved 7 shipments. If we examine the data we find the shrink on the yearlings averaged 9.62% and the calves 9.49%. Shrink is always expressed in pounds or percent although this is not a good index to use. But what other index can you use? It is the only thing we can actually measure and record. We are not able to measure "degree of sickness". For example, we are not able to say an animal is 30%

sick, or he is 80% normal, in his resistance to disease. We use weight simply because we can measure weight and it is one way of expressing the stress that these cattle have been under. I use the word "stress" advisedly and although I've heard it used many times here I am not sure it means the same thing to everyone at this conference. I think we need some definitions as to terms, we need a glossary of terms so that we are all on a common footing. For example, I really don't know what "stress" is but if it were being used by everyone on the basis of some standard definition we would at least know what was meant when it was used. Other examples might be the need to establish some standards for hemotacrits, hemoglobin level, or the range within which body temperature is considered normal. We need some definitions so that everybody using these terms is speaking the same language. Stress as we are using it means the animal is subjected to something other than a normal environment, and this of course, speeds up certain of the physiological processes, in the body.

Now, back to shrink, per se, it was pointed out in Table IV that we saw no difference in the percentage of shrink between calves and yearlings. Pooling the yearlings and calves results in an average shrink of about 9½% shrink over the 3 year period. I mentioned earlier in the example in Table I, that 9% of shrink was not excessive. I'm not going to be able to show you data on slaughter cattle because of time limitations but I assure you the 4½% used in Table I is not excessive for slaughter cattle.

After making observations during this first 3 year period it became obvious that we needed to know considerably more about the basic factors influencing shrink in feeder cattle. The next segment of our studies were conducted on a ranch in North-central Texas, near Granbury, with the cooperation of Mr. Clyde Wells, Manager of the Black Estate Ranch. The procedure required that we arrive a few days ahead of the shipping date and weigh the cattle on pasture. This was done by easing them into the handling facilities with as little disturbance as possible. They were passed individually through a chute, weighed and identified individually with a back tag. They were immediately turned back to pasture so that no animal was held for more than a few hours. Two to four days later the cattle were rounded up and shipped in the same manner as if they had not been handled previously. Shipment was accomplished by one of two procedures. *Procedure I:* The cattle were eased into the corral and loaded on the truck that would transport them to Iowa. The empty truck had been weighed previously in town. It was weighed loaded to obtain pay weights. *Procedure II:* The cattle were brought into the corral at the ranch, loaded into the rancher's trucks for delivery to a weigh station and a loading facility fifteen miles away. Upon arrival at the facility they were unloaded, sorted into groups of 10 to 12 for weighing on foot, and then loaded into the Iowa bound trucks following weighing. In Procedure I, the on-truck weight was shrunk 1½% to obtain the pay weight. It should be pointed out that 1½% is not being recommended as a basis for others to use in trading. It is given here for the purpose of information only. Note in Table V that the weight on pasture on April 13, for one load shipped by Procedure I averaged 609 pounds, and the two loads shipped under Procedure II weighed 631 pounds.

TABLE V

COMPARISONS OF TWO PROCEDURES FOR SHIPPING YEARLING STEERS (Spring '65)

	Pasture Wt. 4-13-65	Pay Wt. 4-15-65	Arrival 4-16-65	Shrink From Past. Wt.	Shrink From Pay Wt.
Procedure I[a]	609	591	551	9.52	6.67
Procedure II[b] 135 head	631	617	559	11.41	9.34

[a] Loaded at ranch and purchase weight obtained "on-truck".
[b] Purchase weight obtained "on-foot" at central weighting and loading point.

Two days later, the day of shipment, the pay weight for Procedure I was 591 pounds, and for Procedure II was 617 pounds. On arrival in Iowa (850 miles and 26 hours later) they weighed 551 and 559 pounds respectively, for a shrink of 9.52% and 11.41% from pasture weights. The shrink from pay weight to arrival weight was 6.67% and 9.34% respectively. Thus, the cattle that were handled several times prior to obtaining the pay weight (Procedure II) shrunk more from pay weight than did those cattle that were rounded up and loaded with as little handling as possible and shrunk 1½ percent to obtain pay weight (Procedure I).

It is well to point out that not more than two hours additional travel time enroute was required for Procedure II than for Procedure I and usually the trucks hauling cattle under Procedure I would wait and travel with the trucks involved in Procedure II.

The second series of shipments were processed in October, 1965. The data are shown in Table VI.

TABLE VI

COMPARISON OF TWO PROCEDURES FOR SHIPPING YEARLING STEERS (Fall 1965)

	Pasture Wt. 10-21-65	Pay Wt. 10-25-65	Arrival 10-26-65	Shrink From Past. Wt.	Shrink From Pay Wt.
PROCEDURE I 116 Head	577	577	533	7.63	7.63
PROCEDURE II 117 Head	578	583	534	7.61	8.40

Procedure I cattle weighed 577 pounds on Pasture, on October 21st. Four days later, they weighed 586 pounds on trucks, resulting in a pay weight of 577 pounds (586 less 1½%). The arrival weight in Iowa was 7.63% less than the pasture weight on October 21. The shrink from pay weight also was 7.63%. Under Procedure II, shipped the same day under the same conditions except for the difference in handling, the shrink was 8.4% from pay weight compared to 7.63% for Procedure I. So, again, as in the first shipment, cattle handled several times (Procedure II) not only lost more weight for the producer, but also lost more weight for the purchaser.

The third series was shipped during a period of severe flooding in early May, 1966. Two loads were shipped on May 2nd, according to Procedure I and shrunk 7% from pay weight. On the same date, but under Procedure II there was 7.6% shrink. The weather was made up of drizzle and intermittent showers. Two days later, on May 4, 1966, two loads were shipped on a clear and dry day, one load by each of the two procedures. The load under Procedure I on May 4 shrunk 8% from pay weight and Procedure II cattle shrunk 9.1%. Thus Procedure II cattle shrunk 1.5 percentage points more than cattle shipped by the same procedure two days earlier.

TABLE VII

COMPARISONS OF TWO PROCEDURES FOR SHIPPING YEARLING STEERS (Spring 1966)

	Wt. on Truck	Pay Wt.	Arrival Wt.	Shrink From Total Wt.	Shrink From Pay Wt.
PROCEDURE I					
5-2-66 (58)[1]	671	661	615	8.4%	7.0%
5-4-66 (57)	673	663	610	9.4%	8.0%
PROCEDURE II					
5-2-66 (57)	—	660	609	—	7.6%
5-4-66 (56)	—	657	598	—	9.1%

[1]Number in parenthesis is total steers hauled per truck.

The Procedure I shipment on May 4, shrunk 8.0% from pay weight, or 1 percentage point more than the cattle shipped by the same procedure two days earlier. This appears to be a weather factor, but we don't have enough data to accurately estimate the magnitude of weather effects or to predict its effect on fill and/or shrink. There is very little doubt, however, but that weather does enter into it. Summarizing those three shipments under each of the two procedures, shows that Procedure I, shrink averaged 7.48% and Procedure II averaged 8.74%. Therefore, if you ignore the 1½% pencil shrink involved in Procedure I and bought the cattle at their total on-truck weight, the shrink from pay weight would be about equal for the two procedures.

Several observations were made during the course of these studies; one that may be of interest to you is in regard to the appearance of cattle during loading. When the Procedure I cattle were loaded at the ranch they were rather calm, they were dry and there wasn't a dirty rear-end in them as they went into the truck. The Procedure II cattle, by the time they had been loaded on the truck, moved into town, unloaded, sorted into groups of 10 and 12, weighed and then sorted a little more to go on the truck were dirty with manure from their hip-bones backward. They were excited, nervous; as evidenced by the twitching of their tails, and they were in considerable irritation. It seems that on the basis of these observations that once cattle get nervous and excited and shrinkage is started, they keep on shrinking throughout the transit period. Some of these shipments regained their pay weight in four days, while others required more time, but I believe in every case they were back to pay weight in seven days or less. On an average, the cattle in the earlier studies as shown in the 3 year summary in Table IV, where they were purchased by order buyers from several states required 13 to 17 days to return to their purchase price per hundred pounds. Calves required slightly less time than yearlings to regain price per hundred pounds.

Within each of the three seasons that cattle were shipped from the Black Estate Ranch 10 pairs of animals (total of 20 animals) were selected. The two animals of each pair were similar as to color, weight, and condition. One member of each pair was slaughtered immediately off the pasture and the various component parts were weighed during the slaughter process.

The second member of the pair was shipped to Iowa with the other cattle and slaughtered immediately upon arrival in Iowa. All the component parts, such as the hide, head, shanks, and liver, intestines (Full) and intestines (empty) and carcass were weighed. The relationships were very consistent from one time to another. The data for the 3 seasons are summarized in Table VIII.

The animals in the Texas slaughtered group weighed 609 pounds, and those in the Iowa slaughtered group weighed 611.8 pounds. That's a difference of only 2.5 pounds, which is very acceptable. It should be pointed out that all of the slaughter data is based on pasture weight because it was the only weight that was common to all individuals. The group slaughtered in Texas yielded a carcass equal to 52.7% of the pasture weight, whereas the Iowa group had 50.8% of the pasture weight as carcass, (321.1 pounds vs. 310.0 pounds of carcass). This is a drop approximately 11 pounds or 1.9 percentage points, a statistically highly significant value. The hide was 0.6% less in the Iowa post-haul group which

TABLE VIII

SOURCES OF WEIGHT LOSS IN YEARLING FEEDER STEERS DURING SHIPMENT

	Texas Slaughter (Pre-haul) %	Lbs.	Iowa Slaughter (Post-haul) %	Lbs.	DIFFERENCE
Live Wt. on Pasture		(609.3)		(611.8)	+2.5 ns
% Carcass	52.7	(321.1)	50.8	(310.7)	−1.9 **
% Hide	8.7	(53.1)	8.1	(49.2)	−0.6 **
% Intestines					
full	21.3	(117.2)	18.1	(93.0)	−3.2 **
empty	9.0	(55.1)[1]	8.6	(52.7)[1]	−0.4 ns
% Head	3.7	(22.6)	3.6	(22.1)	−0.1 **
% Shank	2.2	(13.3)	2.1	(12.7)	−0.1 *
% Pluck[2]	2.0	(12.3)	1.8	(10.6)	−0.2 **
% Liver	1.5	(9.0)	1.6	(9.8)	+0.1 **
TOTAL		(549)		(508)	—

ns: No significant difference
*: difference significant at 5% level
**: difference significant at 1% level
[1] : Also included as part of "full" value
[2] : Included heart, lungs, and trachea

was also highly significant. The only way you can get a difference as small as these to be statistically significant is for all of them to be consistently in that same direction. It is evident, therefore, that when these same small differences exist to a statistically significant degree, the measurement was very consistent in that direction. I don't want to leave the impression that statistics make the data, they simply help you analyze it and interpret it. The full intestines were 3.2% lighter in weight following the long haul and the carcass was 1.9% lighter in weight. Both values were highly significant.

The empty weight of the intestines did diminish some, but not a statistically significant amount. The weight of the head and shanks decreased significantly. These latter items are made up of connective tissue, bone and other non-meaty items. A rather profound factor is at work when these non-meaty parts of the anatomy lose significant amounts of weight. It can be readily detected in examining Table VIII that all items lost weight, except the liver, which increased in weight, (from 9.0 pounds to 9.8 pounds). Keep in mind that these were fresh cattle. They were 24 to 26 hours from the pasture to the feed lot, they were not under stress a long period of time. They were all handled by the same trucking firm, they all came from the same ranch, and they had been together for a period of at least four to five months prior to the time we purchased them. It is evident that anything that can decrease the weight of something as non-muscular as a shank, is exerting a severe amount of otherwise unmeasurable stress on those animals. Here again, we encounter the word stress and we use it without really knowing what it means. Using the term "stress" in connection with these cattle, leads us to conclude that they were under sufficient stress to cause a statistically significant loss of weight in those non-meaty parts of the carcass. A few simple calculations also show that up to 40% of the loss in weight is occurring in the carcass, and the remaining 50 to 60% is due to losses from the digestive tract.

It is not only important to know this, but to learn something of the magnitude of these losses is also very important. This knowledge in itself may not be overly important to the cattle feeder, but it is important to the research worker and also to the person who is selling to slaughter on a carcass basis. If his cattle stand at the packing plant over night he is reducing the pounds of beef on the rail the next day.

We used electrolytes on a random half of the steers shipped from Texas to Iowa, and could not demonstrate any benefit during the post-haul period by this use. We first became interested in this because one group of cattle shipped from Amarillo were destined for two different farms. They were divided so that 35 calves went to one farm and 54 calves went to another farm. The 54 head that went to one farm had a couple of calves that looked a little droopy and were treated by the local veterinarian because he is usually on hand when shipments arrive or he will see the cattle within hours after they arrive

In addition to treating the two droopy calves, he put the entire group on electrolytes for a couple of days. The 35 head that went to the other farm did not appear to need medical attention upon arrival. We had no further trouble with the 54 head that were put on electrolytes. However, the group of 35 head became quite ill and 2 died. Whatever it was that they broke with was transmitted to other cattle that were already on the farm. As a result, our medicine and veterinary bill was excessive, plus losing the value of the two animals lost by death. Since all of these cattle had originally been one group on the same truck and illness occurred only in the group not receiving electrolytes, we thought it within the realm of possibility that the electrolytes staved off illness in the group of 54 head. This stimulated us to use electrolytes in randomly selected groups in later shipments, but we saw no evidence of a beneficial effect from the electrolytes. It is doubtful that the Texas cattle used in these shrink studies had been sufficiently stressed to permit them to benefit from electrolyte treatment.

Although we feel that the data presented to you today are helpful, they are not conclusive. They point out the need for more facts. And, I say in all seriousness, that facts are important to any decision making process. It seems to me that up to now facts presented in this meeting have been far outnumbered by opinions. Facts and opinions are not necessarily the same. Therefore, before a full fledged program with recommendations for preconditioning or any other program is put together we should attempt to get more factual data upon which to base recommendations. There should be more data around and available than we have heard here. Recommendations not based on carefully ascertained facts are always questionable and there is too much at stake to risk losing the faith of those we hope to help. Differences of opinion may occur due to different interpretations of the same data, but this is not the usual situation.

Mr. Chairman, I appreciate the opportunity of participating in the meeting. Our data have thrown a little light on some of the problems at a slightly different angle. In our work we have not attempted to get into the medical problems and there may be some of those areas that should be more carefully examined. If anyone has suggestions to offer, we would be happy to receive them.

SUMMARY OF PROBLEMS AND RESEARCH OPPORTUNITIES FOR THOSE IN ANIMAL SCIENCE RESEARCH

Dr. O. Burr Ross
Vice President For Research
Oklahoma State University

With the array of talent that has been before you at this conference, I doubt seriously that anything I can say will be apropos.

I would like to visit with you just briefly about some of the general problems as I see them related to animal scientists and their activities in the years ahead. We might start with some basic economics. I suppose the first question one might raise is, "Can we make money in the beef cattle business?" I shall not try to answer this because for a day and a half we have been trying to answer this question. Specifically I might ask, "Will we have more product to sell if managerial systems are changed?"

Let's start with marketing as one of the items of management. It seems to me that one of the fallacies that we live with in agriculture is that we expect to be able to sell all of our product at a price, irrespective of quality. I think this, in a sense, is what we ask of our packer friends. If we have a boar that has been casterated and obviously has considerable infection, we frequently rush him to the stockyards, and we are generally disappointed if he doesn't bring top stag prices. How many times have you heard people talk about a particular cow that is "all torn up" inside from calving, perhaps with infection, saying, "If I can just get the fever down a bit, I can get her through the auction sale." These are some of the things that I think are fundamentally wrong. As livestock producers we should try and market a quality product and not try to shift ownership of an animal obviously sick or undesirable for human food just to get rid of the animal. Somehow or other we expect our marketing agencies to take everything we produce, regardless of quality. What about the people who sell their calves, round them up, load them out, get the check and kiss them goodbye? If they don't do well for the person who bought them, they look for another buyer next year.

I'm wondering if we're paying enough attention to the quality, or reputation, of the product we produce. Let's consider the carcass of the cattle we produce. Is it acceptable to modern-day buyers, and adaptable to modern merchandising methods? Is it meaty enough, too fat, wasty, or is it just right? We need to know these things about the end product of our work. Dr. Chambers, at this station some years ago, found that the acceptability of fed beef from animals 15 to 18 months old was essentially equal, regardless of the genetic background. The actual percentage of lean muscle of carcasses, and consequently the actual value, may differ a great deal among cattle of differing genetic backgrounds. What are we doing about the problem of producing muscular high-value carcasses with maximum cutability and retail yeild? It seems to me that we must look this problem squarely in the face and, first of all, admit that we do have a problem, that carcasses differ a great deal in cutability and retail yield of closely trimmed cuts.

As I have studied the industry, it seems to me that this is one of our major problems. If you agree we must have more beef in our beef cattle, let's all admit it and then do something about it. I'm not sure the industry as a whole would agree today that this is a serious problem. I think there is some reluctance on the part of research people to face up to this issue. We could probably get agreement that the meatiness of our product can be influenced to some extent by breeding and to some extent by feeding. Very definitely I feel we could get agreement that our population of cattle today can be fed too long, get too fat and too wasty. We can overcome this to some extent by marketing them at younger ages and lighter weights. Maybe we have reached the limit of this approach. Are we really serious about doing something about the meatiness and cutability of beef carcasses? I ask myself this many times each week. I'm not really sure that we want to face up to this problem because it might upset a status quo, someone's reputation cattle, cast undesirable reflections upon a particular strain or breed of cattle, or create other public relations problems. We went through all kinds of problems in the swine industry, trying to get meat type hogs. We haven't reached our target yet—but, we've come a long way. We have made substantial progress in this area in the sheep industry.

There are some questions that need to be answered which are physiological in nature as we reach for meat type muscular cattle. What happens if we develop a beef animal that does not have subcutaneous fat, does not have the capability of storing large amounts of fat throughout the body, and then ask that cow to weather a blizzard in Montana, Wyoming or some other state?

Is some, or a lot of, fat under the hide desirable under such environmental conditions or conditions of scanty food supplies? This one has not been studied extensively and may present a problem if an animal is bred that is too lean and muscular.

I agree with Dr. Self when he said this morning, "let's define stress." I don't think that we've done this. I've been listening to this word in the poultry industry for fifteen years or longer, and I still don't believe we have it adequately defined, so that we communicate with others when we try to ascertain causes or affects upon the animal. We haven't, in my opinion, scratched the surface relative to environmental research as it pertains to animals. We haven't the technology nor the resources in sight to control the environment of cattle, as we have in the case of the pig or chicken. We are still old-fashioned enough to believe that cattle should go out and harvest grass and other forage crops which man cannot use directly.

Another factor, or facet, which we have not studied extensively, is the efficient use of the offal of our meat animals. I don't know whose specific job it is, whether the job of the experiment station or the job of industry, but it seems to me that someone has dropped the ball; we are not doing much about it. As you know, there was a time when the value of offal would pay for all of the slaughter and chilling costs. Today it won't do this. Certain biologicals that once came almost entirely from the offal of our aniamls are today being made synthetically. The casings for sausages and other products of this type are being replaced by plastic products. Dr. Walters of our Animal Science group inquired within the past few days relative to what the value of offal is today. The current value of offal from a thousand-pound steer is now about ten dollars, or slightly less, and that will not pay for the slaughtering, chilling, and other costs of preparing the product for the consumer.

There has been quite a bit of conversation about hides during the past year, as you know. Everyone comes up perhaps with a different idea about the cause of depressed prices for hides. In August of 1966 heavy steer hides were bringing 17¾ cents, in August, 1967, they were bringing 10¾ cents. What does this mean? It means that this price decrease results in a reduction of 45 cents per hundred weight of a 1,000 pound steer. Whether you think it is because of our export policy or something else is probably immaterial. You are well aware, I'm sure, of competitive products—a product competitive with leather. The point that I would like to raise is—"What land-grant institution, what university, do you know of today that is actually doing any research on hides, or trying to improve leather? This is an important commodity within the livestock business.

We lost the lard market some years ago and never quite regained it. We're in the process of losing the fiber market, specifically, cotton and wool. Is there something that can be done with animal products, leather or wool, that would make it better than the synthetic competitive products?

An amazing set of statistics was called to my attention recently by Dr. Walters of our staff. He bought bones from a major packer, bones coming from the beef boning operation, and hired students to remove all soft tissue. He found that 25% of the total weight of the purchased bones consisted of soft tissue, largely lean meat. On every ton of bones coming from the beef boning department there was five hundred pounds of soft, edible tissue. The question the packing plant faces is how they can recover this tissue economically. Labor cost is a major problem. Who is researching this kind of problem at the present time—who should do the research? Today approximately eight billion pounds of beef goes into sausage and prepared meat products—eight billion pounds. Carcasses that would produce that much boned beef would yield two and two-thirds billion pounds of bones, or 1.3 million tons of bones. If all bones yielded twenty-five percent lean or soft tissue, 325 tons of lean tissue would be lost as human food. At 20¢ a pound, this would be worth $130,000,000. In tankage or meat scraps that lean tissue would be worth only a fraction of that amount, a sizeable cost to the industry. It appears to me that somebody ought to be looking at this particular problem.

It meat protein superior to other kinds of protein? For growth and some body functions perhaps not. What about longevity of human life? Do we know much about the state of mind, whether people eating meatless meals are as satisfied as those fed meat meals? Recently, as I have become more and more involved with an overall research effort, I have asked myself these questions—"How important are animal proteins from the standpoint of the learning process? What is the importance of animal protein from the standpoint of initiative or motivation of the people of the world—or is this question unimportant?" I don't believe we really know the answers to these questions.

I think that we have much to learn about the relationship of nutrition to many of the factors that we talk about as being stress, or that which is harmful to health. I won't dwell very much in regard to researchable areas relative to nutrition because many related areas have been discussed at this meeting.

It seems to me that we know precious little about the relationship of the soil to the plant, to the animal and to the human. We have touched the surface from the standpoint of soil management relative to metabolites

or nutrients in the plant, but what do they do for the animal? In addition, I have been quite interested in what is to be done with all of the refuse from the corn plant in the corn belt. Can it be utilized by animals to produce human food? Can stalks, cobs and shucks be utilized as sources of energy for ruminants? I think we are overlooking a fruitful area of research in regard to products of this kind that are wasted today.

Least cost rations, of course, have been explored pretty much through the years. Today, with computors, I think we can do a much better job of feeding economically.

In the early days of SPF pigs, many of my colleagues told me that they did not want to use SPF pigs for research because they were not normal pigs. I would ask you, what is the normal disease level in pigs? What is a normal disease level in cattle? If we can define this, then I think we can begin to do some very basic and fundamental research regarding breeding, nutrition and even stress, or environmental factors. I doubt that we will even know what abnormal level of disease in our animal is, or whether we should even be concerned. We should do all in our power to practice preventive medicine and produce animals as free of disease as possible within the limits of practicality.

I never cease to be amazed at how little work we're actually doing on bloat. This morning it was mentioned that in the Imperial Valley, alfalfa can be grazed and no hazards, apparently, are incurred from bloat. And I'm wondering how much or how little we should do relative to this malady. Alfalfa is a wonderful forage plant. How can we use it more efficiently, how can we control alfalfa bloat in Oklahoma?

From a physiology standpoint, we are still troubled with problems of sterility of both the male and the female. We certainly need higher percentage calf crops—more calves marketed. Too many producers market too few calves per cow. Maybe we ought to be selecting for twins. I don't know whether anybody's doing this in the case of cattle but, it seems to me, we've got to do something to increase the percentage of marketable calves.

Much has been said about management. The thing that bothers me is—how de we define management, and particularly, how do we define good management? Generally speaking, I suppose good management is anything you do that gives you a high percentage calf crop and heavy selling weight. I think we need to define this particular term more precisely. We need more precise management research.

What I have said this afternoon is certainly not very profound. These are areas of interest that you have been talking about or thinking about for some time. Perhaps some are just biases or prejudices of mine. We need to face up to many of these problems and, in speaking to you animal scientists, are you willing to take on the job that to date we have checked to the food technologists, the chemist, or somebody else, rather than do it ourselves? I am referring to any research which will improve our product, up-grade by-products, or improve efficiency of production.

SUMMARY OF PROBLEMS AND RESEARCH OPPORTUNITIES FOR THE VETERINARY RESEARCH INSTITUTIONS

Nelson B. King, D.V.M.
Cooperative State Research, USDA
Washington, D. C.

I've enjoyed very much listening to these interesting discussions during the past two days, and I think there's one thing most of all that stands out and is quite clear to me; and that is, that there are several questions that we do not have the answers to. What is not clear to me though, is who is going to do the research on this work and where they are going to get the money. I am very much concerned about the trends in veterinary medical research, things that are going on in our colleges and our departments of veterinary science, and I want to take a little bit of time to discuss this with you, because I know there are a good many veterinarians here, extension people, and heads of departments.

Although the United States enjoys one of the highest standards of animal health in the world, total losses to the public from disease and parasites are estimated to be approximately 2.6 billion dollars a year. This loss is approximately twelve percent of the yearly income from livestock and poultry and their products. At present more than two hundred specific diseases and parasite conditions of economic importance to the livestock and poultry producers are under study by research scientists in the experiment stations in our colleges of veterinary medicine and industry. Many of these problems, although of considerable importance have received only superficial or perhaps intermittent study because of the lack of funds for conducting research in depth.

Diseases of feedlot cattle are a major cause of economic loss to producers. For example, James H. Warner, beef cattle specialist at Ohio State University, completed a survey in 1966 and he indicated that Ohio cattle feeders were losing about $6.73 per head annually to disease. This represented a total loss of approximately 3.5 million dollars in Ohio alone. Approximately 826 farms and 119,000 head of cattle were involved in this survey. Shipping fever occured on 67%, IBR on 21% of the farms and BVD on 23%. The results of this survey indicate an urgency to conduct research which hopefully will provide necessary information for reducing such heavy losses. Although not reported in the survey some of the other feedlot diseases gaining prominence are, liver abcesses, the encophalitides and numerous gastro-intestinal problems.

Examples of livestock and poultry diseases that continue to take a heavy economic toll in addition to these are; respiratory diseases of poultry, swine, sheep and horses; intestinal diseases of calves and pigs; reproductive diseases; for example, vibriosis, leptospirosis, viral agents such as BVD, EBA, IBR, and others not yet clearly identified. There are other diseases which could become catastrophic at any time; for example, salmonellosis, mycoplasmosis, anaplasmosis, equine infectious anemia, etc. Exotic diseases, such as foot and mouth disease, African horse sickness, rinderpest, and African swine fever are a constant menace to the producers in this country.

In addition to the economic pressure of continuing losses, the responsibility to increase the effectiveness of animal disease control is made more urgent because many of these diseases are transmissible between animals and man. For example, salmonellosis is gaining quite prominent attention in this country at the present time. Moreover, the increasing incidence of antibiotic-resistant micro-organisms is making the effective treatment of animal diseases more difficult, and may pose an additional public health threat. Furthermore, the presence of certain livestock and poultry diseases, such as hog cholera, salmonellosis, and trichinosis are limiting the demand for our livestock and livestock products on the world market.

The essentials for an expanded livestock production are; first of all, suitable types of livestock for local conditions; two, satisfactory feeding and husbandry practices; three, high level of reproductive performance; and four, maximum degree of animal health. Although great strides have been taken in the United States on all these essentials, the most note-worthy have been in breeding, nutrition and management. The presence of animal disease has been, and continues to be, the most important limiting factor in the continuing livestock and poultry expansion.

In a report of a study sponsored jointly by an Association of State Universities and Land Grant Colleges and the U. S. Department of Agriculture, (report is now known as the National Program of Research for Agriculture), it was pointed out that infectious diseases represent the single greatest hazard to the production of an adequate and wholesome supply of animal protein. Recognizing the seriousness of this problem, this study group recommended that research on livestock reproductive per-

formance be increased by 119% and work on other disease problems be increased by at least 81% during the next ten years. When you speak to livestock producers, as most of you people do, and you ask them what factor or factors limit livestock and poultry production most, they generally point to the exhorbitant losses due to diseases. Many administrators of agricultural research concur in this appraisal. However, there has been little or no increase in the percent of agricultural experiment stations' expenditures for livestock and parasite disease research during the last decade. For example, in 1953 approximately three million dollars in federal grant—this would be Hatch Funds—which our Agency administers, and state funds were expended by the state experiment station for livestock and poultry disease research. This was approximately 17% of the total expended for research on livestock and poultry. Some ten years later, expenditures for disease had expanded to 8.75 million. But, this still represented only 20% of the total expended for research on livestock and poultry. Even though the expenditures have tripled in this time, the number of scientists conducting studies on livestock diseases remained about the same. This indicated to me that the increase in funds reflected an increase in costs of doing research and not in any increase in effort.

In 1965, the State agricultural experiment stations expended about $10.5 million in support of livestock and poultry diseases. This amount included monies from state appropriations, and Federal Hatch funds (U.S. Department of Agriculture). Some of the support from industry and Federal agencies other than the U. S. Department of Agriculture are also included. However, the bulk of the Human Health-oriented funds expended by veterinary medical scientists are not included in this figure. The U. S. Department of Agriculture spent approximately $10.7 million in support of livestock and poultry disease research (exclusive of the Federal Hatch support administered by the Cooperative State Research Service).

The $21.2 million expended by the State agricultural experiment stations and the U. S. Department of Agriculture represented the major part of the support for livestock and poultry disease research. If this estimate is correct, the total cost of research work on animal diseases and parasites is little more than one-half of one percent of the losses. In my opinion, this is not an adequate program and if the support continues at this level, the disease and parasite losses will cost the public approximately twice as much by the year 2000. To be sure, by this time research will have found methods for controlling or eradicating many of the important disease and parasite problems. However, greater numbers of livestock and more intensive production methods will inevitably lead to a substantial increase in disease problems if means for curtailing these losses are not developed.

The lack of research support for work on livestock and poultry diseases has caused research workers to seek other support. Many scientists in the colleges and schools of veterinary medicine and in the agricultural experiment stations have been successful in obtaining support for human health-related research. However, in order to qualify for this support, it has been necessary to direct emphasis toward problems for which these agencies seek answers. With the increased support and emphasis on human health-related research many of our best trained scientists are no longer concerned with the urgent disease problems facing the livestock producer. Our most promising graduate students are being trained in human health-related research.

As a result of these trends, logical questions are being asked by scientists and adminstrators. Who will do the livestock and poultry disease research in the future? What are the consequences resulting from this shortage of funds for support of livestock and poultry disease research?

There are some who feel that increased attention will be paid to livestock disease research in due time, when animal protein shortages become acute. Can we afford to wait and react to this situation or should bold action be taken now to bolster support for livestock disease research and bring about a better balanced research program?

Even though I have geared my comments to the veterinary medical aspects of research, I am glad to see the interest of others with varied background in this problem of maintaining health in feedlot cattle. There are no sharp divisions between livestock disease research and animal science or husbandry research.

This is the reason I am so optimistic about what has been discussed during this symposium pertaining to conditioning cattle for the feedlot. It has been my feeling for some time that while we need more knowledge concerning the disease agents involved in respiratory disease of cattle perhaps the problem is so complex—so many agents involved—that research on means of maintaining resistance and reducing stress and exposure may offer greater chance for success at this time. This approach which has been the subject of this symposium may offer the best probability for success in reducing the severe losses due to these diseases.

Some of the questions which need answering are:

(1) Will conditioned cattle perform better in the feedlot

Can the feedlot operator afford to pay a higher price for these calves?

(2) Can resistance be better maintained and exposure reduced by shipping calves direct from range to feedlots thus avoiding the auction markets? Would this practice be economically feasible?

(3) What management practices need to be adopted by farmers, ranchers and feedlot operators in order to maintain resistance and reduce exposure to disease producing agents? Can operators continually make additions to the feedlots and keep diseases under control?

(4) The cause of feedlot diseases need to be identified, and better diagnostic tools need to be developed for shipping fever, encephalitides, footrot, toxemias, liver abscesses, parasitisms, etc.

(5) Is the prophlactic and therapeutic uses of antibiotics contributing to or alleviating disease of feedlot cattle?

(6) How effective are vaccines, antisera and bacterins in preventing disease?

(7) What is the role of mycoplasma. What is the significance of low-level titers to PI3, BVD?

(8) What is the interrelationship of Pasteurella and other bacterial agents to PI3, reoviruses, adenoviruses, enteroviruses etc?

In closing, I would like to emphasize a need for caution in proceeding with the development of the preconditioning program. I don't believe we have the facts and figures to prove that such a program, as discussed here, is economically sound. To be sure, it seems logical that such a program should offer good possibility for success. But good controlled research is needed to provide the necessary information before the program can be recommended to ranchers and feedlot operators. Proceeding without good reliable information could delay indefinitely the acceptance of what may be a very valuable adjunct to present disease control procedures.

THE OPPORTUNITIES FOR FIELD RESEARCH AND EDUCATIONAL PROGRAMS IN EXTENSION

Dr. Dixon Hubbard
Federal Extension Service
Washington, D. C.

The major function of the Cooperative Agricultural Extension Service is the educational arm of the Land Grant system. In other words, our prime responsibility is to take research findings to the agricultural public. Also, we are responsible for identifying problems that need to be researched and relay this information back to researchers. I am happy to say that Extension has played a major role in the development of this symposium on conditioning cattle to try to identify the problems and stimulate efforts toward solving these problems. Also, when the information that has been presented at this symposium has been evaluated and we have facts to support any improved practices that will be beneficial to beef producers, we will make every effort to get them applied. However, I am sure that if one thing can be said about this symposium, it has revealed that a lot more research will be necessary before the problems we are facing will be solved. Thus, I hope that this symposium will help to stimulate needed research on the problems and that funds can be obtained to do this research.

We have come together to discuss one of many inefficiencies in the beef cattle industry yet it is one of the most pressing problems presently facing the industry. The challenge is to:

First, agree upon the basic problem—identify the critical factors causing the problem so that causes can be dealt with instead of symptoms;
Second, decide what can reasonably be done to solve the problem or problems in a practical way;
Third, establish and agree upon a program and procedures for carrying out; and
Fourth, organize to execute the program.

These, as many of you know, are the basic steps for problem solving.

I don't believe there is any doubt that we can agree on the basic problem and I believe we can identify the critical factors causing the problem. However, the next three basic steps in the problem solving process in this particular case are considerably more difficult. The primary reason for this is that the U.S. cattle and beef economy is a highly complex and interrelated structure. It consists of thousands of diverse production and marketing units ranging from giant corporations to small independent producers and distributors. Although the various segments have certain common interests and goals, it must be recognized, also, that there are many diverse interests within the total industry that sometimes are difficult to understand and reconcile.

It would be possible to reduce the cost of feeding cattle ten to twenty dollars a head by devising methods of controlling one disease syndrome—that group of diseases called "shipping fever". This disease causes 75 to 80 percent of all losses from disease. In comparison, because of routine immunization practices, other diseases are of minor significance. And bloat, which isn't even a disease, is the second largest cause of reduced efficiency in most feedlots. Shipping fever is the big problem. It is causing a gigantic loss to the beef industry and is recognized throughout the industry as the major disease problem. However, the solution to this problem is not as obvious as the problem itself and becomes very complex when factors such as differences in size, age, management, and disease exposure of animals are considered along with the variation in systems of marketing. Also, if death of calves was the big loss, the industry would have little to worry about. Feeders are doing an excellent job of controlling death losses from disease with the tools at hand. However, the relatively small death loss from this disease is not a measure of its economic importance. On the average, it is estimated that from 10 to 30 days are required to get calves over the effects of shipping fever and back to their initial pay weight. Reported incidence among various lots of incoming cattle varies from ten to over forty percent requiring treatment. Thus the major losses from shipping fever are increased time required to start cattle gaining weight, labor necessitated by extra care and treatment, drug cost of treatment, and attempts at prevention.

The major objective of all segments of the beef industry should be to maximize income from the combined use of resources under their control. There is little doubt that all segments of the beef industry from producer to retailer could profit from conditioning cattle. However, each is in a somewhat different economic and competitive position and naturally wants to protect or improve that position. The various segments of the beef industry are interdependent on the success of one another. Each

segment of the beef industry is out to get just as big a piece of the pie as possible. Business is business and if one segment makes a dollar, some other segment frequently loses a dollar. At the present time, there is doubt in my mind whether the various segments of the industry will cooperate to the extent that all of them will profit from a conditioning program.

Before any single problem can be solved, it must be put in its proper perspective as it relates to the total industry. Thus, before we plan a course of action, I think we had better take a closer look at beef production. The beef industry has been a tremendous growth industry and it takes great pride that cattlemen are furnishing 100 plus pounds of beef per capita annually. For the period 1946 to 1966 beef production increased from 9.3 billion pounds to 19.7 billion pounds or 110 percent. However, half the time, this beef was produced at a loss to one or more segments of the industry. Because U.S. beef can be purchased at less cost than ever before as measured by "labor minutes" per pound appears to be nothing but a measure of inadequate merchandising by an industry making approximately two percent on investment.

Presently, there are very few places in the U.S. where a cow unit can be maintained for less than $100 per year if all costs of production are considered. This puts a real squeeze on anyone trying to make a living in this business. The feeders are also having difficulty making money because feeder prices are too high to be compatable with economic feedlot finishing.

We hear a lot about feeders feeding plain or Okie type cattle. The reason for this is these cattle have consistently been more profitable than better grades of feeder cattle because of a lower initial investment, they are less fleshy and make compensatory gain, and they grade well when finished primarily because of age. Even though fresh well managed cattle frequently get just as sick if not sicker than these cattle, there is little doubt that mismanaged cattle are the major contributors to the shipping fever problem.

It is apparent that the cowman producing these cattle is losing money if he considers all fixed and variable cost of production. However, as long as these long age light weight cattle are available at reduced prices, feeders will continue to feed them in preference to better grades of cattle even though they are sick. The reason for this is that the feedlot finishing industry is the most sophisticated business in beef production today. In other words, they are generally better businessmen than cow-calf producers. They have better records and these records have taught them that money is made in the cattle feeding business in three major ways—buying, feeding, and selling. Presently, the combination of cost of cattle in the lot, cost of gain, and value when finished favors feeding plain cattle that have been mismanaged, in spite of the health problem they cause. They are still usually a good buy when they are healed and are on full feed. The statement has been made at this symposium that we want to eliminate this situation. I am sure this is true for the group that has met here to discuss conditioning of cattle. However, I am not sure that it is true for those that are capitalizing on this situation. My own father is one of these individuals and I assure you he likes these cattle and will continue to buy them because they make him money.

What this all really means is that the majority of the money presently being made by cattle feeders (if any is being made) is made by capitalizing on the mismanagement of the cow-calf producer. This is just good business for the cattle feeder and he would be foolish not to capitalize on it. However, when we advocate feeding these cattle in preference to higher quality cattle that have been properly managed, we are basically admitting that there just isn't enough money in the beef producing business for the good cow-calf producer and the good feeder to both have a decent profit. It definitely is not a healthy situation when the only way an industry can make money is by capitalizing on the mismanagement of another segment of the same industry.

This brings out another point. What type of cattlemen are we dealing with? A cattleman that leaves his bull with cows year round, doesn't control external or internal parasites, doesn't castrate or dehorn, doesn't keep any records or performance test, doesn't use proper feeding or marketing practices and many more known management practices that would improve efficiency of production. We have been trying to improve this situation through research and Extension education for a multitude of years and it still exists. These are the producers that make the major contribution to the shipping fever problem. It has already been stated in this symposium that well managed cattle that reach the feedlot fresh are not a major problem.

Thus, we are dealing with the age-old problem of poor management. The question is, if we haven't been able to get this type of producer to adopt other improved management practices, can we get him to condition cattle?

Another paradox in beef production is that cattlemen of this modern age can really be classified into three separate categories: (1) The traditional cattleman. Most of his income is from the cattle business. He probably inherited his ranch or has been in the cattle business most of his life. Thus, in most cases, he owns his land and cattle. Under these conditions, he has a reasonable profit per animal and volume helps him retain a substantial income. (2) The land stocker. This cattleman has land

available for grazing so he stocks it with cattle. Maybe this land contains oil or minerals and/or is being held as an investment for price appreciation. This category, could also include the ex-farmer or rancher who now works in a factory, but runs cattle on the side to supplement his income. Profits might be small, but this beats just wasting time and land resources. Also, many farmers who run small bunches of cattle would be classed in this category. (3) The doctor-lawyer-businessman cattleman. He owns a ranch primarily for its esthetic value, income tax reasons, outdoor recreation, or some other indirect economic return. This group not only includes the very wealthy hobby breeders, but also people like bankers, house builders, feed dealers, drug store operators, college professors, and others who enjoy weekend ranching.

There are many individuals who cannot be placed into only one of these categories. They really might have combined goals or reasons for being in the cattle business. At any rate, this division of cattlemen into categories provides us with a basis for some interesting analysis.

Each of these classifications seems to react differently to price changes. The first group, and even some of the second, either cut back or build up herds according to the profitability of the cattle business. Most of group two and practically all of the last category do not increase or decrease their operations to maximize cattle profits. They have other goals in mind. It might very well be that we must learn how to measure these other values of the cattle business, in order to do any logical industry planning.

It is significant that a major portion of the producers do not respond to normal economic principles and this makes it very difficult to improve efficiency by any means. However, there are cattlemen who are in the business to make money and must make money to survive. Basically, they are the ones that are interested in improving efficiency of beef production and this is what we are really trying to do by conditioning cattle. However, we must not confuse economic efficiency with biological efficiency. The major area of concern at the present time for the beef producers that are tryint to make money in the cattle business is "what is it going to take to make money raising beef cattle?"

Dr. Algeo presented data to show that conditioning would improve feedlot performance. However, the total cost per pound of gain for conditioning plus finishing was higher than cost per pound of gain when cattle were finished without conditioning. Thus, economic efficiency was reduced by conditioning and someone has to pay for this increased cost of gain.

The majority of the problems presently existing in the beef cattle industry would be minor if application of present technology had been made by a major portion of the industry. However, throughout the industry, enthusiasm is depressed by a low rate of return on investment and this is antagonized by those producers that do not respond to normal economic principles. Increased efficiency in production could lower cost substantially. To encourage beef producers to make needed changes in management is a real challenge when they are not properly rewarded for making these changes. Granted that in most cases more efficient production is needed just to maintain a status-quo; nevertheless, this does not reduce the challenge. If anything, it may be increased.

A good many ranchers are resisting the idea of conditioning cattle. Part of this opposition stems from the requirement for extra facilities and labor. Many ranchers do not have the needed pens, feedbunks and waterers. They feel the feeder is asking for an unreasonable investment on the ranchers part.

In many areas, feed is not available. Grazing conditions in many areas of States such as Texas, New Mexico, and Arizona, make any weaning program prior to shipment virtually impossible. Ranchers in Mountain States say weaning ahead of shipment would mean early removal of cattle from mountain grazing land and thus mean a heavier wintering load for the ranch. Also, it has been estimated that in Wyoming less than 20 percent of the ranchers would have the capability of doing even a basic preconditioning job.

Conditioning definitely means extra cost and extra effort for the rancher. Thus, if feeders want cattle conditioned for the feedlot, they must be willing to pay for the extra service and provide an extra profit incentive. Ranchers are not really convinced that feeders will pay enough extra for conditioning to foot the bill and because of the present economic situation existing in the cattle business I must admit I am also a little skeptical on this point.

The payment problem raises a whole series of questions among feeders and ranchers. Ranchers feel if they are going to condition, the feeder should pay a sizable bonus for the calves. I also feel this would be necessary for the rancher to justify a conditioning program; however, I seriously doubt if the feeder could afford it. The question has been raised whether or not a feeder should have to pay a premium for conditioned cattle. The statement was made that he should pay top dollar anyway and premiums are not necessary. There is one thing that I have learned in selling cattle or anything else and that is the buyer will pay the minimum amount necessary. Also, when I go to buy cattle, I try to get them at the lowest possibl

price not top dollar. I assure you I am not the exception to the rule. In contrast, most feeders feel the rancher should do the conditioning and rely on the added value of the calves to offset the extra cost. However, I seriously doubt if the rancher could afford to do this. I also believe that demand for feeder cattle is such that unconditioned cattle will have a ready market. Thus, the rancher will be able to sell his cattle just as profitably without conditioning them. The point I am trying to make has been clearly defined by John Guthrie, President of the American National Cattlemen's Association, who operates both a feedlot and a ranch. He faces the problem with mixed emotions. As a rancher, he says he can't afford to condition cattle he raises on his ranch to go into his own feedlot. However, he buys calves in the Southwest and ships them to a feedlot in California and he is trying to work out a method of getting the cattle conditioned before hauling them to the feedlot.

Some ranchers have suggested that feeders develop lots in areas where feeder cattle are purchased and do the conditioning for themselves or make arrangements with commercial lots in the ranch country to get the calves conditioned before moving them to their feedlot. However, commercial feedlot operators in general say this process would be too expensive. In other words, it is more economical to do things the way they are presently doing them. The question in my mind is, if they can't afford to condition cattle for themselves, can they afford to pay or are they willing to pay the necessary price to have producers to do this job for them?

I do not know what future of conditioning cattle is. However, I do believe at the present time it will be limited to individual arrangements between feeders and ranchers where they can work out an equitable arrangement where each can have a fair share of any advantage brought about by the conditioning process. A program where money is just shifted from one pocket to another or from one segment of the industry to another will not be successful. Buyer resistance is not going to force ranchers into a wholesale conditioning program because there are too many buyers that will buy them as is. Any other progress that is made in conditioning will depend on our ability to obtain a good health and performance history on cattle. This alludes to a point that has been made several times in this symposium and that is, we need a better identification system. Before any major advancements can be made in conditioning, it will be necessary to be able to trace cattle to their origin and develop meaningful morbidity and mortality statistics. This is impossible without a good identification system. The development of such a system would be a tremendous task in itself.

I think if ranchers condition cattle, a situation will arise that closely parallels the one presently existing with ranchers that have developed superior performing calves through performance testing. The only way they can capitalize on their effort is to feed the cattle themselves.

Like I have previously stated, the shipping fever complex is the major problem involved in conditioning cattle for the feedlot. Many of the conditions causing this problem are beyond the control of the feeder. Exhaustion from handling and hauling prior to delivery, overheating, or chilling in transit, and pre-existing nutritional deficiencies that lower resistance can not be changed on an individual basis. These are major problems and we need to try to help the industry solve them. The question to me at the present time is a matter of economics and it is very difficult to arrive at methods that would be satisfactory for all segments of the beef industry that would need to be involved, if conditioning is to be successful. However, I believe some of the important predisposing causes of shipping fever could be effectively eliminated soon after arrival if sufficient information were available. Because of the complexity of the beef industry and the economic situation presently existing in this industry, this is where we have the greatest opportunity. This is where we can expect cooperation and participation. There is little doubt that most feedlot conditions could be improved for receiving cattle to reduce shipping fever. Also, it is commonly supposed that there is a disturbed electrolyte balance in calves as a result of dehydration. Attempts at offsetting this condition have been somewhat empirical based on studies conducted on electrolyte control in humans. Ketosis is common in calves arriving at the feedlot and should also lend itself to control with a limited amount of new information. The two conditions, dehydration and ketosis, could be major factors in calves slow to start eating and in increasing susceptibility to respiratory diseases.

New arrivals at the feedlot are also usually suffering from malnutrition. The type of diet that new arrivals need could be a very important solution to many problems facing the feeder.

As to the primary, or contagious causes of shipping fever, studies in California, Colorado, and Nebraska have demonstrated that the causes are not the same in all areas. There is also evidence to indicate that the predominant causes of shipping fever in Texas are significantly different. More information is definitely needed in this area.

The most effective method of handling incoming cattle would also be considerable significance to cattle feeders.

Methods of handling incoming cattle vary considerably. Most operators remove and treat visibly ill animals immediately on arrival. Many have a definite plan for observing the balance closely for varying periods to detect additional animals that were not ill enough to be observed in the excitement of initial handling. Routine treatment of all incoming cattle with antibiotics seems to be declining. Medication of feed or water for treatment purposes has been discontinued in many cases. Drugs being used are principally tetracyclines or penicillin—streptomycin combinations. Atropine is used as an added agent by some on calves with obvious fluid accumulation in the lungs. In cases not responding to the former drugs one of the sulfonamide preparations is used. Occasional use of corticosteroid preparations are reported for use on critical cases. Furacin seems to be the most common drug used where diarrhea is present. Most feel that treatment should be continued for a minimum of three days. A 90 percent recovery rate following treatment periods of three to seven days seems typical. The ten percent of non-responsive cases are attributed to animals with advanced cases on arrival, or to cases caused by organisms not sensitive to the drugs in use. These are typical treatments and beliefs; however, research is very limited to prove or disprove their validity. Another area that needs to be clarified is working procedures. Should cattle be worked immediately after arrival or should they be rested. Both methods are presently being used by successful cattle feeders. Feeders are presently using the shotgun approach and trial and error procedures. We often criticize them but have we really tried to help them. Also, we probably could learn a great deal by observing some of the practices that feeders have found to be successful in reducing shipping fever problems.

Recommended procedures to reduce losses from shipping fever could include such things as:

1. Use of rapid-reading dial thermometers on all calves as they are put through the branding chute to facilitate earlier spotting of cases for treatment.
2. Care in combining drugs. Penicillin is antagonistic with any of the tetracyclines or sulfonamides. If used in such combinations, the effect will be less than that obtained by using either drug alone.
3. Lots of tender love and care.

Ultimate reduction of cost of shipping fever rests on additional information that must be supplied by research. Most pressing needs are:

1. Rapid means of returning incoming animals to physiological normality.
2. Definition of the microbial agents associated with shipping fever.

With information from such research, it would be possible to design feeding and handling procedures to shorten the interval required to get calves on feed and would assist commercial drug and vaccine producers in preparing agents to prevent or treat the conditions with maximum efficiency.

This has been an excellent symposium and a great deal of information has been presented on various aspects of conditioning cattle. The program committee and especially the staff at Oklahoma State should be commended for the fine job they have done in developing this symposium. However, if we fail to summarize what has been said here to determine where we are in time, what can be done about this situation based on present knowledge, what direction we should take and what we need to do in the future, the value of this symposium will have been greatly reduced.

The challenge we are facing is to maintain a competitive and profitable beef cattle industry. Undoubtedly, much can be done to improve beef cattle efficiency to make a more competitive and profitable industry.

While we are in complete sympathy with the desire to improve the efficiency of beef production by conditioning cattle for the feedlot, any program designed for that purpose must be scrutinized carefully. It must be carefully analyzed with regard to factual information and workability; to cost in relation to benefits and to aggregate effects on the total beef cattle industry. We cannot afford to sponsor programs that would be a discredit to our profession. Conversely we need a program that everyone can be proud of.

That change is in the air is obvious. Where it will lead is not so obvious. The future is not fixed or predetermined. To be sure, there are historic forces that influence it and cannot be disregarded. On the other hand, now, as in days of the past, the future is what we individually and collectively make it. In other words, the future of conditioning cattle is up to us.

PROBLEMS AND RESEARCH OPPORTUNITIES FOR THE PHARMACEUTICAL AND BIOLOGICAL INDUSTRY

Dr. N. H. Casselberry
Vice President, Cutter Laboratories
Berkley, California

The role of our biological and pharmaceutical industry has been and will continue to be to recognize the needs of the livestock industry in disease prevention and try to provide the needed tools. To do this we must glean all the research information available from the scientific community and apply this information to product development and evaluation. Then, we must make the best information we have available for their proper application.

It is encouraging to see the emphasis being placed on preconditioning of feeder animals. It is my belief that the improved overall management steps, with more attention to proper handling and better information on the history of animals purchased, will go a long way to reduce disease problems. At least, I hope this becomes an important part of the program and that preconditioning does not begin to mean only vaccination against certain diseases as a stereotyped routine and routine treatment against certain things before shipment without proper regard to the extreme importance of good handling procedures as part of the program.

We producers of biological products recognize the importance of and encourage immunizing during a stable phase of the management program, if possible, to give the animals every chance to react fully to injected antigens and, also, to accomplish immunization before the most dangerous exposure time. These fundamentals, we realize, are often not possible to apply practically but we would be callous indeed if we didn't fear the abuses in the use of immunizing agents which have been referred to elsewhere in the program in the application of our products.

We feel we must be in a position to guide the livestock man in the selection of those immunizing agents which are required for routine disease prevention under his circumstances. Not all producers have the same problems. We can do this now much better than in past years but, while we have been learning more about the importance of some new diseases within the last decade, we have neglected some old potential troublemakers that still seem to resist adequate control. Pasteurellae were mentioned by Dr. Collier yesterday and he indicated the importance of antigenic types in dealing with this problem. There needs to be much more done to reappraise the importance of this bacterium in the bovine respiratory complex. It seems to me that we also must go back and reappraise the true importance of some other old enemies to profitable cattle feeding as well.

We know that vaccination of cattle on entry to the feedlot leaves much to be desired. It did seem the only practical way in the beginning to apply IBR vaccine as there wasn't time to wait until newly acquired cattle rested up and also it was not a recognized problem except in the feedlot so the range man couldn't be expected to be very much interested in use of vaccine. Vaccination in this way, that is, on arrival in the feedlot, has continued to be a relatively safe and effective method against bovine rhinotracheitis. However, in trials, which have been run by vaccination of cattle after weaning and then allowing them to develop immunity at pasture and then bringing them to the feedlot, the overall results have been better as one would expect. This is just one disease agent and, unfortunately, there are several other potential causes involved in the respiratory or shipping fever complex that may cause trouble too soon after arrival at the feedlot to give hope that they can ever be prevented by vaccination after arrival. The problems of how to use bovine virus diarrhea and para-influenza 3 vaccine have already been discussed on this program and, it appears that if they are to be successfully applied, the timing of the vaccination is of paramount importance.

There are some real holes in our understanding of our clinical problems that need more work to improve our knowledge and, therefore, hopefully allow us to supply better tools to cope with these diseases.

As previously mentioned, Dr. Collier discussed the importance of Pasteurella and I think he would agree that one of the things we need to know is which ones we should concern ourselves with most. That is, is type A of the Carter classification the only type of Pasteurella multocida we need to concern ourselves with? Personally, I doubt this. There are some strains that still resist classification and yet are pathogens. We recognized another distinct species, Pasteurella hemolytica. We know we have several different antigenic types of this species as pointed out to us several years ago by Dr. Biberstein. We feel that some types of these two species of Pasteurella are more important than others but we do

not have good agreement as to which of these different types are essential in preparing biological products to be sure the products we use for disease prevention are as antigenically complete as they should be. Until we have a capability for typing all pathogenic strains isolated from field infections, we are not likely to have this data either.

We need also to know much more about the importance of Mycoplasma (PPLO) and to assess its true importance in the respiratory complex. If Mycoplasma is an important pathogen, even if we couldn't immunize against it effectively, we should at least be able to determine when it is involved to prevent disease control measures directed at other agents from being discredited as being ineffective.

We have long been led to believe that certain types of Corynebacteria are important contributors to the shipping fever complex. Yet, little is known about this bacterium and certainly we don't know much about how to evaluate immunizing agents used to prevent this suspected contributor to clinical disease.

Dr. McKercher, yesterday, also mentioned the problem of the psittacosis lymphogranuloma agent as a cause of respiratory problems in cattle and so far there is no means to immunize against this infection. Fortunately, it seems to be only occasionally encountered.

It would be interesting to know more about the pathogenesis of thrombo-embolic encephalitis which has caused grave concern in some feedlots rather sporadically. The causative organism has been isolated and considered to be Hemophilus-like but has not definitely been classified. We need to know how the organism invades. Is it the result of invasion from the respiratory tract when cattle are going through a respiratory episode and the mucus membrane may permit invasion of these organisms? Does it need some help from a primary virus infection to accomplish this invasion? It may be that if it is truly a secondary infection one could handle it effectively by controlling the primary disease. Attempts at immunization have been made but evaluation is extremely difficult for lack of knowledge on the pathogenesis and because the disease is so sporadic as to cause rather severe death loss for several weeks with a sudden disappearance thus invalidating field exposure trials.

Another real need for information, that makes a problem for us and for the livestock man, is the lack of refined information on the enterotoxemias in the feedlot. This problem seems to have increased greatly as we have made changes in feeding technology employing more concentrated rations. It appears that these hotter rations, while increasing feeding performance also provide an environment very much to the liking of certain Clostridia that are encouraged to produce more toxin than the host can stand without immunization. However, our ability to produce specific antigens to deal with this problem more effectively is hampered somewhat by lack of knowledge as to which of the Clostridia are the important offenders. For example, Clostridium perfringens D and C are considered to be important but no real critical information is available to indicate if they are of equal importance or if only one type of antigen might be required as the immunizing agent. More diagnositc work needs to be done to indicate which of the toxins Beta or Epsilon is the important offender so that immunization could be accomplished with a rifle instead of a shotgun. Diagnosis is also important since related organisms, namely, Clostridium sordellii and Clostridium novyi have also been incriminated or at least suspected as a result of organism isolations. In critical enterotoxemia episodes immunization can be accomplished if the known offenders can be determined. In this whole group of enterotoxemia problems we need to know more also about when to vaccinate, how many times and what interval between doses to attain the most effective protection.

I can't overemphasize the need for good diagnostic service, readily available to service the livestock industry and to guide all of us in our efforts to cope with disease specifically and, therefore, more effectively.

We have many other imperfections in our knowledge that hampers our effort in immunization such as the interference of passive antibody at the time of attempted immunization. We know that immunity passed on from the mother to the suckling calf can interfere with a response to vaccination if the animal is vaccinated while this immunity is still in force. We also know that some types of passive immunity, transmitted through the colostrum, are much more durable than others but we need more information on the duration of such protection in order to be able to program the time of immunization more effectively or when to recommend a second dose after weaning when a calculated risk is taken to attempt early immunization before weaning. We also know that passive antibody injected as antiserum will interfere with the response to active immunizing agents but the duration of this is also quite variable. For example, until recently the common belief was that prophylactic injection of antiserum was considered effective for ten days to three weeks. Now, we know that if antiserum is homologous the anitibody may persist in the injected animal for much longer periods of time so the timing of vaccination after a dose of serum for maximum

effect from the vaccine would be very different if the specific antiserum used was homologous rather than heterologous serum.

After we have a better understanding of these problems that we are already dealing with and recognize as important our next group of potential problems will be disease agents that we don't now recognize as serious. Some of these are already strongly suspected as being involved but without sufficient proof to have received close attention. Tissue culture techniques have enabled the scientific community to uncover many more agents than we can understand easily. That is, they can be recognized as being present but it is often difficult to determine how greatly they contribute to our total disease problem. Dr. McKercher mentioned these yesterday and I'm sure it won't be long before we are hearing more about reo viruses, rhino viruses and adeno viruses. We have already been hearing about Mycoplasma and its importance has been established in the poultry field. We can hope that we can learn enough about it that it is either not a real problem in our cattle population or that we can deal with it without having to resort to the drastic steps taken for its elimination that have to be practiced in the poultry field.