Research Summaries

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Subclinical Coccidiosis Infection in Calves

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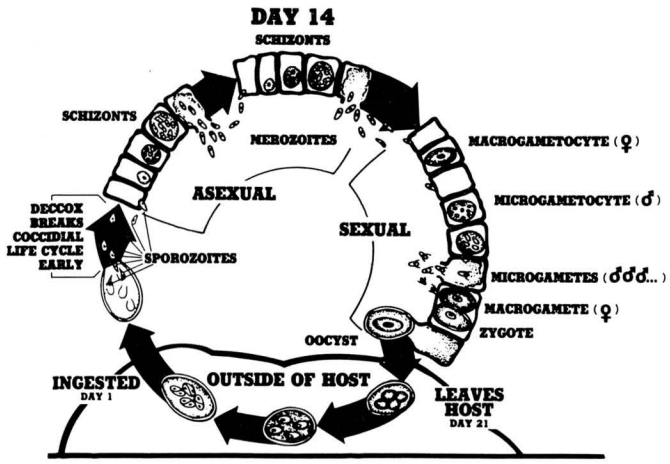
Coccidiosis causes large economic losses from death, labor and treatment cost, morbidity, along with loss of feed conversion, and weight gain in calves and cattle following classical outbreaks. Low level or subclinical infections are proving to be more prevalent and costly than previously thought. Their control can increase performance of cattle not necessarily exhibiting obvious clinical symptoms.

We need to briefly look at the life cycle of the parasite to understand the total disease problem.

Overhead on Cycle

1. Area of major activity of coccidiostats.
2. Area of "minor activity" of sulfa.
3. In ruminants the sexual cycle moves to large intestine.



Damage to the small intestine is not as drastic as in monogastric species, but it does exist to a degree.

Major damage is not proportionate to degree or intensity of the infection. It is a number game without a minimum infective dose—the MIF varies with species, stress, immune response from previous exposure—a series of passes of low infection is believed to produce some immune response, however it is not a good immune response, which with the stress of a steroid, a clinical infection will develop. *An oocyst count at its best* can only be considered a diagnostic aid.

All levels of infection occur in the real world. Research cannot duplicate *natural* infection. Dr. Fitzgerald of the University of Illinois and other researchers have measured the effect of different dose levels of oocysts. They have established that an increase in level of infection (number of oocysts) in cattle in controlled trials, results in an increase in the intensity of the infection. But again I wish to state "There is no way anyone can accurately measure or evaluate this response in the field with all the variables that exist."

The husbandry and production practices of the cattle industry limit the capabilities we have to measure the degree of infection or percent of response, as a result of 1. variable feeding practices, 2. Weight variation, 3. Genetics, 4. Length time fed. The poultry industry have the capabilities of measurement and control of many variables and as a result they regard a low level coccidia infection a problem and a major threat to profitable production. Poultry and swine research has gone one step even further and have demonstrated as disease synergy between coccidiosis and certain viruses (a reoviral infection in poultry and a rotoviral in piglets). This further complicates the effect of a sub clinical cocci. I am very suspicious similar synergist activity occurs in cattle.

To date we have only field data to support our position that low level or sub clinical coccidiosis in cattle constitutes a serious threat to the husbandry of all young cattle.

Since no research model exists to properly measure the accumulative damage we over the past several years have used, the following parameters are used to attempt to measure the results of control of coccidia. We have routinely measured the effect of cocci control on:

1. Feed gain.
2. Weight gain.
3. Morbidity.
4. Mortality.

I would like to present data from:

1. A controlled pen study by academia in a research facility.
2. A controlled pasture study conducted by extension

personnel with stocker cattle.

3. A commercial field study utilizing over 10,000 feeder cattle.

Clinical coccidiosis was not diagnosed or observed in these studies to be a problem. The major disease diagnosed was respiratory problem—probably of viral origin.

The coccidiostat used is very specific in activity and possesses no other known drug activity.

No. 1. Feed intakes from calves housed in pens with individual feed monitoring devices, kg dry matter/dry.

	Treatments					
	Decoquinate			No Decoquinate		
Days	Steers	Heifers	Average	Steers	Heifers	Average
1-7	1.96	2.21	2.11[a]	1.18	1.60	1.45[b]
	+1.73[c]	±1.77	±1.73	±1.47	±1.66	±1.59
1-14	3.11	3.14	3.13	2.48	2.86	2.72
	±1.92	±2.10	±2.01	±2.05	±1.89	±1.93
1-28	4.54[a]	2.55[b]	3.38	4.31[a]	4.03[a]	4.12
	±1.76	±2.75	±2.57	±2.69	±2.07	±2.25
1-56	6.44	6.12	6.31	6.86	5.63	6.02
	±1.58	±2.36	±1.91	±2.15	±1.79	±2.97

[a,b] Means in same row with different superscripts are significantly different ($p < .05$).
[c] Standard deviation.

No. 2. Average daily gains, kg/day, of all experimental calves.

	Treatments					
	Decoquinate			No Decoquinate		
Days	Steers	Heifers	Average	Steers	Heifers	Average
1-28	.62	.49	.56	.64	.36	.50
	±.11	±.10	±.12	±.35	±.07	±.28
28-56	1.49	1.36	1.43	1.47	1.38	1.42
	±.15	±.09	±.14	±.13	±.28	±.21
1-56	1.06	.93	.99	1.08	.91	.99
	±.02	±.02	±.07	±.19	±.14	±.18

No. 3. Feed conversion kg of feed per kg. of gain of all experimental calves.

	Treatments					
	Decoquinate			No Decoquinate		
Days	Steers	Heifers	Average	Steers	Heifers	Average
1-28	7.14	8.43	7.77[a]	8.61	12.29	10.45[b]
	±1.36[c]	±1.57	±1.53	±3.64	±1.73	±3.29
28-56	5.82	6.07	5.94	6.39	6.20	6.29
	±.52	±.52	±.50	±.67	±.70	±.64
1-56	6.13	6.58	6.36	6.59	6.88	6.72
	±.13	±.40	±.37	±.26	±.79	±.56

[a,b] Means in same row with different superscripts are significantly different (p .05).
[c] Standard deviation.

No. 4. Morbidity and mortality of experimental calves.

	Treatments					
	Decoquinate			No Decoquinate		
	Steers	Heifers	Average	Steers	Heifers	Average
Morbidity, %	85.3	73.3	78.2	82.5	83.3	83.0
Mortality, %	10.2	16.4	13.9	28.2	16.7[a]	22.0
Number of Dead	4	10	14	11	10	21

[a] One dead off truck not included in calculations.

TABLE 1. Average Performance of Heifers Fed Deccox-Mineral Supplement Mixes on Native Range Pastures.

	Control	Deccox
Number Heifers	60	59 [1]
Starting Weight	492	498
Final Weight (6/22/83)	633	666
Total Gain	141	168
Animal Days	56.9	56.7
ADG	2.47	2.97

[1] One chronic heifer died. Data removed from study.

Performance of Heifers in Oklahoma Pasture

TRIAL NO. 3

	Control	Deccox
Number of Heifers	50	51
Start Weight, lbs.	388	400
Daily Gain, 1st Period (32 Days)	.73	.80
Daily Gain, 2nd Period (26 Days)	—.14[a]	.78[b]
Daily Gain, Total Period (58 Days)	.34[a]	.79[b]
Final Weight	408	446
Sick Pulls [c]	27 (54%)	19 (38%)
Repulled	16	2
Sick Pulls with Scours	27	2
Dead	0	0

[a,b] Means differ ($P < .01$)
[c] One sick pull signifies that a calf was removed for treatment and treated until deemed well.

Performance of Heifers in Oklahoma Pasture

TRIAL NO. 4

	Control	Deccox
Number of Heifers	49	51
Starting Weight, lbs.	397	379
Daily Gain, 1st Period (29 Days)	.09[a]	.66[b]
Daily Gain, 2nd Period (28 Days)	—.15[a]	.47[b]
Daily Gain, Total Period	—.03[a]	.57[b]
Final Weight	396	412
Sick Pulls [c]	32 (65%)	8 (16%)
Repulled	10	5
Sick Pulls with Scours	32	8
Dead	2	1

[a,b] Means differ ($P < .01$)
[c] One sick pull signifies that a calf was removed for treatment and treated until deemed well.

Large Southwest Field Trial

	Control	Deccox	Deccox Adv.
Number Cattle	5,108	5,089	
Pens	21	21	
Average Weight (lbs.)	450	450	
Weight Range (lbs.)	400-500	400-500	
Pulled for Treatment	1,087	880	18.4%
Diagnosed Cocci	12	11	
Scours (Non-specific)	189	130	30.9%
Retreats (Mostly Respiratory)	310	267	—1.8%
Deaths	113	90	29%
Realizers	80	62	22.2%
Economics		$2,700	$12,000 (Estimate)
Dead $200			
Realizer $175			
Treated $ 12 (3 days x $4)			

Conclusion

1. Low level (sub clinical) coccidiosis constitutes a serious economic loss.
2. The sub clinical disease of coccidiosis interferes with optimum performance.
3. The severity of the coccidiosis symptoms is affected by stress and can as a disease, contribute to stress.
4. Medication with a coccidiostat can remove one of the stress associated with cattle husbandry, and as a result help reduce the severity of some other disease syndromes.

Suppression of Neutrophil Function in Bovine Respiratory Disease

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During the last seven years or so I have been doing research on bovine respiratory disease at Iowa State University. We have been trying to define the immunosuppression that occurs with the animals susceptible to the bacterial pneumonia, with the ultimate goal of identifying or finding drugs that can be used to reverse immunosuppression. I will go fairly rapidly through our data, just hitting the high points and probably not giving you time to digest all of the data, but I'll try to hit the high points as I go along. As you all are aware I am sure, of the bovine respiratory disease syndrome, or shipping fever, the hypothesis for its pathogenesis is that it requires three things—stress, plus a nonbacterial infection, and these two components suppress the host defense mechanism sufficiently to allow the bacterial infections to produce a severe pneumonia. And the economic losses are primarily due to the bacterial infection.

This talk is going to focus on neutrophil function primarily, and I will talk about how each of those things can suppress neutrophil function. First of all neutrophils are white blood cells found in the blood stream. Polymorphonuclear leukocytes are very active in phagocytosis and destruction of bacteria. They ingest bacteria in the blood stream or in the lungs, wherever they come across them and take them internally. The lysis zones within that neutrophil are little sacks with enzymes that merge with the phagozone containing the bacteria. The enzymes then attempt to degrade and destroy the bacteria and control the infectious process. Neutrophils have several potent mechanisms for killing bacteria, in addition to these enzymes. Neutrophils take oxygen and convert it to hydrogen peroxide, as well as other oxygen radicals more potent than hydrogen peroxide. They also cause a generation of aldehyde, and you are all familiar with the bacteriocidal properties of hydrogen peroxide and aldehydes like formaldehydes and gluteraldehyde. Neutrophils figured out long before man did that you can kill bacteria with these things. So they have mechanisms for generating these products.

When we do these experiments, the first thing that we do is isolate neutrophils from the peripheral blood. We bleed the cattle and go through some steps to get pure neutrophils and we evaluate their function. We use a series of tests. Neutrophils go through a lot of steps in killing bacteria and we can assay most of those steps. So we use a battery of functional assays. We don't really have time to get into the specific assays. I'll be doing a lot of generalization in this talk.

First of all we want to look at stress because that is an important component. One thing that universally occurs with stress is an elevation of cortisol levels, plasma cortisol levels. An animal under stress releases ACTH from its pituitary gland, which causes the adrenal gland to release cortisol. These are conditions that have been reported to cause increased cortisol concentration in cattle. You will recognize