Epidemiologic Observations on Bovine Winter Dysentery*

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Introduction

The cause of bovine winter dysentery remains unknown. Until a singular etiology is demonstrated, we must continue to consider the possibilities that several factors operating in concert may be responsible or that several etiologic agents may produce a similar clinical syndrome.

Previous studies on winter dysentery have been directed toward isolation of a causal microbiologic agent (3). In the absence of an isolate, however, advances in our knowledge of the disease can be made by epidemiologic methods. Epidemiologic methodology involves description of the distribution of disease in populations, and subsequent attempts to explain the observed distributions.

In an effort to update bovine practitioners on current thinking about winter dysentery, this paper describes the clinical and epidemiologic pattern observed in two winter dysentery outbreaks and reviews previously reported epidemiologic studies on the occurrence of winter dysentery in cattle inoculated with vaccines prepared for prevention of other diseases.

Lacking serologic or other diagnostic tests, our studies have relied on tabulations of the clinical sign "diarrhea" in herds experiencing outbreaks fulfilling the following seven criteria for winter dysentery. 1) Sudden onset. 2) Diarrhea or dysentery.** 3) Three or more adult cattle affected. 4) Stabled or housed cattle. 5) Occurrence in November through April (in North America). 6) Blood grossly evident in feces of some animals. 7) Few fatalities.

Herd No. 1

Of 61 Holstein and Jersey cattle of all ages on a New York dairy farm, 50 had liquid brown stools and 21 had temperatures greater than 103°F (104°F for calves under three months) during an outbreak of "classical winter dysentery" accompanied by coughing, occasional bloody stools, moderate and variable degrees of anorexia, and some reduction in milk production. There were no fatalities during the outbreak. The distribution of diarrhea was unremarkable with respect to age (Table 1), sex, breed (Table 2) or location in the barn. Three cases were observed simultaneously on the index date (first day of the outbreak) and new cases appeared daily for eight continuous days with a maximum of 15 new cases observed on Day 2.

Table 1

Diarrhea Attack Rates by Age, Herd No. 1

Year of Birth	Age Years	No. of Cattle*	Diarrhea Attack Rates
1963	9	2/3	.666
1964	8	2/2	1.000
1965	7	1/1	1.000
1966	6	1/2	.500
1967	5	5/7	.714
1968	4	5/5	1.000
1969	3	7/7	1.000
1970	2	12/14	.857
1971	1	7/7	1.000
1972	< 1	8/13	.615
Total		50/61	.820

*numerator is number with diarrhea in age group
*denominator is number present in age group

Table 2

Diarrhea Attack Rates by Breed, Herd No. 1

Breed of Cattle	No. in Herd	No. with Diarrhea	Diarrhea Attack Rates
Holstein	42	34	.810
Jersey	17	15	.882
Holstein x Hereford	2	1	.500
Total	61	50	.820

***While definitions differ, diarrhea means abnormal frequency and liquidity of fecal discharge while the term dysentery usually conotes severe diarrhea with the presence of mucous and blood in stools.*

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Table 3

Fever Attack Rates by Age, Herd No. 1

Year of Birth	Age Years	No.* of Cattle	Fever Attack Rates
1963	9	1/3	.333
1964	8	1/2	.500
1965	7	0/1	.000
1966	6	0/2	.000
1967	5	1/7	.143
1968	4	2/5	.400
1969	3	3/7	.429
1970	2	5/14	.356
1971	1	1/7	.143
1972 (less than 1 year)	1	7/13	.540
Total		21/61	.340

Fever is defined as one or more three-time daily rectal temperature readings greater than 103.0 degrees F (104.0 degrees F for calves under three months).
*Numerator is number of febrile cattle in age group.
*Denominator is total cattle in age group.

This distribution (see Figure 1) suggested a propagated disease with cow to cow spread but could have been produced by simultaneous exposure to a common source of infection (or toxin) with variable incubation periods or by variable exposure to a common source and uniform incubation periods.

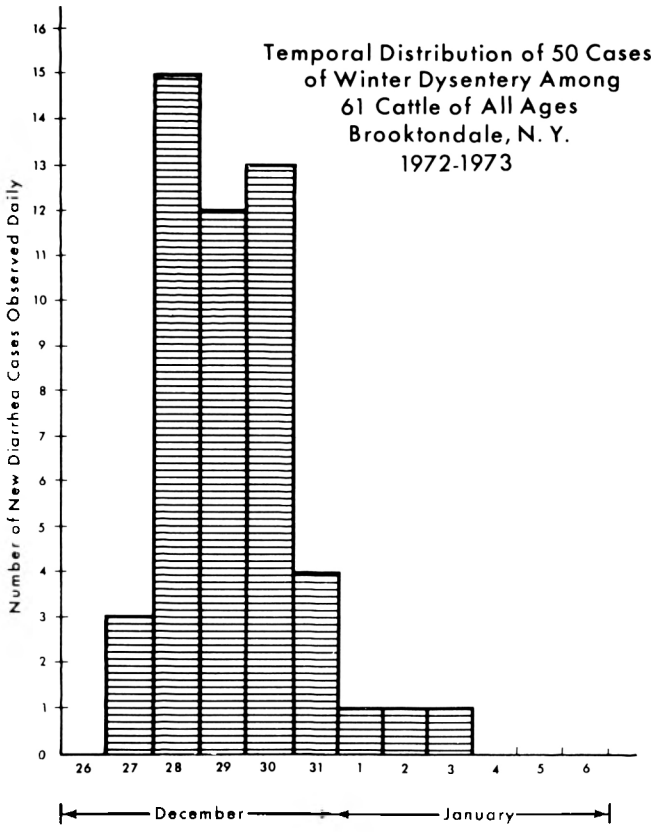

Figure 1. The epidemic curve for herd No. 1 shows the onset of new cases of diarrhea started with three cases on December 27 (the index date) with 15 cases on December 28 and then a gradual decline in new cases. This configuration suggests cow to cow spread but could be explained by simultaneous exposure with variable incubation periods.

The rectal temperature of each cow and calf was recorded three times daily. Twenty of the fifty cattle with diarrhea had detectable fevers and one unaffected calf had a fever. There was no consistent relationship between fever and onset of diarrhea. Calves had a slightly lower diarrhea attack rate (Table 1) than adult cattle.

The immediate source of infection was undetermined, although a worker from a neighboring herd affected with winter dysentery, and a veterinarian who attended a dystocia in one of the index cases six days before the index date, were considered possible sources of infection. A group of commune dwellers who visited the farm four days before the index date were considered as possible sources of infection along with a cattle dealer and an artificial inseminator. The relationship between visitors and the onset of diarrhea is shown in Figure 2.

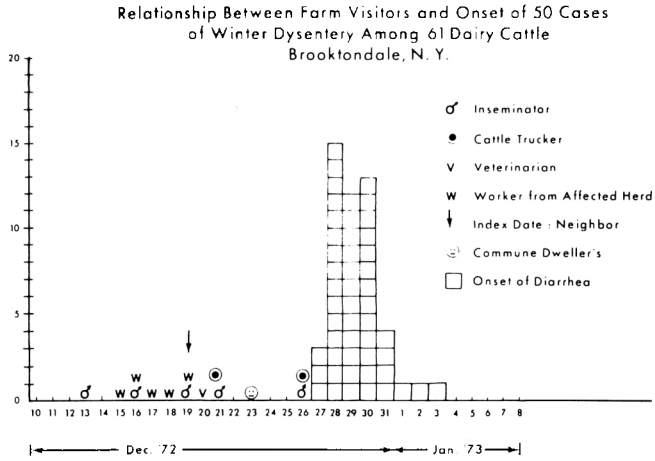

Figure 2. The relationship between visitors (including a worker [W] from a neighboring farm with winter dysentery) and the onset of diarrhea shows the difficulty in clearly designating the immediate source of exposure.

Herd No. 2

Of 33 Holstein cattle of all ages, 17 had brown liquid stools and five had one or more temperatures greater than 103°F. (adults) or 104°F. (calves less than three months old) in two-time-daily temperature readings. In this outbreak, bloody stools were observed in only one case. Slight anorexia, cough, and moderate depression in milk production were observed. Breed and sex comparisons were impossible since the entire herd was Holstein females. Birth dates were unavailable, so cattle were categorized as adult (milking cows) or calves (less than three months of age).

In this herd, the diarrhea attack rate (Table 4) in calves was 75% while that observed in adults was 44%, but the difference was not statistically significant. Like Herd No. 1, the fever attack rate (Table 5) was greater in calves than in adults, but again the magnitude of the difference was not statistically significant.

Table 4

Diarrhea Attack Rates by Age, Herd No. 2

Age Group	No. of Cattle*	Diarrhea Attack Rate
Adults	11/25	.44
Calves (under 3 months)	6/8	.75
Total	17/33	.52

*Numerator is number with diarrhea in age group.
*Denominator is number present in age group.

Table 5

Fever Attack Rates by Age, Herd No. 2

Age Group	No. of Cattle*	Fever Attack Rate
Adults	2/25	.08
Calves under three months	3/8	.38
Totals	5/33	.15

The temporal distribution of new cases resembled that expected in a propagated disease (Figure 3).

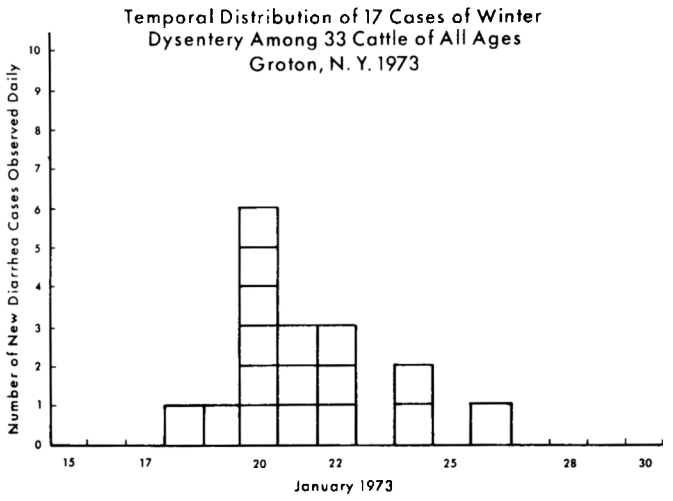

Figure 3. The epidemic curve for Herd No. 2 shows a pattern similar to Herd No. 1, suggesting a propagated disease with cow to cow spread.

Effect on Herd Milk Production and Pregnancy Status

Both herds had individual cows with marked decrease in milk production during the outbreaks but the impact on total herd production appeared slight (see Figures 4 and 5). The actual production loss was obscured in part by addition of milk from newly freshened cattle. Milk production losses were of less significance than expected in light of informal reports of major losses associated with winter dysentery. This apparent discrepancy can be explained several ways. These herds may have had atypically low losses or the addition of newly freshened cows to milking herds during the epidemic may have cancelled out losses. It must also be considered that in outbreaks where production losses were not actually documented, dairymen may have exaggerated losses.

One abortion occurred among 38 cows or heifers pregnant during these outbreaks. The cause of the abortion was unknown.

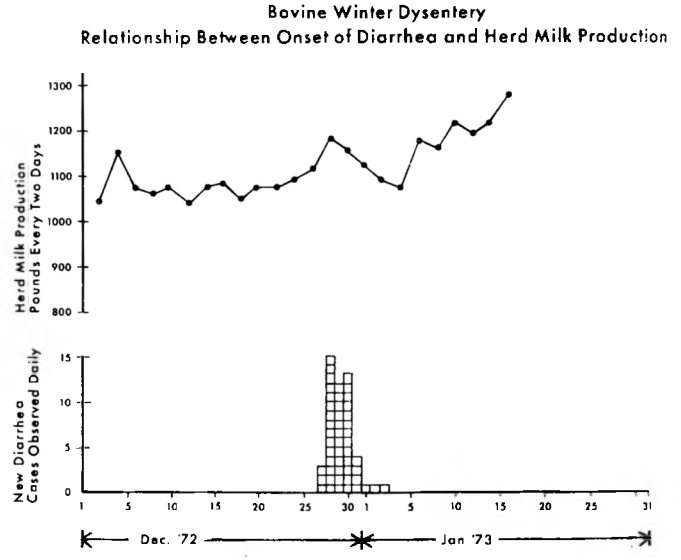

Figure 4. Milk production in Herd. No. 1 dropped slightly after the peak day of the epidemic. Addition of new milking cows on December 18, 20, 22, 25 and 26 made evaluation of production loss difficult.

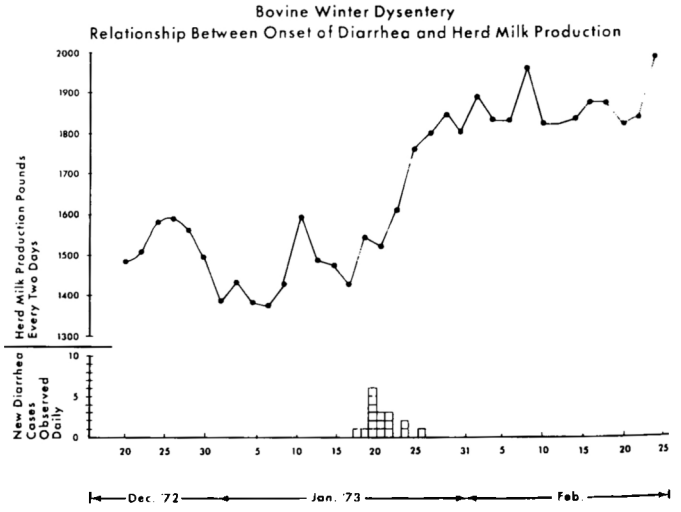

Figure 5. Milk production in Herd No. 2 was erratic prior to, during, and after the winter dysentery outbreak and production loss was difficult to evaluate. One new milking cow was added on January 25.

Winter Dysentery in Vaccinated Cattle

In the absence of specific vaccines for winter dysentery there has been temptation to use available bovine vaccines in hopes of preventing or controlling the disease. In 1965, it was reported that a winter dysentery attack rate of 54.8% was observed in 366 exposed cattle vaccinated previously with *Leptospira pomona* bacterin and live virus vaccines for IBR, BVD, and Parainfluenza-3 while a similar attack rate (53.7%) was observed in unvaccinated herdmate controls (1). It was concluded these vaccines did not prevent winter dysentery. Three of seven herds in that study were vaccinated with bovine mixed bacterin No. 1, one to two months prior to experiencing winter dysentery outbreaks with attack rates of 100%, 33% and 100%. While this observation did not comprise an adequately controlled evaluation of mixed bacterin No. 1 protection, it clearly indicated that winter dysentery can occur in cattle vaccinated with this product (1).

IBR and BVD are not Causes of Winter Dysentery

In the same study (1), significant IBR or BVD seroconversion was not detected in 120 paired serums from cattle which developed winter dysentery. While few have suggested IBR as a contributing factor in winter dysentery, much speculation regarding BVD as a cause of winter dysentery had been informally proposed. The serologic findings tended to lay that speculation to rest but occasionally herd outbreaks of BVD occur which involve adult cattle with diarrhea in which winter dysentery is the initial diagnosis. The diagnosis of BVD becomes evident only after examination of the oral mucosa of many cattle reveals erosions and the recognition of excess nasal and lacrimal discharges, rapid respiration and luecopenia suggests more systemic involvement than is usual in "classic winter dysentery." Fever is more frequent and persistent in BVD than in winter dysentery in which fever is rarely observed unless two- or three-times-daily temperature readings are taken on the entire herd.

Differential Diagnosis of Winter Dysentery

The characteristic acute onset of diarrhea in stabled cattle enables easy herd and individual diagnosis using the criteria outlined above. Careful history taking is necessary to assure the diagnostician does not overlook sudden feed changes or accidental access to succulent feedstuffs or toxic chemicals. Careful physical examination, including examination of the oral mucosa is required to differentiate from BVD or rinderpest. Fecal examination and fecal cultures are needed to differentiate from parasitosis and salmonellosis respectively.

Treatment

Empirical treatments for diarrhea are commonly used and maintenance of electrolyte balance and state of hydration is indicated in the occasional cow that becomes significantly dehydrated. The conclusions of Roberts (2) that various treatment regimens have no effect on the duration or severity of diarrhea have apparently withstood the test of time because convincing evidence to the contrary has not appeared.

Summary

In two herds undergoing winter dysentery outbreaks, the distribution of onset of diarrhea with respect to time and space suggested an infectious and contagious disease with cow to cow spread. In both calves and adults, there was no consistent relationship between fever and the onset of diarrhea. Age and breed appeared inconsequential in diarrhea and fever.

Data reviewed suggest infectious bovine rhinotracheitis (IBR) and bovine viral diarrhea (BVD) viruses are not involved in the etiology of winter dysentery and currently available vaccines and bacterins are not effective in its control.

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Etiologic Studies on Bovine Winter Dysentery

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Introduction

Winter Dysentery (WD) is a highly contagious, acute enteric disease of cattle characterized by a brief attack of severe diarrhea and sometimes dysentery (1). It occurs primarily in stabled dairy cattle in Northeastern and North Central United States and Canada, although similar diseases have been reported in Australia (4), Sweden (3), France (2), Israel (8), and England (10). As the name implies, it occurs in late fall, winter, or early spring. The most severely affected cattle are the two and three year old pregnant and milking heifers, with older cows usually showing less severe involvement. Severe intestinal hemorrhage occurs in 5-10% of affected cattle. Mortality is uncommon and most cattle survive without treatment unless dehydration or hemorrhage occurs. A more detailed description of the clinical signs and epidemiology is presented in the companion paper (7).

Etiology

The etiology of WD is unknown. Scientists in the early 1930's indicated that *Vibrio jejuni* was the etiological agent (5), but more recent studies have failed to substantiate this (2,3,9,). Viruses have been isolated and incriminated in WD-like disease in Canada (9), France (2), and Israel (8).

The apparent transmissibility of the disease by fomites, contact, or fecal suspensions, the extreme contagiousness, the mode of spread through a herd, and their febrile response seen early in the disease in some cattle (7,11) would indicate that it is infectious. Certainly bovine practitioners, cattle dealers, and inseminators who have been incriminated for spreading WD from farm to farm are easy to convince of its contagiousness.

Over the years a number of microbiologists have attempted to determine the etiology of WD. To say the least, it has been a "tough nut to crack."

The senior author has been involved in studies to determine the etiology of WD since 1965. In the fall of 1972, the investigation was expanded to provide a multidisciplined approach with six primary investigators, including a clinician, an epidemiologist, a gastroenterologist-clinician, a physiologist, an immunologist, and a microbiologist. Eight additional investigators contributed to the program in their specialties. While funds are limited, it is the start of a program that hopefully in time will solve the mystery of WD.

This report summarizes attempts to determine the etiologic agent of WD. Approximately twenty herd outbreaks have been investigated, some with only a cursory examination and some with a detailed herd investigation including taking temperatures and an examination of all cattle three times a day for one week. Feces, serum, blood,and sometimes nasal swabs were taken from three to six animals in each herd for bacteriologic, virologic, and transmission studies.

Vibrio

Since the reports in the early 1930's (5) *Vibrio jejuni* was considered to be the etiologic agent of WD for several years. In recent years, attempts at isolation of *V. jejuni* from clinical cases have been consistently negative. Several investigators (2,3,9) concluded from their studies that *V. jejuni* was not the cause of WD.

In our studies, when rigorous microbiologic techniques were applied, vibrios could not be isolated from fecal, gut content, or blood specimens collected from several WD outbreaks. It is our opinion that *Vibrio jejuni* was not involved in these outbreaks. This conclusion is analogous to recent findings that *Vibrio coli* once considered the cause of swine dysentery has a less significant causal role than the spirochete *Treponema hyodysenteriae.*