Rhinitis Syndrome In IBR-Vaccinated Feedlot Cattle

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Infectious Bovine Rhinotracheitis (IBR) is one of the most common virus diseases of cattle. The etiological agent, a member of the herpesvirus family (10), has been associated with vulvovaginitis (12), balanoposthitis (8), abortion (11), conjunctivitis (1), meningoencephalitis (5), and respiratory disease (3, 14). Rhinotracheitis, the respiratory form, is important in western feedlots and it is also associated with shipping fever (6, 7).

A number of vaccines are presently used in nearly all phases of the cattle industry (9, 15). In a major western feedlot, all incoming cattle are vaccinated upon arrival with intramuscular modified live virus IBR vaccines. Despite vaccination, significant respiratory disease expressed mainly as rhinitis occurred during the late fall and winter months. This study was initiated to determine if IBR virus was a primary cause of rhinitis in cattle known to be vaccinated against IBR.

Materials and Methods

Yearling feeder cattle weighing approximately 350 kg were obtained and routinely processed for entry into a feedlot. Management procedures include implantation in the ear of a growth stimulant, a total body dip in an organophosphate insecticide and vaccination against IBR and leptospirosis with a commercial mixed vaccine composed of intramuscular modified live IBR virus and killed leptospirosis bacterin [a-c] . Vaccines against other diseases were not administered. Selection criteria for subjects were those of clinical rhinitis and were as follows: a) subjects must have been vaccinated against IBR at least 21 days prior to day one of illness, b) body temperature of 40 C or over, c) inflamed nasal membranes with vesicles, d) "snoring type" respiration e) respiratory rate above 15/min, f) excess bilateral nasal discharge, g) anorexia, and h) general depression.

A serum sample and nasal swabs were taken from each subject on day one of illness, and also from each of 5 clinically normal pen mates as follows: a) for virus isolation, cotton swabs were placed in 1 ml of cell culture medium with antibiotics and immediately frozen on dry ice for transport, b) for bacterial isolation, swabs were placed in 1 ml transport medium and kept on wet ice, c) for mycoplasma isolation, swabs were placed directly into Hayflick's medium and kept at wet ice temperature. The animal was then placed on antibiotic and supportive therapy. If the animal recovered, serum samples for antibody titration and nasal swabs for virus isolation only were taken 21 days after the onset of illness.

Virus isolation attempts were made on secondary bovine fetal spleen cells. One-tenth ml of the inoculum was placed in each of 2 cell culture tubes containing a cell monolayer. The

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[a] IBR-Lepto, Bio-Ceutic Laboratories, Inc., Saint Joseph, Missouri.
[b] Rhino-Lep, Diamond Laboratories, Inc., Des Moines, Iowa.
[c] Jencine-IL, Jensen-Salsbery Laboratories, Kansas City, Kansas.

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cell cultures were examined each day for cytopathic effect (CPE). When CPE became evident, the cultures were frozen at -70 C until virus identification was attempted. If CPE was not evident by 5 days, the cultures were frozen at -70 C, thawed, centrifuged at 600 X g for 15 min, and the supernatant inoculated onto new cell cultures. Three subpassages were made before samples were considered free of virus. The final subpassage was tested for PI_3 virus by hemadsorption with bovine erythrocytes and for BVD virus by the immunofluorescence test. When IBR-like CPE developed, the samples of the original nasal swab samples were reacted with anti-IBR antiserum to neutralize IBR virus and allow other possible viruses present to replicate and be recognized as above. Serological identification of virus isolates was done by using the constant serum-varying virus neutralization test on secondary bovine fetal spleen cells in microtiter plates. A commercial antiserum monospecific against bovid herpesvirus 1, Colorado strain produced in germfree pigs [d] was used as the reference serum. All samples were run in quadruplicate.

Samples for *bacterial isolation* were cultured on blood, MacConkey, EMB and sodium azide agar plates and incubated aerobically at 37 C. Cystine heart bovine blood agar plates were inoculated and incubated anaerobically for isolation of *Hemophilus somnus.* Isolated bacteria were identified by colony and cell morphology and biochemical reactions according to standard bacteriological procedures.

Hayflick's agar and broth were inoculated with swab samples for the *isolation of mycoplasma.* The plates and tubes were incubated anaerobically for 24 h at 37 C followed by incubation in a candle jar.

Results

Fourteen animals had the clinical criteria for rhinitis syndrome and were used in this study. The virus isolation and serological responses of these animals are summarized in Table 1. Accession numbers are in chronological order. IBR virus, serologically confirmed, was isolated from all of the first 11 subjects (September 25-November 11) on first passage in cell culture. One of these 11 animals died, and of the remaining 10, 9 had seroconverted to IBR virus 21 days later. Three of these first 11 subjects also seroconverted to BVD virus and one to PI_3 virus. However, only IBR virus was isolated from these animals. IBR virus was reisolated from 3 of these 11 subjects 21 days after the onset of illness. IBR virus was not isolated from the remaining 3 of 14 cattle nor did they seroconvert to IBR virus (December 11-January 7). Another virus, not IBR, was isolated from subject 17189. No additional attempts were made to identify this isolate serologically after IBR antiserum failed to neutralize it.

[d] *Miles Laboratories, Inc., Code 66-105, Lot 10, Elkhart, Indiana.*

Table 1 Serological Responses and Virus Isolations

Animal	IBR Antibody Titers Acute	Conval.	IBR Sero-conversion	IBR Virus Isolation	Other Virus Seroconversion	Other Virus Isolation
13606	<2	12	+	+	-	-
14414	<2	6	+	+	-	-
14324	<2	24	+	+	BVD	-
14368	12	128	+	+	PI-3	-
14756	<2	<2	-	+	BVD	-
14999*	<2	ND	NA	+	NA	-
15016	<2	16	+	+	BVD	-
15018	2	48	+	+	-	-
15272	16	64	+	+	-	-
15277	4	96	+	+	-	-
15431	4	64	+	+	-	-
16104	128	128	-	-	-	-
16219	12	ND	NA	-	NA	-
17189	48	48	-	-	-	+

Mycoplasma and bacterial pathogens were not isolated

*Animal died ND = Not done NA = Not applicable

IBR virus was isolated from 8 of 48 (17%) normal pen mates of the clinically ill subjects. Another virus, not IBR but one that produced CPE identical to the virus isolated from subject 17189 (Table 1), was isolated from 6 of 48 (12%) normal pen mates. The normal animals from which IBR virus was isolated were pen mates of the first 11 subjects only. The unidentified, non-IBR virus isolation occurred randomly throughout the study.

While IBR virus was isolated from normal pen mates, the isolation pattern in cell culture was different from that of the clinically ill subjects. IBR virus CPE was evident on first passage, frequently as early as 24-48 h after inoculation onto cell culture for clincally ill subjects, while CPE appeared later (4-5 days first passage or on second passage) for the normal pen mates. This may infer that only small amounts of virus was being shed from normal animals. SN antibodies to IBR virus were negative (<1/2) in 19/51 (37%) of normal pen mates.

Pasteurella haemolytica was isolated from 7 of 14 animals, and *P. multocida* from 1 of 14, but in no case was there large amounts of *Pasteurella. Corynebacterium pyogenes, Escherichia coli, Pseudomonas, Bacillus* and alpha streptococcus were also isolated randomly and in various combinations from most of the subjects. Neither *Hemophilus somnus* nor mycoplasma were isolated from any animal.

Discussion

The occurence of clinical IBR in feedlot cattle vaccinated against the disease was confirmed by the following criteria: a) the development of clinical signs (7), b) isolation of IBR virus and c) seroconversion to IBR virus. We also concluded that other agents can cause a clinical syndrome similar to IBR. Our results indicate that in this feedlot true IBR was most common in early to late fall, whereas another clinically

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similar disease was prevalent in early winter.

Reasons for failure of a vaccine to protect against clinical disease can be manyfold. The animals may fail to respond to vaccination. In a companion study, 20% of vaccinated animals did not have measureable SN antibodies 21-35 days after vaccination (2) and 37% of the normal pen mates (of the subjects) in this study had no measurable SN antibodies. Possible reasons for the lack of antibody stimulation are discussed in the other report (2). IBR virus has been shown to become latent in the body of recovered animals. Stress, administration of corticosteriods and other factors can stimulate a recrudescence of virus shedding with development of lesions in some animals (4, 13). The presence of antibody, even in high titer, does not guarantee against virus reactivation (4). The viruses isolated from clinically ill and normal animals could conceivably be vaccine strains shed from vaccinated animals. Unfortunately, markers that could differentiate between wild type and vaccine strains were not available at the time this study was done. Recently developed nucleic acid oligonucleotide "finger prints" can distinguish between virus strains but these techniques were beyond the scope of this project.

It is most important that the reader not interpret our results to mean that IBR vaccines are generally ineffectual and not protective. Before vaccines were available, IBR epizootics in western feedlots were frequently severe and we believe vaccination of cattle against IBR is very important. However, it is also important to recognize that a variable but significant number of cattle may develop clinical IBR despite vaccination. To obtain maximum protection from a vaccination program, careful handling of a "fragile" vaccine is very important and the vaccine should be administered to cattle at an appropriate age and in the absence of significant stress or concomitant disease.

The purpose of this study was to determine if clinical rhinitis (IBR) could occur in feedlot cattle known to be vaccinated against IBR virus. However, the incidence of IBR in this study should not be confused with the actual incidence of IBR in feedlot cattle. No attempt was made to critically evaluate every animal with rhinitis and many clinically ill cattle lacking one or more of the pre-determined criteria for this study were disregarded.

Summary

Fourteen feeder cattle that had been vaccinated against Infectious Bovine Rhinotracheitis (IBR) at least 21 days previously developed clinical signs of acute respiratory IBR (rhinitis). IBR virus was isolated from 11 of the 14 subjects. Seroconversion to IBR virus occurred in 9 of these 11 cattle and one died acutely. Several of these 11 cattle also seroconverted to BVD virus or bovine PI_3 virus, but neither of these viruses was isolated from any subject. IBR virus was not isolated from nor did seroconversion to IBR virus occur in the remaining 3 of 14 cattle. Another virus, not IBR, was isolated from one of these three. The potential for vaccine failure to protect is discussed.

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