Some Viral Diseases Associated with Cow-Calf Production*

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Fluorescent Antibody
Rabbit + Specific Antigen = Specific Antibody
Specific Antibody + Fluorescein Isothiocyanate = Special Conjugate
Specific Conjugate + Specific Antigen = FA +

The above is the direct FA method, but there are indirect and complement fixation methods. All of these are quite reliable, if properly performed. As with any laboratory technique, errors are possible.

Improper results in the test may be due to:

1. Dead antigen—rotten tissue or animal has recovered
2. Autofluorescence—eosinophil
3. Wrong tissue—(tissue not affected)
4. Improper pH—must be basic 8+
5. Error in preparation and reading

The fluorescent antibody examination is the most rapid and yet reliable test we use in our laboratory. We utilize this procedure for rapid IBR, BVD, TGE, and Clostridial detection. Tissues can be prepared and read easily within two days while, if circumstances warrant, tissues can be prepared and read the same day. This is the one tool for recognizing viral infection whereby an answer can be given to the practitioner in time for use in the case involved.

Infectious Bovine Rhinotracheitis

Recognized forms of the disease are:

1. Respiratory—shipping fever complex
2. Ocular—winter pinkeye
3. Vaginal—IPV
4. Preputial—BP
5. Abortion
6. Central nervous system—not common
7. Fatal diseases of newborn calves

All forms of the disease are caused by a Herpes virus which may have a latent period. Apparently the agent has adapted to various tissues of the body and thus produces the various forms of the disease. Multiple forms of the disease can occur. In our experience, this is the exception rather than the rule, although individual members of the herd may have different forms of the disease at the same time. McKercher (1963) considered forms of IBR as occurring in the absence of viremia, but French (1962) was able to recover IBR virus from washed leucocytes from blood of affected cattle. Abortion form of the disease suggests that there is a viremia at some time after exposure. Variations of the diseases are suggested to be due to intrinsic factors, virus adaptability, changes in environment, and host susceptibility. Other forms of the disease can be expected to develop. Experimental mastitis has been reported by Baker, et al. (1960).

Properties of the virus:

1. Member of Herpes group
2. Acetone and ethyl alcohol inactivate the virus
3. Survival 9 months at -60°C (Griffith, et al., 1958).
4. Virus completely inactivated after:
   a. 50 days at 22°C.
   b. 10 days at 37°C.
   c. 21 minutes at 56°C.
5. Freezing and thawing from -70°C to 37°C five times has essentially no effect, but there was a 25 percent loss after two more cycles of freezing and thawing (Stevens and Groman, 1963).
6. The virus is stable between pH 5-9 but is inactivated at lower ranges (Hahnfield, et al., 1963; Griffin, et al., 1958).
7. This organism is not pathogenic for embryonated eggs, young mice, guinea pigs (Kendrick, et al., 1958). Mule deer are susceptible and may be carriers (Blood and Henderson, 1968).
8. Since it is a Herpes virus, latency is a problem. Infected cattle either by vaccination or

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natural infection can be made to shed the virus later by use of cortico-steroids or by stress.

9. Other viral properties not applicable for this discussion.

Study of the above characteristics of the virus helps explain why infection is more common in the fall and winter.

Respiratory Form of IBR—IPV

This disease was described early in 1956 by Madin, York, and McKercher but had been clinically recognized earlier in Colorado and California, although Schroeder and Moys described the disease in 1954. All of you know the clinical signs, so a limited description will be given here. Mention should be made here of multiple etiology of respiratory diseases in cattle. Our laboratory has recognized cases of pulmonary adenomatosis and IBR in the same herd at the same time.

The respiratory form of IBR may occur in dairy cows (one reported $50 loss per cow) and occasionally in beef cows, but is most common in the fall of the year in feeder calves. This disease in feeder calves is usually associated with another agent or agents.

Experimental incubation period is three to seven days while natural infection is 10 to 20 days after introducing new cattle. Clinical signs are elevated temperature, dyspnea, tenacious nasal exudate, lacrimation, and ulcers of nasal mucosa. Occasionally there is a blood tinged diarrhea. Lactating cows drop in milk production.

Mortality varies but is usually in the area of 10 percent of those affected. Morbidity is difficult to predict because of lack of previous history of animals. That is, some animals may be recovered immune while others are susceptible. Transmission is considered airborne contact.

Uncomplicated cases have mild necropsy lesions but these do not often die. Most animals which die have small erosions of nasal mucosa, which is nearly always red from severe congestion. The trachea usually has a pseudomembrane and contains excessive mucus with froth. The epithelium from these affected mucous membranes serves as excellent tissue for FA examination or virus isolation. The anterior-ventral portion of the lung is usually pneumonic. Pasteurella can often be isolated from the affected tissue. Inclusion bodies are described but I have never seen them. Special fixation is required.

Regional lymph glands (mediastinal), trachea mucosa, and upper sections of lung are good FA specimens.

Recovery from the infection usually requires 10 to 14 days, although complications may prolong the recovery period. Some calves may not recover for three to four months.

Diagnosis. This may be confirmed by a combination of clinical signs, low white cell count (acute cases), paired serum samples, aseptically collected nasal swabs (virus isolated and FA of epithelial cells), necropsy lesions and frozen sections, FA or virus isolation.

Treatment. Routine antibiotic-sulfonamide. Open but protected quarters with access to water and feed. Do not hesitate to use either form of IBR vaccine in the face of a respiratory outbreak in unbred animals. If the nasal form is used, change canulas between each animal. There is some indication of spread of TEME and other respiratory infections by canula. Serologic globulin (IGG), nasal mucosa globulin (IGA), and interferon production are essentially the same with either vaccine (intranasal or intramuscular) (McKercher and Crenshaw, 1971).

Ocular Form of IBR

Some of the early research of this form of IBR was done by Dr. Andrew Gray at Kansas State University under the direction of Dr. Harry Anthony. Most everyone has seen this condition and knows that it occurs most often in the winter (winter pinkeye) but can be observed during other seasons. Almost any age animal can be affected, although it is most often seen in feeder calves. Respiratory signs may or may not be associated. The virus only may be involved or it may be associated with *Moraxella bovis.* If the cause is viral alone, there is a granular-appearing, quite red conjunctiva with no corneal involvement. If there is corneal involvement, the periphery is first involved. Dr. Gray found the conjunctiva to have pseudoepitheliomatosis and postulated that there might be some relationship to squamous cell carcinoma of the eye. Infectious bovine rhinotracheitis virus has been isolated from clinical cases of cancer eye.

In susceptible herds the morbidity reaches 100 percent. We observed such a case in 800 head of backgrounding calves. Response was quite good subsequent to intramuscular IBR attenuated vaccine, but not all herds respond as well as this one. Serologic results are inconclusive in this form of disease. Vaccination is not always successful although it is usually recommended.

Pustular Vaginitis and Balanoposthitis

Daubney, et al., (1938) first described this as a cause of infertility and tagged it "Epivag." This is

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apparently the same IBR virus which produces respiratory and other forms of this syndrome. Kendrick, et al., (1958) and Parsonson (1964) indicated that transmission was primarily venereal but non-venereal method was possible. Infectious bovine rhinotracheitis-infectious pustular vulvovaginitis virus has been isolated from feces of cattle without clinical signs of disease. This suggests another source of infection. The virus may be excreted intermittently into the preputial sac for long periods after apparent recovery (Snowdon, 1965). Virus could be isolated from preputial washings up to 26 days after topical applications of the mucosa of the penis and prepuca (Studdert, et al., 1964). Spradbrow in 1968 reported isolation of IBR virus from semen of infected bulls. With AI as widespread as it is, this has serious implications.

Clinical signs vary from little external evidence other than restlessness and twisting the tail, holding the tail up and to one side, to severe purulent discharge, kicking and frequent urination. The vulva and posterior vaginal mucosa may be red. Usually there are small white pustules which later coalesce to form larger areas. The thick yellow purulent material which developes is odorless. Bulls have the same lesion of the mucosa of the penis and prepuce. Mating behavior is usually normal, although fibrosis may develop to the extent of permanent sterility.

Pustules develop within 48 hours after artificial infection. Temperatures may be elevated to 105°F and last for two to five days.

Paired serum samples are helpful from an academic standpoint but are of little value for the present case and they may not give much information. Infectious pustular vaginitis can be confused with contagious granular vaginitis.

Treatment and Control. None very effective. Variable control may result by stopping breeding. Because of secondary bacterial infection, antibiotic therapy may be indicated.

Abortion Form of IBR

Abortion due to IBR virus was first recognized during the early 1960's when pregnant cattle were vaccinated with attenuated vaccine. There were several papers published to indicate that it could occur, while others went to great lengths establishing that the virus was not the cause of the abortion. Eventually abortion due to IBR vaccines was substantiated.

Canadian researchers reported natural forms of IBR abortion during the middle 1960's while a year or so later South Dakota reported a high incidence of IBR diagnosed abortions during the 1969-70 year. Our laboratory subsequently reached peak years during 1970 and 1971. I suspect that Kansas had their peak year in 1972, because the pattern is for this disease to move from north to south.

Table 1

Comparison of the Clinical Features of IPV (Kendrick, et al., 1958) With Those of Contagious Granular Vulvovaginitis (GVV) (Hunter, et al., 1958; Afshar, et al., 1965)*

Clinical Features	IPV	GVV
Rise in body temperature	To 104-106°F	None
Lesions	Soft, can be ruptured	Firm, translucent when small
Time of appearance of lesions after experimental infection	1-2 days	3-14 days
Vaginal discharge	Straw-yellow color	Whitish-yellow and often sticky
Epithelial cells of the vaginal smears contain intranuclear inclusion bodies, when fixed with Bouin's fluid and stained by haematoxylin and eosin	Yes	No

*Taken from Afshar, 1965. Veterinary Bulletin.

Both dairy and beef cattle of all breeds are affected. Normally the cows do not have clinical signs until abortion occurs. Ormsbee (1963) reported a febrile reaction prior to experimentally induced IBR infection. Our experience and other reports is that the fetus is expelled dead. Abortion can occur at any stage of pregnancy. Artificial abortion occurs 20 to 35 days after inoculation (Faulkner, Chow, 1968). In one instance we did see acute IBR infection in calves at birth. The cows apparently were infected at exactly the correct time for this. If the cow is immune to IBR, abortion does not occur.

Various gross descriptions have been given of aborted fetuses, but I find it nearly impossible to give a correct gross diagnosis of the cause of abortion. Bicknell and Reed were able to find miscroscopic focal necrosis lesions of the liver in 91 percent of one group of cases studied. We have not had that good response. Many of the cases we get have been frozen or are too decomposed to do histopathology, but in those well preserved cases we have not found a high incidence of necrotic foci of the liver.

How do we diagnose the cases? We routinely do frozen sections of the lung, liver, adrenal or kidney using IBR-FA conjugate. Stomach contents are examined by darkfield and phase contrast, while this material plus liver, spleen, and kidney are

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bacteriologically examined in both O_2 and 10 percent CO_2 atmospheres.

Prevention. Prevention is best done by vaccinating open cows and replacement heifers with attenuated vaccine. Aborted cows are thought to be immune for several years.

Killed vaccines have been reasonably successful in the face of an outbreak. We do not recommend any attenuated vaccine during an outbreak. As a matter of fact, we have knowledge of cases where this has been done with exacerbation of an otherwise quieting situation.

Acute IBR of Calves

There is very little written about this condition, but we have observed the condition. Other people report essentially the same findings. The gross lesions could easily be confused with those of bovine virus diarrhea but FA is negative to BVD conjugate and positive to IBR conjugate. Control conjugate of normal bovine serum was negative. Pustules were observed under tongues of calves examined.

Bovine Virus Diarrhea

Acute

First described by Olafson, et al., in 1946, while at the same time the acute form was described in New York, Indiana, Nebraska, and California.

Serologic information indicates that bovine virus diarrhea is widespread, occurring in most countries. Local serologic surveys indicate that over 50 percent of the cattle have had previous exposure to the BVD virus. Incidentally, buffalo and deer are also affected according to serologic sampling. All types of cattle may be affected. We have observed BVD in range cattle as well as feedlot cattle. There is a wide range of reactions in a herd, making a prediction of the seriousness almost impossible. All of us have seen the very mild form of transient diarrhea with little after effects and the extremely acute, severe diarrhea where calves never grow as well as they should. These calves tend to have eye involvement, persistent diarrhea, mild nasal discharge, as well as salivation. In some calves there is a severe reddened mucosal surface followed by ulceration. These calves are thin, yet continue to eat. We have also observed cases of acute death in fat cattle. These cattle were actually dying from hemorrhage through ulcerated Peyer's Patches. There was no evidence of coccidia or Salmonella.

Clinical Signs. By way of review, you will remember there is a febrile response lasting 48 to 72 hours with a subsequent drop for three to four days, followed by a second rise, the so-called diphasic temperature pattern observed with most viral infections. Leucopenia is also typical during the early part of the disease. Calves acutely affected may have signs of severe headache or brain disturbances before lesions can be observed. Diagnosis was confirmed by S-N reaction. We have observed cases where MD type lesions were observed with negative S-N but eventual virus isolation. These are difficult to explain.

Lesions. Principle lesions are erosions of mucosal surfaces of the gastrointestinal tract. These may be very small or extremely obvious and may occur from stem to stern. Erosions are most often, but not always, present in the esophagus mucosa. Erosions of the abomasum must be differentiated from those occurring as a result of being paralyzed or those associated with respiratory embarrassment. Lesions are often observed on the tongue and palate. Intestinal ulceration of Peyer's Patches may be observed.

Chronic Bovine Virus Diarrhea—Mucosal Disease

Older practitioners recall seeing the charts describing the similarities and differences between IBR, Indiana Virus, malignant catarrhal fever, BVD, MD and others. Now we know, thanks to the virologists, that BVD, MD, New York Virus, Indiana Virus, Nebraska Virus, and California Virus are all the same agent. The only differences were geographical location and host response to the virus. Several theories have been given for the MD form of the disease. None of those I have heard fit all circumstances. There seems to be little doubt that it is a result of lack of immunologic competence of the host. Several treatments have been offered for MD. I personally have never seen a recovery.

This form of BVD could readily be confused with blue tongue, chlorinated naphthalene poisoning, MCF, and mycotic stomatitis.

Control of BVD-MD. Vaccines (modified live) are available. These are effective and if given after 6 to 10 months of age confer a lifelong immunity. Incidentally, the IBR vaccines have been contaminated with BVD virus up until recently. **This vaccine should only be used in healthy animals.** We have used it in one herd during an outbreak with satisfactory results, but do not recommend this procedure. Some veterinarians use the vaccine in young calves with varying results.

Treatments. Electrolytes and antibacterials.

Abortion

Some cows experimentally exposed during the first trimester of pregnancy abort but are not so likely if infected during the 2nd or 3rd trimester. Field abortions due to BVD have not been

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important in our laboratory. Manifestations of *in utero* infection include cerebellar hypoplasia, hydrocephalus, mummified fetus, alopecia, lens opacity and weak calves at birth. Ward, et al., (1969) reported 3 of 11 calves born after IV inoculation of the dams which were 5 to 7.5 months pregnant were born with lesions typical of BVD and were able to find S-N titers in four calves prior to sucking colostral milk.

We routinely check frozen sections of "follow up" abortions utilizing FA and have been unable to demonstrate a positive BVD-FA under these circumstances. We have used various organs, including lung, liver, adrenal, lymph nodes, and brain. If the BVD virus is the cause of abortion, it must become inactivated by the time we collected the tissues. We have been suspicious but unable to confirm a relationship of BVD virus to weak, small calves from reasonably well fed dams.

Kahrs, Kendrick and others have found aborted calves to have positive S-N titers of different order from dams and presence of IGM which cannot pass intact placental barrier.

Anomalies

Cerebellar hypoplasia, hydrocephalus and weak calves due to BVD virus infection of the dam have been fairly well documented.

Here, we should be checking serology rather than attempting to isolate the virus. Our viral isolation attempts have been negative.

Calf Diarrhea

Reo Virus

Pathogenesis and transmission is not understood, but we do find positive FA reactions in calves which are only a few hours old. Usually after three days to one week of age we do not find positive reo virus reaction from calves with a diarrhea. Experts have given lesions which are indicative to this infection. Experience in the laboratory finds that lesions are not helpful in making a diagnosis of reo virus infection. The calf tissues apparently have limited responses to multiple irritants.

Clinical signs range from death before clinical evidence of diarrhea to severe, more or less chronic, diarrhea. Blood may or may not be present. Color and consistence of feces vary from extremely water-like to yellow paste-like to white or gray. Depression is the rule rather than the exception. The nose is usually red and crusted, while pneumonia is extremely rare. Bacterial isolates associated vary from *E. coli*, beta hemolytic *E. coli*, Salmonella, occasionally an extremely resistant Streptococcus, Pseudomonas (especially after therapy) and Klebsiella. We routinely check the antibiotic sensitivity of these isolates. Antibiotic resistance may vary from year to year, thus the need for continued monitoring.

This virus was first isolated and reported by Dr. Mebus, et al., in 1969. Since then, numerous reports of field studies and isolation have been reported by Dr. Mebus and his co-workers. The virus appears to infect the epithelial cell of the small intestine causing a sloughing of intact cells. An avenue for bacterial infection is established. With this bacterial infection, we observe generalized infection and often CNS involvement.

As a result of considerable investigation of field cases and use of attenuated oral vaccine, Scour-vax was released last March (1973) by Norden. Doctor Gene White at North Platte Station directed the major part of the field research for this vaccine.

Vaccine is given by mouth as soon as the calf is born. Results of effectiveness in Western Nebraska are of the magnitude of 65 percent. Other areas, such as Northern Kansas, are around 50 percent. This varies with the infection of the area. It may be advantageous to try in a few calves with annual severe diarrhea. **Do not expect this vaccine to eliminate or control all forms of diarrhea in calves.**

Corona Virus of Calves

During the field studies with reo virus, calves which died were brought to our laboratory for pathologic evaluation. Many of these calves had no fluorescent antibody indication of reo virus. These calves were older than those expected of having reo virus infection, being 6 to 14 days of age. Material was collected and saved in the ultra cold Revco for researchers in Lincoln. Electron scope pellets revealed the corona type virus particles. After several attempts, Dr. Mebus finally was able to somewhat reproduce the disease in caesarean derived calves.

We have observed the combination of attenuated reo and corona vaccine to not be effective. Killed combined reo and corona cow vaccine was given to 16,000 cows in 42 herds at 90 days prior to calving to 30 days later with promising results.

Another Virus

Another type of virus has been observed by Researchers Stair and Mebus, but this virus has not been adapted to monolayer. Needless to say, there are probably other viruses involved in this syndrome.

Malignant Catarrhal Fever

Malignant catarrhal fever is a disease of cattle which occurs if the cattle are associated with sheep. Our laboratory has had one case in over four years. We do not consider this an important disease

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but one of which we should be cognizant. You will recall that gross lesions consist of erosions of mucous membranes and corneal opacity. Microscopic sections of the brain usually are those of a nonsuppurative encephalitis with perivascular cuffing.

Blue Tongue

Blue tongue is a viral disease long recognized in sheep. More recently reports of this causing fetal anomalies in cattle have been prevalent. Other reports indicate that this disease may affect adult or growing cattle. Because of the oral mucosa lesions, blue tongue may be confused with mycotic stomatitis or ulcerative stomatitis. Morbidity is less than 5 percent.

Fetal anomalies may be up to 15 to 20 percent. Infection must occur at 30 to 70 days gestation. Doctor Metcalf, Denver Federal Blue Tongue Laboratory, reports that no blue tongue virus has been isolated from cattle in Nebraska or Kansas. Positive serologic evidence (Agar-Jel and complement fixation) has been found in serums from Nebraska and Kansas cattle.

Culicoides is a known vector; thus, it is more likely to occur along creeks and rivers. There is some evidence that *Linognathus vituli* (sucking louse) can transmit the virus. Mosquitoes have not been implicated at this time.

Parainfluenza 3

Parainfluenza 3 experimentally has been shown to cause broncho-pneumonia in feed cattle. More recently this virus has been isolated from bovine fetuses and bull testes. Experimental abortion has been produced by injecting *in utero* fetuses with PI_3 virus. It appears that PI_3 virus requires a longer period to produce abortion than IBR virus.

Aborted fetuses are reported to have skeletal muscle and perirenal hemorrhage. Hemorrhage, cellular infiltration and lymphoreticular hyperplasia are found in the lungs of aborted calves. Inclusion bodies have been described.

Dunne, et al., have recently reported 55 percent positive PI_3 antibody of aborted fetuses. They regard PI_3 virus as a significant agent in sporadic abortions of cattle and suggest the use of modified live virus vaccine in herds where other etiologic agents have been eliminated.

Serum neutralizing antibody titer to PI_3 does not necessarily indicate immunity according to some experts. However, fetal lymphoreticular proliferation of lung tissue, IGM antibody and a different, often high S-N titer of fetal blood than dam blood are indications of the significance of PI_3 as an aborting agent.

Bovine Enzootic Abortion

Bovine enzootic abortion has been indicated but not proven in southern South Dakota. Apparently at this time, this condition is not important in the Middle West.

Polyarthritis Virus of Idaho

Polyarthritis virus of Idaho or weak calf syndrome is a condition first reported in 1964 along the Bitteroot Valley. During 1968 the Southeast of Idaho reported approximately 400 cases and 80 percent death loss. During 1973 over 1,000 cases were reported but treatment reduced the losses to 20 percent.

Doctors A. W. McClurken and E. H. Steaber furnish the material relative to this syndrome. Clinical signs consist of depression, weakness, often inability to stand. Calves may have the condition at birth or develop weakness during the first 10 days of life. Polyarthritis with hemorrhagic synovial fluid, which is somewhat turbid and contains neutrophils, is a common finding. Tenderness and thickness is noted over the joint capsule. Petechiation of the third eyelid is also reported.

Postmortem lesions include edema and hemorrhage in 95 to 100 percent of the cases. Focal erosions and/or extensive ulceration and hemorrhage of the stomach and intestinal mucosa are also observed.

Adenovirus and noncytopathogenic BVD virus have been isolated from the blood, buffy coat and synovial fluid of a calf with the weak calf syndrome. The adenovirus isolate was given intravenously or by aerosol to seven calves nursing BVD immune cows. One calf developed a diarrhea which lasted two days. White cell count remained in the normal range in all instances. Three calves had joint fluid which was typical of that observed in calves with weak calf syndrome. The virus was re-isolated from synovial fluid. Clinical studies indicated calves which are BVD infected may have similar joint reactions.

Serologic studies at the National Animal Disease Center show high BVD titers in herds with weak calf syndrome, but this is probably true of herds not affected by the latter condition. Titers for BVD increase from summer range to calving time; however, pre-colostral serology suggests that BVD is not the cause of weak calf syndrome.

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establishments producing medical preparations, and investigation into the misuse of drugs. At the present time there are eight veterinarians assigned to the district offices who assist the directors with veterinary activities and affairs.

In summary, our total effort is for the single purpose of safeguarding the health of our animal population and the wholesomeness of foods of animal origin. We have available the combined talents of industry, the veterinary medical profession, and government. Each must do his share, and each must be alert to the responsibilities and the legitimate interests of the other. As servants of the people, we in FDA have a public trust which must be met at all costs. As practitioners, you can accept no lesser responsibility in serving the public.

Please note: The Food and Drug Administration has no control over veterinary biologics. These are regulated by the U.S. Department of Agriculture.

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