

BIOSECURITY CONCERNS OF WASHINGTON DAIRIES THAT CAN BE ASSOCIATED WITH EXPANSION

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Coming soon to a dairy near you are diseases that you may have only read about. Nine years ago when I started practice in the Snohomish area, salmonella was a rare problem. *Mycoplasma* was a California disease. We did not know what heel warts were. *Clostridium perfringens* was an occasional issue. Today many herds have experienced the different manifestations of salmonellosis. There has been a tremendous increase in the number of *Mycoplasma* outbreaks. Today *Mycoplasma* is more than a mastitis issue. Most dairies have ongoing programs to control heel warts. *Clostridium perfringens type A*, in addition to types C and D, has recently become an important cause of sudden death.

Our ability to move cattle around the continent along with the unclosed nature of dairies has contributed to the increased frequency of some of these diseases. The activities associated with maintaining a dairy, such as heifer replacement and bull purchases, are facilitating disease movement, even without the added opportunities provided by cattle purchases and herd expansions.

MYCOPLASMA BOVIS

It was uncommon to culture the bulk tank for the contagious mastitis pathogen *Mycoplasma bovis* when I started practice. In 1992 this became routine. True *Mycoplasma bovis* was a rare finding. By the early 1990's there were only two herds in our practice that had experienced *Mycoplasma*. In the last two years 12 dairies in our practice area have had positive cultures for *Mycoplasma bovis*. The disease is most often expressed as an incurable mastitis. Frequently the original concern would be a rising somatic cell count. Just as often, the complaint would be lame cows with severely swollen joints. Sometimes calves with swollen joints are also a concern. These cattle become so lame they will not use the affected limb. Pneumonia has not been an issue in our outbreaks so far.

To date we have not found an effective treatment for the infectious arthritis. These joints are fairly obvious in that the swelling is limited to the joint. It most commonly affects the front knee. It can also involve the hock and fetlock joint (by the dew claw). A joint fluid sample with special preservation media is used to confirm diagnosis. There is an environmental bacterium that cultures similar to *Mycoplasma bovis*. It is important, after initial culture, to determine whether the organism detected in milk, joint or respiratory secretions is indeed *Mycoplasma bovis*.

Mycoplasma mastitis is often characterized as creating meaty udders and sandy milk. However, cows carrying mycoplasma can have completely normal udders, milk, CMT scores and somatic cell counts. Some cows that have the disease may self-cure. Today, there does not seem to be a treatment.

So far cattle are the only identified source of the disease. The classic way that *Mycoplasma* has entered herds is through the purchase of cows with the infection in the udder. Since few of our herds are expanding via cow purchases other means of entry have surfaced. Heifers are probably our most frequent introducers of *Mycoplasma* into herds. This happens either as new purchases or when heifers return from contract raisers. When heifers are exposed to other heifers with *Mycoplasma pneumonia* they develop temporary pneumonia and the great majority recover without incidence of disease. A very low percent of heifers become carriers for mycoplasma either in their lungs and/or udders. When these cattle enter the herd they can shed *Mycoplasma bovis* from either reservoir. If it

is only from the udder then the only disease manifestation that we seem to see is mastitis. A bull could also be a respiratory system carrier. Calves drinking hospital milk can have septic arthritis and possibly pneumonia. However, if the initial introduction is through the respiratory system reservoir then mastitis, septic arthritis and / or pneumonia can happen. Sometimes the proportions of affected animals can be equal between mastitis and arthritis. With the respiratory reservoir flies may be involved in the disease spread. This model for *Mycoplasma bovis* spread is a compilation of disease research and clinical experience. Thus, while not well tested, this serves as our most logical explanation to date and a basis for successful control and prevention programs.

When *Mycoplasma bovis* infection is detected either as mastitis, bulk tank culture, or septic arthritis there are several options for dealing with the disease on a herd health basis. If the herd is fairly closed then testing and culling of all infected animals has been very satisfying provided the interval from initial disease introduction has not been too long. Often the testing is focused on new introductions, mastitis and pneumonia cows that have presented in the last few months. Animals that have recovered during this period are still suspect for *Mycoplasma*. If this does not result in a negative bulk tank and disease incidence then the entire herd can be cultured. Focusing on high somatic cell counts or CMT positive cows has not always been successful, as normal cows can be carriers. Once the bulk tank is negative, culture all cows at freshening that went dry during the outbreak and all mastitis cows for the next three months. This will allow quick elimination of new sources of the disease. With one whole herd culture, some dairies have been able to eliminate *Mycoplasma* without further testing. This approach has been very rewarding for our clinic. The only exception would be those herds with an ongoing exposure if they purchase replacements and/or are in an expansion mode. In this situation as well as during the test and cull phase approach it is important to manage the herd to prevent new cases. It takes 10 days to get definitive culture results for milk or joint fluid. Effective tools are: 1) stop feeding hospital milk to the calves; 2) use a back flush plus good milking hygiene in the parlor; 3) isolate the suspect cows; and 4) control the flies. For expanding herds these management control techniques are mandatory because of the ongoing disease exposure potential. Culturing and/or review of bulk tank history before purchase is a great tool. However, it is not practical for heifers or bulls. We might be able to detect respiratory carriers with culture, although to my knowledge, this has not been attempted as a screening tool.

The most important part of *Mycoplasma* control is back flushing in the parlor. This effectively controls the disease spread from the most significant reservoir, the udder. Using either hot water, iodine or chlorine with either manual or automatic back flush has been very successful. With this control, expanding herds have been able to continue purchases by letting the *Mycoplasma bovis* positive cows either cull themselves or self cure. Perhaps when there is less demand for cattle and better testing procedures, introduction can be prevented. Obviously research to better define this disease process would be helpful.

STAPHYLOCOCCUS AUREUS

This contagious mastitis pathogen has been with us for some time in the Pacific Northwest as well as in the rest of the country. Without careful screening it is possible to purchase infected cows. There is always some risk that a heifer can have it in her udder even if she is from a *Staph aureus*-free dairy. With an aggressive culture program where all fresh cows, mastitis cows and new introductions are examined it is possible to treat early cases and cull the incurable ones before other cows are infected. Again back flushing the equipment helps. Remember that back flush valves wear out allowing vacuum leaks and thus encouraging claw vacuum fluctuation. Plan to maintain those valves. Managing for a *Staph aureus*-free herd is a worthwhile objective that might be difficult to achieve while in an expansion mode. Management effort in the volatile cow market might be better spent controlling the spread, appropriately treating clinicals and using effective dry cow therapy. Using culture to specify antibiotic therapy facilitates the use of both systemic and intramammary administration. Unless you are able to culture all replacements before you buy them, expect to calve a few *Staph Aureus*-positive cows. Be prepared to deal with them.

STREPTOCOCCUS AGALACTIAE

I have yet to see this contagious pathogen on any bulk tank culture. However, it is still out there. Most intramammary tubes effectively treat this form of mastitis. There has been some suggestion with the reduction of *Strep agalactiae* that *Strep uberis* is emerging as a contagious cause of mastitis. It is certainly more difficult to treat.

In summary, it can be difficult to avoid the introduction of mastitis pathogens into the herd with the current volatile cattle market. If the new introductions are isolated, milked last and cultured, their risk to the other cows can be effectively determined. Spread should also be controlled with good milking hygiene that involves a predrip, individual cow towels, clean hands, post dip, back flushing of equipment and properly maintained equipment.

HEEL WARTS

This contagious disease showed up in Washington in 1992. We did not know the exact cause. It sometimes appears as a hairy wart and other times as an erosive skin infection. Scientists have discovered that spirochete-like bacteria cause it. In our practice the herds that have the roughest flooring and poorest manure removal seem to have the worst problems. Treatment and control are not too difficult. Numerous recipes for effective foot baths have been developed. There are some factors to consider that might influence results. The use of lincomycin or tetracycline may become less effective in foot baths as bacteria resistance develops. Foot baths significantly fouled with manure are less effective. A rinse bath before the treatment foot bath improves effectiveness. Topical treatment may give the best results when done on a regular schedule. If you ever eliminate these bacteria from your dairy consider foot bath treatment for all new cows entering the herd. If new cows can be isolated, the foot bath treatment could be repeated. Have everyone trimming feet disinfect his/her equipment before and after working on your dairy. Do not allow manure from other dairies to enter your farm on equipment or people. Depending on your facility design, this is not always that difficult to achieve. To date there have been no effective vaccines to prevent this disease. There is one vaccine approved for treatment. However, we have yet to be impressed with the results.

SALMONELLOSIS

The clinical disease expressions of salmonella can vary tremendously and to a certain extent is associated with the serotype involved. Calves often develop a toxic small intestinal infection that can progress so fast that they never show diarrhea before they die. Sometimes it can cause central nervous system problems in calves. Cows develop diarrhea that can be mild to profuse and sometimes very bloody. Cows can vary from mildly sick to dead in 24 to 36 hours. The disease comes to your farm from other animal reservoirs. This includes not only wild animals and birds but cattle also. So far the serotypes *Dublin* and *DT104* have been shown to create carrier cattle that shed the salmonella bacteria without clinical signs of disease. *DT104* also sheds in saliva. The most susceptible animals on the dairy are those undergoing significant stress. Fresh cows and calves less than one week old are more commonly affected. However, in one dairy *DT104* was so aggressive that even 6-month-old heifers contracted the disease. Controlling the amount of fecal matter in feed bunks, water troughs, calf feeding utensils and the calf environment can help limit the exposure to salmonella. In our older facilities limiting exposure can be a significant challenge and not an adequate means of control. Vaccination against salmonella is not a concept that all veterinarians agree with. For our practice we are 95% effective in protecting calves and cows from salmonellosis with vaccination. Vaccines that contain both *Salmonella Dublin* and *Typhimurium* do well. Endovac Bovi can also be successful and some cross protection can be achieved with J-5 type vaccines. Of the serotypes that we have experienced only *Newport* seems difficult to create immune protection for. Vaccinating heifers and cows one to two months prior to calving and calves just after birth has been the most rewarding approach. In some cases, with severe outbreaks, it is necessary to do the entire herd. Expect salmonella problems on your farm. Vaccinate your cattle and develop good hygiene in your feeding and housing protocols to limit exposure. All unexplained sudden deaths should be investigated with a necropsy and appropriate laboratory tests. Salmonella can cause chronic health problems and/or a dramatic large-scale loss of cattle.

CLOSTRIDIUM PERFRINGENS

This clostridial disease involves several types: A,B,C, and D are of most significance to dairy cattle. The emerging evidence suggests that type A, which is considered to be a normal gut bacteria, is causing a significant death loss. It appears that within type A there are some strains that produce much more toxin than others. Cattle can develop bloody diarrhea and die in 24 to 36 hours. More often the toxemia develops so rapidly that there is no diarrhea. The main finding at necropsy is bloody small intestines and hemorrhage over the surface of the heart. In calves, bloody abomasal fluid is found after death and this might be more associated with types other than A. If detected very early sometimes large doses of penicillin designed for IV use is effective in both cows and calves. There is evidence that herds that are expanding via cattle purchases see more of this problem in adults. In our practice it has been a unique problem for only a select few herds until the last few years. This suggests that some herds do not have the *Clostridium perfringens* type A that produces large amounts of toxin. We have learned that seven or eight-way clostridial vaccines that contain clostridium perfringens types C & D do not adequately protect cattle. However, vaccines specific for types C & D with or without tetanus do give some protection against type A. In the last two years it has become apparent that what we now assume to be type A is very active, and even a yearly vaccination is not adequate. We recommend C/D toxoid every 4 months. Some other countries have vaccines specific for type A. Hopefully we will have one soon.

SUMMARY

Mycoplasma, salmonellosis, and *Clostridium perfringens* all cause diseases that have up and coming significance. We either can not or do not routinely vaccinate for them. By upgrading our management protocols they can be dealt with fairly well. Fortunately live IBR and BVD vaccination programs have been in place in Washington for some time. So far I have not seen any outbreaks of IBR or hemorrhagic BVD. In the future, without proper screening our incidence of BVD, bovine leukosis (cancer) and Johnes could increase as new cattle are introduced into our herds.