## What does Congestive Heart Failure in Cattle Mean to Producers in the Northern Great Plains:

## Dr. Brian Vander Ley

## Season 1, Episode 69

[Intro music]

**Kiernan Brandt:**

Welcome to Cattle HQ, a podcast from industry experts and progressive producers discussing cutting edge info about the cow calf sector to keep cattlemen and women in the know and positively affect their bottom line.

**Robin Salverson:**

Welcome to Cattle HQ, brought to you by South Dakota State University Extension. I am Robin Salverson, a Cow/Calf Field Specialist living the life in Lemmon, South Dakota. I am joined by my guest, Dr. Brian Vander Ley. He’s an associate professor with the University of Nebraska-Lincoln, and he is also the director of the UNL Great Plains Veterinary Educational Center. Welcome Dr. Vander Ley to our Cattle HQ. Actually, I should say welcome back because I know some of my past colleagues have had you in the past, so thank you for joining us again.

**Brian Vander Ley:**

Yes. Thank you for having me. Yes.

**Robin Salverson:**

Absolutely. On this episode, we are going to focus on bovine congestive Heart failure in cattle. I know you’ve done a lot of research, extensive research in this area. Could you share why you have focused so much time on understanding congestive heart failure?

**Brian Vander Ley:**

Sure. I think there’s a couple of important things that have led my colleagues and I, because I’m part of a team of people that have been working on this for a while. I think probably to begin with, when I came here, I was not very familiar with congestive heart failure in cattle. I had the opportunity, Dr. Dale Grotelueschen was the director when I moved here to the University of Nebraska and he asked me, “Would you be interested in working on this?” That gave me the impetus to dig in a little bit and try to get a feel for what was going on, and that included visiting some feedlots that were in Western Nebraska that actually had some pretty significant issues with congestive heart failure. I think that was maybe the big driver and I saw the amount of impact that it was having in yards that were hard hit. Some of our early contacts with feedlots, there was some folks that were losing 3% to 5% of their calves in the feedlot in western Nebraska. It’s a big hit at any time and it was, in particular, it was notable at that time to me. The second thing that I think really has allowed, maybe it wasn’t the starter, but it has allowed the research to progress very nicely, is we have a cool team of people and we’re in kind of a nice location with some resources that are available. The team was started with Dr. Grotelueschen. He had a lot of contacts with feedlots that were struggling with the problem. We needed some animals that we knew were at risk. That was probably the first big step. He understood the problem. The second really important piece is that Dr. Grotelueschen and I both collaborate with Dr. Mike Heaton, who is a molecular geneticist. He’s a research scientist for the USDA stationed here at U.S. Meat Animal Research Center, and he’s very interested in the genetic components of this disease. Then, as an epidemiologist, I kind of have the ability to connect as a veterinarian. I could help with a lot of the - we did a lot of necropsy and stuff like that. We actually assembled a team that had the resources, whole genome sequencing, the analytical capability, and then the skills that it took to actually pursue it here. I think that’s why it’s a big problem and we had some unique capabilities to pursue it. It’s been - yes, we’ve been at it for quite a while now. I think I moved here almost eight and a half years ago.

**Robin Salverson:**

I was going to ask that question and how long has it - has the research been going on? With Dr. Grotelueschen and yourself now, it’s been going on for quite a while, but with - and we’ll get more into some of the things that your current research and future things to help mitigate or manage this issue, but kind of going back to, you said it has a large impact on the yards, do you know the financial impact it has or like a dollar value to that?

**Brian Vander Ley:**

Yes. We worked with a feedlot during - so in 2018, we actually hosted, we called it a collaborator’s workshop, and we had several different researchers who were interested in the problem come to GP VEC. Then, we had some producers and some industry groups show up. One of that actually, that workshop summary is posted on the USDA. It’s on the USMARC website. If you search bovine congestive heart failure, you can find it. In that summary, one of the yards, in their own cattle, they had I think about 1,500 animals that they fed a year. They figured heart failure alone was costing them about a quarter of a million dollars a year. That, again, pretty - higher incidence than a lot people have, but very expensive, very impactful.

**Robin Salverson:**

With the way the cattle prices are here this day and age, we sure don’t want to see some weaned calves that we paid a pretty penny for [Laughter] dying right at the end.

**Brian Vander Ley:**

Yes. Yes. Yes, which is it’s another piece of the puzzle is that they’re - I think the ones that die late are very memorable, but when we actually got into our work, we had cattle that were affected at every stage of the feeding period. We actually have pictures and my PowerPoint presentation, we talk about the hypothesis busters, the ones that don’t fit what we thought, and one of them was a day zero calf. Like they brought them in on weaning day and he already had brisket disease at that point. We had…

**Robin Salverson:**

I was going to ask you how soon you had seen it, so that’s soon. [Laughter]

**Brian Vander Ley:**

Yes. Pretty, pretty young. I think the ones that happen later are super memorable because we have a lot into them. We have a lot into them generally now, but when they go towed up on the way to the load out, that is - sticks in one’s mind.

**Robin Salverson:**

Kind of following up a little bit more on that, where is the highest percentage of congestive heart failure seen in regards to - is it the later, the ones almost getting loaded out? Or is that the highest percentage where you see it? Like you said, you saw some right at day zero when they were weaned. When does it occur typically on the way?

**Brian Vander Ley:**

We actually did a little bit of work, a master’s student, Dr. Adam Bassett, worked on that a little bit when he was working with me a few years ago. What he found in his work is that we actually saw it distributed throughout the feeding period. We definitely saw an increasing rate of cases occurring as the feeding period ramped up, so we looked at it on a percentage of the feeding period basis. We basically said, if an animal, if there’s a lot of cattle that are in a yard and they’re in there for 100 days and we see a case at day 33, we would say that one was 33% finished. So, we tried to correct for the differences in feeding period length. There’s some potential problems with that, but that was the approach we took for that work. What we saw is that up until about a third of the way through the feeding period, we were seeing - we saw a few cases very early, and then we would see a few more in the next 10% of the finish, and then a few more. Then, when we got to about 33%, about a third of the way through, we saw kind of a plateau, and that pretty much carried on. It actually tailed off at the end, but that was an artifact because a lot of yards have developed a lot of ability to spot these. What they do is they just market them early to salvage the value of those animals. That’s actually been published. Dr. Randall Raymond at Simplot Livestock and his team have published some work on their early - like they have kind of a program that they’ve developed to get compromised cattle out before they essentially lose their value. He talks about that quite a bit. Yes, long story getting longer, I guess, is that we see them all the way through the feeding period. It’s not just a late-day problem.

**Robin Salverson:**

I kind of got so excited and I got ahead of myself. I should probably back up a little bit and just describe to our audience what congestive heart failure actually is.

**Brian Vander Ley:**

Yes, that’s a great question because congestive heart failure is a - it’s a syndrome that shows up at the very end of a number of important pathological processes. When we talk about congestive heart failure, what we’re talking about is the end stages of heart disease where we start to see especially a lot of swelling. Brisket disease is a common name that people use and it gets that name because any area, when the heart starts or when it gets to the point where it can’t keep up anymore, it basically has backlog. There’s fluid, blood that can’t - isn’t moving around appropriately, so it creates some back pressure on the blood vessels. Those blood vessels get leaky and that fluid that leaks out of the vessels becomes edema or swelling. It just follows gravity. It goes to the low places. So, we’ll start to see it show up in that brisket area of those cattle and they’ll get a big, swollen brisket. It’s pretty notably different than fat. Once you’ve seen a few, it’s kind of hard to describe, but once you’ve seen a few of them, you’ll be able to appreciate it. We see a few other signs. Although actually they’ll leak a lot of fluid from their liver, and that’s a special kind of fluid. We call it ascites. It’s the ten-dollar word for it, but basically that makes them get a really big pendulous swollen abdomen because it’s full of fluid. Some of them will actually get fluid on their lungs. They’ll be like people with congestive heart failure where they actually get a lot of fluid in their chest cavity. Some of them will even - they’ll get bad enough where you can see their jugular veins stand out on their neck because the heart can’t move enough blood away from those jugular veins, so it backs up and make them distended. In the worst case of those, you can actually see the heartbeat pulsing in their jugular veins. Those would probably be the big characteristics, but there’s a lot of things that can cause congestive heart failure. We actually can see it on both. There’s the right side of the heart, which actually pumps blood to the lungs to get it reoxygenated before it goes back to the body. Then, there’s the left side of the heart which pumps that oxygenated blood out to the rest of the body. If we see right-sided heart failure, we tend to see more of the brisket disease, more of the issues with the big swollen abdomens and stuff like that. If we see left-sided heart failure, we’ll see more lung signs that actually will get lots of edema and fluid built up, not just around their lungs, but actually in their lung tissue. In people, a lot of - I don’t know what the percentage is, but my impression is that a lot of people actually will develop left-sided heart failure, that’s why we tend to see more lung problems with the people. In cattle, it’s easier for us to note right-sided heart failure. Left-sided heart failure is a little bit more difficult to diagnose, and the classic form of heart failure is the high elevation heart failure. Cattle, when they get up to, I think, I’m not exactly sure where the cutoff is, it’s somewhere between five and 7,000 feet of elevation. Their lungs are designed to - all of our lungs actually have this capacity, but if you want to make sure - our lungs are trying to make sure that our blood is well oxygenated. If there’s not a lot of oxygen in the air that’s in a particular part of the lung field, the blood flow actually slows down to make sure that it can harvest all of the available oxygen. The problem at high elevation is because the air is thin, there’s not as much oxygen, is it wants to slow all the blood flow down. That puts tremendous back pressure on the right side of the heart. It basically, it’ll compensate for a while. It’ll increase the force to try to drive blood past that, but after a while, it just can’t do it anymore and it starts to break down. Then, we see those right-sided symptoms. That’s one form, but we can also see heart failure develop from bacterial infections like Histophilus, like H. somnus, we vaccinate for that quite a bit. We can see it from certain kinds of toxicities, so rumensin toxicity can induce heart failure or any other ionophore, monensin, or others. We can see it with gossypol, which is a toxin that comes from cottonseed or cottonseed meal. We can see it from hardware disease. If they get a wire, we can see heart failure developed from that. There’s lots of paths to get there. The one that I deal with is a type of heart failure where we don’t actually - we are not 100% sure what the entirety of the cause is. We can talk about this more. We know there’s a genetic part of it, but there’s the genetics alone don’t predict exactly who’s going to get sick.

**Robin Salverson:**

You know that genetics is part of the cause of those ones that you’re talking about, that you work on. Do you have any inkling or idea of other factors that could be causing some of those or…?

**Brian Vander Ley:**

We have lots of ideas. I don’t know how good most of them are. [Laughter] [[Unintelligible]](https://recordings.civi.com/cgi-bin/player.php?file=Cattle%20HQ%20Ep72%20-69.mp3&starttime=868&duration=20) them down. We’re fairly confident that oxygen is still playing a role, but we’re not sure how. There’s two likely candidates. One of them could be - we do see, if you look at the state of Nebraska, we see them across the state, but we see way more of them in Western Nebraska, which I think it’s fairly similar to South Dakota in that we’re a lot higher on the western end. We are on the eastern end of the state. Right here at Clay Center, Nebraska, where I’m at, we’re at like 1,600 feet of elevation. Most of the panhandle is at or above 4,000, so it’s not in the risk zone for what we would expect for high elevation heart failure, but we think that the difference in oxygen concentration might be driving some of the problems, but we’ve talked about ways that we could work on that. It’s kind of a tough problem. We’d have to move cattle around quite a bit. Then, we introduce new potential problems with how we would identify whether - what the role of oxygen would be. Another thing that we’ve thought about relative to oxygen is that we are pushing cattle to bigger end weights, which creates a lot of metabolic oxygen demand. Those tissues need more oxygen and maybe that’s driving some of it, but it’s not just oxygen. We’re confident that there has to be a genetic predisposition because we have lots of cattle that get really big and don’t have any problems.

**Robin Salverson:**

It’s always fascinating because I know we had sent from our research facility here in Northwestern South Dakota, and this was several years ago, we sent calves down to Western Nebraska to be fed and we had several die and a high percentage. Everybody, they said it was brisket disease. I instantly went to high altitude or - and I’m like, but we’re going from Northwestern South Dakota to Nebraska. How can it be that, right?

**Brian Vander Ley:**

Yes.

**Robin Salverson:**

It was a form of congestive heart failure. My mind went directly to high altitude or high mountain, high altitude, but this - as this conversation we’ve just had with you makes a lot more sense. It may or may not been somewhat due to altitude because I think you guys are slightly higher even than what we are up here, but also then there’s other factors playing into it and not just the altitude. That always just blew my mind. I was like, how, how, how, how, how, how? I don’t know if they ever fully figured it out because I believe actually someone from your crew took some samples, but…

**Brian Vander Ley:**

Yes, probably not. The work we are doing now is very associative. We’re trying to tie an event to other things that are happening at that time or before it, which is a great way. It’s a great start, but where we want to get to is we want to know what causes something and we’re not quite there yet in terms of either the genetics, or the other factors. It could be, there’s just such a long list of things that we could look at, altitude, finish weight, diets. There’s some interesting changes in how we feed cattle over the last - the people that I talked to really talked about the early 2000s being when they started to notice this becoming a more important problem. There’s reports in the literature that go back to the ‘70s, the 1970, talking about this in feedlots, especially in Colorado. They were mentioned. There’s an extension bulletin that came out in Nebraska, I think in the late ‘70s or early ‘80s, talking about congestive heart failure that was kind of an unknown origin problem, that we think might have been this, the same one we’re working on now. There’s a big gap or didn’t seem like anybody was working on it that much. Then, when we started, especially when I got involved and I could interview feedlot owners and managers, they talked about some of the issues with - or some of the timing was 2000, mid 2000, like 2005, ‘06, ‘07, ‘08, that timeframe. The way we [[tend to]](https://recordings.civi.com/cgi-bin/player.php?file=Cattle%20HQ%20Ep72%20-69.mp3&starttime=1145&duration=20) cattle is kind of evolved and there was some notable changes that probably aren’t worth talking about today, but are interesting and I just haven’t had the chance to follow up on them yet.

**Robin Salverson:**

That’ll give you job security.

**Brian Vander Ley:**

Oh, [Laughter] security [[Unintelligible]](https://recordings.civi.com/cgi-bin/player.php?file=Cattle%20HQ%20Ep72%20-69.mp3&starttime=1160&duration=20) security, Robin. We got - not [[a risk]](https://recordings.civi.com/cgi-bin/player.php?file=Cattle%20HQ%20Ep72%20-69.mp3&starttime=1163&duration=20) to running out of problems anytime soon.

**Robin Salverson:**

That’s unfortunate, but slightly good in regards to…

**Brian Vander Ley:**

Yes.

**Robin Salverson:**

…paying your bills. [Laughter] I have another question. I did some reading too in regards to the incidences of congestive heart failure, and I’m not sure if this data or this information is correct or not, so I’d like you to tell me if I’m right or not. There is a higher risk just in straight beef cattle versus the beef dairy, is that correct? Because there’s a lot more beef on dairy coming into our feedyards, especially with the implementation of sexed semen, and then so on and so forth.

**Brian Vander Ley:**

Yes. Yes, the beef on dairy thing is one that we haven’t spent a lot of time looking at, so there, in the area of the genetics of congestive heart failure, Holsteins actually have their own genetic problem with congestive heart failure. It’s not the one that we’re seeing in beef cattle. They’re different genetic markers that are tied to those. I don’t know. I probably can’t speak very - I’m not very well informed on the incidents in beef on dairy cross cattle. I know that we see a pretty big range when we look at beef cattle. We’ve seen estimates from as low as 15 cases per 10,000 head in the literature that’s been published to some of, again, going back to some of the places that we’ve worked with, have been as high as 70 cases per 10,000 head placed. That’s not answering the question about what’s the difference between the beef on dairy, but that’s a - we have reports from consulting veterinarians about straight Holstein steers that have had quite a bit of problem. The whole beef on dairy cross is - it’s a whole bunch of other podcasts, right? That’s [[Crosstalk]](https://recordings.civi.com/cgi-bin/player.php?file=Cattle%20HQ%20Ep72%20-69.mp3&starttime=1283&duration=20).

**Robin Salverson:**

It is. It really is. Yes.

**Brian Vander Ley:**

It’s certainly becoming a - it’s a - they’re a force to be reckoned with at this point. There’s a lot of those calves out there.

**Robin Salverson:**

There are a lot of calves, more research to be able to do. Yes, that’s - it’s - I think the feedyards are - I don’t know the percentage of the number of beef on dairy within feedyards, but like you said, it’s increasing as our dairy start using more beef genetics into their systems.

**Brian Vander Ley:**

Yes. Some of the work that’s coming out, this is totally off-topic, but some of the West Texas A&M, Ty Lawrence and his group, work that they’ve done, looking at the carcass value of those animals, they are pretty close to the native.

**Robin Salverson:**

Yes. It’s very interesting. Is there anything that cow/calf producers can do at the, like the farmer ranch level, to help decrease or reduce those incidences of congestive heart failure?

**Brian Vander Ley:**

Yes, there actually is. One of the big findings that we’ve come across in the last eight years of research is that there is a genetic link. It’s a genetic predisposition. We actually have identified three factors. Two of them we’ve published - we’ve pretty much done the - we’ve done a really thorough job. Then, one of them we have made public, but we’re still working on some of the validation for it. The two first factors that we published on were both risk factors, so they were associated with an increase in the frequency of heart failure in the animals who had them compared to animals who didn’t. Then, the third one is actually a protective factor, so animals that have one copy of this particular gene or marker in a gene are at lower risk than animals that don’t have a copy of it. Those three genetic links are, we think, pretty valuable. The first two – so I always kind of struggle with how to refer to these because they don’t have super concise names, but we’re going to talk about them in terms of the genes that they’re part of. We found two risk factors that are associated with a gene called ARRDC3 and I - that’s arrest in domain-containing, I can’t remember the name, the full name of the gene, but it’s ARRDC3, so I’m going to talk about it. We have another one called NFIA. Then, there’s a gene called jumonji. All of these genes are associated - they have associations with heart health or disease in people and in mice. There’s some links to heart conditions in other species. That’s kind of cool for us because it tells us that we’re - it’s another piece of evidence that we’re on the right track, but what we see with the two risk factors, so ARRDC3 and the NFIA gene is - both of them, by themselves, if an animal has ARRDC3 or NFIA, they’re about eight times more likely to have congestive heart failure than an animal that doesn’t have them. If they have them, if they have both of them together, they’re about 28 times more likely to have congestive heart failure than an animal that doesn’t have them, has either one of them. The problem is, is that because they’re a genetic predisposition and not a genetic determinant, it’s not like coat color where if they have a certain combination, they’re going to be red or black or different color, it just seems to push them closer to the edge. We talk about something called positive predictive value, which is the percentage of animals that have both of the genetic risks that actually will become heart failure cases, and that number is actually really low. It’s like 8% to 10%. What that tells me is that for animals that are at genetic risk, there’s actually - the majority of them will never develop heart failure, which makes testing for a feedlot really impractical, right? If I have to take a pretty big chunk of the population and do something different with them, which we don’t know what the different thing would be anyway, I would only affect about 8% to 10% of that population. So, the positive predictive value is not that good. The value is in its negative predictive value, which is the percentage of animals that don’t have the risk factors that actually get heart failure, or I should say that differently. It’s percentage of animals that don’t have the risk factors that stay healthy. That number is 98%, 99% plus, like it’s really good. Meaning that if they are not at risk genetically, we don’t see heart failure develop in those animals. That makes it actually a pretty strong breeding tool for cow/calf producers, because if they can find genetics that don’t have the risk factors, they can find animals that don’t have the risk factors, then they actually can breed the risk down in their herds. We’re involved with a ranch since working on this. We’ve been doing it for quite a while. We don’t have the final outcomes for that yet, but I think the most important thing I’ll say is that the risk factors are fairly prevalent in some breeds, especially the British breeds and in particular, we see quite a bit of risk in Angus cattle, and that’s been validated independently with some other methods. Dr. Raymond and his group at Simplot actually had a similar take home from some of the work that they did is that the Angus cattle were pretty rich with risk factors, and that fits what we see in the yards as well. The good news is, if you can find animals that don’t have the risk factors, you can start to get rid of the risk, but it’s going to take time. It’s going to take a while. Both of the ARRDC3 and NFIA risks are recessive, so in order to be at risk, they have to have both copies, which is a good thing because that means if we can get rid of one of the risk copies in the offspring, we can alleviate the risk in that generation. They’re still carriers, but it can help. It’s an ongoing process. We’re still learning. The protective factor we have not done quite as much work on, but it’s pretty exciting because it looks like for the type of heart failure we’re looking at, that if they have one copy of this jumonji, this protective jumonji marker, that they are in pretty good shape. They don’t seem to get very much heart failure, but we’re still working on that one.

**Robin Salverson:**

I’d like to just say that’s the best name ever, jumonji. You go from ARRDC3 and NFIA to jumonji. [Laughter]

**Brian Vander Ley:**

I agree. It rolls off the tongue a bit more easily than [[Unintelligible]](https://recordings.civi.com/cgi-bin/player.php?file=Cattle%20HQ%20Ep72%20-69.mp3&starttime=1729&duration=20).

**Robin Salverson:**

Exactly. Just to go back to these, the ARRDC3 and the NFIA, the risk factors, is there a commercial way that producers can start testing, some of our seedstock producers be able to do that?

**Brian Vander Ley:**

Yes. Yes. Those tests, there are a couple - there’s one, there’s for sure one company that I’m aware of that’s doing that kind of testing specifically. It’s a company called Bullseye-Genetics. I would have to do a little bit of follow up. GENEZ or Neogen was doing it. They may have changed, but there are some options that are out there.

**Robin Salverson:**

Great. That’s great that there’s something for producers to start trying to implement if something that they’ve been seeing or having issues with or don’t want to have potentially in the future. That was all on the cow/calf side of things. For our feedlots specifically, can they manage or do anything differently to help reduce those incidences of heart failure?

**Brian Vander Ley:**

At this point, we just don’t have great tools for feedlots. What this problem has turned into is it’s another problem that we see manifest at the feedlot level. It looks like there’s a lot of factors that contribute to it that some of them, and again, I’ll say we just don’t know what those factors are. I’m certain that some of those will be feedlot controllable when we can finally - there’s enough anecdotal evidence that different kinds of feeding programs may have an impact on incidents. Location is probably going to have an impact. It’s pretty hard to move feedlots from one place to another, but it’s one of those problems that they’re - the whole supply chain is involved, the seedstock producers and the commercial cow/calf folks, and then the feedlots are all involved in creating the risk or the lack of risk for disease, but it doesn’t really show up in most cases until they get to the feedlot, which makes it a difficult problem to tackle.

**Robin Salverson:**

Absolutely. [[Crosstalk]](https://recordings.civi.com/cgi-bin/player.php?file=Cattle%20HQ%20Ep72%20-69.mp3&starttime=1883&duration=20) Like you said, it - go ahead.

**Brian Vander Ley:**

It fits right into the BRD problem. It’s like we - respiratory disease, we accumulate risk all the way up to supply chain, but most of the time we actually see the disease when they get to the feedlot.

**Robin Salverson:**

Like you said, it’s definitely the effort of the seedstock producers, the cow/calf, the commercial cow/calf, and the feedyards. Once you guys find out more in the world of research and the implementation of some of those research findings, hopefully things will look better [Laughter] because I know, like you said, you’ve talked about the past, the current research that you’re working on. Is there any future ideas that you would really like to work on?

**Brian Vander Ley:**

Yes. I’ll talk about one. I’m a little hesitant to talk about things that aren’t done yet, but…

**Robin Salverson:**

Absolutely. [Laughter]

**Brian Vander Ley:**

An exciting project that we’re working on, so one of the biggest challenges that people that are working with trying to breed congestive heart failure risk down in their animals is that you have to own the genetics before you can find out what you have. If you want to purchase a bull, be it natural service, natural cover bull, or an AI sire, you have to purchase the bull first, but we’re working on trying to – and we - there’s a lot of pitfalls in making heart failure genetics public, because the problem with publicizing any particular animal’s genetics is that they might have some risk, but they might produce hundreds or thousands of calves that do just fine. That’s the part of this that’s really important to bear in mind, is that we don’t know those other factors that drive disease and they’re - perhaps a bull that might have a lot of risk, if we use them in Western Nebraska, it might produce calves that get a lot of congestive heart failure. If we moved into Eastern Nebraska or Iowa or Missouri, his offspring would be just fine, so we’re trying to be careful about how we do this. What we’re thinking about right now, what we’re actually working on is developing a - we’re calling it the winners list. [Laughter] It’s kind of a heart healthy genetics thing, but we’re not interested in devaluing any bulls, but we are trying to put together some - a list of bulls that for people who really want to breed down the risk, they have an access to this so that they can - it mostly will focus on AI sires for now, but we hope to get some natural cover bulls in there. We’re just trying to give people the opportunity to know something about a bull before they buy it, and that way, they can use it in their system. We also stress a lot, we don’t want to see people completely single-trait selection is - the bull’s got to work in their system. We think that things - something like heart failure is important. If the trait involves death, it probably moves up in the list of things that you might want to select against a little bit, but we are trying to be pretty careful about how we - we don’t want to create - we don’t want to devalue animals. We just want to give people the opportunity, if they’re interested in working on this problem, to have a little broader set of animals to pick from than what they do right now.

**Robin Salverson:**

Are you searching out cooperators to work with you on that project or…?

**Brian Vander Ley:**

Yes. Yes. We’re actively doing that right now, so yes. If anybody has bulls that they will be offering for sale either in semen or natural cover, definitely have them get in contact with me.

**Robin Salverson:**

It sounds like a very practical information that we all need to understand. Like you said, you’re not wanting to devalue a bull or devalue a genetic line, but just trying to make sure you offer or find bulls that reduce that risk.

**Brian Vander Ley:**

Yes. We’re just trying to match people that are trying, that are really struggling with this problem and trying to breed risks down with genetics that will help them accomplish that goal, but also help them maintain a productive, profitable herd otherwise as well.

**Robin Salverson:**

Absolutely. I’m going to go back a little ways in our podcast, and you had mentioned something, and then we’re going to wrap up actually. You said that feedyards were killing the animals earlier to help prevent. Can they do a test or something in the feedyard itself to know if animals are developing congestive heart failure, if they are not seeing any major signs yet, or were they killing animals that were starting to show preliminary signs of congestive heart failure?

**Brian Vander Ley:**

That’s when they start to see the early signs.

**Robin Salverson:**

Okay. Okay.

**Brian Vander Ley:**

So especially animals that early signs will be a little bit slow, a little bit depressed. It’s actually one of the challenges because very early in disease, it’s hard to tell it apart from pneumonia because you’ll see a lot of the same thing. That’s one of the challenges with early marketing, is if it looks like pneumonia and they get treated like pneumonia, now they have to contend with a slaughter withdrawal on the drugs. We have worked a little bit on trying to develop a test that would help feedyards discriminate between pneumonia cases. What we try to do is adapt off the shelf technology, things that already existed to see if we could make that work, and we have been fairly unsuccessful. One of the diagnostic tests that actually does work pretty good is pulmonary arterial pressure or PAP scoring. That’s a great diagnostic for animals that are either developing or in congestive heart failure because those numbers will go sky high. They’ll have really high pulmonary arterial pressure, but it’s an invasive kind of technically demanding test that there are a growing number of people that can do it, but they’re still - it’s not widely available.

**Robin Salverson:**

Yes, it does take a very, like you said, it’s a very technical procedure that, yes, [[Crosstalk]](https://recordings.civi.com/cgi-bin/player.php?file=Cattle%20HQ%20Ep72%20-69.mp3&starttime=2266&duration=20) not everyone can do.

**Brian Vander Ley:**

Dr. Holt is the master. There are other people that can do it. I’ve done it a few times. I would not call myself - I’m not that good at it. We can get it done, but I’m not that good. Yes, it is a good test. It tells you the state of their heart function at that time.

**Robin Salverson:**

Again, thank you. I really appreciate having you on this podcast. We probably should be wrapping this up, but do you have any last-minute thoughts or comments before we end?

**Brian Vander Ley:**

No, I’m always happy, if people are struggling with congestive heart failure in their herds, if they want to understand more of the genetic work that’s been done and what’s out there and how they can apply it in their system, I’m happy to talk about those things. I always tell people, if you search me on the website, the UNL website, I think my cell phone number is there. You’re welcome to call that number. My daughter says, “Dad, you’re not a real vet anymore.” [Laughter] The reason I bring that up is because if you call me at 2:00 in the morning, I probably won’t answer, but if you call, leave me a message, shoot me a text, I’ll call back. Yes, if people, if your listeners want to talk more about it, I am more than happy to engage with them and share more details and options.

**Robin Salverson:**

Aren’t daughters and sons just give you a great reality check, don’t they?

**Brian Vander Ley:**

Oh, yes. [Laughter] Yes. All the time.

**Robin Salverson:**

Again, just a reminder to all the listeners, if you would like to work with Dr. Vander Ley on that last project he was talking about, about lining up some, whether it’s bulls that are going to be for natural service, you’re selling bulls for natural service or even through an AI type situation, to reach out to him. I think he would appreciate having more in his list.

**Brian Vander Ley:**

Absolutely. Yes, we want to make that list as big as we can make it.

**Robin Salverson:**

Excellent. Once again, this has been Cattle HQ, brought to you by SDSU Extension, headquarters for all things beef cattle. Visit extension.sdstate.edu for the latest beef information. Until our next episode, remember to live a great life.

**Kiernan Brandt:**

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[Outro music]