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# Flooring, temperament and foot problems: links to foot issues in the feedlot

Western Canada Feedlot Management School  
February 13, 2024

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# Feedlot lameness – how big is the issue?

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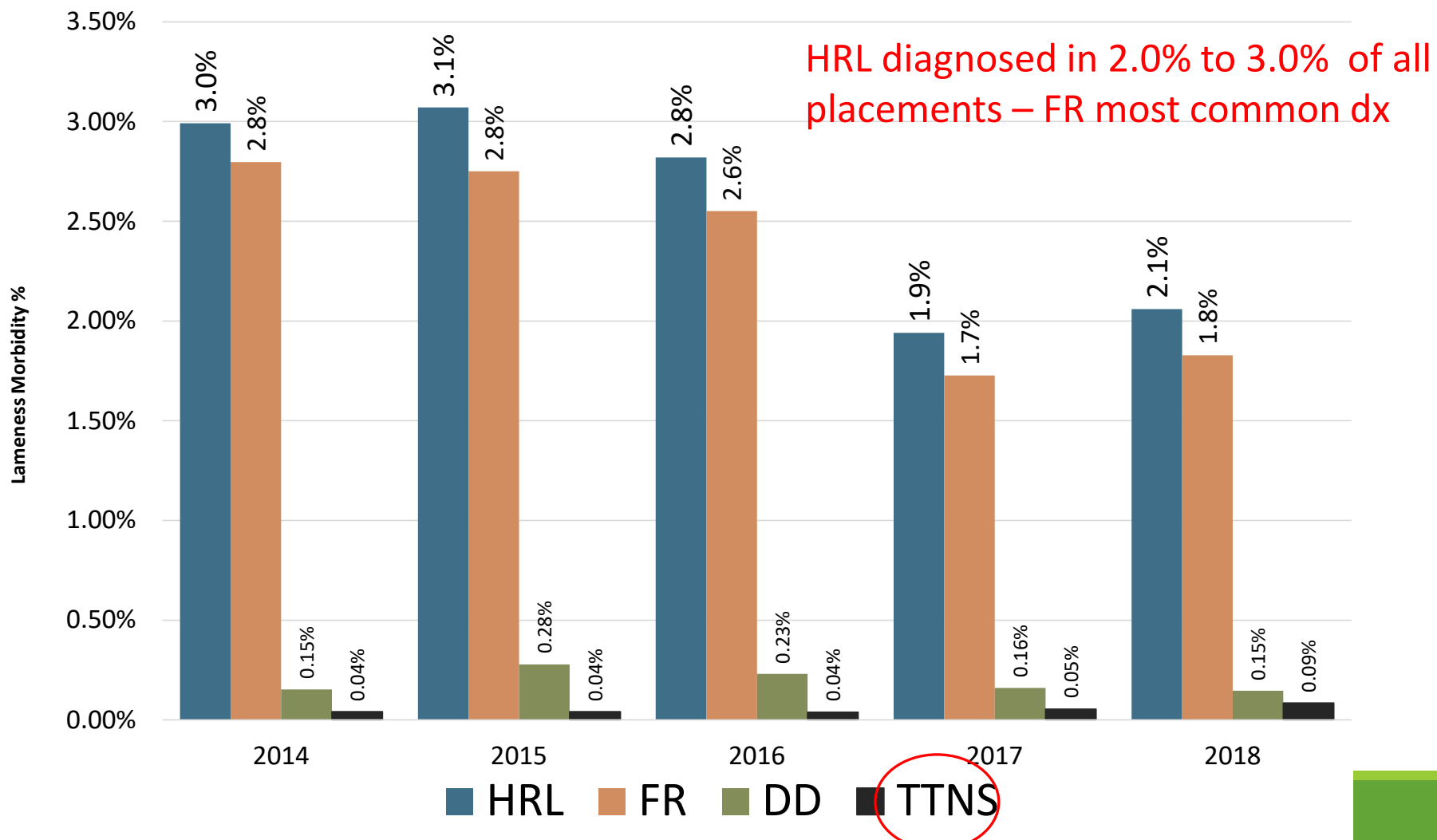
- What percentage of your feedlot treatments are for lameness?
  - Hendrick and Abesykara (western Canada) – multi-year study (2008-2013) found lameness accounted for **40%** of treatments.
  - Davis-Unger *et al*, 2019 - southern Alberta 10-year study of 28 feed lots found that lameness accounted for **32%** of treatments, range 1.3 to 46% .
  - Marti *et al*, 2021 - two-year study of two Alberta feedlots found lameness accounted for **36%** of treatments.
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# Feedlot Lameness

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- Sarah Erickson – 2022 (FHMS by TELUS Agriculture), 5-year study (2014-2018) of 1.77 M head, lameness accounts for **25.7%** of all treatments.
  - **Hoof-Related lameness (HRL): 70.8%** of lameness treatments and **18.3%** of all feedlot treatments, of which ~90% of HRL are foot rot.
- Terrell *et al*, 2013 – Nutritionists, veterinarians and feedlot owners rank **foot rot, injuries** and **toe abscesses** as the most common causes of lameness.

## Hoof-Related Lameness (HRL) as a Percent of Cattle Placed from 2014-2018 in 28 Western Canadian Feedlots (Sarah Erickson)





# Cost of lameness (Davis-Unger *et al*, 2017)

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- Davis-Unger *et al*, 2017
- Health costs = treatments costs, lost performance, and death
- Lame cattle average 48.5 lbs less than their healthy pen mates

Average return on healthy cattle = \$690.

- Average return on cattle treated for **Foot rot** = \$568
- Average return on cattle with **injuries** = \$259
- Average return on cattle treated for **joint infections** = -\$286
- **Average return on cattle treated for TTNS cases (lameness with no visible swelling) = - \$701**

# Toe Tip Necrosis Syndrome (TTNS)

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- A disease known by many names:

- **Toe abscess**
- Toe necrosis
- P3 necrosis
- Foot lesions (FTL)
- Apicus necrotica
- Apical white line disease
- Apical pedal bone necrosis

- Technically an ulcer; however, creates confusion with toe ulcers (TU) and thin sole toe ulcers (TSTU) of dairy cattle.

## Toe tip necrosis syndrome (TTNS)

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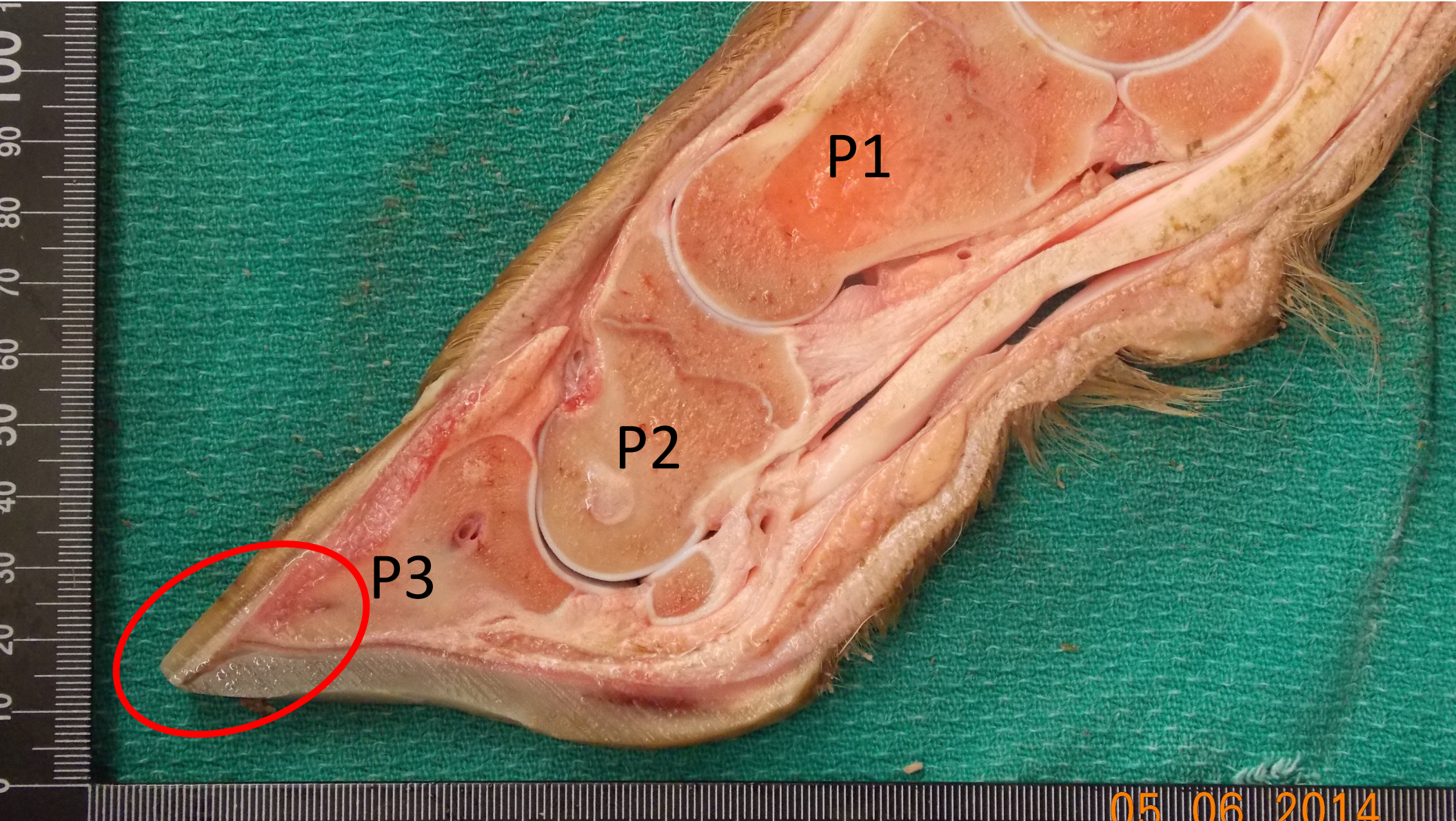
- Describes the continuum of disease presentations
- White line separation resulting in necrosis *and* lameness
- Involvement of the bones of the hoof (P3, Pedal or Coffin bone), tendons, muscles, cellulitis, and evidence of bacteremia such as embolic pneumonia

# Quick anatomy lesson

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P1


P2

P3

05 06 2014

## TTNS – not a ‘new’ disease

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- Dewes HF. *Transit-related lameness in a group of Jersey heifers*. NZ Vet J **1979**
  - Sick FL, Beeker CM, Mouw JK, Thompson WS. *Toe abscesses in recently shipped feeder cattle* (Veterinary Medicine/Small Animal Clinician, **1982**)
  - Miskimins DW. *Bovine toe abscesses* (Proceedings 8<sup>th</sup> International Symposium on the disorders of the ruminant digit, Banff, Alberta **1994**).
- 



# Description of TTNS

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- **Hindlimb lameness** seen within **days to weeks** of arrival to feedlot
- **No obvious swelling** of foot, often misdiagnosed with foot rot or trauma
- Tip of toe is worn, **white line separation**
- Toe lesion has **grey pus, dry-black material**,
- Ascending infection, and on PM examination evidence of **bacteremia**
- Risk factors: “**high-spirited**”, abrasive flooring, handling, soft hooves (wet season)
- Treatment/Outcome – **poor response to antimicrobials**, trim toes to effect drainage, place on soft, dry flooring

What does TTNS look like?

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Courtesy of FHMS



# Diagnosis

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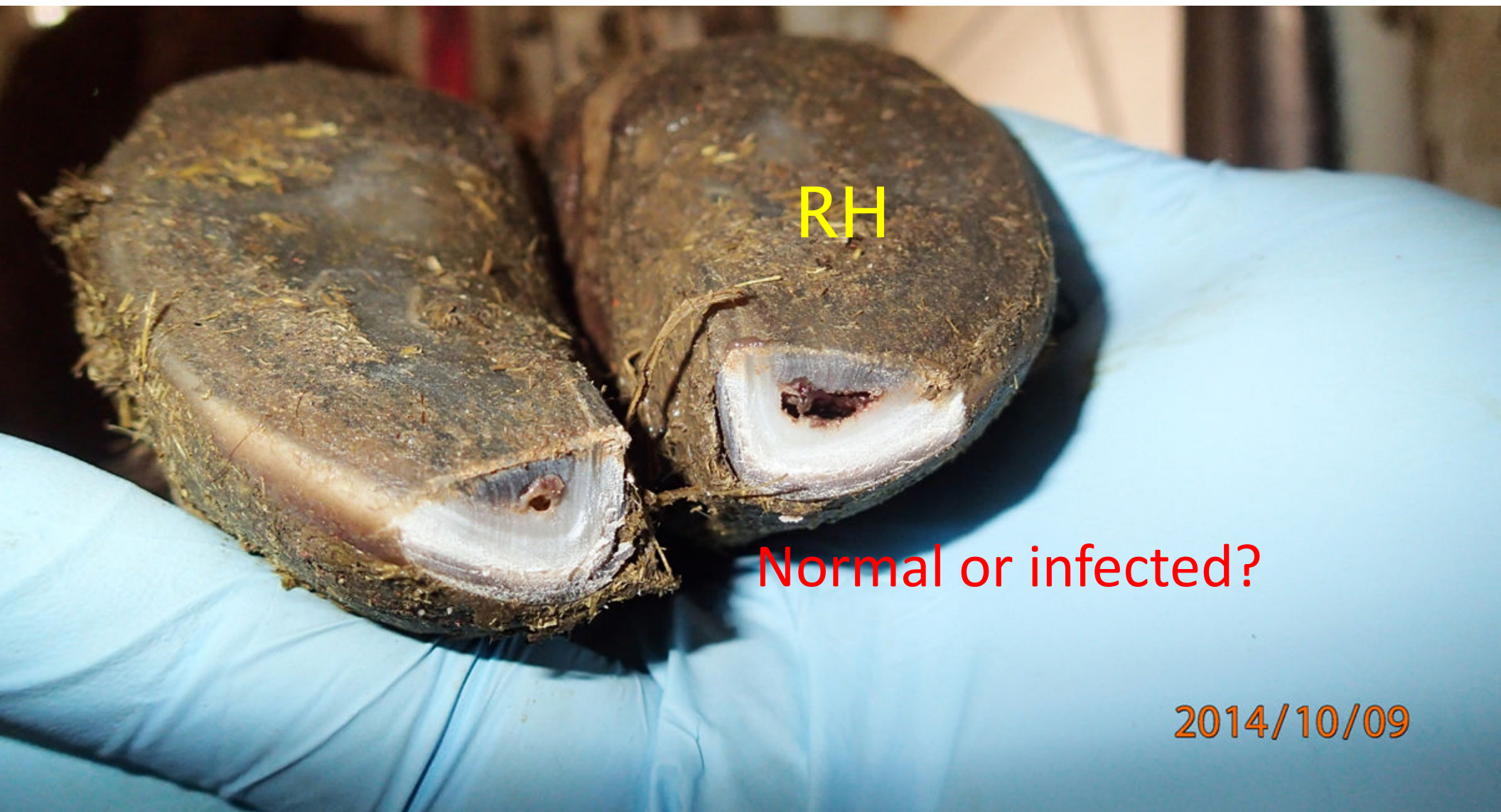


LH

Normal or infected?

2014/10/02





RH

Normal or infected?

2014/10/09





Normal or infected?

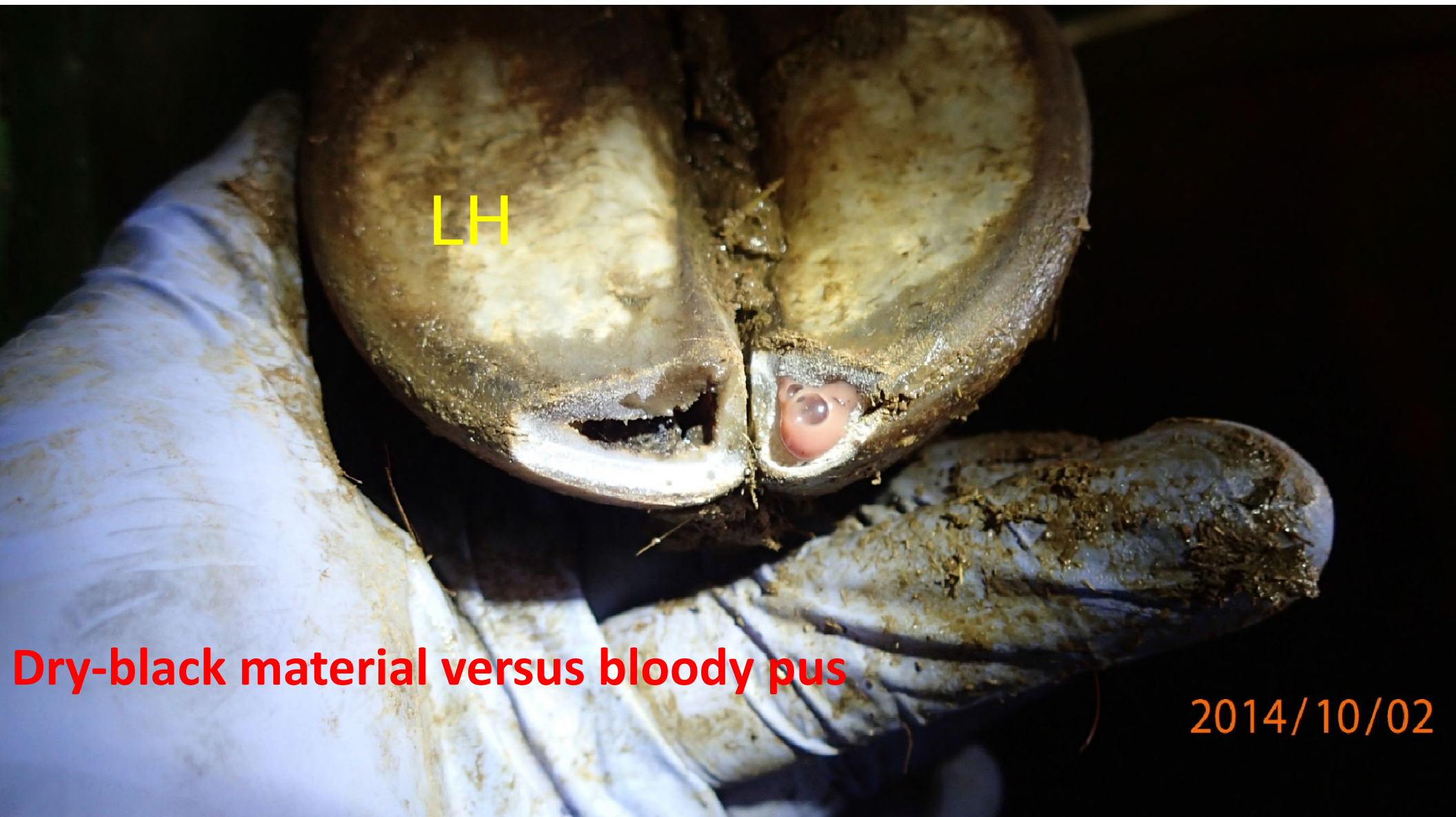
2014/11/06



2014/10/09







LH

**Dry-black material versus bloody pus**

2014/10/02

# Postmortem findings

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Does this fit the diagnosis of Toe Necrosis?











Courtesy of Dr. Andy Allen







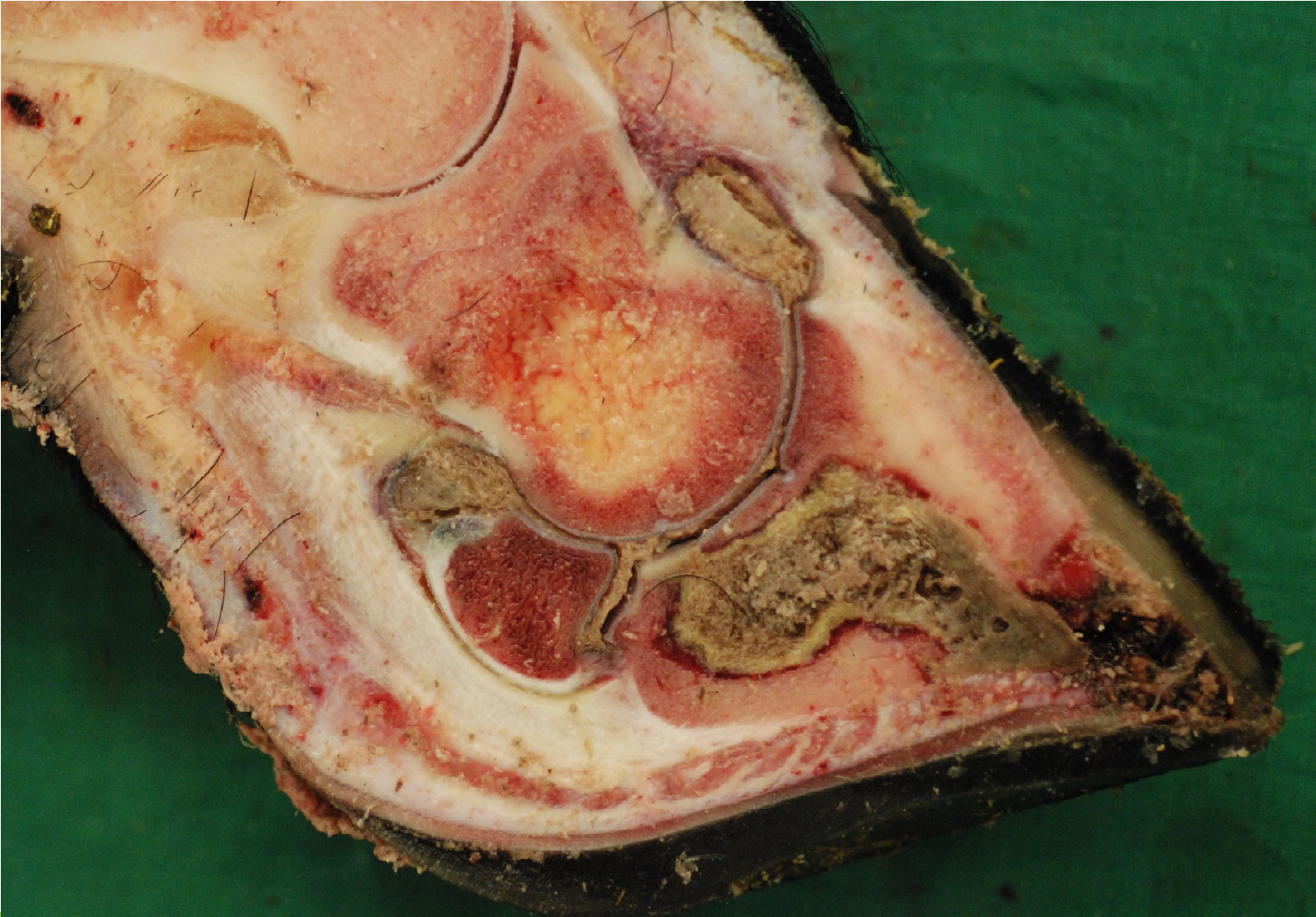






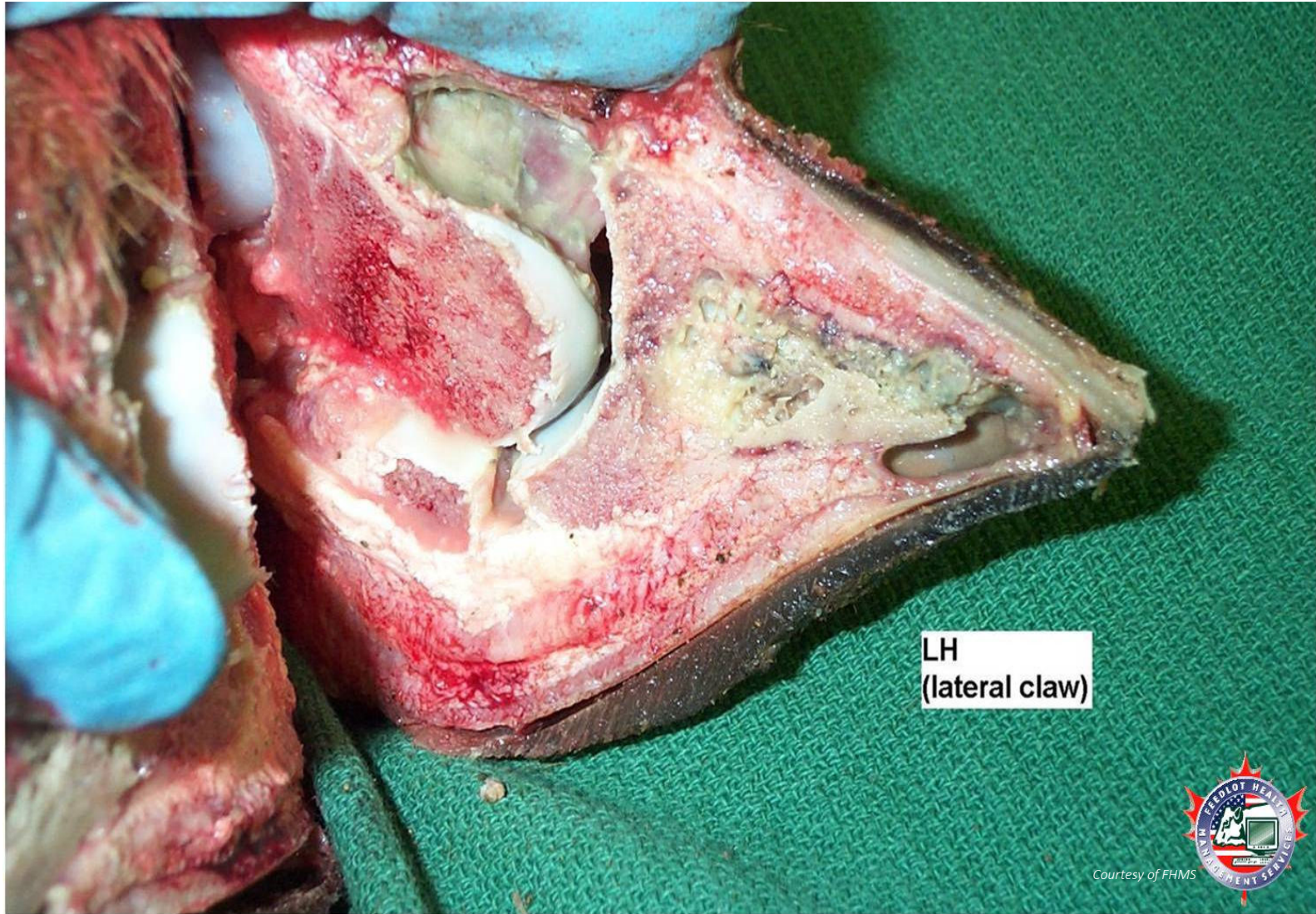
*Courtesy of A. Allen*





*Courtesy of A. Allen*



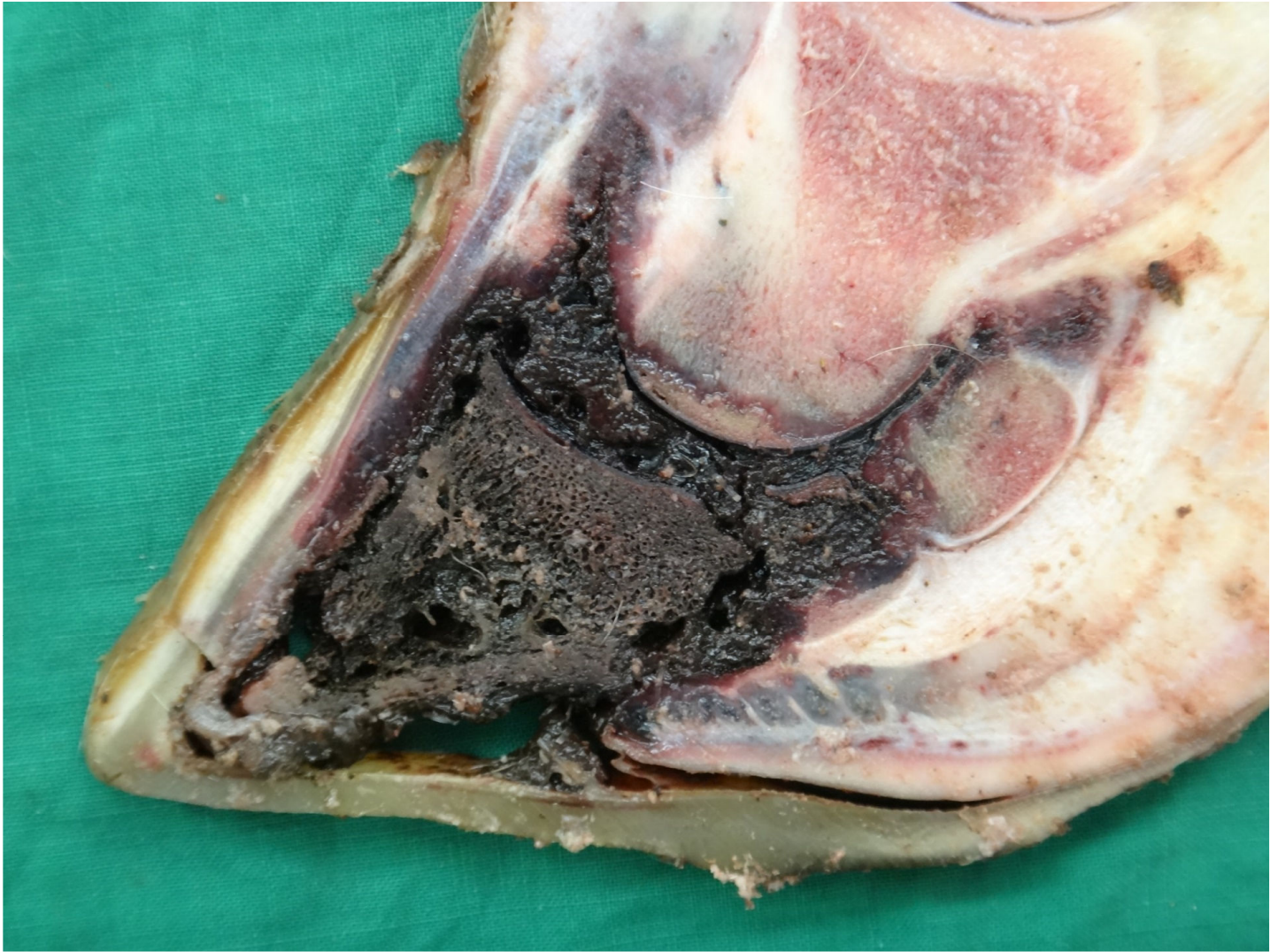


LH  
(lateral claw)



Courtesy of FHMS





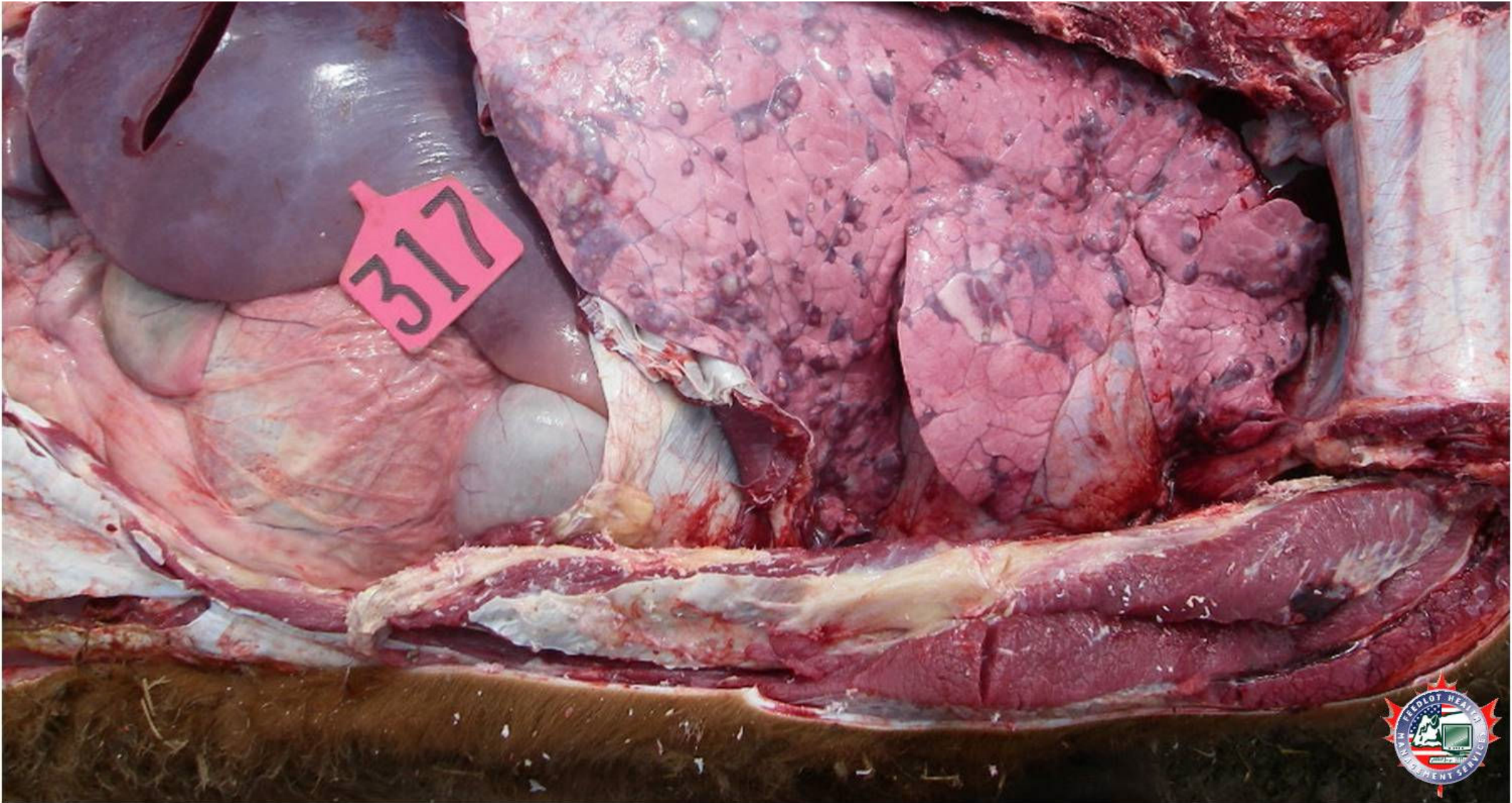













# Start of our research, we asked ourselves

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- Does the infection start from inside the hoof and move out or vice versa?
  - Why does it appear to be related to high-strung cattle?
  - What role does the feedlot play – flooring, handling, etc?
  - Is it more common in groups of cattle (lots) or certain feedlots?
  - Is it a disease of calves/yearlings, heifers/steers?
  - What bacteria/viruses may be related to the disease?
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# Started with describing the disease

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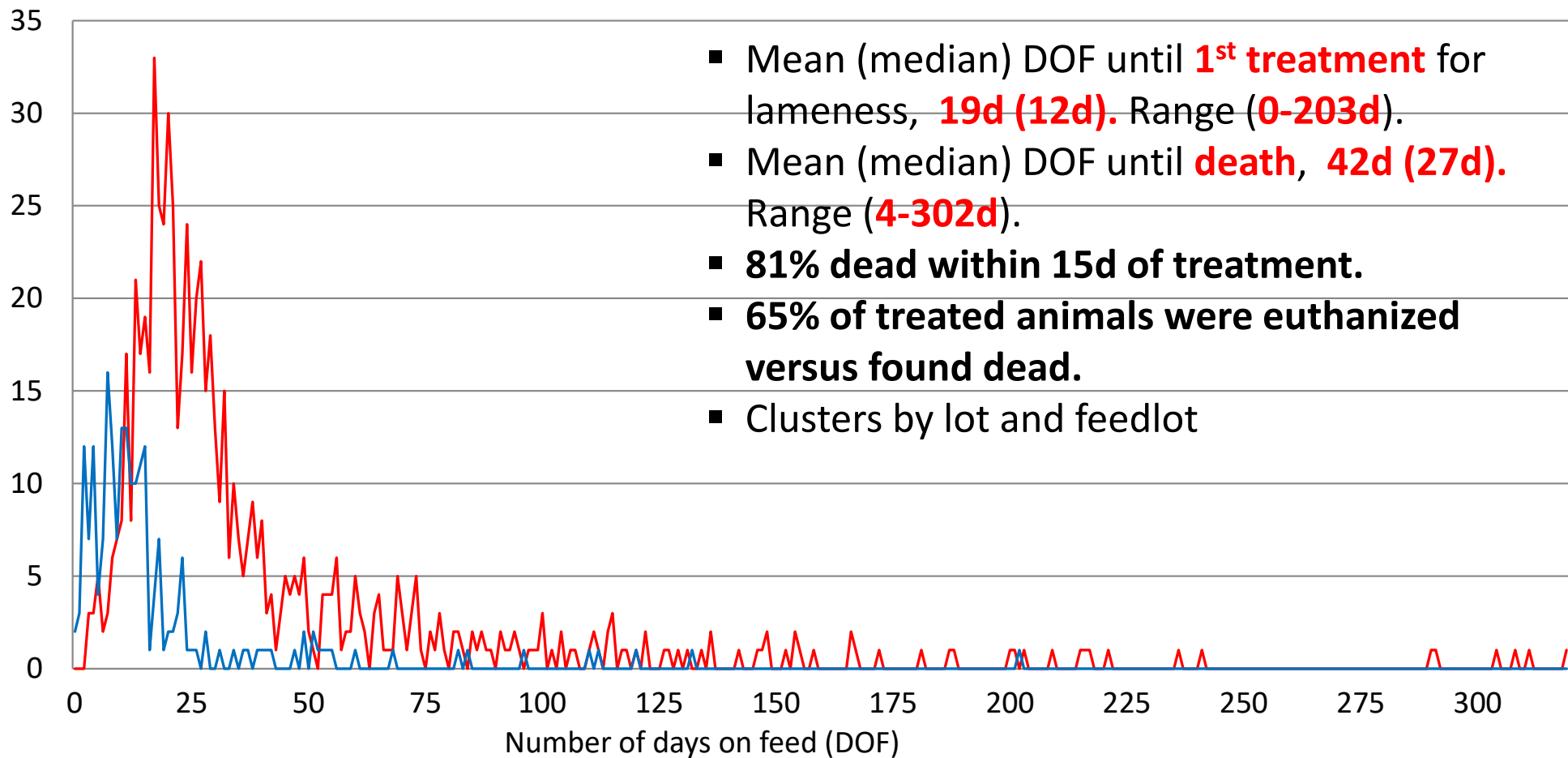
## Materials and Methods

- 1,904 cattle lots (>100 head) from 48 feed yards.
- **702 confirmed cases of TTNS**
- Days on feed until first treatment and death, age class, death date, source of animals, weights, gender, and outcome.

## Results:

- Only **72/1904 lots had  $\geq 1$  case** (lot prevalence **0.50-1.22%**)
- **45% calves, 55% yearlings**
- **78% auction-derived**, 12% ranch direct, 10% background
- **September-November**

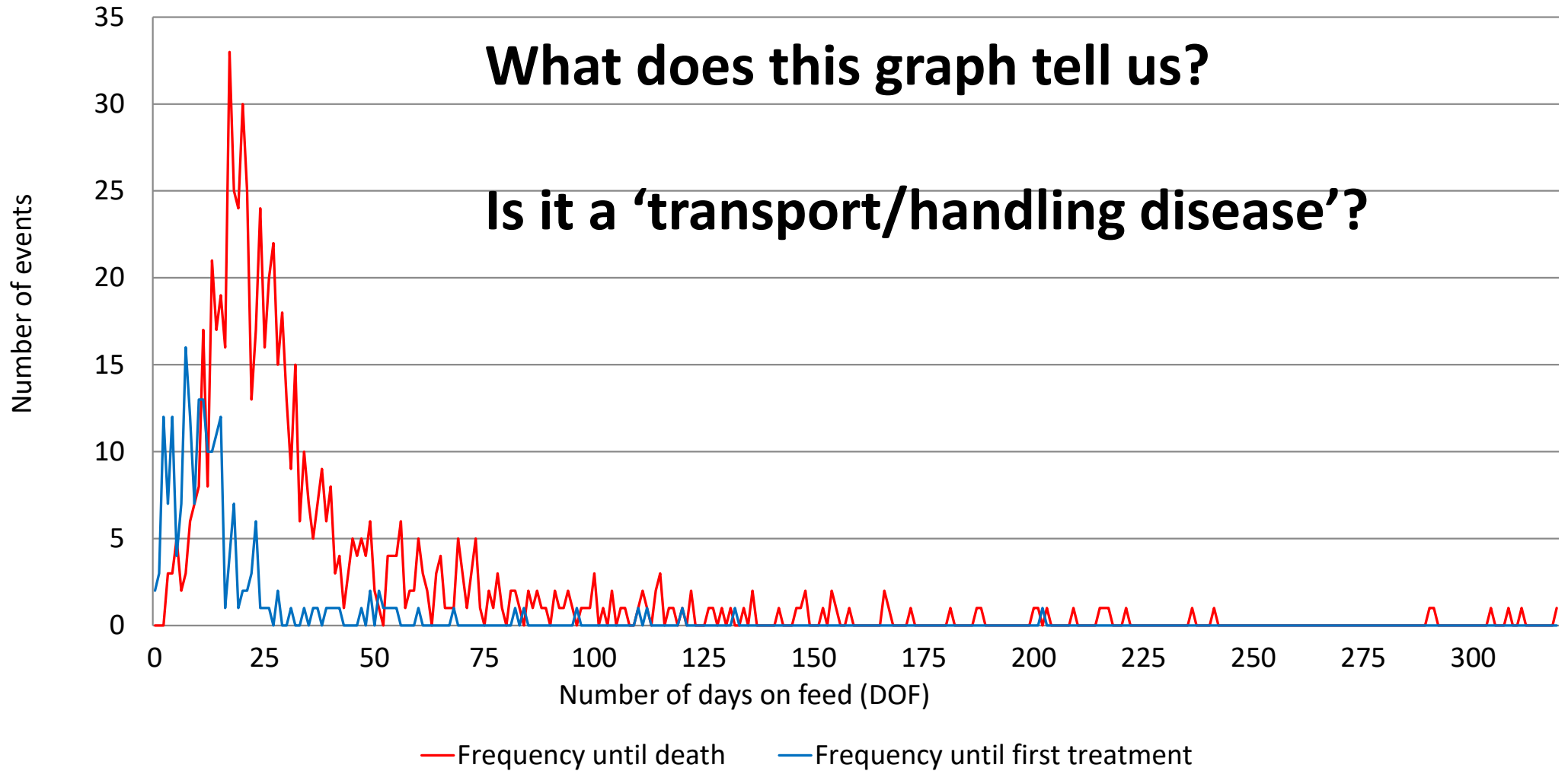
Number of events



- Mean (median) DOF until **1<sup>st</sup> treatment** for lameness, **19d (12d)**. Range (**0-203d**).
- Mean (median) DOF until **death**, **42d (27d)**. Range (**4-302d**).
- **81% dead within 15d of treatment.**
- **65% of treated animals were euthanized versus found dead.**
- Clusters by lot and feedlot

**What does this graph tell us?**

**Is it a 'transport/handling disease'?**

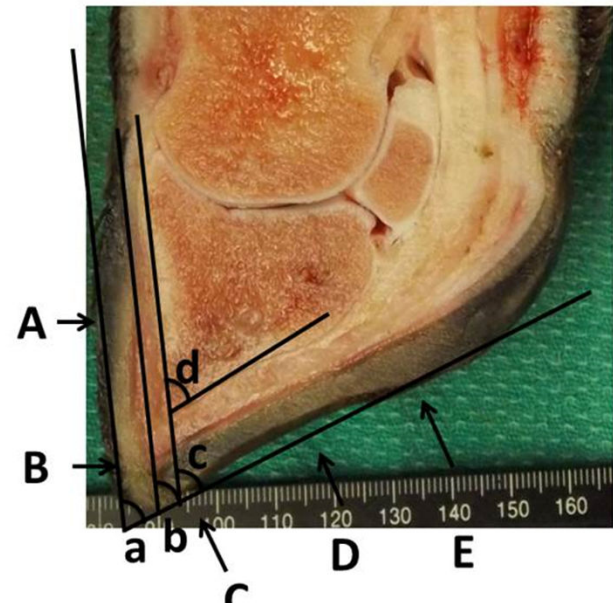




# Sorting out the risk factors for TTNS

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- 94 healthy and 93 diseased feet
- Practitioners from three feedlot practices submitted fresh feet and formalin fixed skin and cardiac tissue
  - Feet were sectioned
  - Culture swab
  - Pathology: vasculitis
  - BVDV, *Histophilus somni*



# Results

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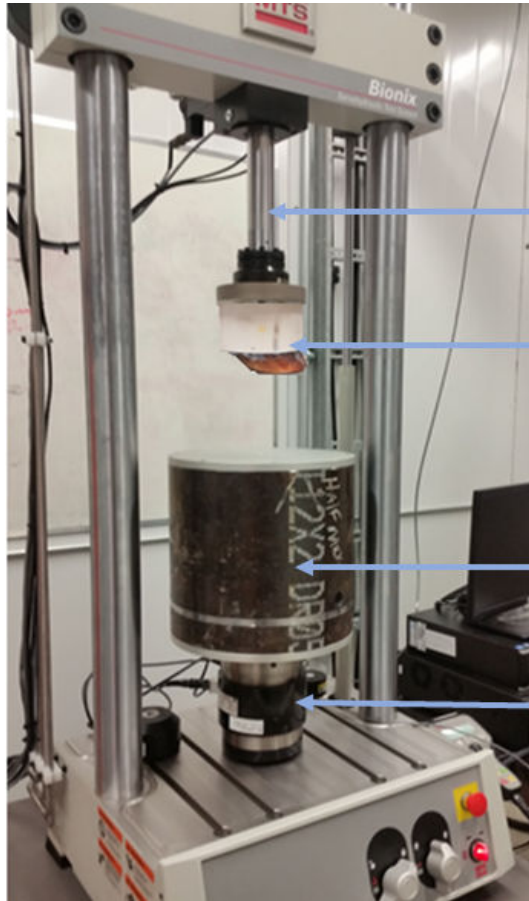
- **Thinner sole thickness at tip of toe**, no P3 rotation
- Cases always progressed from **white line into the P3 bone**
- Cases with heavy overgrowth of *E. coli* and *Trueperella pyogenes*
- Cases 4.4 times more likely to test positive for **BVDV** (immunohistochemistry)
- Cases had **lower Mg** concentrations in both solar and hoof wall horn tissue, could also be an indicator of deficiencies in Ca, S and P?

Next – we needed to explain this?





# 'High tech' approach (HR-pQCT and CLS)

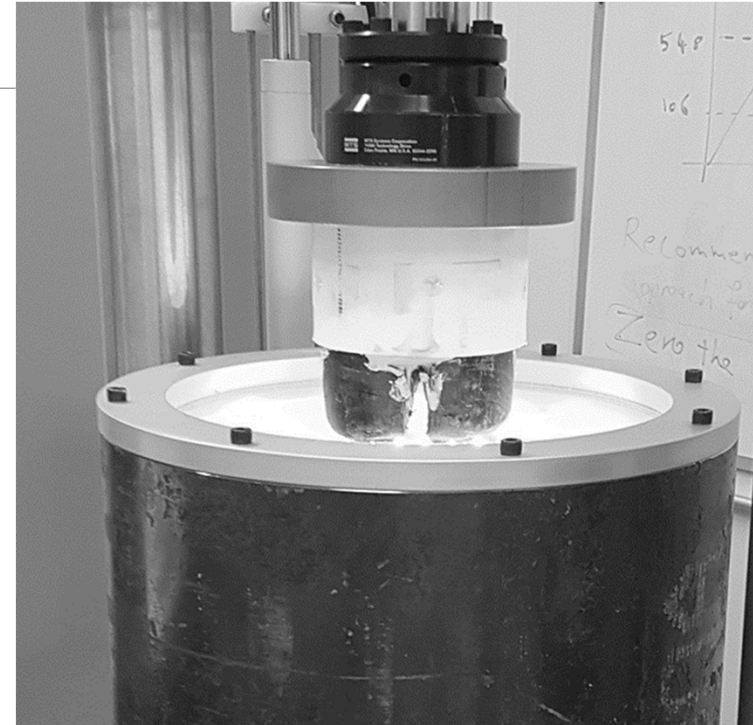


Actuator

Specimen

Camera Housing

Load Cell



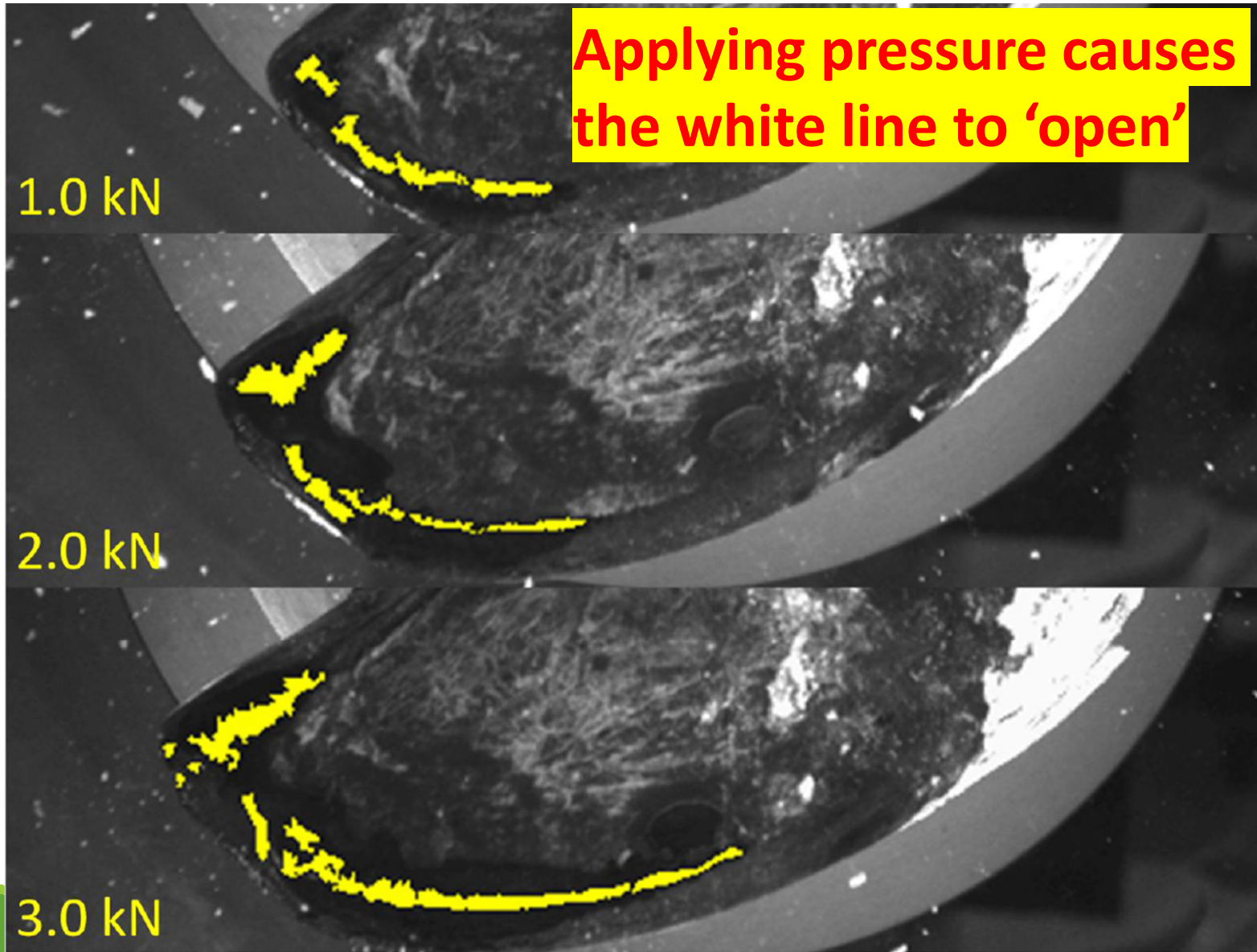
High-resolution peripheral quantitative computed tomography (HR-pQCT)

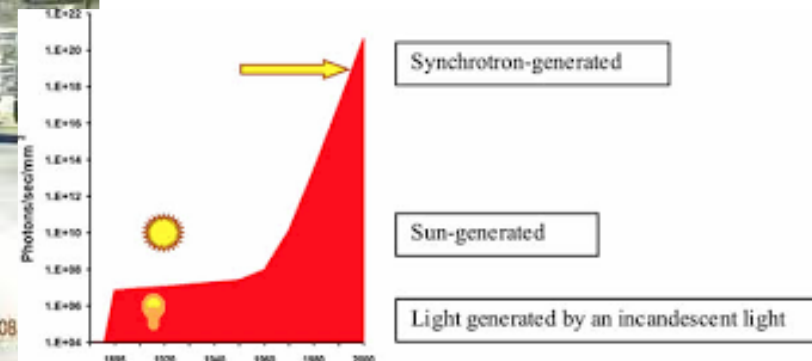
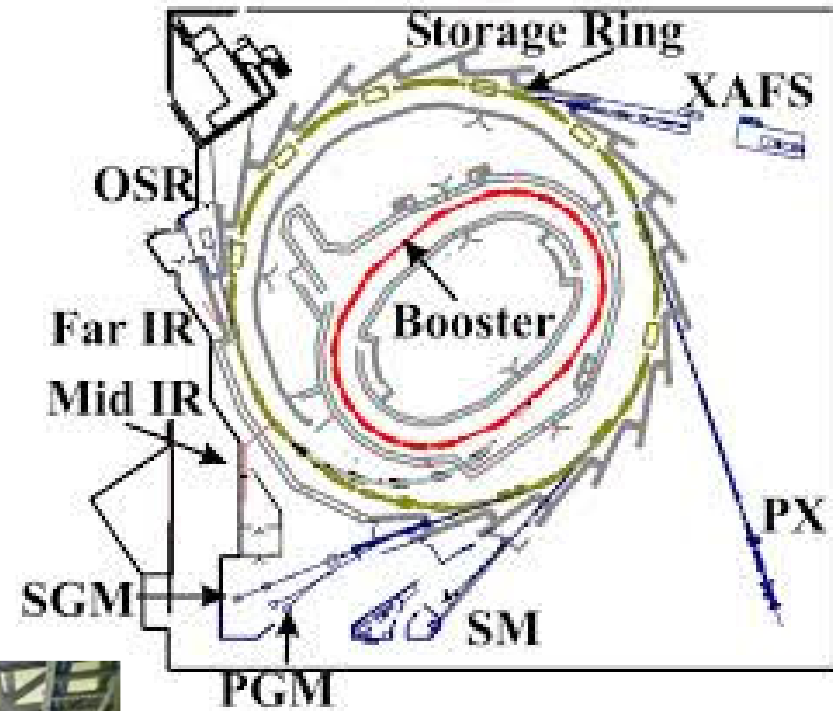
Applying pressure causes the white line to 'open'

1.0 kN

2.0 kN

3.0 kN











Results – visualized the ‘channels’ the bacteria can invade.



# Now what do we know? Or suspect?

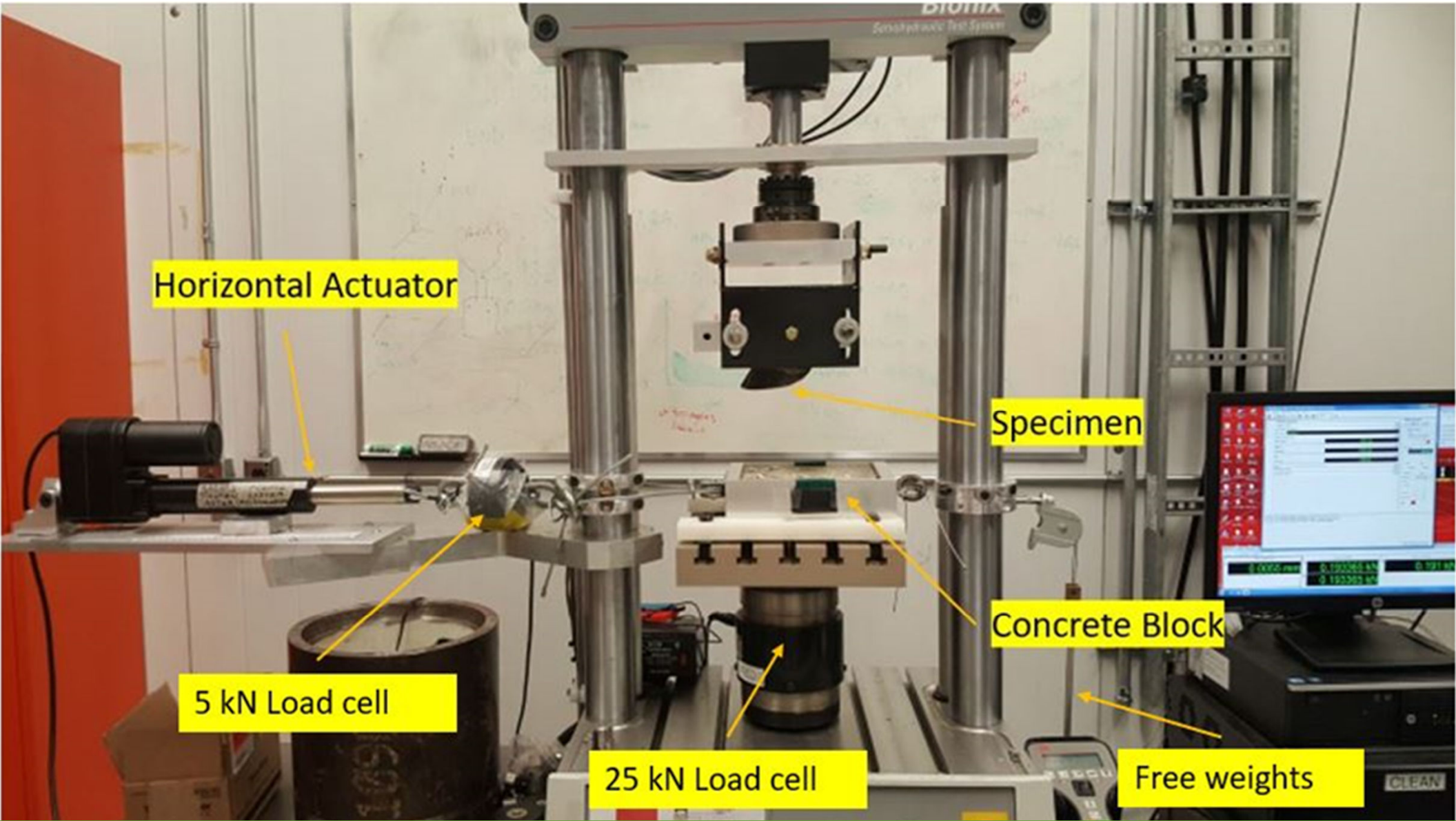
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- Animal develops white line separation
- Bacteria from pen colonize the white line, release enzymes that break-down the white line
- Weight of animal, causes the white line to separate, more bacteria/dirt gets in the lesion, leading to more separation
- The lesion finally begins to 'gape' open when weight-bearing, allowing even more material to get trapped within the foot
- Infection penetrates P3 and 'game over'

But, missing one important piece to the puzzle?  
How/why does the white line separate?

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- “Abrasion theory”
  - Excitable cattle pushing in chutes
  - From heavily muscled back limbs
  - Claws rasp on floor – explains why apex of hoof sole is thinner
  - Clusters by lot – excitable, pasture nutrition, wet environment
  
- **Onto the ‘Drag Test’ to see if we can prove our theory**



Horizontal Actuator

Specimen

Concrete Block

5 kN Load cell

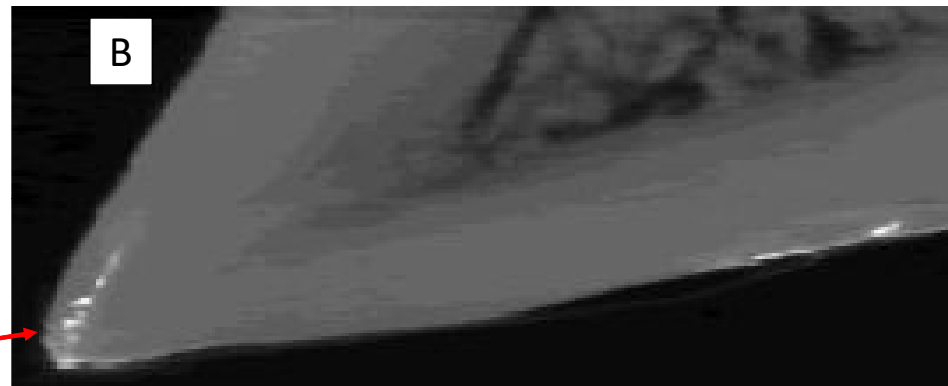
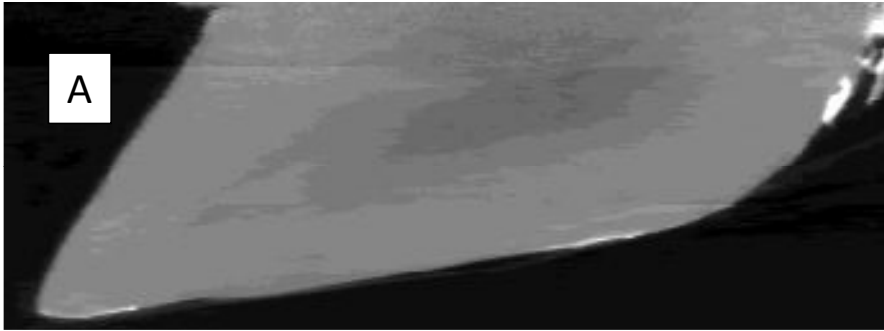
25 kN Load cell

Free weights





*HR-pQCT scan showing no contrast agent before dragging (A) and contrast agent sucked into the claw after dragging occurred (B)*



Pathway created  
for bacteria to enter  
after dragging

# Next – let's go look at flooring!

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- Library of all flooring from trailers to auctions and feedlots





































What is this – what is it used for???



Next – assessed the effects of gravel on claw wear with different floorings (rubber, concrete, sand).






# Results

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- 155% greater wear with concrete surface with presence of gravel
- No difference in wear with rubber surface with or without presence of gravel

# Treatment

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- **Aggressive trimming – diagnosis and treatment in one**
  - Antimicrobials and NSAIDS
  - Claw amputation
  - Euthanasia
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# Case study – treatment and recovery

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- 95 Angus X cattle shipped ~90 km to auction and then ~25 km to feedlot
- Within 48 h high prevalence of lameness
- 21 head returned to auction with degrees of lameness
- All diagnosed with TTNS of varying degrees
- Monitored for 7 months
- 16 of 21 recovered and were sent to slaughter
- EXTREMELY EXCITABLE

1290 – TTN #11 Pink  
Wk 0 – Oct 31



HR



HL



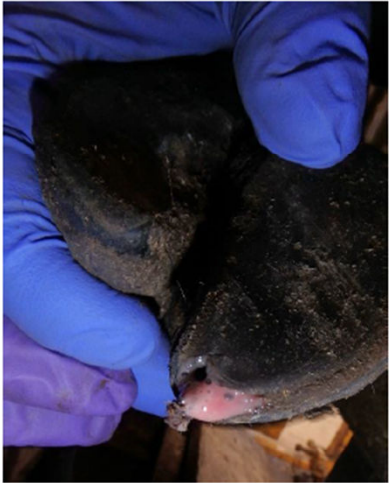
### Left hind

- Primary aggressive bone disease consistent with osteomyelitis of the distal 3<sup>rd</sup> phalanx of lateral (4<sup>th</sup>) digit of both the right and left hind feet.
- Medial digits of both hind feet; Normal.

### Right hind



1290 – TTN #11 Pink  
Wk 0 – Oct 31



HR



HL

Wk 1 – Nov 7



HR



HL

Wk 2 – Nov 15



HR



HL

Wk 3 – Nov 21



HR

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HL



1290 – TTN #11 Pink  
Wk 4 – Nov 28



HR

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HL

Wk 5 – Dec 6



HR

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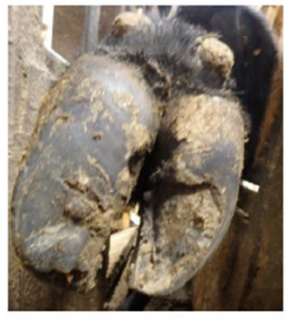
HL

Wk 7 – Dec 20



HR

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HL

Wk 9 – Jan 3



HR

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HL



1290 – TTN #11 Pink  
Wk 11 – Jan 17 2019



HR

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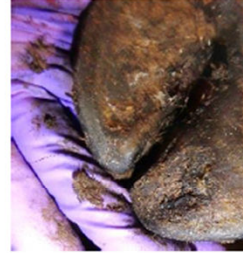


HL

Wk 13 – Jan 31



HR



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HL



Wk 15 – Feb 21



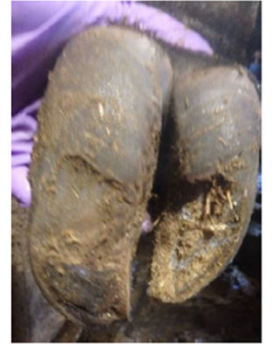
HR

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HL

Wk 18 – Mar 14



HR

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HL




# Take home points

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- TTNS may be starting at the auctions
- Related to excitable cattle – perhaps worse going forward?
- Use low-stress handling
- Avoid gravel - use sand?
- Can be treated with aggressive trimming and antibiotics
- Last resort is claw amputation but need to check other claws because often more than 1 claw affected.

# Acknowledgments

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