Genomic-based identification of host and environmental *Listeria monocytogenes* strains associated with an abortion outbreak in beef heifers

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Introduction

Listeria monocytogenes (LM) is a bacterial pathogen that causes late-term abortions in cattle, and definitive diagnosis and point-source determination may be challenging. In 2014, a diagnostic work-up of listerial abortions was initiated by a novel sample collection protocol. To determine the source(s) of the outbreak, LM strains isolated from cases and their environment were subjected to whole-genome sequencing and compared.

Materials and Methods

Twenty crossbred heifers that aborted from late January into February were identified. Presumptive diagnosis of listerial abortion was based on 2 positive, late-term aborted fetuses. To confirm that LM was the cause of the outbreak, proximal vaginal/distal cervical swabs (VCS) or retained fetal membranes (RFM) from heifers were submitted for LM culture and PCR. Silage and water sources were also submitted for culture and PCR. Resulting strains were subjected to whole-genome sequencing and compared.

Results

Heifer submissions identified 16/20 LM positive samples. Genomic sequencing of fetal, heifer, and environmental isolates demonstrated that 10/18 animal samples were closely related to each other, and to strains isolated from silage and drinking water. The remaining case strains were similar to each other, but genetically distinct from all other case and environmental strains.

Significance

When fetal materials are unavailable, CVS and RFM samples may be an effective means to diagnose abortions caused by LM. In this investigation, 2 LM case strains were responsible for the listeriosis outbreak, 1 causing the majority of the outbreak and linked to contaminated silage and/or water. The source of the other strain remains unknown.

Assessment and comparison of the barriers to interspecies transmission of bovine spongiform encephalopathy and chronic wasting disease prions

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Introduction

Prion diseases are transmissible, neurodegenerative diseases caused by templated conversion of the normal cellular prion protein to a misfolded, neurotoxic form. After conversion, the misfolded protein aggregates into characteristic protease-resistant amyloid plaques, which are hallmarks of prion disease. Species barriers among prion diseases have been observed epidemiologically and experimentally, but the mechanism(s) mediating this phenomenon remains unknown, making it impossible to predict the risk of transmission to new species, including humans, and to formulate