Kansas Agricultural Experiment Station Research Reports

Volume 0
Issue 1 *Cattleman's Day (1993-2014)*

Article 1208

1979

Rumen bacterial endotoxins and their possible role in the sudden-death syndrome

Tiruvoor G. Nagaraja

E. Bartley

L. Fina

H. Anthony

See next page for additional authors

Rumen bacterial endotoxins and their possible role in the sudden-death syndrome

Abstract
We have found that rumen bacteria contain endotoxins that are released into rumen fluid. Also rumen fluid from grain-fed cattle contains considerably more free endotoxin than rumen fluid from hay-fed cattle. Injecting cattle with rumen bacterial endotoxins may be involved with such diseases associated with high-grain feeding as the sudden death syndrome.

Keywords
Cattlemen's Day, 1979; Report of progress (Kansas State University. Agricultural Experiment Station); 350; Beef; Endotoxins; Sudden death syndrome

Authors
Tiruvoor G. Nagaraja, E. Bartley, L. Fina, H. Anthony, and H. Leipold

K S U

Rumen Bacterial Endotoxins and Their Possible Role in the Sudden-death Syndrome

T. G. Nagaraja, Erle Bartley, Lou Fina, Harry Anthony, and Horst Leipold

Summary

We have found that rumen bacteria contain endotoxins that are released into rumen fluid. Also rumen fluid from grain-fed cattle contains considerably more free endotoxin than rumen fluid from hay-fed cattle. Injecting cattle with rumen bacterial endotoxin produced endotoxic or anaphylactic shock. We believe that rumen bacterial endotoxins may be involved with such diseases associated with high-grain feeding as the sudden death syndrome.

Introduction

Bovine sudden death syndrome (SDS) is a fatal disease of unknown origin, chiefly affecting healthy feedlot cattle that have been on high energy grain rations for more than 100 days. Affected animals stop eating, step back, and die with no other clinical sign. We have been trying for seven years to determine what causes SDS. One of the first areas we investigated was rumen metabolism, because we thought some end-products of rumen fermentation in cattle fed high energy rations might be toxic. We found none that could be linked to SDS. Then we investigated the role of rumen bacteria, thinking rumen gram negative bacteria might release endotoxin, which when absorbed might cause shock and sudden death.

Procedure and Results

We obtained samples of rumen contents from hay-fed and grain-fed cattle, extracted the bacterial fraction and tested it by standard procedures for endotoxin. The purified extract was endotoxic based on the following characteristics: 1. Proved lethal to mice and chick embryos. Toxicity in mice was increased by actinomycin D (an established characteristic of endotoxins). 2. Made mice more susceptable to streptococcal infection. 3. Caused a characteristic fever and white blood cell response in rabbits. 4. Caused local tissue degeneration in rabbits after a sensitizing dose. 5. Caused gelation of limulus lysate (a test for estimating endotoxin).

Those tests confirmed that rumen bacteria contained endotoxin. Next we had to determine if the endotoxin was released from the bacteria. Unless it is released, it could not be absorbed or cause a problem.

We looked for endotoxin in rumen fluid from hay- and from grain-fed cattle. Using the tests just described, we consistently demonstrated that

endotoxin had been released from rumen bacteria. Additionally, we found more free endotoxin in grain-fed than in hay-fed cattle. Certain factors, still unknown, may favor release of endotoxin from rumen bacteria when cattle are fed large quantities of grain.

To determine the effect of rumen endotoxin on cattle, we intravenously injected 3- to 7-month-old calves with rumen endotoxin in doses from .5 to 2 mg per lb. body weight. Calves that did not die of endotoxin toxicity were reinjected on the 15th day to see if the second injection caused anaphylactic shock, on the assumption that a nonfatal first injection might sensitize all animals so the next dose caused anaphylaxis.

After the first injection, signs were typical of endotoxin poisoning. Within a few minutes the calves showed rapid and labored breathing and they usually collapsed on their sides in about 30 minutes. The various first doses we tested did not kill any calf.

When the animals were reinjected the 15th day, response was more dramatic. The signs were similar, but happened strikingly sooner. Before the needle was removed from the jugular vein, the animals were down in shock, gasping for breath. Two of 8 calves died, but the remaining 6 recovered in a few hours. Blood samples taken after the first and second injections were characteristic of endotoxin poisoning. The 6 calves that survived the second endotoxin dose were killed after 24 hours and posted. The hemorrhagic lesions we observed were typical of endotoxin poisoning. The most significant damage was in the lungs. Bronchial constriction was the most typical lesion. Because the lungs are the target organ for anaphylaxis in cattle, our findings suggest that we probably sensitized the calves with the first injection of endotoxin and then produced anaphylaxis after the second injection.

Discussion

Free endotoxin in rumen fluid and its higher concentration in grain-fed cattle suggests that rumen bacterial endotoxin is involved in diseases, like lactic acidosis or the sudden-death syndrome, that are associated with high-grain feeding. The fate of free endotoxin from the rumen is not known. It may be absorbed and detoxified in the liver or passed on to the abomasum and small intestine where it may be inactivated by acid or enzymes or absorbed into the portal blood. There is no evidence that endotoxin may be absorbed through the rumen lining. However, absorption from the rumen cannot be ruled out in conditions like ruminitis and lactic acidosis where the rumen lining is inflamed or damaged. The sudden release of large quantities of endotoxin in the rumen, if rapidly absorbed by damaged rumen lining, could produce endotoxic or anaphylactic shock--and sudden death of cattle.