BOVINE ACUTE-PHASE RESPONSE AFTER DIFFERENT DOSES OF CORTICOTROPIN-RELEASING HORMONE CHALLENGE

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Background

The acute-phase response is an important component of the innate immune system. However, it can be detrimental to cattle performance.

Management strategies that lessen the magnitude of the acute-phase response triggered by stressful management practices have been shown to benefit cattle productivity and overall efficiency of beef operations.

However, the physiological mechanisms associated with the bovine acute-phase response are not completely understood.
Background

A preliminary study showed that a steers receiving a non-pathogenic stress stimulus, more specifically an intravenous CRH challenge to increase circulating concentrations of cortisol, experienced an acute-phase response characterized by increased rectal temperatures and plasma concentrations of interleukin (IL)-6, haptoglobin and ceruloplasmin.

However, due to reduced funding available, an adequate control/placebo treatment was not included into the study design, which prevented proper interpretation and field application of these results.
Objective

Compare the acute-phase response of beef steers receiving

- intravenous saline infusion (control),
- or an intravenous CRH infusion containing 0.1 µg of bovine CRH/kg of body weight
- or 0.5 µg of bovine CRH/kg of body weight.

Evaluate subsequent rectal temperatures, and circulating concentrations of cortisol, non-esterified fatty acids, proinflammatory cytokines (IFN-γ, IL-6, and TNF-α) and acute-phase proteins (ceruloplasmin and haptoglobin).
Materials and Methods

14 Angus steers were used that were halter broke and acclimated to humans were housed in individuals pens. On day 0, steers were randomly assigned to receive 1 of 3 infusion treatments (i.v.): 1) 0.1 μg of bovine CRH/kg of BW (CRH1), 2) 0.5 μg of bovine CRH/kg of BW (CRH5), and 3) 10 mL of saline.

Blood samples were collected and rectal temperatures were recorded both before and after treatments at repeated intervals.

Blood samples were analyzed for plasma concentrations of cortisol, NEFA, ceruloplasmin, haptoglobin, and proinflammatory cytokines (IFN-γ, IL-6, and TNF-α).
Serum NEFA

Hours relative to challenge

Serum NEFA, mEq/L

CRH1
CRH5
Saline
Plasma haptoglobin

![Graph showing plasma haptoglobin levels over time for different conditions. The x-axis represents hours relative to challenge, and the y-axis represents plasma haptoglobin levels in 450 nm x 100.]
Results

Plasma cortisol peaked at 0.5 h for CRH1 steers but returned to baseline levels at 1 h relative to infusion. Within CRH5 steers, plasma cortisol peaked at 0.5 h and returned to baseline levels 3 h relative to infusion. Plasma cortisol concentrations did not change after infusion for saline steers.

Serum NEFA in CRH1 steers peaked at 5 h relative to challenge, but did not change for CRH5 and saline steers.

Serum TNF-α concentrations were greater for CRH1 steers compared to saline at 3, 7, and 8 h relative to treatment infusion, and greater for both CRH1 and CRH5 steers compared to saline at 4 and 5 h relative to treatment infusion.
Results

Following CRH challenge, rectal temperatures were greater for CRH1 steers compared to saline steers at 30, 36, and 72 h relative treatment infusion.

Plasma haptoglobin concentrations in CRH1 steers increased significantly and were greater compared to CRH5 and saline steers from 48 to 96 h relative to challenge. Conversely, plasma haptoglobin concentrations were similar and did not change across time for CRH5 and saline steers.
Implications

Steers receiving an i.v. CRH challenge at 0.1 or 0.5 µg/kg of BW experienced, although in different patterns, increased concentrations of plasma cortisol and TNF-α compared to cohorts receiving saline.

However, tissue mobilization (characterized by serum NEFA), changes in body temperature, and acute-phase protein response were only detected in steers receiving 0.1 µg of CRH per kg of BW.

Therefore, the bovine acute-phase response stimulated by CRH infusion is depended on the dose of CRH applied, and likely to the subsequent response in circulating cortisol.