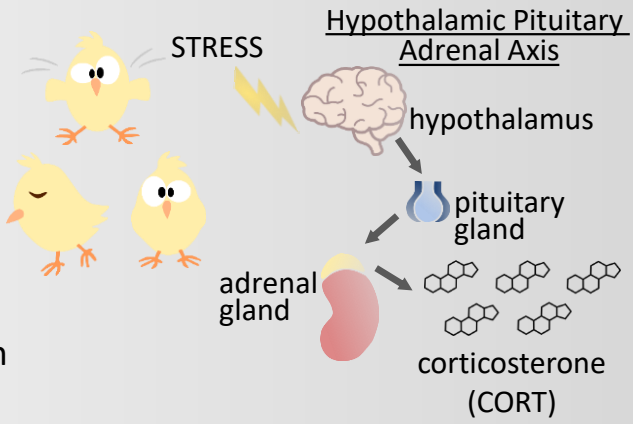
Physiological stress alters intestinal microbial communities in the small intestine and increases the relative abundance of *Clostridium perfringens* in chickens

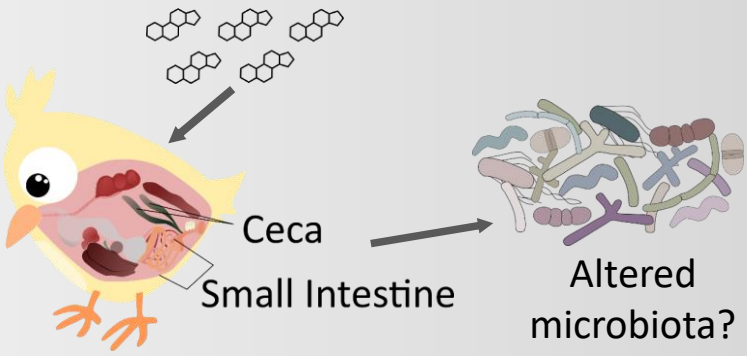
What's the Problem?

- Birds are exposed to various stressors throughout the production cycle
- Corticosterone is a glucocorticoid produced in the cortex of the adrenal glands
- Stressors, such as heat, social, transport, and infection can result in increased corticosterone production
- More research is linking stress to changes in the intestinal microbiota and onset of disease



Study Objective

To mediate a controlled stress response through the administration of corticosterone and examine changes to the intestine microbiota by 16S rRNA gene sequencing and taxon-specific quantitative PCR



Study Setup

- Used specific pathogen free layer chickens
- Administered corticosterone in drinking water at 0, 10, and 30 mg / L doses
- Began administration at 14 days post-hatch
- Sampled birds after 1, 5, or 12 days of continual corticosterone administration
- Collected small intestine (jejunum-ileal junction) and cecum digesta

Control
(standard drinking water)

Ethanol Carrier Control
(0.2% ethanol drinking water)

Low Dose Corticosterone
(10 mg CORT / L drinking water)

High Dose Corticosterone
(30 mg CORT / L drinking water)

Study Findings

1) Stress decreased diversity of bacteria

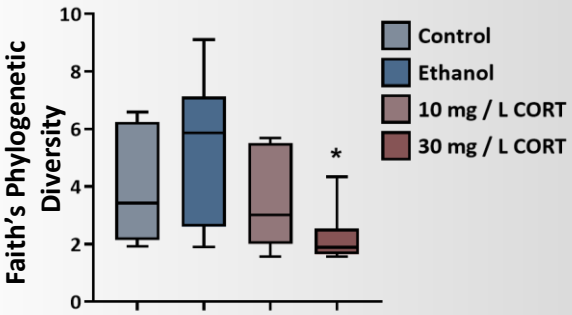


Figure 1. Alpha diversity of small intestine

Less types of bacteria were found with corticosterone treatment in the small intestine. This can reduce protection against pathogens and be a factor that increases susceptibility to disease.

2) Stress altered bacterial communities

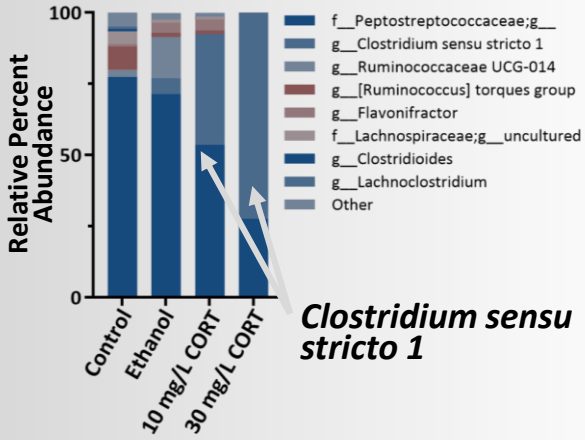


Figure 2. Distribution of bacteria in the small intestine.

The bacterial communities of the small intestine changed more in birds administered corticosterone. The small intestine was more affected than the cecum. Increased abundance of bacteria belonging to the genus *Clostridium sensu stricto 1* were detected in stressed birds.

3) Stress increases densities of *C. perfringens*

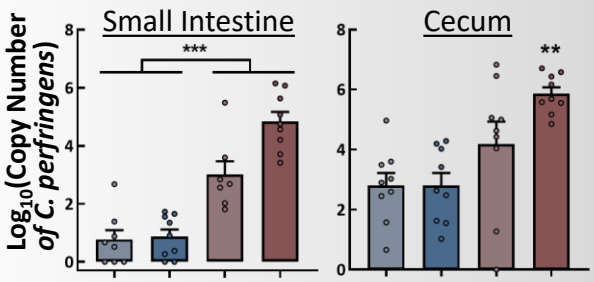


Figure 3. Quantitative PCR of *C. perfringens*.

Clostridium perfringens belongs to the genus *Clostridium sensu stricto 1*. Thus, quantitative PCR was completed and showed that birds administered corticosterone had higher densities of *C. perfringens* in the small intestine and cecum. These results support stress as a predisposing factor to necrotic enteritis

* P<0.05
** P<0.01
*** P<0.0001

What are the next steps?

Complete a challenge experiment with virulent *C. perfringens* and corticosterone

Layer chickens (completed)

Broiler chickens (in-progress)

Resulted in subclinical necrotic enteritis

RESEARCH Open Access

Host responses to *Clostridium perfringens* challenge in a chicken model of chronic stress

Gut Pathogens

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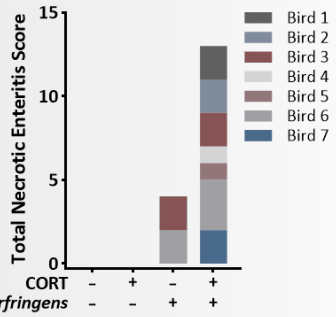
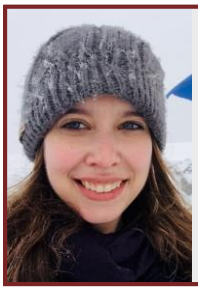
Preliminary Findings:

Birds were scored 0-6 for progression of disease. Broiler birds administered CORT showed more necrotic lesions and disease progression when inoculated with *C. perfringens*.





This research was conducted by Sarah Zaytsoff in the fulfillment of her PhD at the University of Alberta.

Canada

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