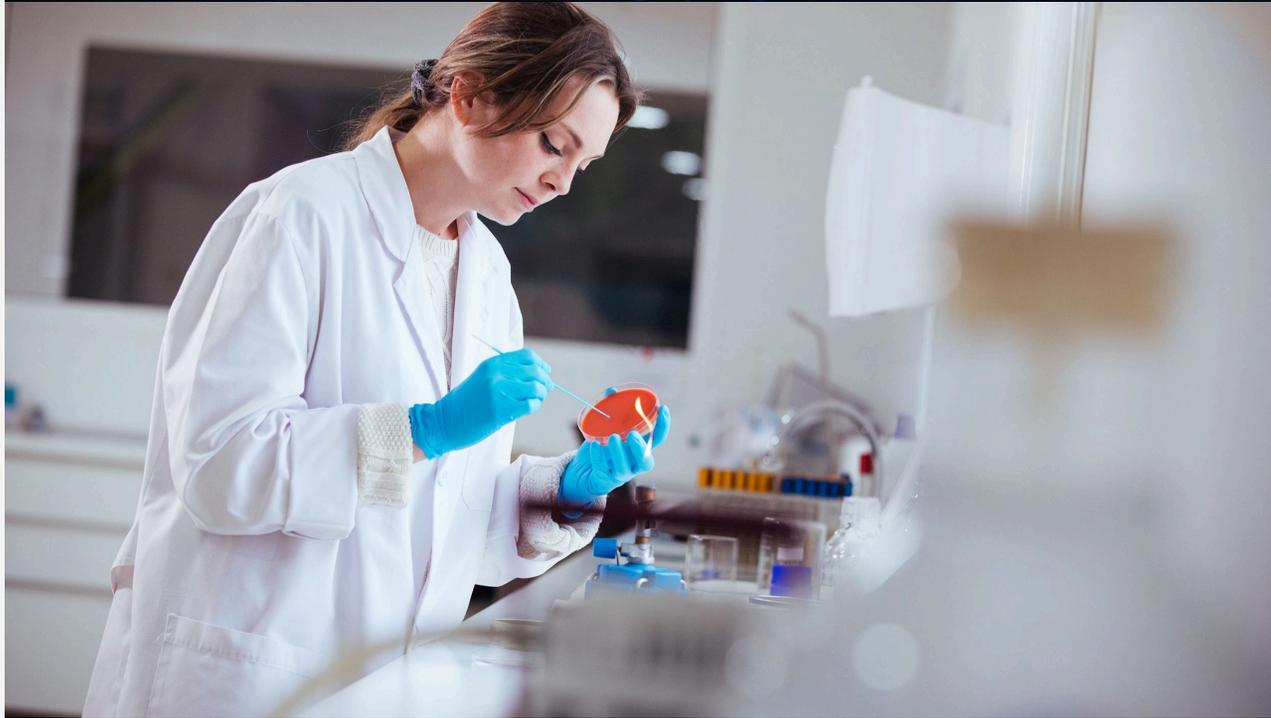


PATHKINEX UPDATE



**PathKinex™ Update: Understanding the Role
of *Clostridium perfringens* in Coinfections and
the Benefits of Novela® ECL**

Clostridium perfringens is a gram-positive, spore-forming anaerobe that causes necrotic enteritis in poultry, neonatal diarrhea in pigs, and enteritis with sudden death in young ruminants, all of which can result in major economic losses for farmers. Disease severity is often aggravated by coinfections, while subclinical cases impair growth and productivity.¹⁻⁵ The pathogenesis of *C. perfringens* involves four major toxins— α , β , ϵ , and ι —with alpha-toxin and NetB produced by certain type-A strains being key to poultry necrotic enteritis.

Another common poultry pathogen, avian pathogenic *Escherichia coli* (APEC), also contributes to costly losses in part by causing colibacillosis, a systemic infection leading to high morbidity and mortality. Together, *C. perfringens* and APEC reduce performance, heighten antimicrobial resistance concerns, and threaten food safety, driving interest in alternatives, such as probiotics, prebiotics, vaccines, and dietary interventions.⁶

United Animal Health conducted a broiler trial under commercial production conditions using litter pretreated with CocciVac[®] B52 to evaluate the effects of bacitracin methylene disalicylate (BMD) at 55 ppm, an antibiotic often used to reduce *C. perfringens*, and Novela[®] ECL (DFM) at 3.68×10^5 CFU/g of feed, on virulence genes linked to necrotic enteritis, coccidiosis, and colibacillosis. Birds were assigned to a fresh litter control (FL) or one of three used litter (UL) groups: control (UL), UL + BMD, and UL + DFM. Ileal swabs and litter samples were analyzed for *C. perfringens* NetB and APEC toxin genes.

The trial model anticipated coinfection with *C. perfringens* and *Eimeria* spp., particularly *E. acervulina* and *E. maxima*, which are major precipitating factors for necrotic enteritis in poultry. Such coinfections increase instances of intestinal lesions, suppress growth, and disrupt immune responses more severely than single infections.⁷

In poultry intestinal health, intact tight junctions and a diverse microbiota restrict *C. perfringens* proliferation (Figure 1, Illustration A). Then, during *Eimeria* infection, epithelial damage and excess mucus release nutrients that favor rapid bacterial growth (Illustration B). Subsequent toxin production compromises intestinal integrity, driving necrotic lesion formation, clinical disease, and mortality (Illustration C).

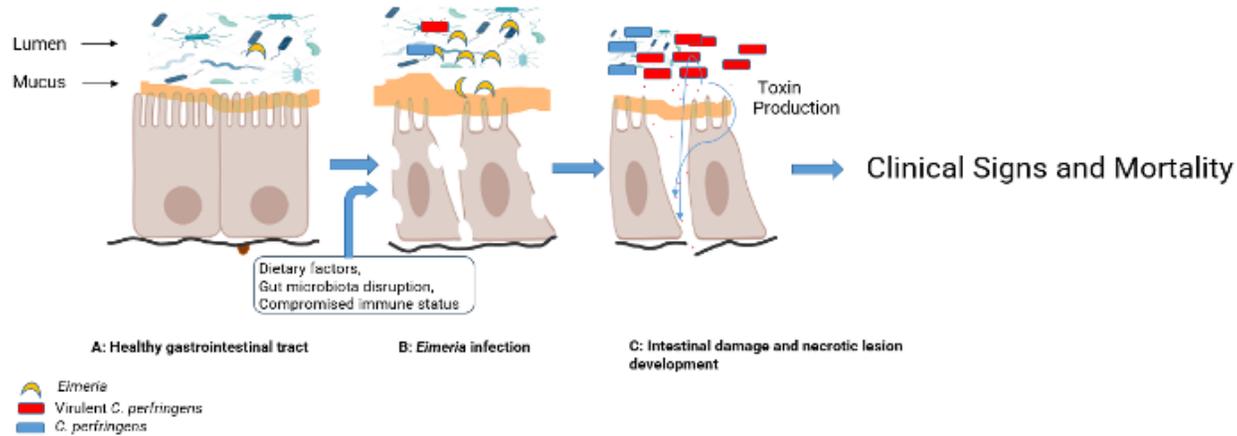


Figure 1: Progression of necrotic enteritis following *Eimeria* infection and *C. perfringens* proliferation. Created using Biorender and adapted from Timbermont et al, 2011.



Trial Results

Performance

Trial results showed that mortality-adjusted feed conversion ratio (FCR) was significantly improved in the UL + DFM group. Feed intake was reduced in UL and UL + DFM relative to FL ($P < 0.05$), while weight gain declined across all used litter groups ($P < 0.05$).

Table 1: Performance results: feed intake, mortality-adjusted FCR, and weight gain at 21 days of age.

Treatment	Diet	Feed intake/pen (kg)	Mortality-adj. FCR	Weight gain (kg)
Fresh litter (FL)	Positive control	26.4 ^a	1.334 ^a	0.917 ^a
Used litter (UL)	Negative control	24.9 ^b	1.338 ^a	0.868 ^b
UL + BMD	UL + BMD	25.3 ^{ab}	1.322 ^a	0.886 ^b
UL + DFM	UL + Novela® ECL	24.1 ^b	1.297 ^b	0.881 ^b

C. perfringens

The *NetB* toxin gene was detected in both ileum and litter across all treatments. Ileal gene levels did not differ, but litter from UL + BMD showed reduced *C. perfringens* genes (*cpa*, *NetB*) compared to FL ($P < 0.05$), while UL and UL + DFM were intermediate. While BMD was expected to reduce *C. perfringens* populations, these findings also suggest that DFM and the established microbial population within used litter play a role in suppressing the overgrowth of necrotic-associated *C. perfringens*.

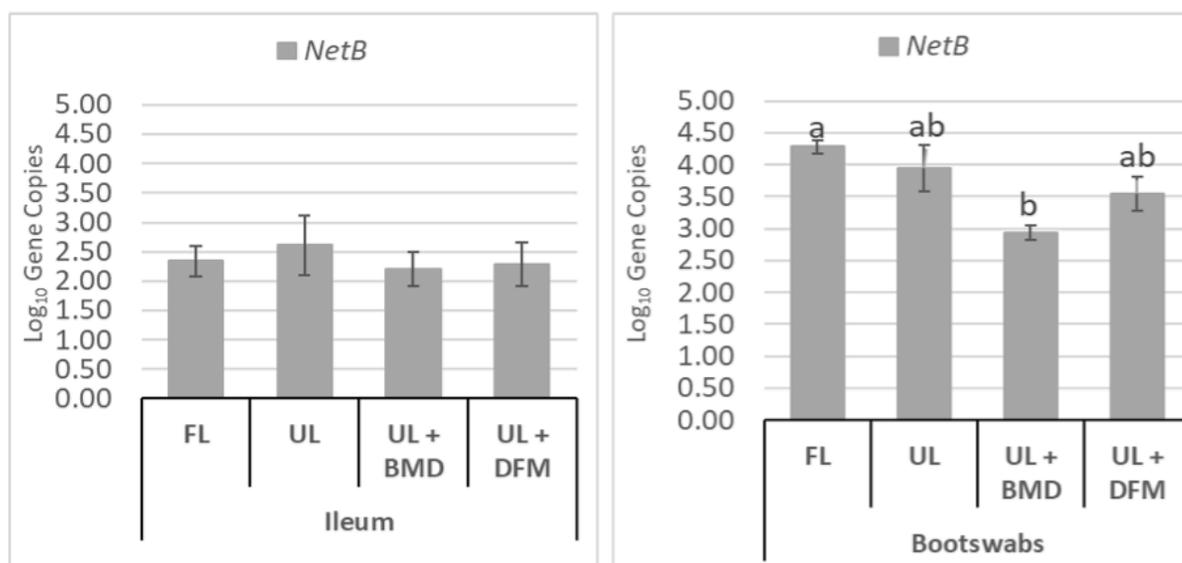


Figure 2: Clostridial *NetB* gene quantities detected in ileum and bootswabs.

Avian Pathogenic *E. coli* (APEC)

Coinfections of *C. perfringens* with APEC, often alongside viral or parasitic agents, are known to exacerbate disease severity. In this trial, the UL + BMD group showed significantly higher APEC gene levels ($P < 0.05$) in the ileum compared to UL, whereas UL + DFM did not differ from controls. Virulence genes (*hlyF*, *iss*, *ompT*, and *iroN*) were reduced in UL + DFM and UL litter samples relative to FL ($P < 0.05$), with UL + BMD showing intermediate values. These findings suggest that broad-spectrum antibiotics like BMD may disrupt beneficial microbiota, creating ecological niches that favor opportunistic pathogens like APEC.⁸

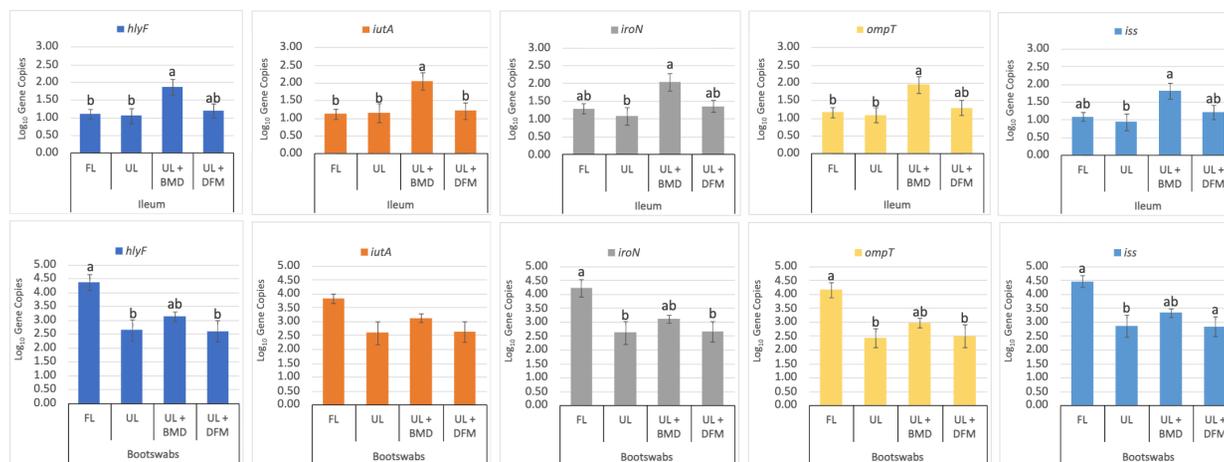


Figure 3: APEC genes quantities detected in ileum (top graphs) and bootswabs (bottom graphs). [Click to enlarge.](#)

Together, these results demonstrate Novela® ECL's potential for improving feed efficiency in broiler chickens while maintaining lower quantities of APEC in birds exposed to a cocci challenge.



Conclusion

Carefully selected DFMs, such as Novela® ECL, can reduce the risk of coinfections by stabilizing the gut microbiota, competitively excluding pathogens, and modulating host immune responses. By promoting a healthier intestinal environment, DFMs can improve nutrient absorption and feed conversion efficiency, leading to better overall livestock and poultry performance.



Call to Action

Interested in learning more about the impact of Novela® ECL during necrotic enteritis? Check out the full versions of poster presentations from past poultry scientific meetings:

ISPF 2025

PSA 2019

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About the Author



Firmin Delago is a Microbiologist II at Microbial Discovery Group. Holding an MSc in biotechnology from UW–Madison, he specializes in poultry direct-fed microbial research and development.



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