Are *E.coli* infections on the rise?

A multi-year quantitative surveillance of nursery pigs reveals increasing carriage of F18+ *Escherichia coli*.

Introduction:
Escherichia coli (*E. coli*) are a main inhabitant of the intestinal tract of mammalian species. Though many strains are harmless, some *E. coli* strains can produce adhesins, toxins, and a suite of other virulence factors that enable them to be pathogenic and cause clinical disease. The onset of clinical *E.coli* infections can be triggered by specific host and environmental stressors, including weaning stress and coinfections with other pathogenic bacteria.

In pigs, the early nursery phase is a critical and stressful period. During this phase, a rapid dietary change, combined with low feed intake, an immature gastrointestinal morphology, and a developing immune system, often contributes to:

- Low microbial diversity in the gastrointestinal tract (risk of dysbiosis)
- Inflammation
- Disruption of the intestinal barrier

Together, these factors allow for proliferation of opportunistic pathogens, such as enterotoxigenic *Escherichia coli* (ETEC), to cause infections that result in post-weaning diarrhea (PWD), reduced growth, or sudden death. PWD is one of the most significant causes of swine health and performance losses globally, with mortality rates reaching 20% to 30% during serious outbreaks.[1,2]

One way MDG and UAH assist in revealing current underlying pathogen dynamics, and aiding veterinary health teams in developing proactive approaches, is by employing tools like the PathKinex microbial surveillance database to monitor the incidence detection rate and gene quantities of ETEC and other current industry pathogens.

Materials and Methods:
Rectal swabs were collected from 962 nursery pigs at 26 commercial farms and classified into three groups by time of sampling: 2018-2019 (n=491), 2020-2021 (n=218), and 2022-2023 (n=253). Genomic DNA were extracted from swab samples and *E. coli* marker genes were quantified using a panel of quantitative PCR assays for toxin and virulence genes involved in swine enteric disease. *E. coli*

genes with less than 25 positive results were excluded from this analysis. Gene data were log-transformed and differences in average gene quantity between two-year periods were determined with the Kruskal-Wallis test, followed by pairwise comparisons using Dunn's test with Bonferroni adjustment. Incidence of *E. coli* marker genes of interest (number of pigs positive) by two years was compared using a Fisher's Exact test with Bonferroni adjustment.

Results and discussion:
PathKinex Surveillance results revealed that:

- A gene marker for the ETEC-associated F18 fimbrial adhesin was detected in increased quantities in rectal swab samples from nursery pigs in 2022-2023 compared to the 2018-2019 and 2020-2021 periods (1.7 vs 1.0 and 0.7 $\log_{10}$ gene copies respectively; $P<0.01$; Figure 1).

- The incidence of *F18* detection was increased in the 2022-2023 period compared to prior years (59% vs 33% and 26% respectively; $P<0.01$; Figure 2).

Together these data indicate that disease pressure from F18+ ETEC is higher than historical levels and may require new, multipronged approaches for prevention and control of PWD. Continued surveillance programs for ETEC and other economically important pathogens will provide veterinary health teams with comprehensive tools for the development of effective strategies for swine health management during single pathogen insults and co-infection scenarios.

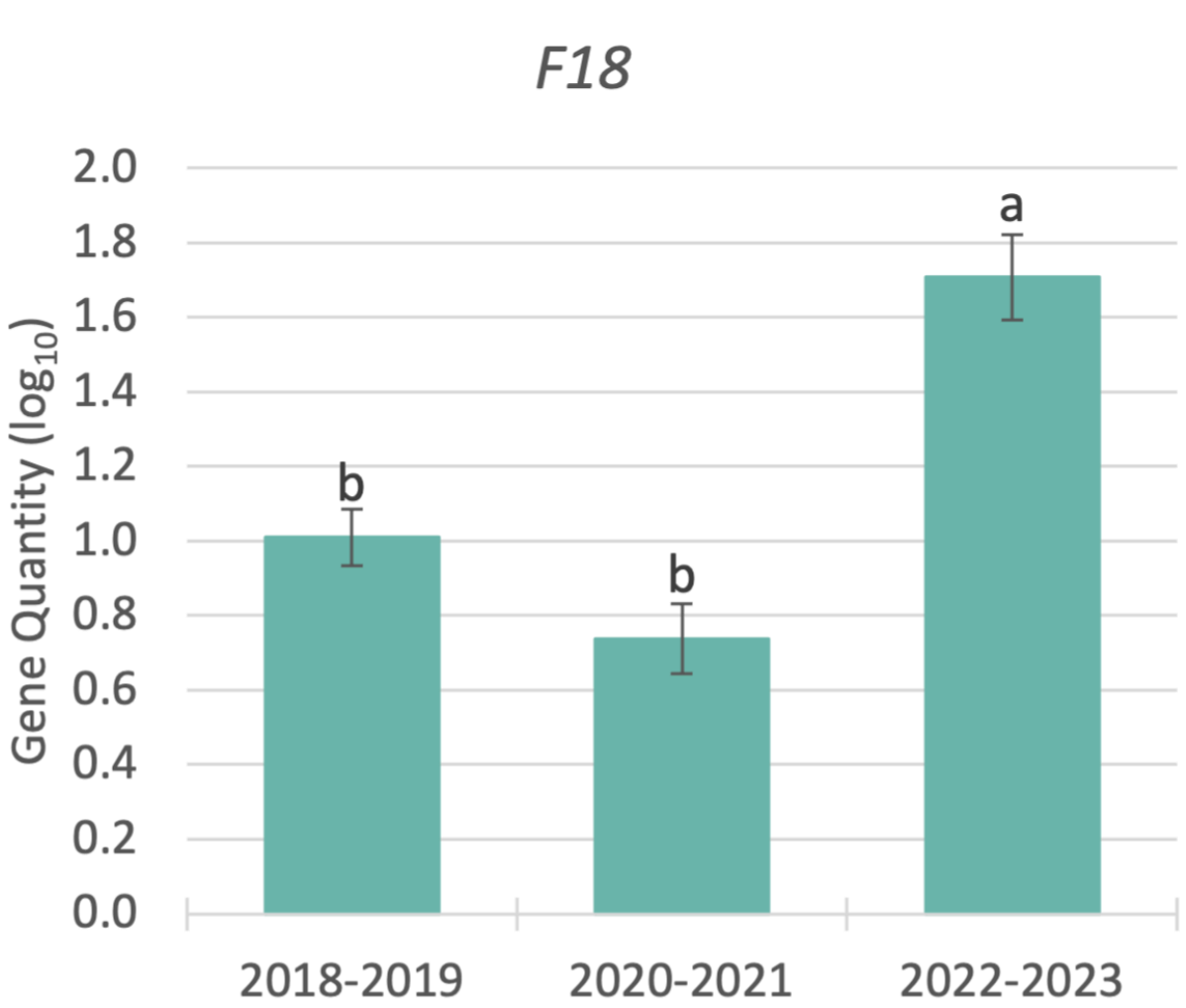

Figure 1. Mean *E. coli* virulence gene quantity in nursery pigs. Superscripts with different letters indicate that mean gene quantities differ $P<0.05$.

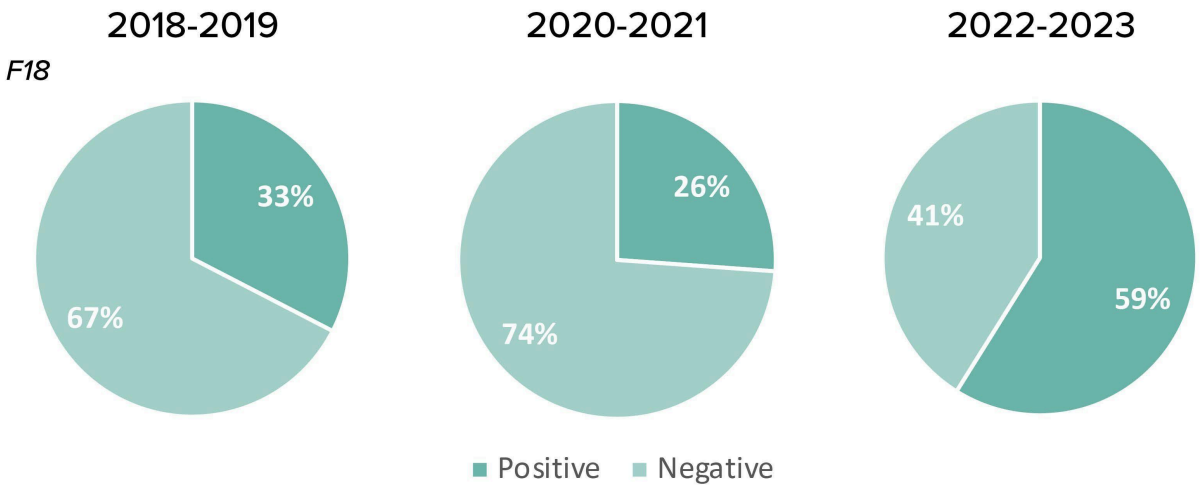

Figure 2. Incidence of F18+ *E. coli* in rectal swabs from nursery pigs over two-year periods.

Consider asking customers these questions:

- Have you observed any similar trends with enteric challenges associated with *F18 E. coli*?
- If yes, what have you done to manage this? What has worked, hasn't worked?

Interested in discussing these F18 findings with your customer?

Consider using these additional key messages and supporting resources for further discussions on ETEC and Provent ECL:

- **Seismic**>Collections>ProVent ECL>All Content>ProVent ECL PPT – Co-Challenge Situations
  - Message: There's more than one pathogen.
  - Message: Multiple pathogens and disruptors compound risk and severity of disease.
  - Message: There's no *"silver bullets."* Multiple interventions and a comprehensive strategy provide greatest success.

- Message: ProVent ECL can be an effective tool in a program approach to managing complex challenge syndromes.

References

1. Castro J, Barros M, Araújo D, et al. Swine enteric colibacillosis: Current treatment avenues and future directions. *Frontiers in Veterinary Science*. 2022; 9:981207.

2. Rhouma M, Fairbrother JM, Beaudry F, et al. Post weaning diarrhea in pigs: Risk factors and non-colistin-based control strategies. *Acta Vet Scand*. 2017; 59(1):1–19.

Is there a topic you'd like to learn more about in a future newsletter? We enjoy hearing from you! We welcome your questions, comments and suggestions on PathKinex updates. Please contact us at AnimalAg@mdgbio.com