



# MedVetPathogens 2025 Draft Program

(Subject to Change)

Monday 26<sup>th</sup> May

1:00 pm Registration Desk Opens

3:00-3:15 pm Welcome address by conference chair Professor John Boyce

## SESSION 1:

Chair: John Boyce

3:15-4:00 pm

### KEYNOTE LECTURE 1

Dr GEORGINA COX (Canada)

Investigating and inhibiting *Staphylococcus aureus* host adhesion.

*Staphylococcus aureus* is the leading global cause of deaths attributable to antimicrobial resistance (AMR), underscoring the urgent need for new therapeutics. Host adhesion is crucial for *S. aureus* colonization and infection, making it an attractive therapeutic target. In this talk, I will describe a whole-cell ELISA-based adhesion assay for genetic and chemical screening of *S. aureus* adhesion to host ligands. This assay was used to delineate the *S. aureus* genetic determinants of adhesion to fibronectin, keratin, and fibrinogen. From this work, we further explored the role of the major murein hydrolase, autolysin (Atl), in host adhesion, demonstrating that the hydrolytic function of this protein is important for the surface display of cell wall-anchored (CWA) proteins. A chemical screen identified a polyunsaturated branched-chain fatty acid, geranylgeranoic acid, exhibiting broad-spectrum anti-adhesive activity in *S. aureus*. This research highlighted the multifaceted role of unsaturated fatty acids in the host-pathogen interaction while providing a better understanding of the regulation of CWA proteins. Overall, these findings provide insight into the mechanisms of *S. aureus* host adhesion, highlighting potential therapeutic targets, and identified two new classes of anti-adhesive agents.

4:00-5:00 pm

Early Career Presentations\_selected from abstracts

4:20-4:40 pm

Early Career Presentation selected from abstracts

4.40-5:00 pm

Early Career Presentation selected from abstracts

5:00-7:00 pm

**WELCOME RECEPTION ON THE PRATO CENTRE TERRACE**

# Tuesday 27<sup>th</sup> May

## SESSION 2

Chair: TBA

8:45-9:30 am

### KEYNOTE LECTURE 2

DR MARK LAWRENCE (USA)

Leveraging comparative genomics to address new pathogen emergence and develop vaccine strategies in a complex aquaculture system

*Aeromonas hydrophila* causes motile *Aeromonas* septicemia (MAS) in fish. In 2009, a clonal type emerged, designated virulent *A. hydrophila* (vAh), that has had significant economic impact on the U.S. catfish aquaculture industry. Mortality rates in ponds ranged up to 50-60%, and predominantly market size fish were impacted. Comparative genomics enabled not only the identification of a phenotypic test to identify the vAh clonal group, but also allowed the identification of potential virulence traits and vaccine antigens unique to vAh. To identify candidate vaccine antigens against this newly emerged pathogen, our team focused on identifying predicted surface-expressed proteins unique to vAh, leading to identification of four candidate outer membrane proteins and four fimbrial proteins. Six of these recombinant proteins provided significant protection of catfish against MAS. Expression of these antigens in live attenuated *Edwardsiella ictaluri* vaccine ESC-NDKL1 provided an effective delivery system for use in catfish aquaculture. Subsequent testing of vAh antigen combinations allowed optimization of an effective dual vaccine against both MAS and enteric septicemia of catfish. Two recombinant ESC-NDKL1 strains (ESC-NDKL1::*fimMrfG::ompA::fimA* and ESC-NDKL1::*atpase::tdr::fim*) showed the best protection, providing relative percent survival of 77.9% and 82.3%, respectively. Comparative genomic analysis revealed putative secreted proteins unique to vAh, including chitinase, enterotoxin, collagenase, sialidase, and an RTX toxin. Isogenic mutant strains for each of these secreted proteins revealed that the RTX toxin, encoded by *rtxC* and *rtxA*, is required for vAh virulence. vAh $\Delta$ *rtxC*, vAh $\Delta$ *rtxA*, and double-deletion vAh $\Delta$ *rtxA-C* mutants were highly attenuated in catfish. The vAh $\Delta$ *rtxA-C* mutant had significantly decreased hemolytic activity against catfish red blood cells and significantly decreased biofilm formation. Furthermore, the RTX-deficient mutant demonstrated efficacy as a live attenuated vaccine candidate. Our results demonstrate that use of comparative genomics can rapidly advance pathogenesis research and vaccine development for newly emerging pathogens, particularly in complex systems such as aquaculture.

9:30-9:45 am

Presentation selected from abstracts

9:45-10:00 am

Presentation selected from abstracts

10:00-10:15 am

Presentation selected from abstracts

10:15-10:30 am

Presentation selected from abstracts

10:30-11:00 am

Coffee/tea break

## SESSION 3:

Chair: TBA

11:00-11:20 am

Early Career Presentation selected from abstracts

11:20-11:40 am

Early Career Presentation selected from abstracts

11:40-12:00 pm

Early Career Presentation selected from abstracts

12:00-12:15 pm

Presentation selected from abstracts

12:15-12:30 pm Presentation selected from abstracts

12:30-1:45 pm LUNCH

**SESSION 4:**

Chair: TBA

1:45-2:00 pm Presentation selected from abstracts

2:00-2:15 pm Presentation selected from abstracts

2:15-2:30 Presentation selected from abstracts

2:30-4:00 pm Poster Presentations

3:30-4:00 pm Continuation of poster session and Coffee/Tea Break

**SESSION 5:**

Chair: TBA

4:00-4:30 pm **Invited Speaker 1 Presentation**

4:30-4:45 pm Presentation selected from abstracts

4:45-5:00 pm Presentation selected from abstracts

5:00-5:15 pm Presentation selected from abstracts

FREE EVENING

## Wednesday 28<sup>th</sup> May

**SESSION 6:**

Chair: TBA

8:45-9:30 am **KEYNOTE LECTURE 3**

**PROF. JEAN-MARC GHIGO (France)**

Causes and consequences of biofilm formation by bacterial pathogens

In most environments, bacteria predominantly exist as surface-attached or aggregated three-dimensional structures known as biofilms. In these communities, bacteria encounter unique physicochemical conditions and undergo profound physiological changes associated with biofilm-specific properties, such as a high but reversible level of tolerance to antibiotics. While biofilms contribute positively to various ecological processes, biofilms formed by bacterial pathogens also lead to frequent therapeutic failures and pose significant challenges in both human and animal healthcare. I will discuss how exploring the nature and functions emerging from bacterial biofilms sheds light on bacterial adaptation to surfaces and how transitioning from classical pure culture studies to multispecies microbiology brings new perspectives for mitigating biofilm-associated bacterial infections.

9:30-9:45 am Presentation selected from abstracts

10:00-10:15 am Presentation selected from abstracts

10:15-10:30 am Presentation selected from abstracts

10:30-11:00 am	Coffee/tea break
<b>SESSION 7:</b>	Chair: TBA
11:00-11:20 am	Early Career Presentation selected from abstracts
11:20-11:40 am	Early Career Presentation selected from abstracts
11:40-12:00 pm	Early Career Presentation selected from abstracts
12:00-12:20 pm	Early Career Presentation selected from abstracts
12:20-12:40 pm	Early Career Presentation selected from abstracts
12:40-2:00 pm	<b>LUNCH</b>
2:00-5:15 pm	TBA

7:00-20:30 pm	<b>CONFERENCE DINNER</b>
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## Thursday 29<sup>th</sup> May

<b>SESSION 8:</b>	Chair: TBA
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9:00-9:45 am

### KEYNOTE LECTURE 4

#### Dr Marty Roop (USA)

Timing is everything – a proposal for how the global regulator MucR functions as a virulence determinant in *Brucella*

*Brucella* strains are major veterinary pathogens and important sources of human zoonotic disease in areas of world where these infections remain endemic in food animals. The Zn finger protein MucR is a global regulator of gene expression and an essential virulence determinant in *Brucella*, but precisely how it contributes to virulence is unresolved. Recent studies in our laboratory provide evidence that MucR functions as a novel type of H-NS-like gene silencer that works in concert with antagonistic transcriptional activators known as ‘counter-silencers’ to ensure the proper temporal expression of virulence genes during infection. For instance, MucR directly represses expression of the gene encoding the polar autotransporter adhesin BtaE and this repression is overridden by the quorum sensing regulator VjbR and MarR-type regulator MdrA presumably in response to host-specific environmental cues. These studies also suggest that MucR works in concert with VjbR, MdrA and other transcriptional activators to coordinate the proper temporal expression of other virulence genes including those encoding the polar autotransporter adhesins BmaC and BtaF and the Type IV secretion system and some of its secreted effectors. It is well-documented that H-NS and H-NS-like gene silencers work in concert with antagonistic counter-silencers in other pathogenic bacteria to ensure that virulence genes are only expressed at the specific stages of their infectious lifecycle in the host when they are needed, and that uncontrolled expression of these genes disrupts the infectious process resulting in attenuation. We propose that MucR and specific transcriptional activators such as VjbR and MdrA are working together to play an analogous role in controlling virulence gene expression in *Brucella*.

9:45-10:00 am

Presentation selected from abstracts

10:00-10:15 am Presentation selected from abstracts

10:15-10:30 am Presentation selected from abstracts

10:30-11:00 am Coffee/tea break

**SESSION 9:**

Chair: TBA

11:00-11.15 am Presentation selected from abstracts

11:15-11:30 am Presentation selected from abstracts

11:30-11:45 am Presentation selected from abstracts

11:45-12:00 pm Presentation selected from abstracts

12:00-12:30 pm Final Address and Awards Ceremony

12:30 pm Conference close

(take-out/take away lunch available for those who pre-ordered)