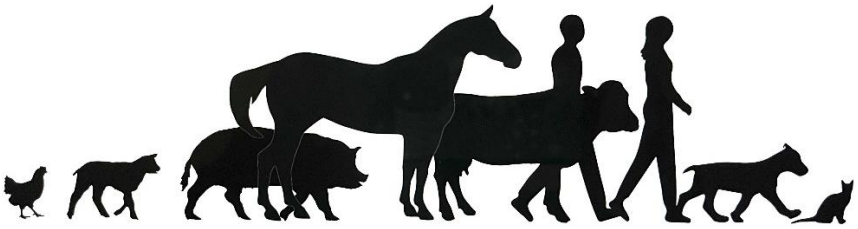

MedVetPathogens 2025 Draft Program

(Subject to Change)

Monday 26th 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 27th 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. vAhΔ*rtxC*, vAhΔ*rtxA*, and double-deletion vAhΔ*rtxA-C* mutants were highly attenuated in catfish. The vAhΔ*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** Dr JACLYN PEARSON Emerging multidrug resistant *Salmonella* and their cunning ability to survive in host cells Non-typhoidal *Salmonella* (NTS) serovars have swept the globe in recent decades, becoming a leading cause of gastroenteritis in humans today. Some serovars of NTS (e.g., ST313; endemic in sub-Saharan Africa) cause invasive disease exclusively in humans, with no known animal reservoir, features reminiscent of typhoidal *Salmonella*. However, a pandemic lineage of NTS, ST34 monophasic *Salmonella*, causes invasive disease in humans, and are endemic in farmed porcine populations. The key features of this emerging pathogen are 1) heavy metal resistance, 2) extensive multidrug resistance (MDR), 3) loss of the second flagella antigen, FljB and 4) increased ability to survive in human macrophages. There are currently no vaccines or therapeutics available to treat human salmonellosis. As the prospect of limited-to-no antimicrobial therapeutic options for MDR invasive NTS (iNTS) looms, it is critical that we understand the pathogenic mechanisms of emerging MDR iNTS and the specific host responses elicited during infection to inform future development of vaccines or host-directed therapies. Our preliminary data indicate that ST34 monophasic *Salmonella* have unique infection outcomes; in human macrophages we observed up to 10-fold higher intracellular replication of ST34 monophasic *Salmonella* compared to matched ST34 biphasic *Salmonella* isolates, as well as clinical isolates of other invasive *Salmonella* including *S.* Typhi. Importantly, the increased intracellular growth did not activate intracellular host cell death pathways typically observed in NTS infections, suggesting ST34 monophasic *Salmonella* evades host cell death activation. Host RNA sequencing and phosphoproteomics revealed unique immune signature suggesting that ST34 monophasic *Salmonella* drive host macrophages into a pro-survival state, supporting robust intracellular bacterial replication and survival. Our ongoing work will provide critical insights into novel host-directed therapies that will aid the development of alternative treatments for zoonotic iNTS more broadly.
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 28th 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	
SESSION 7:		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	**LUNCH**	
2:00-5:15 pm	TBA	
7:00-20:30 pm	**CONFERENCE DINNER**	

Thursday 29th May

SESSION 8: Chair: TBA

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)