Long-read Metagenomics Uncovers the Taxonomic and Functional Profile of Marine Bacteria Colonizing Copper Surfaces

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Marine biofouling of artificial surfaces in seawater, such as ship hulls, can lead to significant economic losses and can introduce invasive species to new environments. The restrictions on using tributyltin-based antifouling paints imposed a need to develop cost-effective and environmentally safe alternatives against biofouling. Currently, copper-based antimicrobial paints are commonly used to prevent biofouling on marine vessels. However, many bacteria can still colonize these surfaces. The initial bacterial population colonizing the marine vessels plays a crucial role by producing extracellular polymeric substances (EPSs), which form a thin layer of organic matter, trapping nutrients and protecting other colonizers by blocking the toxic coatings. There is interest in understanding the factors that drive the initial colonization of copper surfaces to develop new antifouling compounds. Bacteria harbor various regulatory mechanisms to respond to copper and maintain copper homeostasis in cells, but their role in marine copper surface colonization is not yet known. Our goal is to identify the mechanism of surface-associated copper tolerance in marine bacteria. We used a whole-genome PacBio metagenomics sequencing approach to identify the early-stage colonizers of copper surfaces. We identified copper pathways enriched in these early colonizers by evaluating the functional profile of the early-colonizing community of copper surfaces and comparing them to the seawater community. This helped us predict the interaction among marine microbes during the early-stage colonization of copper surfaces. The characterization of the gene abundance profile of the biofilm community, compared to the seawater community, could be used as an indicator of early-stage colonization of copper surfaces.

SroA Modulates The Levels of Staphyloxanthin Pigment in *Staphylococcus aureus*

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Staphylococcus aureus is a pathogen notorious for causing serious infections worldwide. Its ability to evade host immune defenses and develop antibiotic resistance allows it to persist and spread in hospitals and communities. Without new treatment strategies, antibiotic-resistant *S. aureus* infections are projected to become a leading cause of death worldwide. Characterizing novel virulence factors or regulators of virulence factors will assist us in developing new treatments. A critical virulence factor for *S. aureus* immune evasion is the carotenoid pigment Staphyloxanthin (STX). STX enhances the ability of *S. aureus* to cause invasive infections with high mortality, including sepsis and bacteremia. STX protects *S. aureus* from reactive oxygen species released by phagocytic immune cells and is essential for pathogenesis. The multi-step biosynthesis of STX is encoded by the *crtMNOPQ* operon, which is primarily regulated by the alternative sigma factor SigB. However, additional regulatory factors likely exist but remain undiscovered. We recently discovered a novel *S. aureus* protein, SroA, that may act as a major effector of virulence. We created a mutation in *sroA* and noticed that the cells exhibited increased pigment. This led us to hypothesize that SroA may act to modulate STX synthesis in *S. aureus*. To confirm the SroA effect on STX synthesis, we methanol extracted STX from wild type and *sroA* mutants and quantitatively measured them. Our results demonstrate a statistically significant ($P\text{-value} < 0.001$) increase in STX amounts in *sroA* mutants, suggesting that SroA regulates STX synthesis in *S. aureus*.

Enterococcal Strains' Adaptations to Host Immune Response and Fibrinolytic Activity *(maybe grad student so still Lightning)*

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Catheter-associated urinary tract infections (CAUTIs) are the most common hospital-acquired infection accounting for 40% of nosocomial infections worldwide. The causative gram-positive bacteria, *Enterococcus faecalis*, represents 11%-30% of CAUTIs. Previous research on *E. faecalis* CAUTIs have used the oral isolate strain, OG1RF. Currently, there is limited research on *E. faecalis* V583, a strain isolated from a patient with urosepsis. Characterization of this strain will help understand *E. faecalis* CAUTIs as V583 has a specific niche for the bladder environment. The ability for *E. faecalis* to colonize the bladder is dependent on the deposition of fibrinogen (Fg), a healing protein that is recruited to the catheterized bladder and is used as a nutritional source for biofilm formation. Additionally, the *E. faecalis* secreted protease SprE has been shown to degrade C3, plasminogen, and plasmin, which are critical components in immune evasion and the breakdown of Fg. The overall objective of this study is to investigate the differential immune responses and fibrinolytic activity between OG1RF and V583. Here, we showed that V583 is less susceptible to killing by neutrophils and macrophages compared to OG1RF. Additionally, we have found that V583 has a decreased expression of sprE in urine conditions compared to OG1RF; however, similar protease degradation activity was observed in these two strains.These results suggest that SprE contributes to V583's ability to evade the host immune response and dysregulate the fibrinolytic cascade during CAUTI, posing a serious global health threat warranting further study and necessitating novel treatment options.

Strain Specific Outcomes of *Pseudomonas aeruginosa* Catheter-associated Urinary Tract Infections

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Hospitalized patients with indwelling catheters face an increased risk of developing catheter-associated urinary tract infections (CAUTI). Catheters provide a surface for bacterial colonization, leading to either symptomatic infections or asymptomatic bacteriuria. In a previous study using an outbred murine CAUTI model with the PA14 strain, we identified two distinct phases: an acute phase, with host morbidity and mortality within the first week, and a chronic phase, with asymptomatic colonization in the second week. Host mortality is primarily driven by ExoU activity of the type III secretion system (T3SS) in PA14. However, in Pseudomonads, the T3SS effectors *exoU* and *exoS* are largely mutually exclusive. To determine infection outcomes from ExoS activity, we used the murine CAUTI model with the PAK strain. Mice infected with PAK had similar survival rates to those infected with PA14. However, while mortality in PA14-infected mice occurred rapidly within the first three days, in PAK-infected mice, it occurred steadily over a 10-day period. Moreover, mutants lacking either T3SS or type II secretion system (T2SS) did not induce acute outcomes in PAK, suggesting that in *exoS*-containing strains, both T2SS and T3SS contribute towards host mortality. To evaluate the applicability of these findings to clinical strains isolated from CAUTI patients, we tested three strains encoding *exoS* and five strains encoding *exoU* in a murine CAUTI model. We observed similar outcomes with their respective laboratory strains; however, some isolate-specific differences were noted. This study highlights the importance of the T3SS in all strains and the T2SS in strains with ExoS.

Investigating LoaP's Effect on the Proteome and Secondary Metabolite Production by Mass Spectrometry

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The conserved transcription factor NusG forms dynamic interactions with RNA polymerase (RNAP) and other transcription factors (NusB, NusA, Rho and S10) to influence transcription processivity and, in certain cases, termination. While virtually all bacteria encode for a core NusG, many also encode specialized paralogs that enhance expression of target genes (i.e., biosynthetic operons for secondary metabolites or virulence factors). These specialized NusG paralogs affect gene expression by promoting transcriptional readthrough of termination sites located within their targeted operons. We previously discovered a subfamily of NusG paralogs, which we named LoaP, and found that they are widespread in Firmicutes, Actinobacteria and Spirochetes. Unlike other NusG paralogs, which lack RNA-binding activity, our preliminary data also showed that LoaP binds to a conserved RNA hairpin located within the 5' leader region of its targeted operons with both high affinity and specificity. However, we do not know how this ribonucleoprotein complex participates in antitermination activity. In this project, we examined the LoaP regulon for *Bacillus velezensis*, which is a plant growth-promoting bacterium. We used high-resolution mass spectrometry-based quantitative proteomics to examine *B. velezensis* strains that either lacked or over-expressed LoaP. Our data showed that the polyketide antibiotics difficidin and macrolactin are highly dependent on LoaP regulation. Our data also show that LoaP's RNA-binding activity is critical for its regulation in vivo. We hypothesize that this general approach can be used to investigate the LoaP regulons for other bacteria and help discover novel secondary metabolite production pathways.

Pyrrole-Imidazole Alkaloids: Elucidating Their Mechanism of Action

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Bacterial infections are once again becoming a global health crisis. Thus, it is critical to develop not only novel antibiotics, but also novel strategies to combat antibiotic resistance. Marine-derived, pyrrole- imidazole alkaloids can achieve both goals as they have broad spectrum activity, and they can also act as antibiotic adjuvants and inhibit biofilms. There has been some effort to investigate the mechanism of action of a model compound, sceptrin. However, the results of these studies are inconsistent. Furthermore, limited access to multiple pyrrole-imidazole alkaloids has stalled drug development of this class as a whole. To overcome this critical barrier, we have successfully amassed a collection of pyrrole-imidazole alkaloids through a collaboration with the National Cancer Institute's Natural Product Repository, and we have designed a simple, yet powerful strategy to thoroughly assess the therapeutic potential of this class of compounds. Using a combination of unbiased, whole cell chemical genomic analysis and phenotypic fluorescence microscopy in addition to more standard analysis of morphology and bacterial growth, we determined that sceptrin causes membrane depolarization, increases cell permeability, and alters the peptidoglycan cell wall in *E. coli*. Furthermore, in addition to their previously established ability to act as antibiotic adjuvants for meropenem and oxacillin in drug-resistant *A. baumannii* and MRSA, respectively, we have determined that sceptrin and certain analogues increase the potency of ciprofloxacin, erythromycin, and doxycycline in *E. coli*. The findings generated by this project will provide a pathway to generate a new class of urgently needed antibiotics and adjuvants derived from pyrrole-imidazole alkaloids.

Identification and Characterization of an Invasive, Hyper-aerotolerant *Campylobacter jejuni* from a Blood Culture of a Pediatric Leukemia Patient

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Background: *Campylobacter jejuni* (Cj) is a leading zoonotic pathogen causing enteritis. Despite its well-known microaerophilicity, hyper-aerotolerant (HAT) Cj were isolated from intestines in poultry/ humans, which may have enhanced survival/transmission. However, invasive HAT Cj isolates have not been reported in human bloodstream infections (BSI). Recently, we isolated a Cj from aerobic blood culture from a 3-year-old leukemia patient with abdominal pain and neutropenic fever. We aimed to perform microbiological, genetic, and comparative genomic characterizations of this novel Cj isolate (CNH-HAT-1).

Methods: MALDI-TOF-MS and 16S-rRNA sequencing were performed, followed by antimicrobial susceptibility testing. Aerotolerance testing was performed by comparing quantitative culture with control strains after aerobic shaking. Whole genome sequencing (Illumina/Nanopore) of CNH-HAT-1 was conducted, with comparative genomic analysis using ResFinder-4.4.2 and BRIG-v0.95.

Results: CNH-HAT-1 was only resistant to beta-lactam antibiotics, consistent with genome analysis. Aerobic growth assay showed its HAT phenotype. Hybrid genome sequencing yielded four contigs covering the whole genome with no plasmids identified. MLST sequence type and clonal complex of CNH-HAT-1 were ST-454 and ST21, respectively. CNH-HAT-1 has similar iron acquisition systems to other human isolates. Comparative analysis of >50 Cj strains found CNH-HAT-1 phylogenetically close to the recently reported sheep abortion strains (IA3902) and various bacteremia Cj. G250A mutation in major outer-membrane gene *PorA* was identified in CNH-HAT-1, likely contributing to its invasiveness and sheep abortion. CNH-HAT-1 contains CJIE1, a prophage element prevalent in other bacteremia Cj strains.

Conclusions: For the first time, we report a novel, invasive HAT Cj causing human BSI in a pediatric patient.

Per- and Polyfluoroalkyl Substances (PFAS) Induced Mitochondrial Dysfunction and Aging in Neuroblastoma Cells

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This study evaluated the impact of perfluorooctane sulfonate (PFOS) and GenX on mitochondrial dysfunction and cellular aging in neuroblastoma (N18) cells. PFOS and GenX, in the per- and polyfluoroalkyl substances (PFAS) family, are synthetic chemicals found in household products and have been linked to hormonal disruption, biological aging, and oxidative stress. The study hypothesized that increasing concentrations of PFOS and GenX would intensify mitochondrial damage and accelerate aging in N18 cells. N18 cells were exposed to PFOS and GenX at concentrations of 10 µM, 100 µM, and 500 µM for 48 hours. Cell viability was assessed using the XTT assay, while mitochondrial dysfunction was evaluated by measuring mitochondrial membrane potential and ATP levels. Oxidative stress was assessed by analyzing total antioxidant capacity and superoxide dismutase (SOD) activity. Results demonstrated a dose-dependent decrease in cell viability, with 500 µM of PFOS and GenX reducing viability to 69% and 81%, compared to the control group. Mitochondrial dysfunction was indicated by reduced MitoTracker fluorescence and ATP levels, suggesting impaired mitochondrial membrane potential and cellular energy production. Additionally, antioxidant defenses were compromised, with a decline in total antioxidants and SOD activity in cells exposed to higher concentrations of PFOS and GenX. Furthermore, total protein content decreased at higher exposure levels, indicating cellular stress and damage. These findings suggest that elevated concentrations of PFOS and GenX induce mitochondrial dysfunction, oxidative stress, and accelerate cellular aging in N18 cells. Further research is required to explore the broader implications of PFAS exposure on aging and neurodegenerative diseases.

Novel Role of DUF2975-Domain Genes in *Pseudomonas aeruginosa* Biofilm Development and Catheter-Associated Infections.

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Pseudomonas aeruginosa is an opportunistic pathogen forming robust biofilms on many medical surfaces, including catheters. This study explores the roles of two gene pairs (PA5402/PA5403 and PA2784/PA2785) encoding the DUF2975 domain of unknown function protein and a helix-turn-helix protein in *Pseudomonas* biofilm formation. We hypothesize that the PA5403 gene likely encodes a transcriptional regulator of the PA5402 gene, and the PA2785 gene likely encodes a transcriptional regulator of PA2784. The role of DUF2975 is unknown and is the subject of our study. Our findings demonstrate a significant reduction in biofilm formation on catheters in transposon mutant strains of the PA5402 & PA5403 genes compared to the wild type, suggesting that these genes are involved in biofilm development. Colony biofilm assays showed disrupted biofilm formation in PA5402 and PA5403 mutants, with altered morphology whereas the PA5404 and PA2784 mutants formed biofilms like wild type. Catheter-associated biofilm experiment showed much-reduced biofilm formation by PA5402 mutant (PW10114), the PA5403 mutant (PW10116) has less biofilm, whereas the PA2784 mutant (PW5664) formed biofilms like wildtype. Air-liquid interface pellicle assay reveals disrupted biofilm formation in PA5402 and PA5403 mutants, while PA5404 and PA2784 mutants produced pellicles like wild-type. The differences between these results obtained with crystal violet staining and colony biofilm assays suggest that these genes play a crucial role in various stages of biofilm development. The study suggests that targeting these two specific genes could potentially prevent catheter-associated infections and these findings also have broader implications for biofilm formation mechanisms in other clinically relevant pathogens.

The Missing Link: Unlocking the Impact of Uncharacterized msh Genes on *Vibrio cholerae* MSHA Pilus Biogenesis

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The aquatic bacterium, *Vibrio cholerae*, causes the gastrointestinal disease cholera, resulting in ~3-5 million reported cases and ~100,000-140,000 deaths annually. The ability to form multi-cellular biofilms is associated with its environmental survival and persistence. Current circulating pandemic strains of *V. cholerae,* attach to surfaces using the type IV mannose-sensitive hemagglutinin (MSHA) pilus. Loss of MSHA pili attenuates surface colonization and biofilm formation. Components of the MSHA pilus are encoded within two predicted genetic operons; msh-I (mshHIJKLMNEGF) and msh-II (mshBACDOPQ). However, the function of most msh genes remains to be characterized. Understanding the mechanisms of MSHA pilus biogenesis is key to deciphering the environmental survival of *V. cholerae*. To this end, I have generated deletions of each msh gene, along with corresponding complementation plasmids. Analysis of MSHA pilus production for each deletion strain via hemagglutinin (HA) assay, demonstrated that msh-I genes mshIJKLMNEG, and msh-II genes mshACDOP are vital for pilus production. Genes mshH, mshF, mshB, and mshQ were observed to still support pilus production, and might play an accessory role assembly/function. Analysis of major pilin subunit (MshA) protein production via immunoblot, demonstrated similar MshA levels among each deletion mutant (except DmshA), suggesting that pilus components are produced but not assembled among the mutants. Currently, studies are underway to directly visualize MSHA pilus production, and biofilm formation utilizing an established flow-cell model amongst these msh deletion strains. These studies will identify genes crucial for MSHA pilus production, aiming to develop strategies to reduce *V. cholerae* survival in the environment.

Characterizing Skin Microbiome and Genetic Interactions in Infant Atopic Dermatitis

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Atopic dermatitis (AD) is a common skin condition, often beginning in childhood. While genetic loci, such as *FLG* mutations affecting skin barrier integrity, are linked to AD, genetics account for less than 20% of AD heritability, suggesting a significant role for other factors, including the microbiome. Although the skin microbiome in older children and adults with AD is well-studied, research on infants at AD onset and the influence of genetics on the microbiome remains limited. Additionally, up to 40% of infants with AD develop food allergies (FA), yet the microbiome's role in this progression is unclear. The VITALITY trial in Australia recruited breastfed infants at 2 months, assessing AD and FA at 1 year using SCORAD and oral food challenges. Skin swabs were collected from 164 infants with AD alone, 59 with AD+FA, and 53 healthy controls at 1 year. Additional samples were taken from 61 infants at 2 months. The skin microbiome was analyzed using deep shotgun metagenomic sequencing. Our analysis revealed that infants who later developed AD exhibited higher microbial diversity and a more mature skin microbiome at 2 months than healthy controls. Differential abundance analysis revealed that six taxa, including *Staphylococcus epidermidis*, were enriched in AD-only infants, while seven distinct taxa were associated with AD+FA cases. Host genetics analysis linked six genera, including *Staphylococcus*, to *FLG* mutations. A microbial GWAS pipeline identified 194 AD-associated genes in *S. epidermidis*, enriched in tryptophan and biotin synthesis pathways. These findings reveal early microbiome changes and genetics-microbiome interactions in AD.

Role of *carA-carB-pyrB* Operon in *Francisella novicida* U112 Biofilm Formation

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The ability to regulate biofilm formation is key across bacterial species. Using *Francisella novicida* U112 we sought to gain understanding of the effects of alterations of pyrimidine metabolism on *Francisella* biofilm formation, using transposon mutants in the genes *carA*, *carB*, and *pyrB*. We confirmed the phenotype of uracil auxotrophy in each of the mutants, consistent with prior studies in *F. tularensis* SchuS4 and LVS. We assessed whether *carA-carB-pyrB* were co-expressed in an operon using PCR analysis to demonstrate co-transcription. Our results suggest that these genes are regulated by a single promoter, a finding supported by Prokaryote Promoter Prediction v2. To assess the functional relevance of these genes, we examined biofilm formation by each of the transposon mutants. We measured altered levels of biofilm production by *carA*, *carB,* and *pyrB* mutants relative to the parental strain, suggesting that this operon may play a broad role in *F. novicida*. We are exploring the hypothesis that the *carA*-*carB*-*pyrB* locus is potentially influencing bacterial adherence and biofilm community formation. The altered biofilm phenotypes could have implications for *F. novicida*'s survival and pathogenicity in natural environments or host organisms. These findings provide new insights into the genetic regulation of essential metabolic pathways and their broader impacts on bacterial physiology.

LoaP Regulated Operons in Diverse Bacterial Groups

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Antibiotics like polyketides are natural products which are widely synthesized by microbes. However, there is insufficient knowledge regarding the mechanisms of regulation of these metabolites. One of the proteins regulating PKS pathways in *Bacillus velezensis FZB42* is LoaP, which is a paralogue of universally conserved transcription factor, NusG. In *B. velezensis*, LoaP has been shown to interact with an RNA hairpin upstream of the target operon and promote antitermination of the long operons responsible for producing difficidin and macrolactin. It is encoded in the genomes of several bacterial species including Paenibacillus, Bacillus and Brevibacillus, however its regulation of PKS/PKS-like pathways is unknown. We aim to understand if LoaP regulates similar pathways in related bacterial species, like *Bacillus thuringiensis ser. pondicheriensis*. To investigate the regulation of LoaP associated operon in *B. thuringiensis*, we developed antisense RNA based tools to generate knockout and overexpression strains. Through proteomics analysis of these genetically modified strains, we identified potential target genes regulated by LoaP. Additionally, we established molecular genetic tools using conjugation-based methods for genome targeting in diverse bacterial species, facilitating further exploration of LoaP and its homologs.

Impact of Climate Change on Microbial Food Safety

Isabel Walls

US Department of Agriculture Food Safety and Inspection Service.

FSIS developed a Climate Change Adaptation Plan which considered the impacts of climate change (e.g., an increase in heat waves and increased precipitation) on food safety. Foodborne illnesses are more common in the summertime and high ambient temperatures can influence the growth of *Salmonella* during food production, transport, and storage. Heat stress in animals can lead to increased susceptibility to parasites and pathogens and mild winters allow survival of flies which can spread diseases to food production animals, both of which could lead to an increase in zoonotic diseases. Increased drought may reduce water availability for sanitation. Flooding may result in contaminated water entering food processing establishments, carrying *Salmonella* and other pathogens. Natural disasters, which are increasing due to climate change, may cause power outages, or impact transportation of animals, products or samples to the laboratory. Animal food production and processing establishments are required to reassess the adequacy of their prerequisite plans and HACCP plans due to natural disasters, and update them, if necessary, to mitigate any impacts to producing safe and wholesome products. Research is needed to identify additional impacts of climate change on food safety.