Annual Meeting
Ohio Branch of the American Society for Microbiology

The Ohio State University
Pomerene Hall

Columbus, Ohio
September 11, 2022

Official Meeting Program
and
Conference Abstracts

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Acknowledgements

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OBASM Executive Committee

Christine Weingart
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Lubna Abu-Niaaj
Harry Kestler
Chet Cooper
Laura Tuhela-Reuning
Stephanie Miller

Special Thanks to:

Ohio State University – for hosting our meeting
- Our hosts – The American Society for Microbiology Student Chapter at the Ohio State University
- Yalini Ramamoorthy and Simran Chandawarkar – OSU student organizers
- Birgit Alber – faculty advisor

Kim Finer – Region 3 Branch Planning Coordinator
ASM Distinguished Lecturer Program
The American Society for Microbiology

Invited Speakers

Birgit Alber
Patrick Bradley
Steve Diggle

Program Schedule for OBASM 2022

Sunday, September 11

9:00 am **Registration and Poster Set-up –** Grand Atrium, Pomerene Hall

9:30 am **Introduction and Welcome** – Pomerene Hall, Room 160

D.J. Ferguson
President of the Ohio Branch of the American Society for Microbiology

9:45 – 10:15 **Birgit Alber** – Pomerene Hall, Room 160
Department of Microbiology
The Ohio State University

"Probing the central carbon metabolism of *Cereibacter sphaeroides* by random transposon mutagenesis"

10:15 – 10:45 **Patrick Bradley** – Pomerene Hall, Room 160
Department of Microbiology
The Ohio State University

"Gut microbial gene clusters with homology to human enzymes can lead to parallel drug metabolism"

10:45 – 11:00 **Break**

11:00 – 12:45 pm **Podium presentations and judging** – Pomerene Hall, Room 160
Coordinator – Chet Cooper

11:00 – 11:15 am
Genetic determinants of *Klebsiella* phage infection

Marissa R. Gittrich*[1], Courtney M. Sanderson[1], Cara M. Noel[1], Jonathan E. Leopold[1], Erica Babusci[1], Olivia R. Farinas[1], Vivek K. Mutalik[2], Matthew B. Sullivan[1]
[1]The Ohio State, [2]Lawrence Berkeley National Laboratory

11:15 – 11:30 am
Discovery of Anti-Phytopathogenic Phosphonopeptides from *Bacillus velezensis*

Jake Wilson[*1,2], Jerry Cui[1], Toshiki Nakao[1], Happy Kwok[1], Yeying Zhang[1], Chase M. Kayrouz[1], Tiffany M. Pham[1], Hannah Roodhouse[1], and Kou-San Ju[*,1,2,3,4]
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[4]Infectious Diseases Institute, The Ohio State University, Columbus, OH 43210

11:30 – 11:45 am
Siderophore Biosynthesis in *Histoplasma capsulatum*

Chandan Shah*, Chad Rappleye
Department of Microbiology, The Ohio State University

11:45 – 12:00 pm – Break

12:00 – 12:15 pm
Toward understanding the molecular mechanism of novel BPZ derivatives against *Toxoplasma gondii*

Sarah Ritter[*1], Abby Underhill[2], Marisa Prisco[2], and Robert Charvat[2]
[1]College of Pharmacy, University of Findlay; [2]College of Sciences, University of Findlay

12:15 – 12:30 pm
ZFP36 Ring Finger Protein Like 1 (ZFP36L1), a CCCH- Type of Zinc Finger protein significantly suppresses Human coronavirus- OC43 replication

Tooba Momin[*1], Andrew Villasenor[1], Mrigendra K. S. Rajput[1]
[1] Department of Biology, University of Dayton, Dayton, Ohio, USA 45469

12:30 – 12:45 pm
S*-adenosylmethionine byproduct utilization in extraintestinal pathogenic *Escherichia coli

Joshua T. Groves[*1], Katherine A. Huening[1], Justin A. North[1]
[1]Ohio State University

12:45 – 2:00 pm **Mid-Day Activities:**
Lunch: Box or "on your own"

Lunch will be available in Pomerene Hall

OBASM Business Meeting – Pomerene Hall, Room 280

2:15 – 3:15 pm **ASM Branch Lecture** – Pomerene Hall, Room 160

"Understanding the Ecology and Evolution of Chronic Infection"

Steve Diggle
Department of Microbiology
Georgia Institute of Technology

3:15 – 3:30 pm **Break**

3:30 – 5:00 pm **Poster presentations and judging** – Grand Atrium, Pomerene Hall
Coordinator – Chet Cooper

5:10 – 5:30 pm **Meet the Speaker**
An informal meeting with our speakers. Bring your questions and join in the conversation!

Birgit Alber – Pomerene Hall, Room 280
Patrick Bradley – Pomerene Hall, Room 160

5:45 – 6:00 pm **Student Awards Presentations** – Pomerene Hall, Grand Atrium
D.J. Ferguson – OBASM President

Abstracts of OBASM Poster Presentations
Sunday, September 11
3:30-5:00 pm

BOARD 1

Sequencing SARS-CoV-2 Variants from Indoor Dust

John Van Dusen[1*], Haley LeBlanc[2], Nicole Renninger[3,4], Nicholas Nastasi[3,4,5], Jenny Panescu[4], Michael G. Sovic[2], Amanda Williams[2], Seth Faith[2,7], Karen C. Dannemiller[4,5,6]
[1]Department of Microbiology, College of Arts and Sciences, Ohio State University, Columbus, OH 43210
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SARS-CoV-2 still exists in our communities and new variants will continue to evolve and circulate. Individual PCR-based testing is decreasing in frequency, and broader surveillance approaches targeting environmental sources are becoming more widely adopted to monitor these variants. The goal of this study is to demonstrate that SARS-CoV-2 variants can be detected and sequenced from indoor bulk dust samples. From April 2021 to March 2022, we collected vacuum bags containing dust aspirated through normal cleaning procedures carried out by the environmental services staff from selected buildings on The Ohio State University's Columbus campus. We developed a sequencing protocol to detect and estimate the frequency of viral variants in the samples. We demonstrate that sequencing methods can be used to detect SARS-CoV-2 variants in bulk dust samples. Furthermore, we find that using PCR amplification to prepare samples for sequencing results in greater genome coverage compared to methods such as the Illumina Respiratory Virus Oligo Panel, allowing for more sensitive variant identification. Three variants of concern were detected in the dust: Alpha, Delta, and Omicron. Alpha was found in our earliest sample in April 2020 with an estimated frequency of 100%. Delta was the primary variant present from October of 2021 to January 2022, with an average estimated frequency of 91.09% (±1.33%). Omicron became the primary variant in January 2022 and was the dominant strain in circulation through March with an estimated frequency of 87.04% (±3.17%). The detection of these variants on OSU's campus correlates with the circulation of these variants in the surrounding population. Overall, these results support the hypothesis that household dust can be used as a means of tracking SARS-CoV-2 variants and their circulation in buildings.

BOARD 2

Determining the effects of propionate on *Listeria monocytogenes* fitness

Elizabeth Herr*[1], **Jeanne P. Sering***[1], and Yvonne Sun[1]
[1]University of Dayton

Listeria monocytogenes is a harmful pathogen that is transmitted through contaminated food. Listeriosis, the infection associated with *L. monocytogenes*, is rare but potentially fatal, with a twenty to thirty percent mortality rate. For that reason, the lack of safe strategies to prevent infections can be detrimental. Current infection preventative strategies rely on stringent food surveillance and recalls and rely on consumer compliance. To determine alternative tactics to further protect the public from *L. monocytogenes*, we are identifying environmental factors that can compromise the ability of *L. monocytogenes* to persist in the environment and cause infections. In this study, we investigated how propionate, a generally recognized as safe food additive, modulates *L. monocytogenes* survival at cold temperatures as well as susceptibility to lysozyme. Moreover, because propionate exposure likely takes place under low oxygen conditions, we also examined the effects of propionate under anaerobic conditions. Using in vitro growth assays, we observed that the presence of 25 mM propionate did not significantly impact *L. monocytogenes* survival at the cold temperatures over 4 days. Moreover, to determine the potential mechanisms, we also examine the role of the stress response sigma factor SigB in *L. monocytogenes* response to propionate. We found that propionate treatment resulted in enhanced lysozyme survival in anaerobic Δ*sigB* mutant. These results collectively suggest that propionate is not a suitable antimicrobial agent in food under cold storage. Moreover, SigB might be contributing to *L. monocytogenes* propionate responses under anaerobic conditions.

BOARD 3

Feeding the Yeast: The search for *Histoplasma*'s intracellular carbon source.

Stephanie C. Ray* and Chad A. Rappleye
Ohio State University Department of Microbiology

The dimorphic fungal pathogen *Histoplasma capsulatum* infects both immunocompromised and immunocompetent hosts, resulting in disease ranging in severity from a mild flulike illness to a deadly disseminated infection. Unlike other fungal pathogens, *Histoplasma* is almost exclusively intracellular during infection, growing and dividing primarily within the phagosomal compartment of host macrophages. However, it is unknown which carbon substrates are utilized by *Histoplasma* inside the phagosome. Recent data have shown that *Histoplasma* strains which cannot metabolize glutamate or perform gluconeogenesis are attenuated within a murine host, suggesting that glutamate or its related compounds are a major carbon source for *Histoplasma* inside the macrophage. To investigate the possibility of amino acids as *Histoplasma's* intracellular carbon source, we characterized the substrate specificities of *Histoplasma*'s amino acid transporters using heterologous expression in a *Saccharomyces* model. We identified three major amino acid transporters which were then

depleted in *Histoplasma*, creating a strain of *Histoplasma* deficient in transport of most amino acids. The transport-deficient *Histoplasma* yeast were assayed for virulence both in cultured macrophages and in a mouse model of histoplasmosis. Surprisingly, the transport-deficient yeast were only mildly attenuated compared to wild-type *Histoplasma*, suggesting free amino acids are not the primary carbon source during *Histoplasma* infection.

BOARD 4

Concordance of mutations identified by random and high-throughput mutagenesis procedures

Sophia Gomez*[1], Marissa Gittrich[2], Vivek Mutalik[3], Matthew Sullivan[2], and Paul Hyman[1]
[1]Ashland University; [2]The Ohio State University; [3]Lawrence Berkeley National Laboratory

Bacteriophages(phages) are viruses that infect bacteria and need a host to survive. Successful infection requires the presence of a number of bacterial gene products such as receptors for attachment, transcriptional and translational regulators, and other host factors. Mutations in any of these host genes can allow the host to become resistant to bacteriophage infection. Using a genome-wide loss-of-function mutant library of *Klebsiella* sp. M5a1 generated via a random barcode transposon site sequencing system (RB-TnSeq) method, we have identified genes that provide resistance to a group of M5a1-infecting bacteriophages. We are generating resistant M5a1 bacteria by spontaneous mutagenesis and determining if these mutations map onto the genes identified in the RB-TnSeq genetic screen. We have used two phages, KLEB 17 and KLEB 20, to select resistant M5a1 strains. These two phages had very similar RB-TnSeq profiles and we found that the resistant strains showed complete cross-resistance. Recently, updated genomic sequencing showed that these were actually independent isolates of the same phage. A total of 15 resistant strains were isolated, all but one of which displayed complete resistance while the final strain was partially resistant. We designed PCR/sequencing primers to amplify the genes identified in the RB-TnSeq screen that conferred resistance to these two phages. So far, we have completed sequencing of three of the nine target genes in all 15 resistant strains. One gene had no mutations in any of the fifteen resistant bacterial strains. We identified mutations in the two other genes including missense, nonsense and frameshift mutations that might be the cause of the phage resistance. Through this work we may be able to see if the RB-TnSeq genetic screen identifies the locations of all random mutations or if we will find bacteria with resistant mutations in genes that were not identified by the RB-TnSeq screen.

BOARD 5

Patterns in Microbial Production of Phosphonate Natural Products

Jerry J. Cui[1] and Kou-San Ju[1-4]
[1]Department of Microbiology, The Ohio State University
[2]Division of Medicinal Chemistry and Pharmacognosy, The Ohio State University
[3]Center for Applied Plant Sciences, The Ohio State University
[4]Infectious Diseases Institute, The Ohio State University

Phosphonates (Pn) are a class of NPs characterized by a C-P bond. This bond enables mimicry of phosphate esters and carboxylic acids which play essential roles throughout metabolism. The antibiotic fosfomycin, the antiviral foscarnet, and the herbicide glyphosate are all Pn NPs. But despite their structural diversity, the biosynthesis of all characterized Pns begins with the conversion of phosphoenolpyruvate (PEP) to phosphonopyruvate (PnPy) by PepM. This fact also facilitates genome mining for Pn BGCs, as the presence of pepM highlights each cluster. However, these BGCs are often silent, leading to roadblocks in the discovery of novel Pn compounds. **As all known Pn NPs share a metabolic starting point, we hypothesize that there are global patterns in their production which can be observed and applied towards the design of growth media for laboratory production**. To this end, we have cultured a diverse array of bacteria which are known to produce Pn NPs, generated extracts, and quantified Pn production via 31P NMR. Here we show that some strains behave as “generalists,” producing on every media tested, while others are “specialists” which produce only in select conditions. Our initial findings suggest that select media and additives have the best chances of eliciting Pn production from “specialist” strains.

BOARD 6

Identifying the effects of anaerobic exposure on Listeria monocytogenes infection of the central nervous system

Rebecca Rudd[1], and Yvonne Sun[1]
[1]University of Dayton

The foodborne pathogen *Listeria monocytogenes* is capable of crossing the gastrointestinal epithelium and invading macrophages and non-phagocytic cells. As an intracellular pathogen, *L. monocytogenes* replicates inside the host cell cytosol to be transported throughout the bloodstream and avoid any extracellular immune defenses. In this mechanism, the bacteria can reach and cross the blood brain barrier, resulting in bacterial meningitis that can be severe in immunocompromised patients. In this project, the goal of the research is to determine how anaerobic exposure, a typical process during the intestinal phase of infection, affects *L. monocytogenes* invasion of the central nervous system. In the primary aim, Neuro-2A cells, acting as the model host cell for neuronal cells, will be grown and infected with *L. monocytogenes* that will be exposed to anaerobic or aerobic conditions for different lengths of time. The 10403s strain, a neurotropic strain, and a cardiotropic strain will be used to identify strain-dependent variations. The results will show the effect anaerobic exposure has on invasion success and intracellular growth once the bacteria has crossed the membrane of the

cells. From these results, we will identify intestinal conditions that can potentially influence *L. monocytogenes* neural invasion to better understand this particular pathogenic process.

BOARD 7

A Toxin-Antitoxin System Controls Rho Termination During Stress

Mark Finazzo[*1], Bing Wang[1], and Irina Artsimovitch[1]
[1]Ohio State University

Rho, a bacterial termination factor, ensures that useless, toxic, or defective RNAs are not synthesized by RNA polymerase. Rho blocks synthesis of antisense and xenogeneic RNAs, as well as mRNAs that contain early stop codons or ribosome-stalling signals. A recent model in which Rho is recruited to, and travels with, RNA polymerase throughout RNA synthesis explains how Rho selectively targets and surveils the nascent RNAs, triggering premature release of RNAs that are not translated by the ribosome. However, it remains unclear how indiscriminate termination is avoided when translation is inefficient, e.g., during slow growth or under stress. Here, we investigate the mechanism of YaeO (Rof), a small 87-residue protein proposed to inhibit Rho function. We show that Rof reduces Rho-dependent termination in vitro and supports Escherichia coli survival during translational stress, consistent with its proposed role as an antiterminator. However, we also show that the expression of Rof is lethal, unless yaeP is present. The yaeP gene partially overlaps the rof gene and encodes a 66-residue protein of unknown function. The relief of Rof toxicity by yaeP, together with the genomic organization of the yaeP-O locus, suggest that these regulators comprise a toxin-antitoxin pair that modulate Rho-dependent termination. We found that YaeP expression does not block Rof synthesis and that the two proteins do not interact, excluding two of the most common modes of antitoxin action. We are currently investigating how YaeP inhibits Rof and how their expression is controlled.

BOARD 8

Using DMPG as a Possible Treatment for Psoriasis

Mindi Klaus[*1], Wendy Bollag[2], Vivek Choudhary[2\]
[1]Ohio Wesleyan University; [2]Augusta University

Psoriasis is an immune-mediated skin disease that affects a large population of people worldwide. It is characterized by abnormal differentiation and excessive proliferation of keratinocytes, as well as an overactive immune response that causes inflammation. Previously, it was shown that Dioleoylphosphatidylglycerol (DOPG) inhibits toll-like receptors and can reduce inflammation in psoriasis in both an in vitro and an in vivo model. However, DOPG is quite large and does not permeate the skin very easily. This led to the possibility of using an alternative phosphatidylglycerol: Dimyristoyl phospatidylglycerol (DMPG), which is somewhat smaller and more permeable than DOPG. To test this, cells were treated in vitro with a damage-associated molecular pattern (DAMP) called PAM and then DMPG. RNA was isolated, cDNA made, and qPCR ran with inflammatory markers, showing that DMPG similarly inhibits toll-like receptors and decreases psoriasis-like inflammation. The Imiquimod

mouse model was also executed, in which 20 male mice were shaved on their backs and placed into one of four groups: control, DMPG, Imiquimod (IMQ), and IMQ+DMPG. In the morning, each mouse was treated topically with either Vaseline or IMQ, and then in the afternoon, treatment with PBS or DMPG was administered topically. On the seventh day, the animals were sacrificed and the back skin, the right ear, and spleen were harvested. Preliminary results indicate that DMPG was effective in decreasing inflammation and skin thickness in the IMQ model, although some trends did not reach significance, suggesting too low of a DMPG dose was used. The phenotype of the mice improved with DMPG as well. Other data analysis, such as western blotting, is still being completed, but preliminary results so far suggest that DMPG may have the potential to be used in treating psoriasis in a clinical setting.

BOARD 9

Determining the Ability of *ccr5 delta 32* to Influence Expression of Cell Surface Co-receptors *in vivo* HIV-1 Infection

Tatiana A. Fuentes*[1,2] Lyndsy R. Hamilton[1,2], Juliana J. Seder[1,3], Eva M. Wanek[1], Arianna E. Diaz[1], Aliya Z. Ali[1], Mehdi M. Ali[1] Kristin Padgett[1,4], Mia Diaz[1], Noor Deif[1,2], Mariana A. Heru[1,2], Yaw Agyemang[1], Mallory N. Alvarez[1,2], Gary R. Dodson[1], and Harry W. Kestler[1]

Lorain County Community College[1], Early College High School[2], Cleveland State University[3], Bowling Green State University[4]

AIDS viruses such as HIV-1 require two receptors to enter a cell. The principal receptor CD4, as well as one of two co-receptors, CCR5 or CXCR4, are required for HIV-1 infection. A mutation in *ccr5*, known as *ccr5 delta 32*, has been shown to confer resistance to viral infection. That mutation, a deletion of 32 base pairs, appears to alter the cell surface expression of the co-receptors CCR5 and CXCR4.

The ability to down-modulate co-receptors by the delivery of lentiviral particles containing *ccr5 delta 32* will be determined. A Lentiviral vector system was employed to construct viral particles that contained either *ccr5* wild-type or *ccr5 delta 32*. HEK293-ft served as a packaging cell line by co-transfecting three plasmids: pLentiPuroHA-ubiquitin, psPAX2 that encodes HIV metabolic genes, and pMD2.G containing an envelope gene for Vesicular Stomatitis Virus. Cells were cultured for three days, and retroviral particles were harvested.

Stable T-cell lines will be constructed by transducing various retroviral particles containing *ccr5 wild-type* or *ccr5 delta 32* into Jurkat, H9, CEMX174 cells. Flow cytometry and immunofluorescence will be employed to determine the surface expression of CCR5 and CXCR4 on these cells. A novel *ccr5 delta 33* gene with an internal 11 amino acid deletion at the same location as CCR5 delta 32 has been designed. The ability of this protein to bind natural ligand and confer resistance to HIV infection will be determined.

Three individuals have had their HIV reduced to undetectable levels through a bone marrow transplant from *ccr5 delta 32* homozygous donors. This treatment lacks wide accessibility due to its high-risk nature and compatible donor availability. Lentiviral particles that modulate cell infectibility have the potential to treat many patients without the need for donor histocompatibility or the destruction of existing immune cells.

BOARD 10

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) - non-structural protein 3 suppresses the pro-inflammatory cytokines in host cell

Andrew Villasenor*[1], Tooba Momin[1], Mrigendra Rajput[1]
[1]Department of Biology, University of Dayton, Dayton OH

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is an enveloped, single-stranded positive-sense RNA virus with genome size ~30 Kb. SARS-CoV-2 genome encodes13–15 open reading frames (ORFs). Among them ORF1a encodes a non-structural protein 3(Nsp3). Nsp3 is one of the largest proteins encoded by virus with an average molecular mass of about 200 kD. Nsp3 plays an important role in virus replication which includes creating virus replication/transcription complex and processing viral proteins. Nsp3 with papain-like protease activity cleave Nsp1-Nsp2, Nsp2-Nsp3 and Nsp3-Nsp4 in viral polypeptide. However, there is very little known about the role of Nsp3 on host innate immune response. To determine the role of SARS-Cov2- Nsp3 on host innate immune response, we transfected the HEK-293 Cell with plasmid containing SARS-CoV2-Nsp3 gene with green fluorescent protein (GFP). Transfected cells were analyzed for interleukin 6 (IL-6) and tumor necrosis factor alpha (TNF alpha) expression. Our result showed that SARS-Cov2-Nsp3 significantly reduced IL-6 and TNF alpha expression, indicating the role of SARS-Cov2-Nsp3 in virus induced immunosuppression. However, we need to confirm our results with more replicates.

BOARD 11

Cataloging of Environmental Isolates at Hikma Pharmaceuticals

Madeline Russell*[1]
[1]Ohio Wesleyan University

The manufacturing and packaging environment in which various pharmaceutical products are produced is strictly controlled since it could have a direct impact on the final product and subsequently, for the consumer. Environmental monitoring of the manufacturing and packaging areas allows for a check that ensures that the areas are in a state of control and ultimately, the safety of the consumer using the product. Having a catalog of microorganisms will assist in determining normal microbiological flora in these areas. This will ultimately aid in a better control strategy for these environments. Through the course of the research, multiple samples were collected from several different areas across Hikma's facilities. Samples were grown on either Dey-Engley neutralizing agar from a solid surface or grown on a Tryptic Soy agar plate using an air sampler. Once the isolates had grown, the microorganisms were subcultured using a sterile loop and streaked for isolation onto Tryptic Soy agar plates. These

plates were incubated at 30-35◦C for 24-48 hours. After this, the isolates were transferred to test cards containing a variety of nutrients, antibiotics, and other compounds. These test cards were incubated at 30-35◦C for 24 hours. After incubation, the test cards were loaded into a computerized identification system, their data was input into the software, and an identification was generated. If no identification was generated, the plates were either allowed to incubate an additional 24 hours or tested again utilizing a different inoculating fluid medium. All identifications were recorded by area and a final report summarizing the findings of the facility microbiome was produced.

BOARD 12

Ecology of Quaternary Amine Dependent Methanogenesis

Cole C. McKishen, **Powell T***., Ferguson D.J.
Miami University

Leading climate scientists collectively agree that methane is a significant contributor to global climate change. Methane is primarily produced by microbes called methanogens in anoxic soil sediments, animal guts, and other extreme locations. However, the effects of this production are not fully understood and in particular a newly identified group of substrates for some methanogens, the quaternary amines (QAs), are not well understood. QAs are naturally present in soil sediments and include tetramethylammonium (QMA), glycine betaine (GB), choline, and carnitine. As byproducts of biological activity, these compounds are present in the environment, leading to the belief that the production of methane from these compounds may contribute significantly to global methane production. For this study, sediment samples were collected from different locations with varying salinity concentrations along the Crystal River in Florida ranging from freshwater to marine. The samples were used to inoculate enrichment cultures in the lab under anaerobic conditions with the control substrates trimethylamine and methanol or a quaternary amine substrate QMA or GB. The enrichment cultures were monitored for growth by turbidity and methane production. All of the enrichments showed growth and methane production, regardless of growth substrate or salinity of the sample site, indicating that the QA enrichments were successful and suggesting that QA-dependent methanogenesis occurs in freshwater, brackish, and marine sediments. Genomic DNA was extracted from the enrichment cultures to determine the relative percentage of the methanogens in the overall population in the enrichment cultures via 16S rDNA next-generation sequencing. The 16S sequencing showed many of the samples to be significantly enriched with methanogenic organisms. We identified one particular methanogen, *Methanosalsum zhilinae*, dominated most enrichments across samples and substrate conditions. Metagenomic sequencing will be conducted to get strain level data and genomic sequence of methanogens in the sample.

BOARD 13

Characterization of a Potential Chromosome Segregation Gene in *Streptomyces coelicolor*

Olivia Brickey[*1], Dr. Jennifer Bennett[2]
Otterbein University

Streptomyces coelicolor is a gram-positive bacterium located in the soil, commonly used to study antibiotic production and as a model for multicellular prokaryotic development. *S. coelicolor* has a complex life cycle, forming filaments with multi-genomic compartments leading to single linear chromosomes within mature spore compartments. In this bacterium, chromosomes segregate through the ParABS system. ParA and ParB proteins organize subcellular space to play a role in segregation. While ParAB plays a major role in chromosome segregation, mutations of each gene only contributed to small phenotypic growth changes. Using transposon mutagenesis, a novel mutant was previously identified in our lab with a chromosome segregation defect using propidium iodide staining with fluorescence microscopy. The mutated gene resulted in 22% anucleated spores compared to the less than 1% anucleated spores found in wild type. Extending this study into another species, propidium iodide staining with fluorescence microscopy was used on an *S. venezuelae* single deletion strain for the novel gene ortholog and a double deletion mutant of the novel gene ortholog and adjacent gene positioned in a potential operon. Preliminary results showed the *S. venezuelae* single mutant and the wild type had 0.78% anucleated spores, whereas the double mutant had 1.18% of anucleated spores. Both mutants also displayed shorter sporulated chains in comparison to wild type *S. venezuelae*. Currently, the novel gene is being deleted in *S. coelicolor* by using REDIRECT. The deletion mutant will be compared to the wild type and transposon insertion strains. This study will allow us to determine if the identified gene is the cause of the *S. coelicolor* chromosome defect or whether the strain should undergo whole genome sequencing to identify the gene of interest. In addition, the deletion of the novel gene will lead to further understanding of the chromosome segregation process in filamentous bacteria.

BOARD 14

Atypical Key Enzymes in Phosphonate Natural Product Biosynthesis

Ivan Vore*[1], Jerry Cui[2], Kou-San Ju[2]
[1]Ohio Wesleyan University, [2]The Ohio State University

Phosphonate natural products are antimetabolites defined by their carbon-phosphorus bond. This class of compounds exhibits cytotoxic properties by mimicking common molecules in essential metabolism, making them potent antibiotic, herbicidal, antifungal, and anticancer compounds. The mining of genomic data for phosphonate natural products in microorganisms has provided us with a rich source of newly available compounds in medicine and industry. Genome mining for phosphonate natural product biosynthetic gene clusters (BGCs) is conducted by locating the protein-coding gene *pepM*. The product of this gene, phosphoenolpyruvate mutase (PepM) is responsible for the first step of all known

phosphonate biosynthesis: creating the carbon-phosphorus bond that defines all phosphonates. Our analyses of microbial genomes have revealed potential homologs of PepM that lack a defining motif, which has been utilized to distinguish the phosphonate-forming PepM enzyme from ancestral relatives. Yet, their encoding biosynthetic gene clusters contain other known genes for phosphonate biosynthesis within their neighborhoods, suggesting they may indeed catalyze the formation of the carbon-phosphorus bond. Here, we test this hypothesis by examining the biochemical function of two atypical PepM homologs from *Mesorhizobium sanjuanii* and *Streptacidiphilus rugosus*. Understanding the function of these two enzymes will lead to a greater definition of phosphonate biosynthesis and potentially expand the number of pathways for this family of natural products.

BOARD 15

Environmental *Pseudomonas* inhibit multi-drug resistant human and animal pathogens

Jeremiah Adesanya* and Hans Wildschutte
Bowling Green State University

Bacterial pathogens are evolving resistance to all available antibiotics and predicted to contribute to over 4 million deaths worldwide. Thus, the discovery of new antibiotics that inhibit multi-drug resistant (MDR) pathogens is urgent. We hypothesize that environmental *Pseudomonas* strains that can inhibit the growth of MDR pathogens may be source of novel antibiotics and have new or multiple mechanisms of action. We isolated 288 environmental *Pseudomonas* strains from the Portage River in northwest Ohio, which represents a polluted water source. *Pseudomonas* strains were isolated and phylogenetically characterized using the *gyrB* housekeeping gene. To test our hypothesis, the environmental strains were competed against a collection of human and animal pathogens including nine MDR *Burkholderia* species, seven methicillin resistant *Staphylococcus aureus*, and nine MDR *P. aeruginosa* strains, all isolated from lungs of cystic fibrosis patients, and five *Aeromonas* fish pathogens. Results from the 8,640 individual antagonistic assays showed that 115 of the environmental *Pseudomonas* strains demonstrated ability to inhibit at least one pathogen. This suggests that these environmental strains may produce novel compounds which effectively inhibits these MDR pathogens. To identify genes involved in antibiotic production, transposon mutagenesis, genome sequencing and bioinformatic tools will be used to identify specific loci involved in the compound production. Genetic analysis will provide insights on the gene region and predicted novel compounds will be purified and characterized. The overall goal of this work is to identify novel antimicrobial compounds.

BOARD 16

Exploration of Neisseria colony morphologies following transposon mutagenesis of a macaque-derived Neisseria species

Jacob Thompson*[1], Lorna Vizmeg[1], Eliza Thapa[1], Don Holzschu[1], and Nathan Weyand[1, 2, 3]
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Isolate AP312 was cultured from a rhesus macaque bite wound. Phylogenetic analysis of AP312 and additional monkey-derived *Neisseria* isolates indicate that AP312 likely represents a new commensal species in the genus. The isolate was found to form wrinkled colonies upon blood agar. We made a transposon library to search for mutant strains with altered colony morphology and transposon insertion sites were identified using inverse PCR and Sanger sequencing. Our poster will review the classes of colony morphology mutants that have been identified in addition to transposon insertion sites in some mutated genes identified thus far. For example, some colonies appear smooth, or manifest weaker wrinkling compared to the parent strain. Some of the mutant strains have transposon insertion sites in genes linked to polysaccharide synthesis or export. Future efforts will assess mutant strains for *in vitro* phenotypes including biofilm formation and exopolysaccharide secretion. Several of the mutated genes we have identified are conserved in pathogenic *Neisseria* species. Genetic analysis of colony morphology mutants in isolate AP312 provides a tool for identifying the function of orthologs of factors that have not been studied in great detail in the pathogenic *Neisseria* species.

BOARD 17

The Impact of Phosphate Limitation on *Pseudomonas aeruginosa* Phage Infection

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Phage therapy is a promising tool to combat rising antibiotic resistant infections, and uses bacteriophages (phages), viruses that infect bacteria, to treat infections. Most research on phage infection is done under nutrient abundant conditions, but in the environment, nutrients are commonly limited. In natural and clinical settings where *Pseudomonas aeruginosa* is pathogenic, phosphate is commonly found to be a limiting resource. Due to the necessity of phosphate for microbial survival, *P. aeruginosa* has been shown to increase its virulence and exhibit altered behavior under phosphate stressed conditions. Despite these characterizations, it remains unclear how phosphate limitation impacts the interactions between *P. aeruginosa* and its bacteriophages. Here we used PhiKZ and PEV2, two phylogenetically distinct phages, to examine the impact of phosphate limitation on phage infection by measuring the rate of adsorption, length of infection, and burst size in phosphate

replete and phosphate deplete conditions. Both phages exhibited a longer latent period and a shorter burst period under limited phosphate. There was no statistically significant difference in the adsorption rate and the burst size between phosphate replete and phosphate deplete conditions for both phages. This study aimed to explore how phosphate limitation impacts the interactions between *P. aeruginosa* and its relevant phages, to not only characterize phage-host interactions under phosphate-limited conditions, but to provide foundational information for the application of phage therapy on *P. aeruginosa* and two of its relevant phages.

BOARD 18

Characterization of the effects of IP6 on Jaagsiekte Sheep Retrovirus (JSRV) assembly

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Jaagsiekte Sheep Retrovirus (JSRV) is a retrovirus that causes ovine pulmonary adenocarcinoma. As for other retroviruses, the structural protein of JSRV is Gag, which is a multi-domain protein. In assembly of a virus particle, several thousand molecules of Gag come together to form the immature lattice. During the maturation process of JSRV, the viral protease cleaves the Gag on the interior side of the viral membrane releasing the CA subdomain, which is necessary for formation of an infectious virus. Studies of HIV-1 showed that the lattice structures formed by Gag and by the released CA domain differ from each other, but less is known for JSRV. To better understand the transition between the two distinct lattices, we wish to determine the structure of the immature lattice of JSRV, which can be formed *in vitro* from a truncated Gag protein including CA and the adjacent NC domain. And we want to understand how cofactors like inositol hexakisphosphate (IP6) affect maturation of the CA lattice. We used the following approaches to address these two unknowns. To study the JSRV CA-NC lattice assembly *in vitro*, we introduced an expression plasmid encoding CA-NC into *E. coli*, purified the translated immature CA-NC protein, incubated the protein under conditions where it will form immature virus-like particles, and then will solve the structure of the lattice utilizing single particle cryo-electron microscopy. To study the role of IP6 on JSRV assembly, budding, and maturation *in vivo*, we transfected a plasmid expressing Gag into both wild-type and IP6-deficient mammalian cells and measured the release of virus particles from cells and the level of maturation by immunoblotting. Attempts for both the structural study of the JSRV immature lattice and the role of IP6 on JSRV assembly are currently underway.

BOARD 19

Impact of oxidative stress on the community structure of ammonia-oxidizing enrichment cultures

Madelynn D. Spencer*, Madisen P. Kimbrel, and Annette Bollmann
Miami University

Ammonia-oxidizing bacteria (AOB) carry out the first step of nitrification. In nature, these bacteria are found in communities with heterotrophic microorganisms. Oxidative stress due to reactive oxygen species can cause damage to DNA, proteins, and lipids in the cell. Proteomics studies showed that the presence of heterotrophic bacteria reduces the oxidative stress for AOB. We investigated the effect of chronic hydrogen peroxide exposure on the heterotrophic community in two AOB enrichment cultures. The AOB enrichment cultures were obtained from Lake Acton, an eutrophic freshwater lake in Ohio and contained one AOB and a community of heterotrophic bacteria. The cultures were exposed to varying concentrations of hydrogen peroxide for up to six growth cycles. Growth and activity of AOB was monitored by measuring nitrite production. Samples for molecular analysis were collected and the community composition was determined by 16S rRNA amplicon sequencing. Exposure to hydrogen peroxide increased the lag phase of the AOB and changed structure of the heterotrophic community in the enrichment cultures. *Pseudoxanthomonas* sp, and *Bosea sp.* increased in abundance while *Azospirillum* sp. decreased in abundance. The largest changes in the heterotrophic community structure were observed when the culture was incubated in the presence of high hydrogen peroxide concentrations. Removing hydrogen peroxide stress resulted in a reduction of the lag phase and a partial recovery of the heterotrophic community.

BOARD 20

CodY is required in enhancing LLO production by anaerobic propionate treatment in *Listeria monocytogenes*

Allison Herceg*[1], and Yvonne Sun[1]
[1]University of Dayton

Listeria monocytogenes is a prevalent food-borne pathogen, and a clear understanding of its pathogenesis and virulence gene regulation can enhance our capability to prevent and treat infections. *L. monocytogenes* is ingested through contaminated foods, enters the intestinal lumen, and can spread throughout the rest of the body as an intracellular pathogen. The intracellular life cycle of *L. monocytogenes* requires the regulated expressions of a variety of virulence genes. We previously found that exposure to short chain fatty acids (SCFAs), fermentation byproducts present in the intestines, resulted in significant changes in *L. monocytogenes* pathogenesis. More specifically, we reported that aerobic propionate treatment resulted in a significant down regulation of the toxin listeriolysin O (LLO) production while anaerobic propionate treatment resulted in a significant up regulation of LLO production. To further investigate the mechanism underlying *L. monocytogenes* response to SCFAs, this study explored the role of CodY, a transcription factor in response to levels of

branched chain amino acids, in the opposing effects of propionate on LLO production. While the pathogen spreads to tissues outside of the intestines, it resides in macrophages and travels through the bloodstream. Using strain 10403s and a mutant with a *codY* gene deletion (Δ*codY*), culture supernatant LLO activity was determined with hemolytic assays. We found that while the suppression effects of aerobic propionate treatment on LLO production was observed in the Δ*codY* mutant, the enhancement effects of anaerobic propionate treatment were not. These results suggest that CodY is required for mediating the effects of anaerobic propionate treatment on LLO production. Moreover, these results suggest that anaerobic propionate treatment could potentially alter the regulation of branched chain amino acid synthesis.

BOARD 21

TOR-Mediated Regulation of Enolase in Encystment in *Naegleria*

Anushkaa Parwade[*1], Cassie Gohar[1], Kyla Doan[1], Keen Wilson[1], and Yoshi Odaka[1]
University of Cincinnati

Naegleria fowleri is a pathogenic unicellular protist free living ameba commonly found in warm freshwater. Depending on the environment, it undergoes three stages of life cycle - trophozoite (infectious), flagellate (motile), cyst (dormant). The trophozoite form infects the central nervous system by entering the brain through the olfactory nerve in the nasal cavity, causing primary amoebic meningoencephalitis (PAM). The infection is rapid and fatal, with a mortality rate of 97%. Our central idea is to promote cyst formation in Naelgeria, followed by targeting drugs to treat PAM. In this study, regulatory role of target of rapamycin (TOR) kinase in Naegleria gruberi, a non-pathogenic species, was assessed. Phosphoproteomics analysis identified phosphorylation of glycolytic enzyme enolase and enolase-associating proteins when TOR was activated, making it an attractive candidate as a downstream target of TOR. Therefore, GFP-tagged *Naegleria* enolase was engineered and expressed in N. gruberi and murine MK4 cells, and subsequently, point mutations were introduced to the TOR-sensitive phosphorylation sites. We are currently investigating the effects of the mutations on enolase subcellular localization.

BOARD 22

Testing aspartate availability in the *Histoplasma*-containing phagosome of macrophages

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Histoplasma capsulatum can survive and proliferate inside the phagolysosome of macrophages, meaning the phagolysosome must have a utilizable carbon. Earlier studies suggest intracellular *Histoplasma* catabolizes amino acids as the carbon source within macrophages. The expression and specificity of amino acid transporters by the pathogenic yeast form of *Histoplasma* suggest aspartate could serve as the intracellular carbon source. To functionally test whether aspartate is available in the phagolysosome, we will test whether

an aspartate auxotroph can proliferate after infection of macrophages. *Histoplasma* has three proteins predicted to be aspartate aminotransferases (AATs). Deletion of the mitochondrial AAT, which causes aspartate auxotrophy in *Saccharomyces*, did not cause aspartate auxotrophy in *Histoplasma*. We created a triple AAT mutant strain using both CRISPR-mediated gene deletion and RNAi systems. Loss of all three AATs functions did not cause an aspartate auxotrophy *in vitro*. Using a biochemical assay, we found the triple AAT mutant has approximately 23% of AAT activity as wild type. This suggests either the RNAi system is not sufficiently depleting all AAT activity or another aminotransferase beyond the three identified by bioinformatics contributes to the synthesis of aspartate. To test this, we are generating deletions in all three genes encoding the predicted AATs.

BOARD 23

Examining the mechanism by which IFITM3 enhances SARS-CoV-2 infections

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Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infections remain a global health issue, and a better understanding of the virus-host interaction is needed. Interferon induced transmembrane protein 3 (IFITM3) is a cellular protein that inhibits virus-to-cell membrane fusion reactions. We found that IFITM3, which primarily localizes to endosomes and lysosomes due to the presence of a tyrosine-based sorting motif, inhibits SARS-CoV-2 infections of cells. However, IFITM3 mutants that lack the sorting motif and accumulate at the plasma membrane enigmatically enhanced SARS-CoV-2 infection. We further confirmed enhancement of infection by IFITM3 occurring at the plasma membrane by utilizing a SARS-CoV-2 plasma membrane viral fusion inhibitor. To explore this infection enhancement effect further, we engineered mutants of IFITM3 at specific functional regions of the protein. We observed that S-Palmitoylation is not required for endosomal inhibition of SARS-CoV-2 infection but is required for the plasma membrane enhancement of infection. Similarly, the C-terminus of IFITM3 is not required for the endosomal inhibition of infection but is required for the plasma membrane infection enhancement. Investigating this C-terminal region further, we found that its hydrophilic residues were not required for either enhancement or restriction activities. Furthermore, a previously described phenylalanine-based trafficking motif is not required for endosomal inhibition but is required for the plasma membrane enhancement of SARS-CoV-2 infections. In contrast, its GxxxG dimerization motif is not required for endosomal inhibition or plasma membrane enhancement of SARS-CoV-2. Overall, we have identified important functional regions of IFITM3 that contribute to its SARS-CoV-2 infection enhancement versus inhibition activities.

BOARD 24

A Natural Mouse Model for Neisserial Transmission in the Upper Respiratory Tract

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Gonorrhea is considered the second most prevalent sexually transmitted bacterial infection worldwide and is caused by *Neisseria gonorrhoeae* (Ngo). Ngo frequently colonizes asymptomatically the mucosal surfaces of the genital tract, rectum, and pharynx. However, mechanisms of gonococcal transmission between hosts are poorly understood. Studies of *in vivo* transmission of Ngo are challenging due to a lack of natural animal models. We have developed the first murine model of *Neisseria* transmission using the model organism *Neisseria musculi*. We aim to use this model to identify and study neisserial persistence and transmission factors. *In vivo* models are needed to develop approaches to combat reservoirs of *Neisseria* that contribute to persistence and dissemination of disease and antimicrobial resistance. *N. musculi* is a commensal of wild-caught house mice that encodes many orthologs of Ngo virulence factors. For this study we evaluated the ability of *N. musculi* to transmit between colonized and uninoculated laboratory mice. Transmission was detected in two inbred mouse strains, A/J and C57BL/6, following single oral inoculations. Mice were inoculated and three weeks later co-housed with uninoculated mice. Transmission to uninoculated mice was detected as early as one week after co-housing. Transmission kinetics were found to differ depending upon which colonized mouse strain was co-housed with uninoculated mice. We have established a rodent model capable of investigating factors that influence neisserial transmission. Mutation of host and bacterial genes will allow future investigation of molecular determinants that influence transmission between hosts.

BOARD 25

The transcription factor CodY is involved in mediating the effects of aerobic, but not anaerobic, propionate treatment on *Listeria monocytogenes* entry and survival in macrophages

Natalie Narcelles[1] and Yvonne Sun[1]
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Listeria monocytogenes is an intracellular pathogen that can replicate inside macrophages. We previously reported that propionate pretreatment of *L. monocytogenes* could modulate subsequent infection outcomes. To better understand the underlying mechanisms of this propionate response, we compared intracellular infections of wildtype strain 10403s and the isogenic *codY* deletion mutant in RAW264.7 macrophages. Using the gentamicin protection assay, we noted that at two hours post infections, propionate pretreatments did not affect WT *L. monocytogenes* entry and survival. However, aerobic pretreatment of the *codY* mutant significantly reduced entry and survival in macrophages. These data suggest that the transcription factor CodY can mediate *L. monocytogenes* aerobic response to propionate.

Moreover, because CodY activity is mainly regulated by the levels of branched chain amino acids (BCAAs), aerobic propionate exposure in *L. monocytogenes* may alter BCAA levels.

BOARD 26

Characterization of an *FtsQ-Null* Suppressor Gene in *Streptomyces coelicolor*

Feng-Thea Lee*, Alexandra Sherman, Jennifer Bennett
Otterbein University

Streptomyces coelicolor is a Gram-positive filamentous soil-dwelling bacterium. Its growth is similar to that of fungi. *S. coelicolor* grow using a mycelium-like structure which produces aerial hyphae above the media surface for sporulation. During cell division of these aerial filaments, evenly-spaced crosswalls are developed using division genes, including *ftsZ* and *ftsQ*. These genes are essential for growth in common bacteria. However, if these genes are silenced or deleted in *Streptomyces*, these bacteria will retain their ability for growth. When *ftsZ* or *ftsQ* are deleted in *Streptomyces*, it causes a loss of septum formation in the aerial hyphae, and therefore a loss in spore formation, which can be visualized under the microscope.

Previous research performed in this project has contributed to the discovery and characterization of three new ftsQ-null suppressor strains using visual phenotyping and bioinformatics. It was discovered that all three strains contained a mutation within the same gene, *sqnA* (suppressor of *ftsQ-null*). Bioinformatic databases were used to determine that the gene encodes a histidine kinase and is located next to a gene encoding a response regulator.

S. coelicolor deletion strains for these genes of interest are being constructed using the Lambda REDIRECT recombinase system in both the wild type and *ftsQ*-null mutant background. We have successfully designed long PCR primers for the gene disruption process. We are currently working on introducing the *S. coelicolor* cosmid clone into *E. coli* BW25113/pIJ790 with electroporation. Further research involving the deletion and overexpression of this gene will produce more novel information on this newly discovered gene and our understanding of the bacterial cell division process. Once these strains are successfully constructed, they will be compared phenotypically on plates with different media types and by using phase-contrast microscopy.

BOARD 27

Natural antibodies mediate protection against *A. baumannii* infections

Aminul Islam*, Luis A. Actis, Timothy J. Wilson
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Acinetobacter baumannii, an opportunistic human pathogen, causes a range of infections with ventilator-associated pneumonia being the most common and severe form of infection. Treatment of *A. baumannii* infections is challenging due to the emergence of pan-drug resistant strains resulting in increased morbidity and mortality. There is an urgent need to

develop alternative therapeutics in clinical management of *A. baumannii* infections and for this purpose understanding the host immune responses against this pathogen is crucial. However, host immune responses, especially the humoral immune response to *A. baumannii* infections, is poorly understood. This study aimed to investigate lymphocyte-mediated innate immune resistance to *A. baumannii* pulmonary infection using B- and T-cell deficient (*Rag2*$^{-/-}$) C57BL/6 mice. Our results suggest that intranasally infected *Rag2*$^{-/-}$ mice are impaired in clearing *A. baumannii* from lung, liver, and spleen at 24 hours post infection (hpi) compared to WT mice. Because 24 hours are not sufficient to induce adaptive immune response, we hypothesized that natural antibodies (NAb) are required to protect against *A. baumannii* infection at 24 hpi. Our study showed that animal pretreatment with normal mouse serum or purified NAbs rescue *Rag2-/-* mice from infection. Analysis of C3 complement protein binding by flow cytometry demonstrated that NAbs induce increased deposition of C3 protein on *A. baumannii,* indicating activation of the classical complement pathway. Furthermore, our study indicates that NAb-mediated increased deposition of C3 on *A. baumannii* promote phagocytosis by murine macrophages. Overall, our study indicates protective function of NAbs against *A. baumannii* pulmonary infection in murine model. Results from this study provide novel information regarding the involvement of natural antibody-mediated innate immune response against *A. baumannii*, an observation that may lead to the development of effective therapies against *A. baumannii* infections.

BOARD 28

Assembly and screening of nitrogenase-like gene clusters of unknown function for volatile organic sulfur reduction and hydrocarbon synthesis

Elizabeth A. Morgan[*1] and Justin A. North[1]
[1]The Ohio State University

Under anaerobic, sulfate limiting conditions some organisms turn to volatile organic sulfur compounds in the environment for needed sulfur acquisition. We recently discovered that this process can be performed by previously unidentified relatives of nitrogenase that we call methylthio-alkane reductases. These nitrogenase-like enzymes reduce volatile organic sulfur compounds into biologically available sulfur for methionine synthesis and hydrocarbons. For example, dimethylsulfide is reduced to the potent greenhouse gas, methane, and 2-methylthioethanol to the key plant regulatory hormone, ethylene. We initially discovered the methylthio-alkane reductases in the photosynthetic bacterium, *Rhodospirillum rubrum*. However, upon further phylogenetic analysis, gene homologs were identified in numerous additional bacteria. To explore the diversity of bacteria with methylthio-alkane reductase activity and the role it may play in each organism's respective environment, we used a gene synthesis and complementation approach in *R. rubrum*. Using homology searches of genome and metagenome databases, our collaborators mined for nitrogenase-like gene clusters including methylthio-alkane reductases, which were then synthesized by the Joint Genome Institute. We then introduced these synthesized genes via a plasmid into an *R. rubrum* methylthio-alkane reductase gene deletion strain. Growth experiments in the presence of different volatile organic sulfur compounds were performed and hydrocarbon production was quantified by gas chromatography to measure methylthio-alkane reductase

activity. These activity screens revealed that sequences with methylthio-alkane reductase activity for known volatile organic sulfur substrates are localized to the nitrogenase-like clade IV-C. Additionally, a gene cluster from *Siculibacillus lacustris* belonging to a different clade resulted in methane production from a currently unknown substrate. Moving forward, additional methylated compounds present in the environment will be explored to understand the functional diversity of nitrogenase-like enzymes. In addition, identified methylthio-alkane reductase sequences with high activity for ethylene production will be further employed in bioengineering efforts to produce large yields of ethylene for bioplastic synthesis.

BOARD 29

Exploring Protein Interactions Within the Cyclic di-GMP Pathway of *Streptomyces*

Mason Nolan[*1], Evan Shelton[2], Jennifer A. Bennett[3]
Otterbein University

The species *Streptomyces coelicolor* is a member of a genus that is particularly interesting for antibiotic research. *S. coelicolor,* when exposed to stimuli, enacts the use of a second messenger, called cyclic di-GMP, which signals for the natural production of antibiotics, biofilm formation, life cycle progression, and several other major functions within the cell. It is believed that the signaling within bacteria is largely dependent on the local concentration of cyclic di-GMP. Effective elimination of phosphodiesterases and diguanylate cyclases has been observed to greatly affect the cells of *E. coli* without changes in the level of cyclic di-GMP present. The significant proteins for this study are the cyclic di-GMP phosphodiesterases, RmdA and RmdB.

The major purpose of this research is to isolate and test for interactions between the two major cyclic di-GMP phosphodiesterases (RmdA and RmdB). Addressing the biochemistry focus of this project, these proteins have been overexpressed within *E. coli*. From there, purification of individual proteins will allow for the testing of interactions among RmdA with itself, RmdB with itself, and between the two using Surface Plasmon Resonance (SPR). The other avenue of this investigation involves a Bacterial Two-Hybrid system, to further confirm these findings and serve as a more visual representation of protein interaction. Preliminary SPR experiments show an interaction between RmdB and itself, suggesting homodimer formation is important for protein function.

There is still much to understand about the functioning of the cyclic di-GMP pathway in *Streptomyces*. This project seeks to discover the relationships between cyclic di-GMP metabolizing proteins to further build understanding of the *S. coelicolor* cyclic di-GMP interactome.

BOARD 30

Architecture and assembly determinants of *Bordetella pertussis* biofilms on primary human airway epithelium

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Resurgence of pertussis in many countries is largely the result of asymptomatic infections. Majority of individuals who contract pertussis are either asymptomatic or show very mild symptoms. We propose that biofilms enhance the survival, prolong the human carriage, and facilitate transmission of *Bordetella pertussis* (*Bp*). Compared to abiotic surfaces, the mechanisms that drive bacteria to form biofilm, the biofilm architecture, and matrix composition in humans are ill-defined. There is a critical need to study biofilms in model systems that replicate the human environment.

Well-differentiated primary human bronchial epithelial cultures (HBECs) grown at the air-liquid interface were infected with the wild-type strain (Bp536), and its mutant-derivatives (lacking production of Bps polysaccharide, filamentous hemagglutinin, and adenylate cyclase toxin). Scanning electron microscopy and confocal laser scanner microscopy were utilized to analyze biofilm formation and the extracellular matrix (ECM) composition. Biofilm parameters e.g., particle size, biomass, thickness, and roughness coefficient were determined.

BOARD 31

Restraining Rho termination during stress

Irina Artsimovitch*[1], Nelly Said[2], Bing Wang[1], Tarek Hilal[2], and Markus C. Wahl[2]
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In *Escherichia coli*, RNAs that are not protected by ribosomes or antitermination complexes are prematurely terminated by Rho, a hexameric ATP-dependent RNA helicase. As a genome sentinel, Rho ensures that only "worthy" RNAs are transcribed: Rho blocks synthesis of antisense and xenogeneic RNAs, as well as mRNAs that contain early stop codons or ribosome-stalling signals. A model in which Rho rides along with elongating RNA polymerase explains how Rho selectively targets and surveils the nascent RNAs, but raises a key

question: how is indiscriminate termination avoided during translational stress or even slow growth? Rho cellular levels are constant, suggesting that Rho activity is modulated. Since *rho* is essential, we selected for partial-loss-of-function mutants that enable expression of *waa*, one of the most tightly silenced *E. coli* operons. We identified two mutants in a flexible linker that connects the N-terminal RNA-binding domain to the C-terminal, RecA-type motor domain of Rho. In these mutants, substitutions of adjacent Gly residues for Asp lead to termination defects *in vivo* and *in vitro* and, most remarkably, promote formation of long helical filaments that resemble those of RecA. Filamentation is a widespread mechanism of adaptation to stress in all domains of life, and we hypothesize that wild-type Rho may form hibernating filaments in response to yet-unknown cellular cues.

BOARD 32

Deep Genome Annotation to Understand an Under-studied Pathogen & to Train Undergraduates

Brad Goodner*[1], Derek Wood[2], Katie Houmiel[2], & Genetics Course Students 2010-2020[1]
[1]Hiram College, [2]Seattle Pacific University

Genomics and bioinformatics are now key areas of conceptual understanding and technical skill used throughout biology, but especially in microbiology. For the past 15 years, we have been involved in developing a free Web-based platform for undergraduate annotation of microbial genomes (IMG-ACT morphing into GENI-ACT) and training programs to help faculty bring genomics and bioinformatics into their courses. As one example of what is possible using this strategy, undergraduates at Hiram College have been involved for the past decade in deeply annotating the genome of *Streptobacillus moniliformis* strain DSM 12112, first sequenced by the Joint Genome Institute back in 2010. This member of the division Fusobacteria is the cause of rat bite fever in the Western Hemisphere and there is very little lab-based information available on the pathogen and its virulence mechanisms. Students working in groups and as individuals have verified the presence/absence of biochemical pathways, searched for homologs to known virulence proteins from other pathogens, and annotated genes suspected of origin via horizontal gene transfer as well as genes unique to the genus. We have found that *S. moniliformis* has lost the ability to make de novo any of its own amino acids, pyrimidine nucleotides, and most vitamins. It appears to scavenge nutrients from host tissues using a wide variety of secreted proteases and other degradative enzymes that target components of the mammalian extracellular matrix. The pathogen has multiple ways of counteracting the mammalian immune system including IgA proteases. There are several large multi-gene families, most of which encode outer membrane proteins that might as adhesins or other virulence determinants. All of this in a genome encoding roughly 1500 proteins.

BOARD 33

Disruption of peroxisome function reduces iron scavenging and virulence in the human fungal pathogen *Histoplasma capsulatum*

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Department of Microbiology, Ohio State University

The fungal pathogen *Histoplasma capsulatum* causes pulmonary and systemic disease among both immunocompromised and immunocompetent individuals. During infection, *H. capsulatum* is found almost exclusively inside the phagosomal compartment of macrophages. A forward genetic screen for *H. capsulatum* mutants which were unable to replicate in macrophages identified four peroxisome-associated genes (*PEX5, PEX10, PEX11, PEX33*) suggesting an important role for fungal peroxisomes in *H. capsulatum* virulence. Peroxisomes are organelles of nearly all eukaryotic organisms, and house enzymes for beta-oxidation and glyoxylate metabolism. However, β-oxidation and glyoxylate pathways as well as the peroxisomal catalase are dispensable for *H. capsulatum* infection, indicating a novel function for peroxisomes in virulence. Pex5, Pex10, and Pex33 comprise the import machinery for peroxisomal matrix proteins, which are targeted to the organelle by a peroxisomal targeting sequence (PTS1). Accordingly, a PTS1-tagged GFP fails to localize to peroxisomes in *pex5*, *pex10*, and *pex33* mutants suggesting that PTS1-containing peroxisome protein(s) is/are required for pathogenesis. A bioinformatic search of the Histoplasma genome for PTS1-containing proteins identified enzymes involved in siderophore biosynthesis, which *H. capsulatum* uses to acquire iron in scarce conditions in the host. Consistent with this, low iron conditions impaired growth of the *pex5, pex10* and *pex33* mutants similar to that of a siderophore knockdown strain, and addition of exogenous siderophores restored growth of pex5, pex10, and pex33 mutants. Together, these data show that pathogenesis-enabling siderophore production relies upon peroxisomes. However, the *pex11* mutant showed near wild-type growth in low iron conditions, and peroxisome targeting of the PTS1-tagged GFP was normal, yet pex11 mutants still have attenuated virulence. These results indicate that the virulence function of the peroxisome is not limited to siderophore synthesis. To identify this function important for virulence, we are defining the peroxisome proteome through proximity labeling and capture of peroxisome proteins and interrogating the diverse functions represented.

Abstracts of OBASM Podium Presentations

Sunday, September 11
11:00 am – 12:45 pm

11:00 – 11:15 am

Genetic determinants of *Klebsiella* phage infection

Marissa R. Gittrich*[1], Courtney M. Sanderson[1], Cara M. Noel[1], Jonathan E. Leopold[1], Erica Babusci[1], Olivia R. Farinas[1], Vivek K. Mutalik[2], Matthew B. Sullivan[1]
[1]The Ohio State, [2]Lawrence Berkeley National Laboratory

Bacteriophages (phages) are being cataloged at unprecedented rates and are now broadly credited with driving nutrient and energy cycling across many of Earth's ecosystems. However, little is known about the host genes required for infection beyond a few model phage-host systems, and such data are critical for modeling phage-host interactions in complex communities. Here we map bacterial genetic determinants of phage infection using a randomly barcoded, genome-wide loss-of-function transposon mutant library (RB-TnSeq) of a plant growth-promoting rhizobacterium (*Klebsiella sp.* M5a1) separately challenged by 14 diverse, double-stranded DNA phages, spanning four phage families, at three multiplicities of infection (0.1, 1, and 10). Our genetic screen uncovered a multitude of host factors involved in phage infection, such as genes involved in receptor formation, transcription regulation, electron transport, and genes with unknown functions. When disrupted, some of the host genes, such as those encoding putative glycosyltransferases involved in LPS biogenesis, conferred resistance to up to 50% of the phages across multiple phage families, potentially due to preventing phage adsorption. Other host genes involved in intracellular functions, such as the electron transport chain and transcriptional regulation, were phage specific indicating such cellular processes are differentially required across our diverse phage set. This supports previous findings that genes involved in intracellular functions are phage specific while genes encoding for receptors required for phage adsorption are more broadly required across phages. Together these findings provide a foundation to develop predictive models for phage infection that can be applied to environmental and therapeutic systems.

11:15 – 11:30 am

Discovery of Anti-Phytopathogenic Phosphonopeptides from *Bacillus velezensis*

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Phosphonic acids are a class of microbial natural products that have potent inhibitory activities. A carbon-phosphorous bond allows phosphonates to mimic primary metabolites which contain phosphate esters and carboxylic acids, leading to the inhibition of essential metabolic pathways. Consequently, numerous members of this class have been commercialized as antimicrobials and herbicides. Of microbes with the potential to produce phosphonates, actinobacteria have been the focus of recent discovery efforts due to the diversity of their biosynthetic gene clusters (BGCs) and their storied ability to produce pharmaceutically relevant compounds. Despite the widespread taxonomic distribution of phosphonate BGCs, their metabolism in other bacteria has been largely underexplored. Here we describe the isolation, chemical characterization, and bioactivity of phosphonopeptides produced by *Bacillus velezensis*. Bioinformatic analyses identified a phosphonoalamide-like BGC in a *Mycobacteroides abscessus* subsp. *massilience* genome shown to be heavily contaminated with DNA from *Bacillus velezensis*. Purification and structure elucidation of phosphonate metabolites from *Bacillus velezensis* revealed two new phosphonopeptides, which we name phosphonoalamide E and F. Both compounds exhibited antimicrobial activity against plant and human pathogens, highlighting their potential for development as pesticides or antibiotics.

11:30 – 11:45 am

Siderophore Biosynthesis in *Histoplasma capsulatum*

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Histoplasma capsulatum (Hc) is a dimorphic pathogen. The pathogenic yeast infects macrophages as an intracellular pathogen. *Hc* yeasts not only survive in the harsh conditions of the phagosome, but they also proliferate within this compartment. Siderophore production facilitates *Hc* yeasts replication within phagosomes. Siderophores are iron-chelating compounds that enable growth in limited iron conditions. *Hc* yeasts produce three related hydroxamate-type siderophores: dimerum acid, coprogen B, and fusarinine. The *Hc* genome has a putative siderophore biosynthesis gene cluster with eight genes. One member of this gene cluster, *SID1*, catalyzes the first step in siderophore biosynthesis, and loss of Sid1 attenuates *Hc* growth in low-iron media and in a mouse model of infection. To understand the biosynthetic pathway for *Hc* siderophores, we are investigating the function and subcellular

localization of each gene in the siderophore gene cluster. Using CRISPR/Cas9 methodology, we have generated deletion alleles of each gene and tested the mutant strains for the ability to grow in limiting iron as an indication of siderophore production. Only a subset of genes within the siderophore biosynthesis cluster are required for growth in low iron, and include *SID1*, *SID3*, *SID4*, *NPS1*, and to a lesser degree the putative *ABC1* transporter. RFP-protein fusions were constructed to examine the localization of each siderophore biosynthesis gene product. Sid1 and Sid3 localize to peroxisome organelles (i.e., co-localization with peroxisome-targeting GFP), and indicating some biosynthetic reactions are compartmentalized.

11:45 am – 12:00 pm - Break

12:00 – 12:15 pm

Toward understanding the molecular mechanism of novel BPZ derivatives against *Toxoplasma gondii*

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The apicomplexan parasite *Toxoplasma gondii* is a ubiquitous pathogen of both animals and people, infecting approximately one-third of the human population. The parasite's ability to infect nearly any nucleated cell within warm-blooded animals, combined with multiple routes of transmission, make *T. gondii* one of the most successful parasites on Earth. Despite the significant burden of disease, therapeutic strategies for combating toxoplasmosis are seriously lacking due to undesirable side effects as well as the propensity of antiparasitic medications to force the parasite into long-lived tissue cysts that persist for the lifetime of the host. Moreover, the development and proliferation of drug-resistant pathogens is an ever-growing area of concern, and increasing pressure is being placed on discovering new compounds to circumvent the expanding trend in pathogen resistance. One particular facet in the search for new therapeutic strategies is understanding the mode of action of novel compounds and their derivatives. To this end, we endeavored to evaluate the efficacy of several bisphenol Z (BPZ) compounds and began investigating the cellular consequences of challenge in *T. gondii*. Data collected from doubling assays demonstrated that four of the nine tested compounds exhibited potent inhibitory activity, limiting the parasite's ability to replicate via endodyogeny. In addition, immunofluorescence microscopy revealed that the sole mitochondrion lost its ability to retain a MitoTracker stain, suggesting that membrane potential was disrupted. Current efforts are focused on determining the molecular mechanisms of action by mutagenizing parasites, selecting for isopropyl-BPZ-resistant parasites, and completing whole-genome sequencing to identify single-nucleotide polymorphisms. The findings from these studies will broaden our understanding of how BPZ and its derivatives lead to parasite death and which factors contribute to drug resistance, with the goal of exploiting these targets to improve upon or develop more efficacious therapeutic modalities for combating parasitic infections.

12:15 – 12:30 pm

ZFP36 Ring Finger Protein Like 1 (ZFP36L1), a CCCH- Type of Zinc Finger protein significantly suppresses Human coronavirus- OC43 replication

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There is strong evidence that severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), a betacoronavirus can spill over from humans to animals such as mink, dog, lion, tiger, and domestic cats which showed SARS-CoV-2 like symptoms. Additionally, free-living white-tailed deer in the United States also revealed the presence of SARS-CoV-2 antibodies in them. In the current study, we used another member of human betacoronavirus, strain OC43, and measured the effect of zinc finger protein 36L1 (ZFP36L1) on virus replication with a future goal that ZFP36L1 could be used as a potential intervening target for SARS-CoV-2 replication. ZFP36L1 is a member protein of CCCH type Zinc finger protein family. It is well characterized as an RNA-binding protein which control cellular mRNA turnover by degrading it poly A tail. We overexpressed or knockdown both zinc finger domains of ZFP36L1 in HCT-8 cells, infected them with HCoV-OC43. Results showed that HCoV-OC43 replication was significantly reduced with overexpression of ZFP36L1 while knockdown of ZFP36L1 significantly enhanced the virus replication compared with wild type HCoV-OC43 infected cells ($p<0.05$). Knocking down ZFP36L1 facilitated the infectious virus production as early as 48 hours post-infection p.i. while wild-type or ZFP36L1 overexpressed cells start producing infectious virus at 72 hours p.i. Virus titer at 96 hours p.i. in ZFP36L1knockdown cells (5.85 ± 0.01 log 10/ml) were significantly higher than virus titer in wildtype cells (5.42 ± 0.10 log10/ml) or ZFP36L1 overexpressed cells (4.32 ± 0.00 log 10/ml) ($p<0.05$). The comparison of virus titer in wild-type and ZFP36L1overexpressing cells also showed that ZFP36L1 overexpressing significantly suppressed virus titer at 96 hours p.i as compared to wild type cells. Overall, the current study revealed that ZFP36L1overexpression suppressed the HCoV-OC43 replication. Future studies are needed for its protentional application in mitigating the HCoV-OC43 mediated adverse effects in host and the role of ZFP36L1 in another human coronavirus replication.

Keywords: CCCH type Zinc finger protein, ZFP36L1, RNA binding protein, human coronavirus OC43, SARS-CoV-2

12:30 – 12:45 pm

S-adenosylmethionine byproduct utilization in extraintestinal pathogenic *Escherichia coli*

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S-adenosylmethionine (SAM) is an essential, versatile co-substrate for numerous cellular processes. This includes synthesis of signaling compounds like polyamines and acyl-homoserine lactones, generation of adenosyl radicals for radical SAM enzymes, and methylation of proteins, DNA and RNA. Utilization of SAM as a co-substrate results in the

formation of inhibitory byproducts. For example, methylation reactions form *S*-adenosylhomocysteine (SAH), polyamine synthesis results in 5'-methylthioadenosine (MTA), and radical SAM reactions result in 5'-deoxyadenosine (5'dADO) as the byproduct. To prevent inhibitory effects of SAM byproduct accumulation, cells could excrete these compounds at the loss of valuable organic sulfur and/or carbon or could recycle them. Recently, a recycling pathway called the DHAP shunt was identified that allows for the conversion of SAM byproducts, MTA and 5'dADO, into the central carbon metabolite dihydroxyacetone phosphate (DHAP). The DHAP shunt is found in a wide distribution of bacteria and is intriguingly enriched in pathogenic species of several genera, including *Bacillus, Clostridia,* and extraintestinal pathogenic *Escherichia coli* (ExPEC) species. ExPEC strains account for >85% of urinary tract infections (UTI) annually within the United States. How they utilize available compounds found in the blood and urine for growth are largely unknown. Here, we show the ability of a DHAP shunt-containing ExPEC strain to grow with SAM utilization byproducts, MTA and 5'dADO, as carbon substrates. Additionally, internal production levels of SAM byproducts are found to be similar between ExPEC and commensal *E. coli* strains, suggesting this is not an internal salvage pathway. This work seeks to provide exploratory insights into the biological relevance of the DHAP shunt within ExPEC strains, with aims of identifying future therapeutic targets.

Description of Awards for Presentations

N. Paul Hudson Award for Research Excellence

N. Paul Hudson, MD, was chairman of the Microbiology Department at Ohio State University from 1935 – 1950. Dr. Hudson was recruited from the University of Chicago Medical School where he already had established a research/teaching reputation and was told to bring several colleagues with him to serve as faculty members in the department. In 1950 he was appointed Dean of the Graduate School at Ohio State University and served until his retirement in 1957. During the 1930's he was responsible for the successful development of a vaccine against Yellow Fever, which was necessary for the war effort in the tropical countries during the 1940's. Dr. Hudson died at the age of 95 in 1993 in Florida after retiring to his second home in Sarasota in 1970.

Donald C. Cox Award for Research Excellence

Donald C. Cox, PhD, was professor and chair of the Department of Microbiology at Miami University from 1978-1989. After he earned his PhD at University of Michigan in 1965, he joined the microbiology faculty at University of Oklahoma. He later moved to Miami, where he became well known as a charismatic and highly effective teacher, researcher and leader. Dr. Cox received Miami's Distinguished Educator Award, and fostered the growth and development of the Department of Microbiology. Throughout his research career, he focused on the biochemistry and molecular biology of replication of human viruses, and ultimately studied utilization of reovirus in cancer therapy. Dr. Cox was a strong advocate for attracting young people into scientific careers and mentored many students who have gone on to highly significant research careers.

J. Robie Vestal Award for Research Excellence

J. Robie Vestal, PhD, was professor of both biological sciences and environmental health at the University of Cincinnati. He earned his MS in Microbiology at Miami University and his PhD in Microbiology at North Carolina State University. His postdoctoral research at Syracuse University involved the biochemistry of *Thiobacillus ferrooxidans*. Dr. Vestal's research interests focused on how microbial communities function in nature. He studied microbial communities in Arctic lakes and in soils contaminated with hazardous waste, cryptoendolithic (hidden within rock) communities in Antarctica, mangrove-degrading communities in the Bahamas, and decomposer communities in municipal solid waste compost. He also investigated microbial survival under simulated Martian conditions. Dr. Vestal served on many local and national committees and chaired the Divisional Advisory Committee of the National Science Foundation's Division of Polar Programs.

Ohio Branch ASM Award for Research Excellence

This award traditionally recognizes excellence in graduate research and presentation at the annual Ohio Branch ASM meeting.

Allan A. Ichida Undergraduate Research Award

Allan Ai Ichida, PhD, earned his BA from Ohio Wesleyan University in 1953 and went on to study botany, mycology, and bacteriology at the University of Tennessee where he earned his MS in 1955 and the University of Wisconsin in Madison where he earned his PhD in 1960. Dr. Ichida returned to Ohio Wesleyan in 1961 as a faculty member in the Department of Botany and Microbiology where he taught botany and mycology until he retired in 1995. During his career, Dr. Ichida served as president and advisor of the Ohio Branch of the American Society for Microbiology and on the Olentangy Scenic River Commission where his water quality research helped to secure the river's "Scenic River" status. Dr. Ichida also conducted research in the OWU Bohannan and Kraus nature preserves and mentored numerous undergraduates who went on to become research scientists.

The Ohio Branch ASM Award for Pre-college Research Excellence

Established in 2017, this award recognizes excellence in pre-college research and presentation at the annual Ohio Branch ASM meeting.

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