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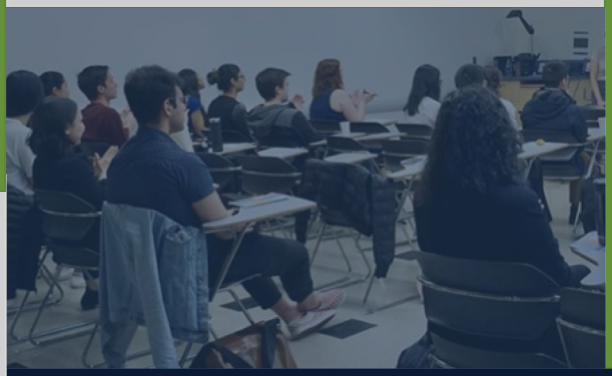
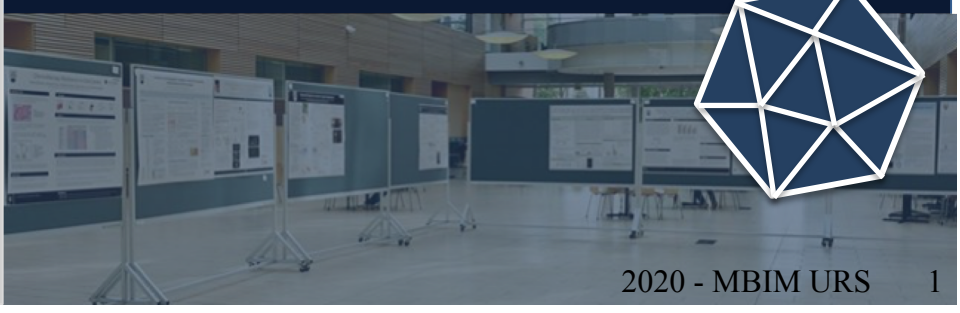


2020 MBIM UNDERGRADUATE RESEARCH SYMPOSIUM

Live Online Symposium
University of British Columbia
April 30th, 2020



WELCOME

It is with great pleasure we welcome you to the 2020 Microbiology and Immunology Undergraduate Research Symposium (MBIM URS)! After the success of the last two years, we are delighted to share with you our students' hard work at the third annual MBIM URS. Although an in-person event is not possible this year due to the COVID-19 pandemic, we believe a live online event is an excellent way to ensure that students have a chance to showcase their work. The organizing committee has been working diligently to make this year's event a possibility and to set a precedent for the first ever online undergraduate symposium, and we are excited to be able to share this experience with you all.

This year's symposium will explore the diverse and evolving topics under clinical microbiology, antibiotic resistance, gut microbiome and biofilms, and mechanisms of adaptation. These sessions will provide students with the opportunity to broaden their knowledge and participate in scientific discourse. With the times that we are in, we believe it is more important than ever to continue pursuing advancements in science.

This symposium would not have been possible without our hardworking undergraduate student and faculty organizing committee. We appreciate Dr. David Oliver for his leadership and support in the planning of this event. A special thank you to Dr. Parvin Bolourani for providing guidance and support, Dr. Marcia Graves and Dr. Evelyn Sun for providing insight at meetings, Eric Lee for his IT expertise, and Craig Kornak for helping us reach out to students. We are honoured to have Dr. Santa Ono as our keynote speaker, and we would like to thank Dr. Michael Murphy and Dr. Julian Davies for their welcome addresses. Lastly, we would like to thank all of the symposium participants and attendees for supporting us and seizing this opportunity to advance their scientific experience and education.

We hope you enjoy this symposium and continue supporting undergraduate research in future years. We are looking forward to your virtual presentations!

Yours Remotely,

Mahta Amanian and Wesley Hunt
Co-Chairs | 2020 MBIM Undergraduate Research Symposium

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ABOUT

The MBIM Undergraduate Research Symposium provides undergraduate students with a forum to share their research findings, interact with scientists with related interests, and develop communication skills.

The 2020 MBIM URS will be held as a live online event due to the COVID-19 pandemic. It will include opening remarks from the URS Co-chairs, a welcome by Dr. Michael Murphy and Dr. Julian Davies, and a keynote speech from Professor Santa J. Ono. We will also have oral and poster presentations featuring the work of our undergraduate students done in research laboratories (MICB 448/449/Co-op), some of our experiential learning courses (MICB 406/421/447), as well as Research Experience (REX) projects. Awards will be presented to the top oral and poster presentations.

http://urs.microbiology.ubc.ca

LETTERS

Letter from Dr. Michael Murphy

A message from the Department Head

Welcome to the Undergraduate Research Symposium hosted by the Department of Microbiology and Immunology at UBC. The symposium is a showcase of the range and depth of research performed by undergraduate students both in lab courses and in research labs at UBC.

The presentations will demonstrate the high-quality of science performed by the talented students in our programs. Moreover, this student-led and organized symposium is evidence of student curiosity to learn and desire to communicate their science discoveries with each other and the larger UBC community. This drive to connect and work as a community of researchers and learners was not to be suppressed by the inability this year to meet in person. Instead, the first virtual undergraduate research symposium has arrived and will celebrate the many research successes of undergraduates in the Department. I look forward to learning about your research projects and meeting you in person in the future.

Michael Murphy, PhD
Professor and Head
Department of Microbiology & Immunology
University of British Columbia

Letter from Dr. Julian E. Davies

I am pleased to have the opportunity to address the 2020 Graduating Class. Before I start, have you washed your hands? How far away are you from your neighbour?

I cannot give you much advice. You should not accept recommendations from anyone approaching 90 years of age!

This graduating class can be called the "Viral Pandemic" class! My hope is that you will be a new generation who know what a pandemic viral infection is.

You are surely going to become involved in the effort to eliminate this new microbial threat.

Incidentally, how many of you know the definition of a virus? This was given by a very famous British virologist: "bad news, wrapped in protein".

However, I am supposed to speak words of encouragement and opportunity for your future careers.

There are so many opportunities for you, you just have to look.

I am not a good example of a sage microbiologist, ostensibly giving you words of encouragement for your career. I have had too many academic and industrial positions in my life. I have tried them all! And I am still looking!

Fortunately, my most recent position was in academia and in this department. UBC Microbiology has treated me well and I trust that I will retire with some accomplishments. You cannot go wrong at UBC!

My only words of encouragement are as follows: even if you are happy in a university faculty or industrial position, and you are offered something scientifically creative that excites you (and usually pays more), take it!

You can do it! I wish you well!

Julian E. Davies, PhD
Professor of Microbiology and Immunology
Life Sciences Institute
University of British Columbia

CONTRIBUTORS

UNDERGRADUATE ORGANIZING COMMITTEE

Mahta Amanian (Co-Chair)
Wesley Hunt (Co-Chair)
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Baria Choudry
Yasmine Chung
Antyrah de Guzman
Nikola Deretic
Ameena Hashimi
Al Rohet Hossain
Helen Hsiao
Cai Lan Jennifer Huang
Cynthia James
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Amelia Tjoa
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Jennifer Tong
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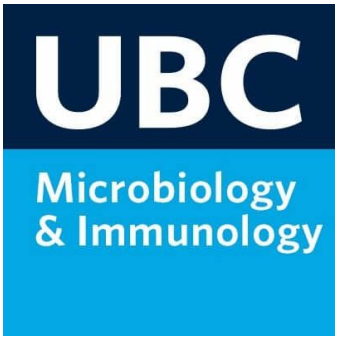
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Microbiology
& Immunology


UBC

OUR KEYNOTE SPEAKER...

Dr. Santa J. Ono

UBC President and
Vice-Chancellor

Ono's research encompasses the immune system, eye inflammation and age-related macular degeneration.

SYMPOSIUM PROGRAM

April 30, 2020 - Live Online Presentations over Zoom

Time Period & Events

9:00AM - 10:00 AM - Opening Remarks & Keynote Speech

- **9:00AM - 9:30AM:** Opening Remarks (URS Co-chairs, Dr. Michael Murphy, and Dr. Julian Davies)
- **9:35AM - 9:55AM:** Keynote Speech by Dr. Santa J. Ono
- **9:55AM - 10:00AM:** Transition to Presentations (URS Co-Chairs)

10:00AM - 10:55AM - Presentations Session 1: Clinical Microbiology

- **10:00AM – 10:15AM: Oral Presentation**
  - Comparisons of clinical isolates of *Pseudomonas aeruginosa* (LESB58 and LESB65) and establishing a chronic *In-Vitro* lung infection model
    - Pavneet Kalsi, Ka-Yee Grace Choi, Robert EW Hancock
- **10:15AM – 10:25AM: Poster Presentation**
  - A systematic review and meta-analysis of patients' knowledge of anti-thrombotics
    - Elaine Hu, William Shen, Jinny Choi, Amelia Choy
- **10:25AM – 10:35AM: Poster Presentation**
  - The potential role of lactobacillus in treatment of colorectal cancer
    - Tran Hoang Anh (Emma) Le, Tiffany Wai
- **10:35AM – 10:45AM: Poster Presentation**
  - Optimal treatment for glioblastoma multiforme (GBM) using oncolytic virus
    - Chin-Yueh Cheng, Yucella Liu
- **10:45AM – 10:55AM: Poster Presentation**
  - TUDCA and Tr1 cell supernatants as potential therapeutics to am:eliorate intestinal epithelial cell ER stress
    - Rene Tandun, Enoch Yau, William D. Rees, Theodore S. Steiner

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11:00AM - 11:25AM - Presentations Session 2: Antibiotic Resistance

- **11:00AM – 11:15AM: Oral Presentation**
    - Generation of an *acrAacrE* double-knockout in *Escherichia coli* and its role in kanamycin resistance
        - Ada Ang, Cathy Park, Joshua De Guzman, Shaneel Kumar
- **11:15AM – 11:25AM: Poster Presentation**
    - Deletions in the capsular polysaccharide *wzy* Cassette genes differentially affect susceptibility to nitrofurantoin in *Escherichia coli* K-12 compared to K30
        - Rehanna Thobani, Gillian Savage, Malhar Shah

11:30AM - 12:00PM - Company Talk: 10X Genomics

- **11:30AM - 12:00PM: 10X Genomics**
    - Crucial Applications of Single Cell Gene Expression and Immune Profiling for Infectious Disease Research

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12:15PM - 1:55PM - Presentations Session 3: Gut Microbiome and Biofilms

- **12:15PM – 12:25PM: Poster Presentation**
    - Increasing concentrations of commercial stevia inhibit growth and biofilm formation of *Escherichia coli* MG1655
        - Al-Rohet Hossain, Derek Lee, Thomas Soroski, Boyan K. Tsankov
- **12:25PM – 12:35PM: Poster Presentation**
    - Biofilm production in *Escherichia coli* K30 with group 1 capsular gene *wza* and *wza-wzb-wzc* deletions is not correlated with erythromycin resistance phenotypes in liquid media
        - Cynthia James, Christine Kim, Catherine Pan, Douglas Zhong
- **12:35PM – 12:45PM: Poster Presentation**
    - Pre-induction of *rodentium* in mildly acidic gut-mimicking media increases attachment to epithelial cells
        - Laurel M. Neufeld, Sarah E. Woodward, B. Brett Finlay

- **12:45PM – 12:55PM: Poster Presentation**
    - Resistance to sucralose in *Escherichia coli* is not conferred by mutations in the quinolone resistance determining regions of *gyrA*
        - Solana Cheng, Sara S. Dalkilic

1:00PM - 1:55 PM - Presentations Session 4: Mechanisms of Adaptation

- **1:00PM – 1:15PM: Oral Presentation**
    - Trehalose does not appear to protect *Escherichia coli* from SDS-EDTA-induced outer membrane stress
        - Mackenzie W. Gutierrez, Laurel M. Neufeld, Xin Wei, Yi Han Yin
- **1:15PM – 1:25PM: Poster Presentation**
    - The rate of T7 phage-mediated lysis of *Escherichia coli* growing in exponential phase is not affected by deletion of *rpoS*
        - Jingyao Zhu, Cai Lan Jennifer Huang, Kamand Doraki, Bryan Lee
- **1:25PM – 1:40PM: Oral Presentation**
    - Limiting phosphorus in M9 minimal media results in impaired growth and decreased glucose content in *Escherichia coli* MG1655
        - Niloufar Benam, Sahi Hajirawala, Jasper Hoi Chun Luong, Charlene Yang
- **1:40PM – 1:55PM: Oral Presentation**
    - Genome-wide association study of transient versus chronic infections in *Burkholderia multivorans*
        - Kendrew SK. Wong, James EA. Zlosnik, Mark A. Chilvers

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2:00PM - 2:15PM - Thank You & Explanation of Awards

May 1, 2020 - Awards announced on the URS website:
http://urs.microbiology.ubc.ca

TALKS

Generation of an *acrAacrE* double-knockout in *Escherichia coli* and its role in kanamycin resistance

AUTHORS: Ada Ang, Cathy Park, Joshua De Guzman, Shaneel Kumar
COURSE: MICB 421/447
SESSION: Antibiotic Resistance
PRESENTATION ID: 1

Comparisons of clinical isolates of *Pseudomonas aeruginosa* (LESB 58 and LESB 65) and establishing a chronic *in-vitro* lung infection model

AUTHORS: Pavneet Kalsi, Ka-Yee Grace Choi, Robert EW Hancock
COURSE: MICB 448/449
SESSION: Clinical Microbiology
PRESENTATION ID: 2

Trehalose does not appear to protect *Escherichia coli* from SDS-EDTA-induced outer membrane stress

AUTHORS: Mackenzie W. Gutierrez, Laurel M. Neufeld, Xin Wei, Yi Han Yin
COURSE: MICB 421/447
SESSION: Mechanisms of Adaptation
PRESENTATION ID: 3

Bacterial genome-wide association analysis hints towards the involvement of potential operons in determining transient versus chronic infection among *Burkholderia multivorans*

AUTHORS: Kendrew SK. Wong, James EA. Zlosnik, Mark A. Chilvers
COURSE: BIOL 448
SESSION: Mechanisms of Adaptation
PRESENTATION ID: 4

Limiting phosphorus in M9 minimal media results in impaired growth and decreased glucose content in *Escherichia coli* MG1655

AUTHORS: Sahi Hajirawala, Niloufar Benam, Jasper Hoi Chun Luong, Charlene Yang
COURSE: MICB 421/447
SESSION: Mechanisms of Adaptation
PRESENTATION ID: 5

POSTERS

Deletions in the capsular polysaccharide *wzy* cassette genes differentially affect susceptibility to nitrofurantoin in *Escherichia coli* K-12 compared to *Escherichia coli* K30

AUTHORS: Rehanna Thobani, Gillian Savage, Malhar Shah
COURSE: MICB 421/447
SESSION: Antibiotic Resistance
PRESENTATION ID: 6

A systematic review and meta-analysis of patients' knowledge of anti-thrombotics

AUTHORS: Elaine Hu, William Shen, Jinny Choi, Amelia Choy
COURSE: Research Experience Program (REX)
SESSION: Clinical Microbiology
PRESENTATION ID: 7

The potential role of Lactobacillus in treatment of colorectal cancer

AUTHORS: Tran Hoang Anh (Emma) Le, Tiffany Wai
COURSE: Research Experience Program (REX)
SESSION: Clinical Microbiology
PRESENTATION ID: 8

Optimal treatment for glioblastoma multiforme (GBM) using oncolytic virus

AUTHORS: Chin-Yueh Cheng, Yucella Liu
COURSE: Research Experience Program (REX)
SESSION: Clinical Microbiology
PRESENTATION ID: 9

TUDCA and Tr1 cell supernatants as potential therapeutics to ameliorate intestinal epithelial cell ER stress

AUTHORS: Rene Tandun, Enoch Yau, William D. Rees, Theodore S. Steiner
COURSE: Co-Op Placement
SESSION: Clinical Microbiology
PRESENTATION ID: 10

Increasing concentrations of commercial stevia inhibit growth and biofilm formation of *Escherichia coli* MG1655

AUTHORS: Al-Rohet Hossain, Derek Lee, Thomas Soroski, Boyan K. Tsankov
COURSE: MICB 421/447
SESSION: Gut Microbiome and Biofilms
PRESENTATION ID: 11

Biofilm production in *Escherichia coli* K30 with Group 1 capsular gene *wza* and *wza-wzb-wzc* deletions is not correlated with erythromycin resistance phenotypes in liquid media

AUTHORS: Cynthia James, Christine Kim, Catherine Pan, Douglas Zhong
COURSE: MICB 421/447
SESSION: Gut Microbiome and Biofilms
PRESENTATION ID: 12

Intestinal pH modulates the growth, stress, and virulence of murine pathogen *C. rodentium*

AUTHORS: Laurel M. Neufeld, Sarah E. Woodward, B. Brett Finlay
COURSE: MICB 448/449
SESSION: Gut Microbiome and Biofilms
PRESENTATION ID: 13

Resistance to sucralose in *Escherichia coli* is not conferred by mutations in the quinolone resistance determining regions of *gyrA*

AUTHORS: Solana Cheng, Sara S. Dalkilic
COURSE: MICB 421/447
SESSION: Gut Microbiome and Biofilms
PRESENTATION ID: 14

The rate of T7 phage-mediated lysis of *Escherichia coli* growing in exponential phase is not affected by deletion of *rpoS*

AUTHORS: Jingyao Zhu, Cai Lan Jennifer Huang, Kamand Doraki, Bryan Lee
COURSE: MICB 421/447
SESSION: Mechanisms of Adaptation
PRESENTATION ID: 15

ABSTRACTS

1 Generation of an *acrAacrE* double-knockout in *Escherichia coli* and its role in kanamycin resistance

Ada Ang, Cathy Park, Joshua De Guzman, Shaneel Kumar

*AcrA*B and AcrEF are operon-encoded, multidrug efflux pumps found in *Escherichia coli* that are thought to mediate the export of kanamycin, and therefore contribute to kanamycin resistance. These efflux pumps are composed of a tripartite system consisting of the periplasmic components, *AcrA* and AcrE, which connect TolC to the inner membrane proteins, AcrB and AcrF respectively. Previous studies found that expression of acrE increased in response to increasing concentrations of kanamycin. Another study found that the loss of *acrA* or acrE was shown to have no effect on kanamycin resistance, indicating that AcrAB and AcrEF may have compensatory effects on each other. For example, deletion of *acrA* may result in upregulation of acrE, making up for the loss in efflux activity. To study the effect of these efflux pumps on kanamycin resistance, any compensatory effects must be eliminated by deleting both *acrA* and acrE. The goal of this study was to generate an *acrAacrE* double-knockout in *E. coli* and determine if this double-knockout had any effect on kanamycin resistance when compared to single-knockouts and the wild-type. We hypothesized that by knocking out both the *acrA* and acrE genes, no compensatory effects would occur to support kanamycin resistance. Therefore, an *acrAacrE* double-knockout should be the most susceptible to kanamycin and have the lowest minimum inhibitory concentration (MIC). To test this, we first generated an *acrAacrE* double-knockout by using lambda Red (λ-Red) recombination to remove *acrA* from an acrE single-knockout strain. To determine differences in kanamycin resistance, MIC assays were performed on all the strains. The *acrAacrE* double-knockout was successfully generated and Sanger sequencing confirmed the deletion of *acrA*. However, MIC results were not conclusive as they did not show any difference in kanamycin resistance between strains. Based on these results, it is still unclear if AcrAB and AcrEF have compensatory effects on each other, since there was no significant difference in MIC between the strains.

2 Comparisons of clinical isolates of *Pseudomonas aeruginosa* (LESB 58 and LESB 65) and establishing a chronic *in-vitro* lung infection model

Pavneet Kalsi, Ka-Yee Grace Choi, Robert EW Hancock

Cystic Fibrosis (CF) is a debilitating condition of the lungs and is progressed upon chronic infection by *Pseudomonas aeruginosa*. Further, the Liverpool Epidemic Strains (LES) of Pseudomonas have been associated with even worse prognosis and higher mortality among patients. Previous models for studying LES infections have primarily been conducted in acute mice or rodent models but not in a chronic *in-vitro* cell line system. Since LES isolates have been strongly associated with chronic infection, this study aimed to model and establish a long-term *in-vitro* infection system while retaining host and bacterial interactions. First, phenotypic traits of LESB 58 and LESB 65 were investigated by comparing growth, swarming motility, and drug susceptibility in bacterial optimized and host-like conditions. Growth of the strains during a 24-hour time period indicated that LESB 65 enters stationary phase earlier than LESB 58 in both bacterial and host environmental conditions. In addition, LESB 58 is more motile on 0.3% (wt/vol) agar KB (King's B Broth) and tissue culture media swarming conditions compared to LESB 65. Further, the minimum inhibitory concentration (MIC) of azithromycin varied from 4-fold to 20-fold when tested between tissue culture media and traditional MHB (Mueller Hinton Broth) and 0-fold to 4-fold between LES isolates. Infection capacity of both LES isolates were tested with a human bronchial epithelial (HBE) cell model system for 18 hours. When comparing bacterial-mediated host cell damage, LESB 58 leads to significantly higher cellular cytotoxicity, while LESB 65 is non-toxic to host cells. However, LESB 65 mediated significantly higher levels of IL-6 and IL-8 production by host cells, quantified by ELISA's, when compared to LESB 58. Overall, directly comparing the LES isolates phenotypic traits and establishing an *in-vitro* cell line system, to model chronic bacterial infection, allows for further analysis of virulence differences between the strains.

3 Trehalose does not appear to protect *Escherichia coli* from SDS-EDTA-induced outer membrane stress

Mackenzie W. Gutierrez, Laurel M. Neufeld, Xin Wei, Yi Han Yin

Trehalose, a glucose disaccharide, has been reported to play a role in stabilizing the cell envelope of *Escherichia coli* under numerous forms of abiotic stress. The biosynthetic pathway of trehalose in *E. coli* has been shown to require the actions of *OtsA*, trehalose-6-phosphate synthase, as *otsA* mutants are unable to synthesize trehalose. The exact manner by which trehalose contributes to cell envelope stability remains under investigation, though a recent study suggested that it stabilizes the outer membrane (OM) component of the cell envelope in response to sodium dodecyl sulfate and ethylenediaminetetraacetic acid (SDS-EDTA)-induced stress. That study used SDS-EDTA assays to assess OM stability by growing cells in different SDS concentrations with a constant EDTA concentration or vice versa. We sought to validate these findings with a more rigorous approach to the SDS-EDTA assays, by incorporating the correct parent wild type (WT) strain, consistent growth media, OD600 readings throughout the assay, and multiple biological replicates. We hypothesized that *otsA* mutants would be hindered in their ability to stabilize the OM compared to the WT, increasing their susceptibility to SDS-EDTA-induced stress. Under both SDS and EDTA conditions over 18 hours, the mutants with the disrupted trehalose biosynthesis pathway did not have significantly reduced cell densities (OD600) compared to the WT. From this result, we conclude that the mutants did not present higher sensitivities to either form of membrane stress (SDS or EDTA). These findings suggest that trehalose may not promote OM stability of *E. coli* during SDS-EDTA stress. Further investigation is needed to elucidate which cell envelope structure of *E. coli* trehalose protects under abiotic stress.

4 Bacterial genome-wide association analysis hints towards the involvement of potential operons in determining transient versus chronic infection among *Burkholderia multivorans*

Kendrew SK. Wong, James EA. Zlosnik, Mark A. Chilvers

Burkholderia cepacia complex (Bcc) is a group of bacteria that can cause deadly infections in people with cystic fibrosis (CF), an incurable genetic disease that predisposes individuals to bacterial infection and other serious health problems. Infections with *Burkholderia* bacteria in people with CF cause unpredictable outcomes, ranging from acute/transient infection to chronic infection, which can be relatively mild to quickly fatal. We do not understand the basis of these differential outcomes. Through 30 years of data obtained from *Burkholderia* infections in people with CF in the Vancouver population, one of the two species of Bcc which most commonly cause infections, *Burkholderia multivorans*, has been observed to exhibit particularly high rates of transient infections at 55%. To gain insight into possible factors that may help characterize chronic versus transient infections, a genome-wide association study (GWAS) approach was employed in analyzing 19 isolates comprised of 6 and 13 isolates from transient and chronic infections, respectively. Isolates were whole-genome sequenced using the Illumina MiSeq System. Trimmed paired-end sequences underwent *de novo* assembly using Shovill and were then annotated with Prokka. Assembled genomes were screened for antimicrobial resistance and virulence genes using ABRicate. Pan-genome association of accessory genes to infection phenotype was accomplished through Roary and Scoary. The phenotypes show little to no association to resistance or virulence genes and are instead more reflective of genetic differences on a strain-level. Genetic clusters are observed among the top 24 associated genes ($p<0.002$, Benjamini $p< 0.4$) with 23 of those genes being specific for chronic infection and 1 gene being specific for transient infection. Despite an insignificant adjusted p-value due to the small sample size, the presence of clusters hint towards the involvement of potential operons in influencing the prognosis of *B. multivorans* infection. Further assessment of these genetic clusters, along with additional variant calling analyses, will be performed to better understand the potential of these clusters, or other additional single nucleotide polymorphisms, in predicting infection outcomes. This may help guide physicians in their approach to treating *Burkholderia* infections.

5 Limiting phosphorus in M9 minimal media results in impaired growth and decreased glucose content in *Escherichia coli* MG1655

Sahi Hajirawala, Niloufar Benam, Jasper Hoi Chun Luong, Charlene Yang

Inorganic phosphate is an important source of phosphorus for *Escherichia coli* metabolism. In addition to carbon and nitrogen, the presence of these molecules within the bacterium's environment contributes to its survival and growth. As such, extended periods of nutrient starvation results in metabolic and physiological changes to allow the organism to survive and adapt. Such a change is the activation of the stationary phase stress response primed by *RpoS* expression that results in glycogen accumulation. We hypothesized that the response occurs when phosphorus is limited to conserve metabolic energy in the form of glycogen as essential nutrients are limited. To determine the effects of phosphorus and nitrogen limitation on cellular growth and stationary phase glycogen accumulation of *E. coli*, MG1655 wild-type *E. coli* cells were subjected to nutrient limitation by limiting available environmental phosphorus and nitrogen, and subsequent cellular glycogen quantification was attempted using a previously-established enzymatic glucose assay. Bacterial growth curve analysis showed that phosphorus-limiting conditions significantly decreased growth yield across all growth phases compared to non-limiting conditions and nitrogen-limiting conditions. Likewise, cellular glucose levels quantified after amyloglucosidase/hexokinase treatment may indicate a reduction of cellular glucose levels when phosphorus was limited compared to non-limiting conditions. Similarly, a possible reduction in cellular glucose levels relative to non-limiting conditions is observed in the nitrogen-limiting conditions but only when treated with amyloglucosidase. Altogether, the results of the investigation did not implicate any conclusive findings between phosphorus or nitrogen limitation and changes in cellular glycogen levels, but rather suggest that limiting phosphorus may lead to a reduction in bacterial growth and cellular glucose levels.

6 Deletions in the capsular polysaccharide *wzy* cassette genes differentially affect susceptibility to nitrofurantoin in *Escherichia coli* K-12 compared to *Escherichia coli* K30

Rehanna Thobani, Gillian Savage, Malhar Shah

The *wzy* cassette is a set of genes responsible for capsular polysaccharide and exopolysaccharide polymerization and export. It has been shown that knockouts of the *wzy* cassette in *Escherichia coli* K30 strain increases susceptibility to the antibiotic nitrofurantoin, but a similar effect has not been observed in *E. coli* K-12. Based on this, we hypothesized that deletion of genes within the *wzy* cassette would change the susceptibility to nitrofurantoin differentially in *E. coli* K-12 compared to *E. coli* K30. To test this, susceptibilities to nitrofurantoin of mutants with knockouts in the *wzy* cassette compared to wildtypes were determined with disc diffusion assays at 28℃ and 37℃, as well as through a minimum inhibitory concentration (MIC) microtitration assay. Furthermore, the presence of capsule in all variants was determined with india ink staining to investigate the role of capsule in nitrofurantoin resistance. Our results show an increase in susceptibility to nitrofurantoin in only the *E. coli* K30 *wzb* and full *wzy* cassette knockouts at 37℃, but an increase in resistance in the *E. coli* K-12 *wza* knockout at 28℃. The MIC assay in liquid media showed the MIC for *E. coli* K30 *wza* was higher than the MIC of the *E. coli* K30 wildtype whereas the *wzb* and full cassette knockouts showed no change. Furthermore, capsule was only visualized in the *E. coli* K30 wildtype. Altogether our results suggest that the *E. coli* K30 and K-12 *wzy* cassette mutants respond differentially to nitrofurantoin and this effect occurs through a capsule-independent mechanism.

7 A systematic review and meta-analysis of patients' knowledge of anti-thrombotics

Elaine Hu, William Shen, Jinny Choi, Amelia Choy

Background: Oral anticoagulants (OACs) are a group of medications that reduce the risk of thrombosis, the formation of clots in blood vessels. Anticoagulants such as warfarin increase bleeding risk, which patients must be properly educated to mitigate and detect. Identifying patient knowledge gaps is the first step towards improving patient education, adherence and ultimately, health outcomes.

Objective: To synthesize the evidence on patients' knowledge of antithrombotic medications in order to identify their knowledge gaps and misconceptions.

Methods: Following Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines, PubMed, CINAHL, and PsychINFO databases were searched from inception to November 2018 for studies that assessed patients' knowledge of antithrombotics. The proportion of participants who correctly answered questions about their medications were extracted from quantitative studies. Data for similar questions were grouped into knowledge domains and a random-effects meta-analysis was conducted for each knowledge domain to pool the results. Any domains where the pooled mean proportion of knowledgeable participants was ≤50% was deemed a knowledge gap. Qualitative data were summarized narratively.

Results: A total of 54 studies (n= 11930 participants) were included. Studies were mostly published after 2010, conducted in North America or Europe, and focused on warfarin knowledge. Knowledge domains identified as knowledge gaps were: drug-drug interactions, drug-food interactions, underdosage or overdosage management, factors that may affect INR levels, side effects of antithrombotics, side effects management, increased risk of bleeding, preparation required on the day of prothrombin time/international normalized ratio test, action to take in case of a missed dose, action to take if in possession of both generic and brand warfarin.

Conclusion: Patients taking antithrombotics are likely to have knowledge gaps in clinically significant areas which can potentially contribute to nonadherence and poor therapeutic outcomes.

8 The potential role of Lactobacillus in treatment of colorectal cancer

Tran Hoang Anh (Emma) Le, Tiffany Wai

Colorectal cancer (CRC) is characterised as highly mutated colon and rectum cells that have become more aggressive, resulting in modified bowel habits, gastrointestinal irritation, and reduced life expectancy. 12% of newly diagnosed cancer cases in 2019 were CRC, thus, research into novel therapeutic methods could save money, resources, and hopefully the patients' lives. The human gut microbiome plays a major role in regulating the immune system and is an area of interest in CRC research. Lenoir et al. (2016) and other studies have shown a positive correlation between the presence of Lactobacillus, a gut-resident group of bacteria, and reduction of cancer cells growth. Therefore, this research aims to investigate how Lactobacillus and its metabolites may be assessed for the prevention, diagnosis, and therapeutic treatment for CRC. This study will be done by assessing current literature regarding Lactobacillus's role in the gut to uncover the possible intrinsic and extrinsic apoptosis cellular pathways as well as interaction with the immune system to hinder tumour growth. These findings may provide insights in understanding the protective role of Lactobacillus against CRC, drawing attention to the nuanced approach of microbiome-focused diagnosis and to optimise future treatment. In addition, understanding Lactobacillus's mechanism in CRC could contribute to the development of adjuvant therapy to achieve maximum efficacy in treatment.

9 Optimal treatment for glioblastoma multiforme (GBM) using oncolytic virus

Chin-Yueh Cheng, Yucella Liu

Oncolytic Virus Therapy (OVT) uses viruses to infect, replicate within, and kill cancer cells. The release of antigens through tumour cell lysis can stimulate antitumor immune responses. OVs offer greater specificity through the direct targeting of tumour cells without harming normal-functioning cells.

This study examines glioblastoma multiforme (GBM), a form of malignant brain cancer developed from glial cells. It features a distinct extracellular matrix (ECM) and unique brain-resident cell types, including microglia and tumor-associated macrophages (TAMs). These cells secrete anti-inflammatory cytokines to suppress antigen presentation, making the tumour strongly immunosuppressive. In addition, irregular vascular growth and high levels of oxygen consumption induce tissue hypoxia. This triggers the release of vascular endothelial growth factor (VEGF) and fibroblast growth factor (FGF) which drastically increases tumor invasion and recurrence, potentially leading to a higher fatality rate of the cancer cells.

The standard of care for GBM includes surgical dissection, high-dose radiation, and chemotherapeutic drugs, which are associated with serious adverse effects. Through conducting literature review along with evaluating results and observations from clinical trials, we assessed the efficacy of traditional treatments in addition to the potential of OVT and designed an optimal treatment plan to increase cancer cell apoptosis and minimize side effects to normal tissues in the brain.

We propose the use of engineered Herpes simplex virus-1 (HSV-1) as the OV, following surgical removal of GBM coupled with radiotherapy. With this combination therapy, we hope to decrease side effects and increase the efficacy of cancer cell killing, ultimately leading to a better treatment strategy for patients with GBM.

10 TUDCA and Tr1 cell supernatants as potential therapeutics to ameliorate intestinal epithelial cell ER stress

Rene Tandun, Enoch Yau, William D. Rees, Theodore S. Steiner

Inflammatory bowel disease (IBD) pathogenesis is associated with a number of cellular and molecular perturbations, including altered inflammatory chemokine and cytokine production, microbial community dysbiosis, and risk-conferring genetic polymorphisms that affect the function of intestinal epithelial cells (IECs). IECs rely on danger signals such as extracellular ATP and endoplasmic reticulum (ER) stress to have a coordinated and robust immune response to pathogens. ER stress in IECs activates the unfolded protein response (UPR), a cellular protection pathway that allows ER stressed cells to return to homeostasis. However, an overactive UPR has been shown to lead to IBD. Recent evidence suggests that Type 1 regulatory (Tr1) cells and the pharmacological inhibitor Tauroursodeoxycholic acid (TUDCA) may have a protective role against ER stress in IECs, although the exact mechanisms of how they ameliorate ER stress, as well as the impact on downstream adaptive immune responses remain largely unknown. We propose that Tr1 supernatant and TUDCA treatments may have a protective effect on cellular ER stress by dampening the UPR, and decreasing flagellin-induced inflammation. Caco-2 cells were pre-treated with Tr1 cell supernatants or TUDCA prior to thapsigargin (Tg) (ER stress inducer) treatment. Subsequently, UPR markers GRP78, CHOP, and Xbp1s/us, were measured via qPCR, and IL-8 and CCL20 chemokine release in response to TLR agonist flagellin (FliC) was measured via ELISA. Caco-2 cells pre-treated with Tr1 supernatants 1 hour prior to Tg treatment did not alter mRNA expression in the UPR pathway, but partially reversed ER-stress-mediated changes in IL-8 and CCL20 expression. Caco-2 cells pre-treated with 100mM- 2μM TUDCA for 2-6 hr prior to overnight Tg treatment and FliC stimulation showed similar protective effects. Collectively, these data suggest that TUDCA or Tr1 cells could be used as potential therapeutics in mitigating ER stress and inflammation in IBD patients.

11 Increasing concentrations of commercial stevia inhibit growth and biofilm formation of *Escherichia coli* MG1655

Al-Rohet Hossain, Derek Lee, Thomas Soroski, Boyan K. Tsankov

Stevia is a readily available, non-caloric sugar alternative with demonstrated non-carcinogenic and non-genotoxic activity. The sweetener is gaining worldwide popularity as a non-nutritive sweetener, with the global stevia market growing about 8% yearly since 2016. However, recent findings suggest that some non-stevia sweeteners may cause adverse health consequences by altering the gut microbiome's composition and diversity, raising concern about stevia. Growth and adhesion are fundamental processes in the microbiome, and previous observations suggest that stevia may impact these processes. To shed light on sweetener-mediated microbiome effects, we investigated the effect of commercial stevia on growth, adhesion, and biofilm formation of *Escherichia coli* MG1655. *E. coli* growth and biofilm formation were significantly inhibited when treated with 40% (v/v) stevia. Gene expression analysis suggested that fimB upregulation may contribute to the observed stevia-mediated biofilm dysregulation. These findings suggest that relatively high concentrations of stevia affect *E. coli* surface-based biofilm formation, and planktonic growth.

12 Biofilm production in *Escherichia coli* K30 with Group 1 capsular gene *wza* and *wza-wzb-wzc* deletions is not correlated with erythromycin resistance phenotypes in liquid media

Cynthia James, Christine Kim, Catherine Pan, Douglas Zhong

The *Wzy*-dependent capsular polysaccharide export system consists of an outer membrane channel (*Wza*), inner membrane channel and tyrosine autokinase (*Wzc*), and regulatory phosphatase (*Wzb*), and is responsible for the export and assembly of extracellular polysaccharide capsules. In *Escherichia coli* K30, this system is critical for the formation of group 1 capsules. Previous studies have shown that *E. coli* K30 Δ*wza* and Δ*wza-wzb-wzc* have greater resistance to erythromycin, a macrolide antibiotic, on solid media compared to wild-type K30; however, the opposite phenotype was observed in liquid media. Therefore, we hypothesized that the different erythromycin resistance phenotypes in *E. coli* K30 wild-type, Δ*wza*, and Δ*wza-wzb-wzc* are due to differences in biofilm production in solid and liquid media. In this study, we investigated whether biofilm formation is correlated with the differential erythromycin resistance phenotype of wild-type *E. coli* K30, Δ*wza*, and Δ*wza-wzb-wzc* in solid and liquid media. Erythromycin disc diffusion assays on solid media, minimum inhibitory concentration assays in liquid media and crystal violet biofilm assays were performed on all three strains. We found that Δ*wza* and Δ*wza-wzb-wzc* were more resistant than the wild-type strain on solid media, but all three strains had similar levels of resistance in liquid media. In contrast to previous studies, we obtained consistent erythromycin MIC values in liquid media and determined that it was between 125 and 250 μg/mL, but we were unable to consistently quantify biofilm formation among our three trials. Thus, we propose that there may not be a correlation between biofilm production in liquid media and erythromycin resistance in *E. coli* K30 wild-type, Δ*wza*, and Δ*wza-wzb-wzc*.

13 Intestinal pH modulates the growth, stress, and virulence of murine pathogen *C. rodentium*

Laurel M. Neufeld, Sarah E. Woodward, B. Brett Finlay

Citrobacter rodentium is an important mouse pathogen model for pathogenic *Escherichia coli*, although much remains unknown about how it navigates the murine gut. As *C. rodentium* travels from the external environment to its niche in the cecum and colon, it encounters distinct gut environments that vary in pH, mucous thickness, nutrient availability, microbial community composition, and much more. Some of these factors represent barriers to infection that *C. rodentium* must overcome in order to colonize its host. Others act as cues that signal the bacteria to upregulate virulence factors and initiate an infection. This thesis aims to characterize how the intestinal environment affects the behavior and virulence of *C. rodentium*. To do this, we screened *C. rodentium* in a variety of previously uncharacterized in vitro conditions that recapitulate aspects of the gut environment, with a particular focus on pH, as closely-related enterohemorrhagic *E. coli* has been observed to increase attachment to host cells and expression of virulence genes in response to acid-induced stress. We identify that gastric pH poses a significant but survivable barrier to *C. rodentium* infections. *C. rodentium* growth dynamics varied across the intestinal pHs tested, with the range of pH 5–7 representing conditions most optimal for growth across varying base media. *C. rodentium* upregulated virulence genes in response to acid-induced stress which, up to a certain threshold, increased attachment to epithelial cells possibly through a mechanism independent of the type III secretion system. However, at a lower pH, the bacteria suffered decreased growth that eclipsed any acid-induced benefit to virulence. The pH range that enabled attachment was extended in gut-mimicking media. Overall, we propose that the response of *C. rodentium* to varying physiological pH throughout the gut involves a careful balance between regulation of growth, stress, and virulence.

14 Resistance to sucralose in *Escherichia coli* is not conferred by mutations in the quinolone resistance determining regions of *gyrA*

Solana Cheng, Sara S. Dalkilic

Sucralose, a synthetic non-caloric sweetener (NCS) commonly consumed in North America, is associated with an increasing number of metabolic disorders that may be the result from interactions between consumed NCS and the gut microbiome. While sucralose has been found to cause bacteriostatic action on various species including *Escherichia coli*, a bacterium commonly found in the lower intestinal tract, the mechanism of action for the bacteriostatic effects of sucralose in the absence of sucrose has not been characterized. However, studies have revealed increased resistance to quinolone antibiotics, inhibitors of DNA *gyrA*se, in sub-inhibitory concentrations of sucralose. Here, we aim to investigate the mechanism of action of sucralose resistance in *E. coli* as it relates to quinolone resistance, by targeting the quinolone resistance-determining region (QRDR) of the *gyrA* gene, the DNA-binding component of DNA *gyrA*se, in *E. coli*. We hypothesized that if *E. coli* DH5α displays resistance in high sucralose concentration, then it is conferred by a mutation in the nucleotide sequence of *gyrA* QRDR. Based on our hypothesis, we selected for a strain of high sucralose concentration (100mM) resistant *E. coli* DH5α and sequenced the *gyrA* gene of our strain. The high sucralose resistance strain was obtained by routine subculturing in TSB with progressively higher concentrations of sucralose. Strains of different sucralose resistance were selected for genomic DNA isolation, amplification, and sequencing for comparison and analysis of our target *gyrA* gene. Our results reveal distinct phenotypic differences between non-sucralose resistant and high sucralose resistant strains, and that the high sucralose resistant strain phenotype and growth pattern was maintained in the absence of sucralose. Additionally, no mutations were found in the QRDR of the *gyrA* gene of the three samples of high sucralose resistant strains. Despite a lack of direct association between sucralose resistance and quinolone resistance in *E. coli*, our study provides insight into alternative mechanisms of action for sucralose resistance.

15 The rate of T7 phage-mediated lysis of *Escherichia coli* growing in exponential phase is not affected by deletion of *rpoS*

Jingyao Zhu, Cai Lan Jennifer Huang, Kamand Doraki, Bryan Lee

RpoS is an *Escherichia coli* sigma factor that has been shown to regulate genes in response to various environmental and internal stressors, particularly in the stationary phase. Previous studies have investigated the role of *RpoS* in a mechanism known as a cross-protection, in which prior exposure to one stressor leads to increased tolerance to a subsequent stressor. Pre-treatment with subinhibitory levels of antibiotics have been used in several studies in an attempt to elicit a delay in phage-lysis, with varying and contradicting results. We propose that the delayed T7-mediated lysis phenotype may be growth phase dependent as the stress response sigma factor *RpoS* is known to be expressed in stationary phase. Here, we attempted to perform phage lysis assays on wild-type (WT) and *rpoS* knockout strains of *Escherichia coli* strains growing in stationary and exponential phase, to look for differences in the time to observe bacteriophage-mediated lysis. We did not observe differences in the time of phage-mediated lysis between wild type and *rpoS* knock-out strains of *E. coli* growing in exponential phase. An attempt was made to compare phage-mediated lysis time of *E. coli* grown in exponential and stationary phase, however, technical issues related to normalizing optical density prevented meaningful comparisons. In conclusion, our study characterizes the *E. coli rpoS* knock-out strain JW5437-1 at the nucleotide level, compares the growth curves of a wild type and *rpoS* knock-out strain of *E. coli*, and shows that phage-mediated lysis times are not different between these strains when growing in exponential phase, which suggests *RpoS* plays no role in this phase.

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