2022
August 12th

VANDERBILT INSTITUTE OF CHEMICAL BIOLOGY
STUDENT RESEARCH SYMPOSIUM

MORNING SESSION (Board of Trust Room, Student Life Center)	
7:30 am – 8:30 am	**Coffee and Light Breakfast**
8:30 am - 8:35 am	**Welcome Remarks: Gary Sulikowski (Director, Vanderbilt Institute for Chemical Biology)**
Oral Session I *Therapeutics, Translation and Chemical Synthesis* (Ruben Torres, Chair)	
8:35 am – 8:55 am	**Eli McDonald (Plate Research Group)** *Molecular Chaperone Recognition of CFTR Measured by Site-Specific Incorporation of Photo-Crosslinking Unnatural Amino Acids*
8:55 am – 9:00 am	**Break**
9:00 am – 9:20 am	**Paige Thorpe (Johnston Research Group)** *Development of an RyR2-Selective Inhibitor as an Antiarrhythmic Agent*
9:20 am – 9:25 am	**Break**
9:25 am – 9:50 am	**Dr. Rebecca Ihrie (Associate Professor of Cell and Developmental Biology and Neurological Surgery)** *Dissecting mTOR Signaling Nodes in Stem Cell Activity*
9:50 am -10:00 am	**Break**
Oral Session II *Molecular Discovery and Systems Analysis* (Emilio Rivera, Chair)	
10:00am-10:20 am	**Mathew Munneke (Skaar Research Group)** *Clostridioides difficile scavenges an unconventional nucleobase during gut colonization*
10:20am-10:25 am	**Break**
10:25am- 10:45 am	**Jonathan Davies (Plate Research Group)** *Comparative Interactomics of SARS-CoV-2 and Homologous Coronavirus Nonstructural Proteins*
10:45am- 10:50am	**Break**
10:50am-11:15 am	**Dr. Dylan Burnette (Associate Professor of Cell and Developmental Biology)** *How Does the Heart Grow? A Cell Biologiest Wants to Know*
11:15am-11:20 am	**Break**
11:20am-11:35am	VICB Citation Winner Flash Talk: **Dr. Robert Coffey** *Reassessment of Exosome Composition*
11:35am – 12:05pm	**Dr. Ian Hardy (Vice President of Chemistry, Manufacturing & Controls, Deerfield)** *Developability - Building Drug-Like Properties Into Molecules to Enable Clinical Success*
LUNCH (Lanford Auditorium Lobby & Light Hall North Lobby)	
12:05 pm – 1:00 pm	**Lunch and Poster Viewing/Judging**
AFTERNOON SESSION (Board of Trust Room, Student Life Center Board of Trust Room)	
Oral Session III *Richard Armstrong Prize for Research Excellence* (Samantha Grimes, Chair)	
1:00 pm - 1:30 pm	**Matthew O'Neill, Prize Runner Up (Roden Lab)** *Contributions of Aberrant Splicing to Inherited Arrhythmia Syndromes*
1:30 pm - 2:00 pm	**Emma Guiberson, Prize Winner (Caprioli Lab)** *Discovery of Bile Acid-Associated Molecular Changes in the Murine Gastrointestinal Tract During C. difficile Infection*
2:00 pm - 2:10 pm	**Break**

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Oral Session IV *Keynote* (Emilio Rivera, Chair)	
2:10 pm - 3:10 pm	**Dr. Christopher Chang (Professor of Chemistry & Molecular and Cell Biology at the University of California, Berkeley; Member of the Helen Wills Neuroscience Institute)** *Activity-Based Sensing and Proteomics Approaches to Decipher Single-Atom and Transition Metal Signaling*
3:10 pm – 3:15 pm	**Break**
3:15 pm – 4:00 pm	**Intellectual Property Enhancing the Impact of Academic Research Panel**
RECEPTION (Langford Auditorium Lobby & Light Hall North Lobby)	
4:00 pm – 5:00 pm	**Poster session**

"Activity-Based Sensing and Proteomics Approaches to Decipher Single-Atom and Transition Metal Signaling"

Christopher J. Chang, Ph.D.
Class of 1942 Chair Professor
Departments of Chemistry and Molecular and Cell Biology
Director, Chemical Biology Graduate Program
University of California, Berkeley

Chris Chang is the Class of 1942 Chair Professor in the Departments of Chemistry and Molecular and Cell Biology at UC Berkeley, as well as a Faculty Scientist in the Chemical Sciences Division of Lawrence Berkeley National Laboratory. He was born in Ames, Iowa and completed his B.S. and M.S. degrees from Caltech in 1997, working with Prof. Harry Gray on spectroscopy of metal-nitrido and metal-oxo complexes. After spending a year as a Fulbright scholar in Strasbourg, France with Nobel Laureate Dr. Jean-Pierre Sauvage on chemical topology, Chris earned his Ph.D. from MIT in 2002 under the supervision of Prof. Dan Nocera, where his graduate work focused on proton-coupled electron transfer and oxygen catalysis. He stayed at MIT as a Jane Coffin Childs postdoctoral fellow with Prof. Steve Lippard, working on zinc biology and then began his independent career at UC Berkeley in 2004.

Research in the Chang laboratory focuses on the study of metals in biology and energy, with particular interest in neuroscience, fat metabolism, cancer, and solar-to-chemical conversion. His group has made fundamental discoveries in inorganic and biological chemistry through developing the concept of activity-based sensing, which exploits molecular reactivity rather than molecular recognition to achieve high selectivity for deciphering chemical processes in biological systems. Probes based on this method have opened a field of transition metal signaling, exemplified by identifying dynamic copper and hydrogen peroxide signals that regulate processes spanning neural activity to fat metabolism. The discovery of copper as a cell signal establishes a new paradigm for metals in biology, expanding the roles of transition metals beyond static metal cofactors in active sites to signaling agents that regulate protein activity at exosites by metalloallostery. His group has also identified single-atom signaling through deciphering reactive oxygen species writers and enzymatic erasers on methionine sites as post-translational modifications. The Chang laboratory has also advanced artificial photosynthesis through bioinorganic catalyst design. His group's work in catalysis has shown that simple molecular mimics of complex enzymes and materials can be used to create new classes of catalysts for solar hydrogen production that feature cheap, earth-abundant elements and operate under environmentally friendly conditions.

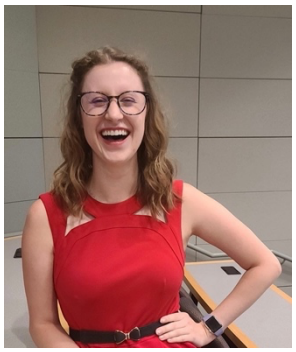
Chris has published over 220 papers (h-index 104) with 15 issued patents, and has given over 360 invited lectures worldwide. He has mentored 41 graduate students, 52 postdocs, 50 undergraduates, and 35 visiting scholars in his laboratory. All trainee alumni are employed in STEM-related fields, and over 40 former group alumni are now in independent faculty positions, including UC Berkeley, Stanford, Chicago, Cornell, and Johns Hopkins in the United States, and Univ. of Hong Kong, Fudan Univ., and Univ. of Sydney abroad. He currently serves as Director of the Chemical Biology Graduate Program and as a founding Senior Editor of ACS Central Science, the flagship open access journal for ACS. His group's research has been honored by awards from the Dreyfus, Beckman, Sloan, and Packard Foundations, Amgen, Astra Zeneca, and Novartis, AFAR, MIT Technology Review (TR35 Award), ACS (Cope Scholar, Eli Lilly Award in Biological Chemistry), RSC (Transition Metal Chemistry), and the Society for Biological Inorganic Chemistry. Included are the 2013 Noyce Prize at UC Berkeley for Excellence in Undergraduate Teaching, 2013 ACS Nobel Laureate Signature Award in Graduate Education, 2013 Baekeland Award, 2015 Blavatnik National Award in Chemistry, election to the American Academy of Arts and Sciences in 2017, 2018 RSC Jeremy Knowles Award, 2019 Sackler Prize in Chemistry, 2020 Humboldt Award, and 2021 Guggenehim Fellowship.

Reassessment of Exosome Composition

Robert Coffey, M.D., Dr. Coffey Received his Bachelors degree in Political Science from Princeton University and went on to obtain his Medical Degree from Georgetown University. The focus of research within the Coffey lab is the study of the role of the EGF receptor (EGFR) and its ligands in gastrointestinal neoplasia. The lab has a particular interest in the trafficking of EGFR ligands in polarizing colonic epithelial cells. This work has led to the identification of a new mode of EGFR ligand signaling via exosomes and the development of FAVS (fluorescence-activated vesicle sorting) to isolate and characterize these extracellular vesicles. The lab discovered that Lrig1, an inducible negative regulator of the EGFR, marks proliferative and quiescent intestinal stem cells, and acts as a tumor suppressor. The lab has recently used CRISPR/Cas9 gene editing to make an Egfr-Emerald reporter mouse that enables direct visualization of the endogenous Egfr. Using a novel 3-D culture system, the lab recently identified a non-genetic cause of cetuximab resistance due to overexpression of a long non-coding RNA, MIR100HG, that confers cetuximab resistance due to increased WNT signaling.

Discovery of Bile Acid-Associated Molecular Changes in the Murine Gastrointestinal Tract Druing C. *difficile* Infection

Emma Guiberson

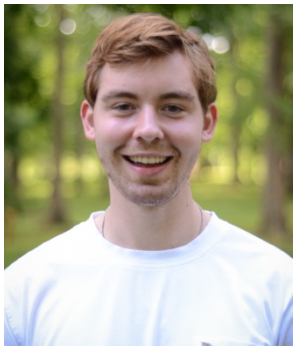


Emma Guiberson, Winner

Clostridioides difficile is a spore-forming pathogen that impacts half a million people annually in the U.S. C. difficile infections (CDI) are characterized by the release of toxins that attack the intestinal linings, disrupting gut flora. Previous C. difficile studies utilized primarily histological techniques, relying heavily on the bacterial impact of CDI rather than the host. Primary bile acids are known germination factors for C. difficile spores, and preliminary data shows increased levels of the bile acid taurocholate in infected species compared to noninfected using imaging matrix-assisted laser desorption/ionization imaging mass spectrometry (MALDI IMS) and liquid chromatography tandem MS (LC-MS/MS). We used these technologies to investigate bile acid abundance changes during infection, and the impact of cholestyramine on spore germination efficacy. MALDI IMS analysis shows a dramatic increase in taurocholate (m/z 514.2946) abundance in the luminal content of infected tissue compared to mock-infected samples and control tissue. LC-MS/MS analysis of liver, small intestine and ceca tissues showed a rapid influx of primary bile acids into the gut, with dramatic differences in abundance of various bile acids occurring as early as 24 hours post infection for both conjugated and unconjugated species. Unconjugated bile acid concentrations decreased significantly by day 3 in the liver, while conjugated bile acids remained consistent throughout the infection. The changes in bile acid abundance are independent of inflammation, and dependent on C. difficile toxins, determined using DSS and toxin knockout models of infection. Toxins alone, however, are not sufficient to cause these bile acid changes without bile acids. To determine the potential impact of this influx of bile acids on C. difficile sporulation, we investigated bile acid abundances, and spore germination, in the presence of a bile acid-sequestering resin, cholestyramine. Results from cholestyramine-treated samples show reduced and delayed host colonization and spore germination when access to elevated bile acid pools is not available. These data indicate that C. difficile infection induces a rapid influx of bile acids into the gut that begins as early as 24 hours post infection, and this influx is necessary for colonization of the host and spore germination.

Contributions of Abberant Splicing to Inherited Arrhytmia Syndromes

Matthew ONeill



Matthew ONeill, Runner up

Inherited arrhythmia syndromes are a major cause of morbidity and mortality in the US. Large-scale genomic studies have helped resolve the genetic basis of these diseases, commonly linking loss-of-function variants in ion channels to clinical phenotypes. While protein-altering missense variants have historically been most studied, splice-disrupting coding and non-coding variants outside the 2-bp canonical splice sites are an increasingly recognized cause of disease. Enabling precision medicine approaches to care for individuals with putative splice-altering variants demands increasingly robust functional assays. We have used minigene and CRISPR-Cas9 edited-induced pluripotent stem-cell cardiomyocytes to functionally annotate the role of these variants in three arrhythmia syndromes – Brugada Syndrome, Long QT Syndrome, and Arrhythmogenic Cardiomyopathy. These functional assays, in conjunction with other variant characteristics, have enabled the reclassification of variants within the American College of Medical Genetics and Genomics framework. To enable high-throughput variant functional interrogations, we are developing Parallel Splice Effect-sequencing (ParSE-seq) to rapidly annotate functional outcomes by leveraging barcoding and Next Generation Sequencing technologies. To move beyond diagnosis, we are exploring chemical genetic interactions in parallel with a variety of therapeutic modalities. We anticipate that these findings will improve precision medicine efforts and provide leads towards a therapeutically tractable class of genetic variation.

Chris Chang, Ph.D., Class of 1942 Chair Professor, Departments of Chemistry and Molecular and Cell Biology, University of California Berkeley

Charleson Bell, Ph.D., Director of Biomedical Innovation, Biodesign, and I-Corps at the Wondry, Vanderbilt's Innovation Center

Dr. Bell is a "Triple 'Dore" earning his Bachelors in Engineering, Masters in Science, and Doctor of Philosophy in Biomedical Engineering – the first African-American to earn a PhD in Biomedical Engineering at Vanderbilt. Dr. Bell's Department of Defense funded research seeks to improve point-of-care deployability, interoperability, and smartphone-compatibility. Dr. Bell's studies on multilayered nanotheranostic approaches to impede the progression of drug resistant bacterial infections, led him to innovate the rapid bacterial diagnostic approach that led to the launch of his startup, BioNanovations Corporation. Dr. Bell has always possessed an entrepreneurial spirit and embraces new endeavors and innovations with great enthusiasm and passion. The first graduate student at Vanderbilt to receive an investment of venture capital to launch a startup, Dr. Bell uses his innovative mind to combine his engineering knowledge to create novel technologies of great impact. As proof of this, he invented BioNanovations Corporation's TestQuick™ and Crystal Innovations' Flash Crystal® in the same year, thus innovating the future of both medicine and media distribution, simultaneously. Thereafter, he was an early innovator in consumer-based Internet of Things (IoT) technology where he was the principal inventor the bluField Network, "the Internet of Bluetooth Technology," which forms fluid networks of low-energy Bluetooth® (BTLE) peripherals for proximity-based, city-wide, smart-tourism applications. He has garnered multiple patents across many fields and co-authored multiple peer-reviewed publications. Most importantly, he is a staunch believer that the value and practice of inclusive innovation is critical to optimize the way innovators empathize with humanity and ideate solutions that create positive change across the world.

Michael Villalobos, Ph.D., Manager, Biotech Licensing, Vanderbilt Center for Technology Transfer and Commercialization

Michael is a seasoned licensing professional with 19 years of experience bringing early-stage life science & medical device technologies to commercial partnerships. He joined CTTC in 2011 and leads a team of licensing professionals responsible for commercializing life science technologies. Prior to joining CTTC, Mike managed the commercialization of pharmaceutical, biotechnology, diagnostic assay and medical device technologies for The Cleveland Clinic and prior to that for Purdue University. During this time frame, he is responsible for over 100 such agreements executed with companies ranging from start-up to multinational large-cap in size and these transactions have resulted in over $20M in near term value. Prior to his career in licensing, he was a Senior Process Scientist at Abbott Laboratories where he oversaw the manufacturing of FDA regulated diagnostic assays for the detection of human blood borne pathogens namely hepatitis B and C. Michael brings an extensive background in the life sciences to his position including a B.S. in Microbiology from the University of Illinois - Urbana and a Ph.D. in Molecular Biology from Loyola University Chicago. He is a member of the Association of University Technology Managers and the Licensing Executives Society.

Ian Hardy, Ph.D., Vice President, Chemistry, Manufacturing & Controls, Deerfield Management

Ian Hardy, Ph.D., joined the Firm in 2019. He is responsible for leading CMC development, manufacturing and supply chain activities for all therapeutic modalities for Deerfield's academic translator companies and some early stage portfolio companies. Dr. Hardy has over 25 years of industry experience leading CMC development activities from preclinical through commercialization and lifecycle management in both large pharma at GlaxoSmithKline, AstraZeneca and Merck, as well as emerging biopharmaceutical companies, most recently Lycera. Dr. Hardy holds a Ph.D. degree in Pharmaceutics from the University of Nottingham and an M.B.A. from Warwick Business School.

Moderator: Tom Utley, Ph.D., Senior Licensing Officer, Vanderbilt Center for Technology Transfer and Commercialization

Tom joined CTTC 10 years ago as a Licensing Analyst and is now a Senior Licensing Officer for life sciences where he is responsible for managing technologies and has front-line responsibility for drafting and negotiating license agreements. This includes working with large and small pharma partners, as well as our larger collaborations with Bayer Pharmaceuticals and Ancora. Prior to joining CTTC, Tom was a post-doctoral fellow at Vanderbilt University in Jeff Conn's Laboratory within the Center for Neuroscience Drug Discovery where he performed *in vitro* compound characterization as well as high-throughput screens for GPCRs. Tom has a diverse background, which include a bachelor's degree in Biochemistry from the University of Illinois at Urbana-Champaign (2002), and a Ph.D. from Vanderbilt University in Microbiology and Immunology (2008). In 2013, he became a registered U.S. Patent Agent and in 2016 he became a Certified Licensing Professional. In 2021, he received his MBA from the Kelley School of Business at Indiana University.

THERAPEUTICS AND TRANSLATION

Molecular Chaperone Recognition of CFTR Measured by Site-Specific Incorporation of Photo-Crosslinking Unnatural Amino Acids

Eli F. McDonald, Minsoo Kim, Hope Woods, Carleen Subusap, Jens Meiler, and Lars Plate

Pharmacological chaperones represent a class of therapeutic compounds for treating protein misfolding diseases. The FDA-approved pharmacological chaperone elexacaftor (VX-445) transformed cystic fibrosis (CF) therapy and represents the first generation of such drugs for CF. CF is a fatal genetic disease caused by mutations in the cystic fibrosis transmembrane conductance regulator (CFTR), an epithelial anion channel comprised of two nucleotide binding domains (NBDs), two transmembrane domains (TMDs), and a regulator domain (RD). The most common CF patient mutation ΔF508 CFTR folds to a near-native state with properly folded domains but is thermodynamically unstable due to impaired domain-domain assembly. Molecular chaperones recognize ΔF508 CFTR as thermodynamically unstable and coordinate premature degradation[1]. By contrast, pharmacological chaperones stabilize ΔF508 CFTR sufficiently to change molecular chaperone recognition[1], yet how unfolding gives rise to molecular chaperone recognition remains unclear. We developed a chemical biology approach for identifying molecular chaperones interactions on a domain and subdomain basis using affinity purification mass spectrometry.

We site-specifically incorporate photo-crosslinking amino acids into different domains and subdomains in CFTR to determine which domain are unfolded in ΔF508 and subsequently recognized my molecular chaperones. The photo-crosslinking groups covalently capture interacting proteins. We use Tandem Mass Tag (TMT) labeled quantitative affinity purification mass spectrometry (AP-MS)-based interactomics to identify and quantify crosslinked proteins. Each TMT label gives a sample a unique quantitative signature that allows us perform pair wise statistical analyses between cross linked and non-crosslinked samples. This allows us to quantitatively compare relative site-specific chaperone binding between WT and ΔF508 CFTR. Our method reveals distinct protein interactions for WT and ΔF508 CFTR. Specifically, we show preferential binding of Hsp90 to ΔF508 as shown in previous studies. Site specific incorporation of photochemical crosslinkers into CFTR can covalently capture protein interactions identifiable and quantifiable by AP-MS. This method allows for quantitative distinction of chaperones involved in evaluating ΔF508 stability. Since CFTR stability is increased by VX-445, we plan to combine VX-445 treatment with site-specific crosslinking to determine which chaperone interactions are changed under drug conditions.

CHEMICAL SYNTHESIS

Development of an RyR2-Selective Inhibitor as an Antiarrhythmic Agent

Madelaine P. Thorpe, Abigail N. Smith, Daniel A. Blackwell, and Jeffrey N. Johnston

Sudden cardiac death due to ventricular arrhythmias is a major public health concern, accounting for 10-20% of all deaths in adults in the United States. Current therapeutics for these fatal arrhythmias are underdeveloped, fail to target the underlying disease cause, and lack the mechanism of action to increase longevity and quality of life for patients. ent-Verticilide is a selective and potent inhibitor of an intracellular calcium channel, RyR2. By probing the SAR of this cyclic oligomeric depsipeptide which lies in BRo5 (Beyond Rule of 5) chemical space, we are engaged in hit-to-lead studies while attempting to better understand its passive permeability via PAMPA assay. Our progress in the development of ent-verticilide as a therapeutic will be described.

SYSTEMS ANALYSIS

Comparative Interactomics of SARS-COV-2 and Homologous Coronavirus Nonstructural Proteins

Jonathan P. Davies, Katherine M. Almasy, and Lars Plate

Human coronaviruses (CoVs) are a threat to global health and society, as evident from the SARS outbreak in 2002, the MERS outbreaks in 2012 and 2014, and the most recent COVID-19 pandemic. Despite the sequence similarity between severe disease-causing CoVs, each strain has distinctive virulence. A better understanding of the basic molecular mechanisms mediating changes in virulence is needed. Here, we profile the virus-host protein-protein interactions of three CoV nonstructural proteins (nsps) that are critical for virus replication. We use tandem mass tag-multiplexed quantitative proteomics to sensitively compare and contrast the interactomes of nsp2, nsp3, and nsp4 from three severe disease-causing CoV strains (SARS-CoV-2, SARS-CoV, MERS-CoV) and two common-cold CoVs (hCoV-OC43, hCoV-229E). This approach enabled us to identify both unique and shared host cell protein interactors and quantitatively compare the enrichment of interactions between homologs. We find both nsp2 and nsp4 are enriched for proteins associated with ER-mitochondria contact sites, ER membrane remodeling, and protein biogenesis, and these interactions are conserved across multiple CoV homologs. Further functional genetic screening reveals these as pro-viral factors for CoV infection. Nsp3 homologs showed more strain-specific interactor profiles, including interactions with nuclear import machinery for hCoV-229E and ribosomal RNA processing for MERS-CoV. Lastly, we find that SARS-CoV-2 nsp3 interacts with the transcription factor ATF6, a regulator of the Unfolded Protein Response, and can suppress the ATF6 stress response. Our results shed light on new roles for CoV nsps in modifying host proteostasis processes to drive viral infection, as well as host factors that may mediate the divergent pathogenesis of common cold CoVs from SARS/MERS strains. Our mass spectrometry workflow enables rapid, robust comparisons of multiple virus proteins and identification of common CoV-host dependencies to be targeted by host-directed anti-viral therapeutics.

MOLECULAR DISCOVERY

***Clostridioides difficile* Scavenges an Unconventional Nucleobaseduring Gut Colonization**

Matthew J. Munneke, Valerie de-Crecy Lagard, & Eric P. Skaar

Clostridioides difficile is the leading cause of antibiotic-associated nosocomial infections and is responsible for nearly 500,000 infections and 20,000 deaths annually in the United States. *C. difficile* infection (CDI) typically proceeds following disruption of the gut microbiota. The incidence and recurrence of CDI has increased in the last decade due to the limited efficacy of current therapeutic strategies. This highlights the need to develop an understanding of the factors that allow *C. difficile* to colonizethe gastrointestinal tract. In the vertebrate host, *C. difficile* faces competition for nutrients that involves both the gut microbiota and the host immune system. Vertebrate colonization requires mechanisms to maintain metabolic homeostasis in these dynamic nutrient environments. Amongst the nutrients depleted following perturbation of the gut microbiota are nucleobases, and synthesis or salvage of nucleobases is critical for pathogenesis. We hypothesize that *C. difficile* possesses a unique repertoire of metabolic mechanismsto salvage nucleobases during infection. We have discovered that *C. difficile* is capable of utilizing 4-thiouracil (4-TU) as a uracil source, an abundant unconventional nucleobase in the vertebrate gut. Growth of *Escherichia coli* is inhibited by 4-TU, anduracil supplementation alleviates lethality. These data indicate that 4-TU is misincorporated into RNA, leading to toxicity. The metabolism of 4-TU is mediated by proteins containing domain of unknown function 523 (DUF523). Notably, *E. coli* lacks a DUF523 homolog, while *C. difficile* expresses two DUF523 paralogs, CD196_RS03875 (3875) and CD196_RS15345 (15345). Using a *C. difficile* strain auxotrophic for uracil, we provided 4-TU as the sole source of uracil to a strain lacking either *3875* or *15345*. We discovered that *15345* is dispensable for utilization of 4-TU as a uracil source but *3875* is required.These data suggest that paralogous DUF523 domain containing proteins in C. difficileare functionally distinct, andwe hypothesize that *3875* functions to detoxify 4-TU to uracil. Indeed, heterologous expression of *3875* in *E. coli* is sufficient to protect from 4-TU cytotoxicity. These data increase our understanding of the relationship between 4-TU and DUF523, anunderstudiedmolecule and enzymefamily, respectively.Experiments are on-going to investigate the role of 4-TU and DUF523 during CDI.

CHEMICAL SYNTHESIS

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Zach Austin
Zihang Deng
Rachana Tomar
Karen Arora
Payton Stone

SYSTEMS ANALYSIS

Adam Ebert
Amanda Cao
Donald Stec
Emanuel Zilbut
Hualiang Pi
Kayla Hess
Velia Garcia

MOLECULAR DISCOVERY

Jonah Zarrow
Katherine Clowes
Crissey Cameron
Valeria Garcia Lopez
Jenny Tran
Raven Dean
Jeanette Miller
Yufan Shan

THERAPEUTICS & TRANSLATION

Sarah Zelle
Catherine Leasure
Christopher Hansen
Brynn Roman
Julie Talbert

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