

# IBASM NEWSLETTER

Volume 20, Issue 1 August, 2017

### Greetings from the President: Tanya Soule

hope you are all having a great summer! Thank you to everyone for making our meeting at Turkey Run State Park a great success! In addition to our branch lecturer Dr. Vin-

cent Young from Michigan State University we hosted ASM Past-President Dr. Lynn Enquist from Princeton University and our Research Award recipient, Dr. Frank Yang from Indiana University School of Medicine. It was great to learn about the microbiome from Dr. Young, herpesvirus particles and imaging from Dr. Enquist, and spirochete diseases from Dr. Yang. It was a well-attended meeting with diverse topics from all subdisciplines in microbiology and immunology.

Mark your calendars for April 6-7, 2018 for our next annual meeting at the University of Indianapolis. We encourage you all to arrive in time to set up your posters for viewing during the Friday night session. We are currently finalizing speakers for the meeting but I'm certain you will not be disappointed!

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#### **Message from the President-Elect - Douglas Stemke**

Welcome, fellow microbiologist, to another year of Hoosier microbiology!

In the face of emerging infectious diseases, antibiotic drug resistance, industrial developments using novel microbes (GM or otherwise), professional needs for microbial sciences in health care, food, and material sciences, and as a body dedicated to the principles of discovery science, IBASM continues the tradition of gathering Indiana microbiologists for our annual meeting. The 2018 IBASM meeting will be held April 6-7 at the Heath Pavilion building on the campus of the University of Indianapolis (UIndy).



As we do every year, we are looking forward to seeing the research being conducted by undergraduate, graduate, and postdoctoral microbiologists from across the state. It is a wonderful opportunity to show off and discuss your findings in a supportive environment. Poster and oral presentations are encouraged to apply, however due to time constraints, the meeting will only be able to present a limited number of oral presentations. Instructions for applying for presentations will be in the next issue of the newsletter. Even if you aren't presenting, faculty, students, health care professionals, those in industry (and beyond) with interests in microbiology are encouraged to attend the annual IBASM meeting.

Our 2017 meeting reflected this philosophy. A round table discussion led by speakers from industry and the State Health Lab suggested several career choices, outside of academia, that require highly trained microbiologists. The speakers discussed both practical information how someone can attain such careers and the personal satisfaction such careers have had in their lives. Our invited speakers were equally engaging. Dr. Vincent Young (University of Michigan), and past ASM president Dr. Lynn Enquist, traveled to the meeting from their respective labs to enlight-ened IBASM attendees of findings from their labs where as Dr. Frank Yang's talk "How Bacterial Pathogens Adapt to Both Vector and Mammalian Hosts" concluded the invited talks from a Hoosier perspective. Last, but certainly not least, the many fine posters and talks presented by students made for an outstanding 2017 meeting.

A challenge for our newsletter readers. Do you know, or is there on your campus, an outstanding microbiology professor that you would like to be considered for the annual "Microbiology Teacher of the Year?" We encourage nominations for this prestigious award. Details are in this newsletter.

Congratulations to Janine Bennett and Ahmed Hassan as our new student representative for the 2018 year. IBASM would not be the success that it is without the dedication and hard work by our student representatives and volunteers.

Finally, as the newly elected President-Elect, I want to thank current and past presidents Dr. Tanya Soule and Dr. Nancy Magill for their years of service to IBASM. I am humbled to continue the work done by these hard-working members of IBASM as well as all the other members through the years.

Look forward to seeing you at UIndy, 2018!

### **Special Thanks to All Judges!**

We would like to extend a special note of thanks to all those who gave of their time to judge posters for the Spring meeting at Turkey Run State Park. Students were evaluated in five categories: professional appearance, scientific thought, creativity, thoroughness, and presentation (abstract, oral and poster). This was no easy task! Next time you see any of these persons please thank them for sweating through a very difficult challenge:

Judging Team #1: Undergraduate Division = R. Gregory (IUSD), N. Magill (IUB), M. Sattley (IWU), D. Wu (PU), S. Daniel (EIU), F. Yang (IUPUI) Judging Team #2: MS Division = G. Hardy (IUB), D. Stemke (UIndy), L. Enquist (PU, NJ) Judging Team #3: Ph.D. Division = S. Bates (Purdue Northwest), R. Sparks-Thissen (USI), R. . Daniel (IPWF)

# From the Desk of John McKillíp...Educational

# Representative



We had a total of 21abstracts, including a wonderful showing from Purdue University for the second year in a row! The number and level of undergraduate presentations was outstanding.

Please see our accompanying research paper summations in this issue and the next issue of the newsletter from winners in each category (Undergraduate, M.S., & Ph.D. Divisions).

Congratulations to all! We hope that you and your students will plan to submit an abstract for our meeting next Spring. The discussion which occurred during the open poster session was outstanding, and a great opportunity for students to visit with each other and also interact with professionals who can provide valuable ideas and advice for future education and employment. All of us who viewed the poster segment and attended oral presentations look forward to even a greater number of participants next year. Winners received a certificate and most have already received a monetary gift when their research summation is published in the IBASM newsletter.

As a note of interest to undergraduate students and their mentors – if you are interested in submitting your research for publication, consider *Fine Focus* (see advertisement in this issue of the IBASM Newsletter)!

#### Hello all,

My name is Ahmed, I am starting my third year of my PhD degree at Purdue University working on antibacterial drug discovery. I am very happy and excited to serve with Janine as graduate student representatives for the IBASM during the next term. We will be glad to work with all IBASM members to improve our experience throughout the different career paths. I am sure you all will have great contributions and excited to hear, report and try to apply these thoughts through the IBASM committee. Please feel free to contact us and we will be there to help and support.

Cheers,

Ahmed.

Hassan23@purdue.edu



#### Message from the Undergraduate Student Representative - Janine Bennett



Hello everyone and welcome to a new academic year! I hope you all had a fantastic summer. My name is Janine Bennett. I am an undergraduate student in my senior year majoring in Biology with a concentration in Microbiology/Immunology at Indiana University-Purdue University (IPFW). This will be my first year serving on the Indiana Branch of the American Society for Microbiology (IBASM) committee and I look forward to serving as the voice of the student community. If you have any suggestions, questions, or concerns, please feel free to contact me at bennjl02@students.ipfw.edu.

# Call for Nominations: IBASM Academic Teaching Award 2018

Do you have a great professor who deserves a teaching award? We call on all student IBASM members to nominate their favorite lecturer/instructor for the IBASM Academic Teaching Award 2018. Your nomination letter should explain why you think your teacher deserves the award. Please provide as many details as possible. Also, your letter will carry more weight if you can get some of your peers to co-sign it.

The IBASM Awards Committee consisting of Dominique M Galli (IU School of Dentistry), John McKillip (Ball State University), and Doug Stemke (University of Indianapolis) will select the awardee based on your letter and additional information obtained from the nominee's departmental chair. The award will be presented at the IBASM Annual Spring Meeting in 2018 where the awardee will be expected to give an oral presentation. Note that the awardee must be a member of the IBASM at the time the award is received. Please send your nominations to dgalli@iupui.edu on or before November 3, 2017.



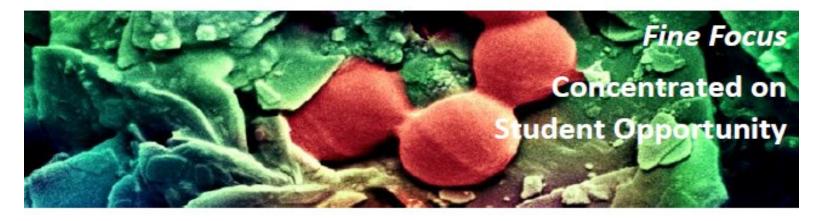
We call on all IBASM members to nominate their colleagues for the IBASM Academic Research Award 2018. At the time of nomination candidates should have been actively engaged in a nationally recognized research program for at least 5 years as a faculty member of an Indiana college or university. Evidence of a track record of scholarly work and significant external funding as a Principal Investigator will need to be provided. Please attach the nominee's short CV to the nomination letter. Note, the nominee does *not* have to be a member of the IBASM.

The IBASM Awards Committee consisting of Dominique M Galli (IU School of Dentistry), John McKillip (Ball State University), and Doug Stemke (University of Indianapolis) will select the awardee based on the nomination letter and information provided in the CV. The award will be presented at the IBASM Annual Spring Meeting in 2018 where the awardee will be expected to give an oral presentation. Please send your nominations to <u>dgalli@iu.edu</u> on or before <u>November 3</u>, 2017.

# 2017 IBASM Membership Application/Renewal

If you have not done it already, it is time to pay your IBASM dues for 2017. You can do it either online when you pay your dues to the ASM National Organization (<u>www.asm.org</u>) or by using this form. Dues are \$15.00 for non-students and \$5.00 for students (per year). Please return the completed form with check, payable to IBASM, to

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2300 South Washington Street		
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Phone: (765) 455-9371; email: <u>cchauret@iuk.edu</u>		
Please check:		
New Member Application		
Renewal for 2017		
and		
Student Member for 2017 (\$5)		
Full Member for 2017 (\$15)		
Name:		
Current Position & Title:		
Institution:		
Mailing Address (new address: Yes/No?)		
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Email:		
Fax: National ASM Member #:		
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Highest Degree:		
Institution:		
Professional Interests:		



#### -----CALL FOR MANUSCRIPT SUBMISSIONS------

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#### Reversal of Azole Resistance in Candida albicans by Sulfa Antibacterial Drugs

Hassan Eldesouky\*, Abdelrahaman Mayhoub, Tony Hazbun, Mohamed N. Seleem

Department of Comparative Pathobiology, Purdue University College of Veterinary Medicine, West Lafayette, IN.

#### **Summary**

Invasive candidiasis presents an emerging global public health challenge due to the emergence of resistance to the frontline treatment options, such as fluconazole. Furthermore, the development and approval of new antifungal agents has not been able to contend with the emergence of *Candida* resistance to current antifungal drugs, thus compounding the problem. Hence, the identification of other compounds capable of pairing with fluconazole and averting azole-resistance would potentially prolong the clinical utility of this important group. In an effort to repurpose drugs and explore new leads in the field of antifungal drug discovery, we explored sulfa antibacterial drugs for the purpose of reverting azole resistance in *Candida*.

In this study, we assembled and investigated a library of 21 sulfa antibacterial drugs for their ability to restore fluconazole sensitivity in *Candida albicans*. The results of Checkerboard assays showed that 15 sulfa drugs were able to exhibit synergistic relationships with fluconazole ( $\Sigma$ FIC values ranged from < 0.0312 to < 0.25). Interestingly, five sulfa drugs, were able to revert azole resistance in a clinically achievable range blew their achievable plasma therapeutic concentrations. A growth kinetic curves were utilized to confirm the in vitro activity of different sulfa-fluconazole. The growth curves showed that neither sulfa drugs nor fluconazole were solely able to inhibit the growth of yeast cells, only sulfafluconazole combination did reduce the growth significantly.

The growth curves showed that neither sulfa drugs nor fluconazole were solely able to inhibit the growth of yeast cells, only sulfa-fluconazole combination did reduce the growth significantly. In addition, we examined the structure-activity-relationships (SAR) of the amino benzene sulfonamide scaffold as antifungal agents. We found that two chemical features must be present in sulfa drugs, in order to act synergistically with fluconazole. The sulfa molecule must maintain the anile amino group unsubstituted, in addition, the N1 must be substituted with aromatic system. Also, it was found that the electron properties of the aromatic system plays a major role in the potency of the interaction between different sulfa drugs and fluconazole.

We also identified the possible mechanism of the synergistic interaction of sulfa antibacterial drugs with azole antifungal drugs. By using a rhodamine efflux assay, sulfa drugs didn't attenuate the efflux machinery of the cell and failed to accumulate the dye intracellularly, indicating that the mode of synergy doesn't involve efflux inhibition. On the other hand, we found that, in a dose dependent manner, the addition of para amino benzoate to the assay medium (PABA), resulted in a complete loss of the synergistic activity between sulfa drugs and fluconazole. Both sulfa drugs and PABA are structurally similar and compete each other for the active site of the dihydropteroate synthase enzyme (DHPS), which plays an important role in the folate biosynthesis pathway. These results suggest that sulfa drugs in combination with fluconazole are able to interfere with folate pathway in a similar way as seen in bacteria. This hypothesis was supported by the fact that, both bacterial and fungal DHPS has a similar active site [1]. Furthermore, the ability of sulfa antibacterial drugs to inhibit *Candida* biofilm was confirmed. In addition, the effect of sulfa-fluconazole combination on *Caenorhabditis elegans* infection model, was utilized and it was found that sulfa-fluconazole combinations were able to significantly reduce the fungal burden per worm.

Animal study in higher organisms is required to further confirm the clinical importance of sulfa drugs in overcoming the azole resistance in *C. albicans*. Also, intense molecular studies are required to fully understand the folate pathway in order to be utilized as an important selective target in yeast.

#### References.

1.Otzen, T., et al., Folate-synthesizing enzyme system as target for development of inhibitors and inhibitor combinations against Candida albicans-synthesis and biological activity of new 2,4-diaminopyrimidines and 4'-substituted 4-aminodiphenyl sulfones. J Med Chem, 2004. **47**(1): p. 240-53.

### McClung First Place Graduate (M.S. Division) Winner

#### Biochemical Characterization of a Putative Glycerol-3-Phosphate Acyltransferase from Mycobacterium tuberculosis

**Jamison Law** and Jaiyanth Daniel Department of Biology, Indiana University-Purdue University Fort Wayne, Fort Wayne, IN.

#### Introduction

Tuberculosis (TB) is a disease caused by *Mycobacterium tuberculosis* (*Mtb*) which latently infects 2-3 billion people and was responsible for 1.4 million deaths in 2015 [1]. After infecting alveoli, the immune system isolates *Mtb* within a hypoxic granuloma, forcing *Mtb* into dormancy where it survives by synthesizing TAG [2,3]. Glycerol-3-phosphate acyltransferase (mGPAT) is responsible for esterifying the first fatty acid to the glycerol-3-phosphate backbone in the TAG biosynthetic pathway and is probably essential for the survival of dormant *Mtb* [3,4]. Inhibition of mGPAT would prevent phospholipid and TAG biosynthesis, thus starving the bacteria. Therefore, our research will focus on the biochemical and functional characterization of mGPAT.

#### Methods

**Protein Expression:** *E.*  $coli \pm$  mGPAT were induced with IPTG, cells were lysed, and equal protein concentrations, as determined by Bradford assay, of each sample were resolved by SDS-PAGE.

**Lysate Assay:** *E. coli*  $\pm$  mGPAT were induced with IPTG, cell lysates were incubated in reaction mix containing <sup>14</sup>C-glycerol-3-phosphate and palmitoyl-CoA, lipid products were resolved on a TLC plate, the plate was exposed to photographic film, and radioactivity of lipids was quantified.

**Pure Enzyme Assay:** *E. coli*  $\pm$  mGPAT induced with IPTG, cells were lysed, mGPAT was solubilized from the lysate pellet, mGPAT was purified using a metal-affinity chromatography column and a molecular weight cutoff, and pure mGPAT was assayed as described above.

**Metabolic Labeling:** *E. coli*  $\pm$  mGPAT were induced with IPTG in LB or media containing fatty acids, aliquots were incubated with <sup>14</sup>C-palmitate or <sup>14</sup>C-acetate, and lipids were extracted, resolved, and quantified as described above. Cells grown in media containing fatty acids were washed to remove fatty acids prior to incubation with radiolabeled substrates.

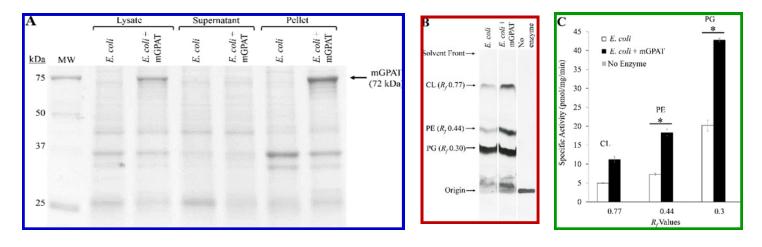
**Growth Experiments:** *E. coli*  $\pm$  *mGPAT* were cultured in either LB broth or media containing fatty acids at 37°C in a shaker, cultures were grown to an optical density (OD<sub>600</sub>) of 0.6 to 0.8 whereupon they were induced with IPTG, and growth was monitored up to 72 hours by OD spectrophotometry and dilution plating.

#### Results

mGPAT was observed in the lysate and pellet fractions of *E. coli* expressing mGPAT, but not in any of the fractions from *E. coli* lacking mGPAT (Fig. 1a). *E. coli* lysates expressing mGPAT catalyzed increased formation of phospholipids (Fig. 1b,c).

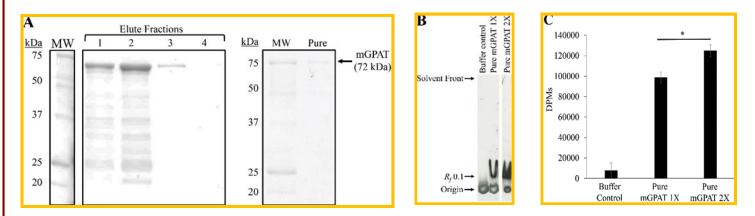
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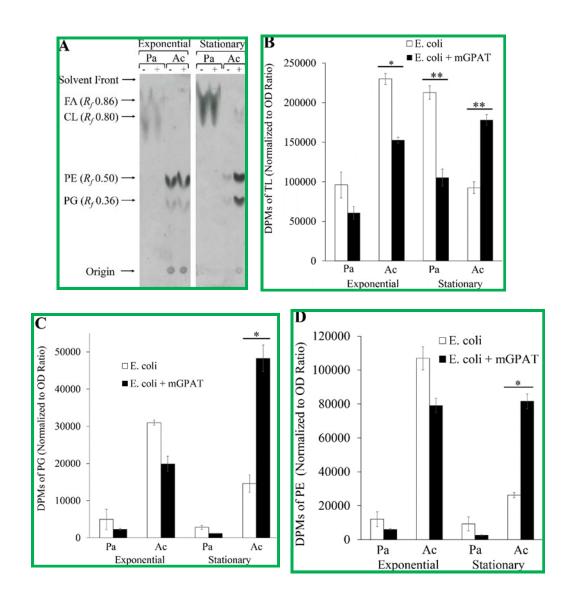
**Figure 1:** A) SDS-PAGE of *E. coli* + mGPAT lysate, supernatant, and pellet fractions. B) Autoradiogram and C) specific activity of *E. coli* + mGPAT lysates showing formation of cardiolipin (CL), phosphatidyleth-anolamine (PE), and phosphatidylglycerol (PG). Averages + standard deviation. A student's t-test was performed: \*, p < 0.05.

Bound mGPAT was eluted from the metal-affinity chromatography column with impurities and low concentrations of pure mGPAT were obtained, with minor impurities, following buffer-exchanging (Fig. 2a). Purified mGPAT formed an unknown product that was not present in the buffer control (Fig. 2b,c).



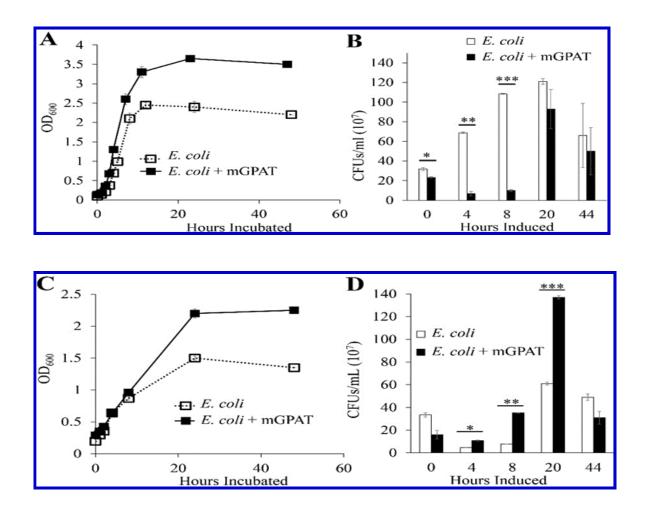
**Figure 2:** A) Purification of mGPAT, B) autoradiogram showing radiolabeled lipid product formed by pure mGPAT, and C) quantification of radioactive products. Average + standard deviation. A student's t-test was performed: \*, p < 0.05.

Incorporation of <sup>14</sup>C-palmitate into total lipids appeared to be decreased during exponential and stationary phase and incorporation of <sup>14</sup>C-acetate into PE and PG was significantly increased during stationary phase when *E. coli* expressing mGPAT were grown in LB (Fig. 3).



**Figure 3:** A) Autoradiogram showing incorporation of 14C-palmitate (Pa) and 14C-acetate (Ac) into lipids by E. coli lacking (-) and containing (+) mGPAT when grown in LB and quantitation of B) total lipids (TL), C) PG, and D) PE. Average + standard deviation. A student's t-test was performed: \*, p < 0.05; \*\*, p < 0.01.

Optical density data showed a significantly higher growth for *E. coli* expressing mGPAT during the stationary phase regardless of the media (Fig. 4a,c). Dilution plating demonstrated that the growth of *E. coli* expressing mGPAT was decreased during the exponential phase when cultured in LB (Fig. 4b), and increased when cultured in LEM as compared to *E. coli* lacking mGPAT (Fig. 4d).



**Figure 4:** A) OD and B) colony forming units per ml (CFUs/ml) of *E. coli* + mGPAT when grown in LB and C) OD and D) CFUs/ml of *E. coli* + mGPAT when grown in LEM. Averages + standard deviations. A student's t-test was performed: \*, p < 0.05; \*\*, p < 0.01; \*\*\*, p < 0.001.

#### Discussion

- \* Assay of *E. coli* lysates expressing mGPAT suggests that mGPAT is an active enzyme that probably forms precursor substrates used for phospholipid metabolism.
- \* Assay of relatively pure mGPAT led to the formation of an unidentified product, which may be due to protein misfolding or could have resulted from the activity of minor contaminants.
- \* Metabolic labeling suggests that the presence of mGPAT enhances the ability of *E. coli* cells to incorporate <sup>14</sup>C-acetate into PE and PG during stationary phase when grown in LB media.
- \* Growth experiments suggest that mGPAT enhances the ability of *E. coli* cells to incorporate exogenously supplied fatty acids to achieve greater growth when cultured in media containing fatty acids.

#### References

- 1. World Health Organization. 2016. Global tuberculosis report 2016. Geneva, Switzerland.
- Korb VC, Chuturgoon AA, Moodley D. 2016. *Mycobacterium tuberculosis*: manipulator of protective immunity. Int J Mol Sci 17(3):131.
- Daniel J, Maamar H, Deb C, Sirakova TD, Kolattukudy PE. 2011. *Mycobacterium tuberculosis* uses host triacylglycerol to accumulate lipid droplets and acquires a dormancy-like phenotype in lipid-loaded macrophages. PLoS Pathogens 7.
- **4.** Crellin PK, Luo C-Y, Morita YS. 2013. Metabolism of plasma membrane lipids in Mycobacteria and Corynebacteria, Lipid Metabolism doi:http://dx.doi.org/10.5772/52781.



### Second Place Undergraduate Division Winner

# The *E. coli* Inner Membrane Protein YhiM is Necessary for Efficient Attachment of Bacteriophage T4

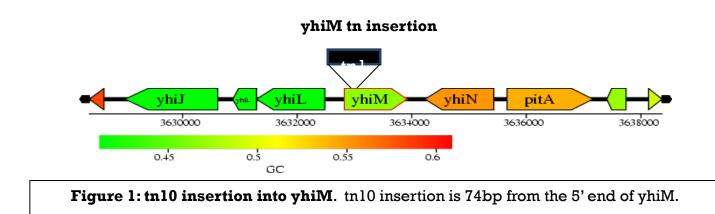
**P.T. Spieth**, M.A. Evans, and R.L. Sparks-Thissen Department of Biology, University of Southern Indiana, Evansville, IN.

### Introduction

Previous research has shown that YhiM is a conserved inner membrane protein found in *E. coli*. It is upregulated under conditions of acid stress (*J. Bacteriol* 184: 6551), low temperature (*J. Bacteriol*. 191:1106), and high osmolarity (*Cell* 90: 43). Additionally, YhiM is necessary for growth in low pH (*Arch. Micro.* 194: 637), low osmolarity, and high temperature environments (*Arch. Micro.* 199:171). We previously found that YhiM is required for replication of a number of bacteriophages, leading us to wonder what role it plays in T4 replication in *E. coli*. We chose to study attachment of T4 to YhiM-deficient bacteria because attachment is the first stage of infection.

### Methods

In order to evaluate T4 attachment, we performed attachment assays to compare T4 attachment in wild type and YhiM-deficient ( $\Delta$ YhiM) *E. coli* at various multiplicities of infection. Wild type and  $\Delta$ YhiM mutant cells were grown overnight in H-broth. They were then diluted 1:1000 into H-broth and grown to an OD<sub>600</sub> of 0.1 to 0.2 at 37°C. These cells were added to T4 phages and T-broth. At 0, 5, and 10 minutes post-



### Results

Our data indicates that YhiM is necessary for T4 replication. T4 was not able to infect  $\Delta$ YhiM cells, as seen in Figure 2. At an MOI 0.1, we see a significant decrease in attachment in YhiM-deficient cells compared to wild type. A one thousand fold increase in virus concentration was then used in an attempt to rescue the phenotype; attachment was still minimal (Figure 3). This demonstrates that regardless of the amount of virus present, T4 does not attach efficiently to *E.coli* cells without functional YhiM. By adding a plasmid containing functional YhiM to YhiM-deficient cells, the attachment phenotype is rescued (Figure 4). This indicates that the inefficiency in attachment is due to a lack of YhiM.

FIG. 2. Dilutions of T4 were titered on plates containing either wild type or  $\Delta$ YhiM cells. The absence of plaques on the  $\Delta$ YhiM plate demonstrates that T4 did not infect the  $\Delta$ YhiM cells. The presence of plaques on the wild type plate indicates that T4 infection successfully occurred in the wild type cells.

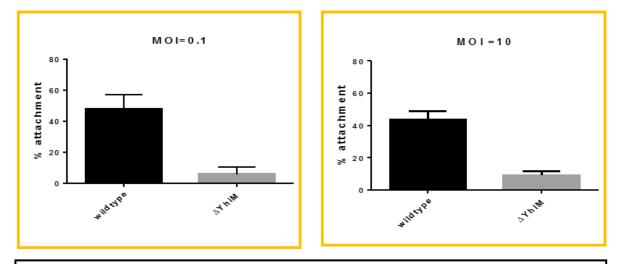


FIG. 3. % attachment of T4 bacteriophage to wild type *E. coli* (black) compared to YhiM-deficient ( $\Delta$ YhiM) cells (grey) 10 minutes after infection at a ratio of 1 T4 phage to every 10 WT or  $\Delta$ YhiM cells (MOI=0.1) p=0.0013 or at a 10 T4 phages to every 1 WT or  $\Delta$ YhiM cell (MOI=10) p=0.0026.

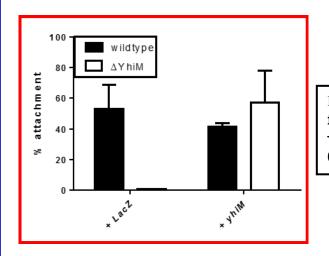


FIG. 4. **T4 attachment is rescued in**  $\triangle$ YhiM cells, when functional YhiM is present, supporting that YhiM is involved in T4 attachment. Lac Z: p= 0.0420, YhiM : p= 0.4119.

### Conclusions

The data in Figures 2 and 3 demonstrates that a lack of functional YhiM significantly decreases T4 attachment to *E. coli* cells. This suggests that YhiM may be involved in one or more attachment-related pathways. The data in Figure 4 indicates that the addition of functional YhiM rescues the  $\Delta$ YhiM phenotype, allowing T4 to efficiently attach to these cells.

### **Future Experiments**

- OmpC has previously been shown to be involved in T4 attachment to *E. coli* (Calendar 2006). We are interested in determining the level of gene expression in ∆YhiM cells compared to wild type cells.
- T4 can attach to *E. coli* through an OmpC-independent pathway involving heptose residues of LPS (Calendar 2006). The genes rfaE and rfaD are involved in heptose residue metabolism of the LPS core (Schnaitman and Klena 1993). We plan to look at the levels of gene expression of rfaE and rfaD in ∆YhiM cells compared to wild type cells.
- We plan to assess attachment of T7 to ∆YhiM cells and compare this data to our T4 attachment data in order to determine if a lack of YhiM also affects T7 attachment.



Additional award papers

will be published in the next

issue of the newsletter.

# Photos from the 2017 Annual Meeting



Dr. Frank Yang, recipient of the 2017 research award

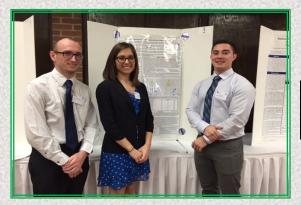


Attendees from the IU School of Dentistry



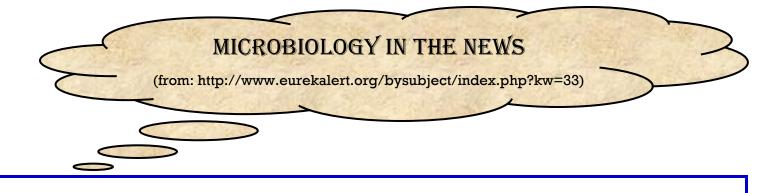


ASM Past-President **Dr. Lynn Enquist's** presentation on mechanisms of herpesvirus pathogenesis



T. Danek, P. Patinich, and C. Carter from Eastern Illinois University with their poster

# Additional photos will be published in the next issue



### Compounds in desert creosote bush could treat giardia and 'braineating' amoeba infections

PLOS Neglected Tropical Diseases August 15, 2017

Researchers at Skaggs School of Pharmacy and Pharmaceutical Sciences at University of California San Diego and the University of Colorado Anschutz Medical Campus have found that compounds produced by the creosote bush, a desert plant common to the Southwestern United States, exhibit potent anti-parasitic activity against the protozoa responsible for giardia infections and an amoeba that causes an often-lethal form of encephalitis.

### NIAID herpesvirus study in mice leads to discovery of potential broadspectrum antiviral

#### mBio

#### August 15, 2017

After herpesviruses infect a cell, their genomes are assembled into specialized protein structures called nucelosomes. Many cellular enzyme complexes can modulate these structures to either promote or inhibit the progression of infection. NIAID scientists studying how one of these complexes (EZH2/1) regulated herpes simplex virus (HSV) infection unexpectedly found that inhibiting EZH2/1 suppressed viral infection. The research group, from the National Institute of Allergy and Infectious Diseases (NIAID) at the National Institutes of Health, then demonstrated that EZH2/1 inhibitors also enhanced the cellular antiviral response in cultured cells and in mice.

### Test uses nanotechnology to quickly diagnose Zika virus

#### *Advanced Biosystems* August 10, 2017

Currently, testing for Zika requires that a blood sample be refrigerated and shipped to a medical center or laboratory, delaying diagnosis and possible treatment. Although the new proof-of-concept technology has yet to be produced for use in medical situations, the test's results can be determined in minutes. Further, the materials required for the test do not require refrigeration and may be applicable in testing for other emerging infectious diseases.

# **Important Dates**

March 2018:	Registration & abstract forms due for 2018
	Annual IBASM meeting
April 6 - April 7, 2018:	Annual IBASM meeting, UIndy
June 7 - June 11, 2018:	ASM Microbe 2018, Atlanta, GA

### 2017-2018 IBASM OFFICERS

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