BIOGRAPHICAL SKETCH

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NAME: Hyungjin Eoh

eRA COMMONS USER NAME (credential, e.g., agency login): hye2002

POSITION TITLE: Assistant Professor

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Seoul National University, Korea	B.S/D.V.M	02/1996	Veterinary Medicine
Yonsei University, Korea	M.Sc.	08/2001	Microbiology
Colorado State University	Ph.D.	01/2008	Microbiology
Colorado State University	Postdoc.	08/2009	Microbiology
Weill Cornell Medical College	Postdoc.	08/2014	Infectious Diseases

A. Personal Statement

I have worked on application of a LC-MS based metabolomics to study adaptive metabolism of various biological systems used to adapt to rapidly changing environments (Eoh and Rhee, *PNAS*., 2013; Eoh and Rhee, PNAS., 2014; Eoh et al., *Nat Microbiol*., 2017). My group has expanded the research areas: **I** the metabolic networks essential for bacterial, fungal, and viral pathogenesis (Lee et al., *Sci Rep*., 2018; Dutta et al. *Sci Adv*., 2019; Lee et al., *Nat Comm*., 2019; Lim et al., *PNAS*., 2021; Quinonez et al., mBio., 2022; Choi et al., *Cell Rep*., 2022; Sharma et al., *mBio*), disease specific biosignatures (Wu et al., *PNAS*., 2017; Seo et al. *Nat Comm*., 2018; Lee et al., *Nat Neuroscience*., 2021; Kim et al., *Gut Microbes*., 2022) and immunometabolome of oncogenic transformation (Zhu et al. *MBio*., 2017; Choi et al. *PNAS*., 2020), **II** the unannotated pathways related with metabolic remodeling (Lim et al., *PNAS*., 2021), **III** discovery of the new antibiotic candidates, and **IV** modes-of-action of new inhibitors (Luna et al., *Nat Microbiol*., 2020). For these studies, I have developed a collaborative network with a diverse array of national and international research scientists. As demonstrated, my laboratory has been relentlessly working on studying KSHV infection mediated metabolic topology as a new source of antitumor targets. In summary, Eoh lab is particularly well suited and strategically posed to perform the studies in the antitumor discovery projects because Eoh lab has extensive expertise, energy, and determination to plan and execute the effective strategies for accomplishing the innovative goals.

Ongoing and recently completed projects that I would like to highlight include:

Ongoing

1R01AI168088, NIH/NIAID, PI: Eoh, 03/16/2023-02/28/2027

Interplay of *M. tuberculosis* trehalose metabolism and its pathogenesis and drug resistance

The goal of this proposal is to examine the role of trehalose catalytic shift activity in *M. tuberculosis* persister formation, drug tolerance, and drug resistance.

Role: PI

R01 DE028521, NIH/NIDCR, PI: Jung, 01/07/2019-11/30/2023

KSHV mediated regulation of proline metabolism

The goal of this proposal is to understand the impact of KSHV infection on host proline metabolism during tumorigenesis.
Role: Co-investigator

1R21AI167027, NIH/NIAID, PI: Karakousis, 11/10/2022-08/31/2024
Characterizing CaeA-mediated rifampin tolerance in MTB
The goal of this proposal is to elucidate the impact of CaeA deficiency on *M. tuberculosis* cell wall integrity and rifampin sensitivity.
Role: Co-investigator

Completed
1R56AI143870
Hyungjin Eoh (PI)
09/05/2019-12/31/2021
PknG mediated tailoring *Mycobacterium tuberculosis* adaptive metabolism is required for the persister formation
1R21AI139386, NIH/NIAID, PI: Eoh, 03/01/2019-02/28/2023
Metabolic compensation in *Mycobacterium tuberculosis* for the formation of persisters
The goal of this proposal is to elucidate the *M. tuberculosis* pathways that are functionally interactive to survive antibiotic stresses
Role: PI
American Lung Association
Hyungjin Eoh (PI)
07/01/2020-12/31/2022
Drug resistance of *Mycobacterium tuberculosis* is enhanced by the trehalose metabolism remodeling
Donald E.& Delia B. Baxter Foundation
Hyungjin Eoh (PI)
07/01/2016-06/30/2017
An unappreciated role of internal carbons at entry into the latent state of *Mycobacterium tuberculosis*

The following publications reflect my experience in applying metabolomics and studying host-pathogen interactions with regards to new drug development.

a. Choi UY, Lee JJ, Park A, Zhu W, Hee HR, Choi YJ, Yoo JS, Yu C, Feng P, Gao SJ, Chen S, **Eoh H**‡, Jung JU‡ (2020). Oncogenic human herpesvirus hijacks proline metabolism for tumorigenesis. *Proc Natl Acad Sci U S A.* Apr 7;117(14):8083-8093. PMCID: PMC7149499 ‡, co-corresponding authors.
b. Seo GJ, Kim C, Shin W-J, Sklan EH, **Eoh H**, Jung JU. (2018). TRIM56-mediated monoubiquitination of cGAS for cytosolic DNA sensing. *Nat. Commun*. Feb 9;9(1):613. PMCID: PMC5807518.
c. Zhu Y, Li T, Ramos da Silva S, Lee JJ, Lu C, **Eoh H**, Jung JU, Gao SJ (2017). A Critical Role of Glutamine and Asparagine γ-Nitrogen in Nucleotide Biosynthesis in Cancer Cells Hijacked by an Oncogenic Virus. mBio. 2017 Aug 15;8(4). *mBio*.01179-17. PMCID: PMC5559638.
d. Wu WL, Grotefend CR, Tsai MT, Wang YL, Radic V, **Eoh H**, Huang IC (2017). Δ20 IFITM2 Differentially Restricts X4 and R5 HIV-1 *Proc Natl Acad Sci U S A.* Jul 3;114(27):7112-7117. Epub 2017 Jun 19. PMCID: PMC5502592.

B. Positions, Scientific Appointments, and Honors

Positions and Scientific Appointments

09/2022 ~ current	Assistant Professor Dept. Ophthalmology, Keck School of Medicine, University of Southern California LA. CA.
08/2015 ~ current	Assistant Professor Dept. Molecular Microbiology and Immunology, Keck School of Medicine, University of Southern California, LA. CA.
08/2014 ~ 08/2015	Instructor Dept. Medicine, Weill Cornell Medical College, NY., NY.
08/2009 ~ 07/2014	Postdoctoral Associate Dept. Medicine, Weill Cornell Medical College, NY., NY.
08/2008 ~ 07/2009	Postdoctoral Fellow College of Veterinary Medicine & Biomedical Sciences, Colorado State University, Fort Collins, CO.
03/2003 ~ 07/2008	Research Graduate Associate College of Veterinary Medicine & Biomedical Sciences, Colorado State University, Fort Collins, CO.
08/2002 ~ 02/2003	Research Associate College of Veterinary Medicine & Biomedical Sciences, Colorado State University, Fort Collins, CO.
08/2001 ~ 07/2002	Research Associate National Research Laboratory (Tuberculosis Drug Discovery & Diagnostics). Yonsei University. South Korea.
08/1998 ~ 07/2001	Research Graduate Associate Dept. of Medicine, Yonsei University. South Korea.

Honors

2023-	Ad hoc Reviewer, DMPA & AIRT, NIAID, NIH
2021-	Early Career Reviewer, BACP, NIAID, NIH.
2021-2022	Keck School of Medicine, USC, Dean Pilot Fund Award.
2020-2022	American Lung Association, Innovation Award.
2020	Burroughs Wellcome Trust PATH Finalist.
2018-2019	Wright Foundation, Los Angeles, CA.
2017-2018	L.K Whittier Foundation, South Pasadena, CA.
2017-	HCPR (Hasting center for pulmonary research) member (USC).
2016-2017	Donald E.& Delia B. Baxter Foundation Faculty Award, Los Angeles, CA.
2015-	Emerging pathogens and immune diseases member (USC).
2011-2013	Postdoctoral Research Fellowship, Stony Wold-Herbert Fund, New York, NY.
2009	Gordon Research Conference for Travel Award.
2006	Rocky Mountain Branch ASM for Research Presentation (Runner-up).
2003	Rocky Mountain Branch ASM for Research Presentation (1st Place).
2000-2001	Brain Korea (BK-21) Graduate Student Scholarship.

C. Contributions to Science

1) **Identification of *Mycobacterium tuberculosis* (Mtb) metabolic pathways necessary for host adaptation and antibiotic resistance.** Control of the tuberculosis (TB) pandemic remains hindered by a lack of simple and rapid cures. Unlike other bacterial pathogens, Mtb spends its life-time in a state of non- or slowly replicating state so that it evolves to better manage its lifestyle under stresses including adverse environmental factors and even antibiotics. One of the research areas has focused to reveal the metabolic remodeling of Mtb, with which Mtb seeks to mitigate the harmful consequences occurred during adaptation to hosts. My lab uses LC-MS

metabolomics based tools and studies the intracellular chemistry, biochemistry and metabolism while using intact Mtb. My lab has discovered specific Mtb metabolic activities that are required to survive host adverse environment and resist antibiotic effects.

a. Quinonez CG, Lee JJ, Lim J, Odell M, Lawson CP, Anyogu A, Raheem S‡, **Eoh H‡** (2022). The Role of Fatty Acid Metabolism in Drug Tolerance of *Mycobacterium tuberculosis*. *mBio*. Jan 11;13(1):e0355921. PMCID: PMC8749430 ‡, co-corresponding authors.
b. Lim J, Lee JJ, Lee SK, Kim S, Eum SY, **Eoh H** (2021). Phosphoenolpyruvate depletion mediates both growth arrest and drug tolerance of *Mycobacterium tuberculosis* in hypoxia. *Proc Natl Acad Sci U S A.* Aug 31;118(35):e2105800118. PMID: 34426499.
c. Lee JJ, Lee SK, Song N, Nathan TO, Swarts BM, Eum SY, Cho SN, **Eoh H**. (2019). Transient drug-tolerance and permanent drug-resistance rely on the trehalose-catalytic shift in *Mycobacterium tuberculosis*. *Nat. Commun*. Jul 2;10(1):2928. PMCID:PMC6606615.
   - Highlighted by Nat Comm editors; Focus: Therapeutics
d. **Eoh H**, Wang Z, Layre E, Rath P, Morris R, Moody DB, Rhee K. (2017). Metabolic anticipation in *Mycobacterium tuberculosis*. *Nat. Microbiology*. May 22;2:17084. PMCID: PMC5540153.

2) **Identification of host immunometabolic remodeling and evasion tactics by viruses to overcome host defenses**. Host protective immunometabolism is initiated by the recognition of pathogens by pattern recognition receptors (PRRs). After recognizing specific pathogen associated molecular patterns (PAMPs) with host PRRs, PRRs activate intracellular signaling pathways to induce anti-viral immunometabolic remodeling. To avoid host immune responses, viruses also have evolved elaborate mechanisms to modulate various aspects of host defense system. Thus, our study is focused on understanding anti-microbial responses by discovering host immunometabolic remodeling and various evasion tactics of cancer-causing viruses and emerging viruses.

e. Choi UY, Lee JJ, Park A, Zhu W, Hee HR, Choi YJ, Yoo JS, Yu C, Feng P, Gao SJ, Chen S, **Eoh H‡**, Jung JU‡ (2020). Oncogenic human herpesvirus hijacks proline metabolism for tumorigenesis. *Proc Natl Acad Sci U S A.* Apr 7;117(14):8083-8093. PMCID: PMC7149499 ‡, co-corresponding authors.
f. Seo GJ, Kim C, Shin W-J, Sklan EH, **Eoh H**, Jung JU. (2018). TRIM56-mediated monoubiquitination of cGAS for cytosolic DNA sensing. *Nat. Commun*. Feb 9;9(1):613. PMCID: PMC5807518.
g. Zhu Y, Li T, Ramos da Silva S, Lee JJ, Lu C, **Eoh H**, Jung JU, Gao SJ (2017). A Critical Role of Glutamine and Asparagine γ-Nitrogen in Nucleotide Biosynthesis in Cancer Cells Hijacked by an Oncogenic Virus. mBio. 2017 Aug 15;8(4). *mBio*.01179-17. PMCID: PMC5559638.
h. Wu WL, Grotefend CR, Tsai MT, Wang YL, Radic V, **Eoh H**, Huang IC (2017). Δ20 IFITM2 Differentially Restricts X4 and R5 HIV-1 *Proc Natl Acad Sci U S A.* Jul 3;114(27):7112-7117. Epub 2017 Jun 19. PMCID: PMC5502592.

3) **Application of LC-MS metabolomics for clinical diagnostics and drug discovery.** While LC-MS metabolomics holds great promise to advance our understanding of basis of nature, it also holds considerate potential for application in a wide range of clinical settings, ranging from identification of new diagnostics technique to, combined with genomics and proteomics, the discovery of new drug targets. That includes cost-effective and productive platform of **i.** drug target identification, **ii.** new antibiotics discovery and **iii.** diagnostic tool development, and **iv** disease progression prediction and/or outcome of therapeutics.

a. Lee H, Lee JJ, Park NY, Dubey SK, Kim T, Ruan K, Lim SB, Park SH, Ha S, Kovlyagina I, Kim KT, Kim S, Oh Y, Kim H, Kang SU, Song MR, Lloyd TE, Maragakis NJ, Hong YB‡, **Eoh H‡**, Lee G‡. (2021). Multi-omic analysis of selectively vulnerable motor neuron subtypes implicates altered lipid metabolism in ALS. *Nat Neurosci*. Dec;24(12):1673-1685. Epub 2021 Nov 15. PMCID: PMC8639773. ‡, co-corresponding authors.
b. Luna B, Trebosc V, Lee B, Bakowski M, Ulhaq A, Yan J, Lu P, Cheng J, Nielsen T, Lim J, Ketphan W, **Eoh H**, McNamara C, Skandalis N, She R, Kemmer C, Lociuro S, Dale GE, Spellberg B. (2020). A nutrient-limited screen unmasks rifabutin hyperactivity for extensively drug-resistant *Acinetobacter baumannii*. *Nat Microbiol*. 2020 Sep;5(9):1134-1143. Epub 2020 Jun 8. PMCID: PMC7483275.

c. Dutta NK, Klinkenberg LG, Vazquez MJ, Segura-Carro D, Colmenarejo G, Ramon F, Rodriguez-Miquel B, Mata-Cantero L, Porras-De Francisco E, Chuang YM, Rubin H, Lee JJ, **Eoh H**, Bader JS, Perez-Herran E, Mendoza-Losana A, Karakousis PC (2019). Inhibiting the stringent response blocks *Mycobacterium tuberculosis* entry into quiescence and reduces persistence. *Sci Adv*. Mar 20;5(3): eaav2014. PMCID:PMC6426458.

d. Puckett S, Trujillo C, Wang Z, **Eoh H**, Ioerger TR, Krieger I, Sacchettini J, Schnappinger D, Rhee KY, Ehrt S. (2017) Glyoxylate detoxification is an essential function of malate synthase required for carbon assimilation in *Mycobacterium tuberculosis*. *Proc Natl Acad Sci U S A*. Mar 14;114(11): E2225-E2232. PMCID: PMC5358392.

4) **Identification of novel drug targets for TB**. The MEP pathway considers a rich source of new TB drug targets. To clinically apply, four consecutive steps in MEP pathways were characterized and high throughput screening platforms were developed to identify MEP pathways inhibitors. To achieve the goal, chemical synthesis of substrates was also successfully conducted. Thus, the results broke considerable new ground in these research efforts.

a. **Eoh H*,** Narayanasamy P*, Brown AC, Parish T, Brennan PJ, Crick DC. (2009). Expression and characterization of soluble 4-diphosphocytidyl-2-C-methyl-D-erythritol kinase from bacterial pathogens. *Chem Biol*. Dec 24;16(12):1230-9. * co-first authors. PMCID: PMC4020808.

b. **Eoh H‡,** Brennan PJ, Crick DC‡. (2009). The *Mycobacterium tuberculosis* MEP (2C-methyl-d-erythritol 4-phosphate) pathway as a new drug target. *Tuberculosis* (Edinb). Jan;89(1):1-11. ‡, corresponding authors. PMCID: PMC2646905.

c. **Eoh H**, Brown AC, Buetow L, Hunter WN, Parish T, Kaur D, Brennan PJ, Crick DC. (2007). Characterization of the *Mycobacterium tuberculosis* 4-diphosphocytidyl-2-C-methyl-D-erythritol synthase: potential for drug development. *J Bacteriol*. 2007 Dec;189(24):8922-7. PMCID: PMC2168624.

Complete List of Published Work in MyBibliography:

https://www.ncbi.nlm.nih.gov/myncbi/hyungjin.eoh.1/bibliography/public/