聖路易台灣學者協會 2017 年會
2017 Taiwanese Scholar Association in St. Louis (TSA-STL) Symposium

Beauty of Immunology
and Opportunities in Biotech Industries
認識免疫學，了解工業界

April 8, Saturday, 9:30 am to 5:30 pm
Cori Auditorium, McDonnell Medical Sciences Building., 1st Floor
Medical Campus, Washington University in St. Louis
4565 McKinley Ave., St. Louis, MO 63110

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President's Greeting

On behalf of Taiwanese Scholar Association in St. Louis (TSA-STL, 聖路易台灣學者協會), we would like to welcome you to the 2017 TSA-STL Symposium "Beauty of Immunology and Opportunities in Biotech Industries 認識免疫學，了解工業界". I sincerely hope you all will enjoy this event and benefit from this Symposium.

TSA-STL (https://www.facebook.com/groups/633943866712758/) is a non-profit organization founded in 2014. Our mission is to organize Taiwanese graduate students, postdocs**,** clinicians, physician scientists, and faculty and life science professionals in the Greater St. Louis area and promote communication and collaboration among these biomedical professionals. We hold monthly scientific seminars and workshops focusing on emerging trends and cutting-edge technologies in the academic and biotech industries and sharing science news, career experience, and job opportunities.

Thanks to the 2016-2017 board members (listed below), speakers, panelists and volunteers (see acknowledgments). Without their effort, we will not be able to present this wonderful symposium to you all.

Sincerely yours,

Lon-Fye (George) Lye

2016-2017 TSA-STL Officers and Board Members:

President:	Lon-Fye Lye (賴榮輝)
Treasurer:	Meei-Hua Lin (林美華)
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Program

09:30 am **Registration Opens**

09:30 – 10:00 **Breakfast**

10:00 – 10:20 **Opening Remarks**
Dr. Hui-Kuang "Tiffany" Yu (游慧光博士)
Director of Science and Technology Division
Taipei Economic & Cultural Office (TECO) in Houston
Let me help you!

Beauty of Immunology 認識免疫學:

10:20 – 11:00 **Keynote Address**
Dr. Ta-Chiang Liu (劉大強博士)
Assistant Professor
Department of Pathology and Immunology, Washington University in St. Louis
Harnessing Big Data to Refine Translational Research of Complex Immunologic Diseases

11:00 – 12:20 **Morning Scientific Session**
Moderator: Chin-Wen "RC" Lai (賴勁文)
Department of Pathology and Immunology, Washington University in St. Louis

Dr. Li-Hao "Paul" Huang (黃立豪博士)
Post-doctoral Research Scholar
Department of Pathology and Immunology, Washington University in St. Louis
Impaired Interstitial HDL Transit in Psoriasis: an Immunological Clue in Atherosclerosis

Chin-Wen "RC" Lai (賴勁文)
PhD candidate
Department of Pathology and Immunology, Washington University in St. Louis
The Role of Mesothelin in Tissue Repair

Yi-Chieh "EJ" Perng (彭義傑博士)
Post-doctoral Research Associate
Department of Pathology and Immunology, Washington University in St. Louis
Identification of BET Bromodomain Inhibitors as Novel Antivirals of Human Cytomegalovirus

12:20 – 01:30 **Group Photo and Lunch**

01:30 – 03:20 **Afternoon Scientific Session**
Moderator: Rachel Wong (黃之妤)
Department of Pathology and Immunology, Washington University in St. Louis

Dr. Chih-Cheng "Stanley" Huang (黃景政博士)
Post-doctoral Research Associate
Department of Pathology and Immunology, Washington University in St. Louis
Metabolic Controls in Macrophage Innate Immunity

Dr. Chih-Hao "Lucas" Chang (張志豪博士)
Instructor
Department of Pathology and Immunology, Washington University in St. Louis
Tumor Microenvironment Sugar Fighting: Metabolic Competition Can Determine Cancer Progression

Rachel Wong (黃之妤)
PhD candidate
Department of Pathology and Immunology, Washington University in St. Louis
B Cell Receptor Affinity-Based Signals and Fate Decisions into the B Cell Memory Compartment

Chih-Chung "Jerry" Lin (林致中)
PhD candidate
Department of Pathology and Immunology, Washington University in St. Louis
Finding B cells: Characterization of a Mouse Strain Lacking B cells

03:20 – 03:30 **Coffee Break**

Opportunities in Biotech Industries 了解工業界:

03:30 – 04:30 **Panel Discussion**
Moderator: Dr. Stephen Wu (吳宏爲博士)
Panelists: Dr. Yie-Hwa Chang (張義華博士), Mediomics, LLC
Dr. Adekunle Onadipe, Pfizer Inc**.**
Mr. Min Zhang, Pfizer Inc**.**
Dr. Kelly A. Robinson, Monsanto Company
Dr. Cheng-Hung Yeh (葉澄鴻博士), Monsanto Company

04:30 – 04:35 **Closing Remarks**
Dr. Lon-Fye "George" Lye (賴榮輝博士)
Senior Scientist
Department of Molecular Microbiology, Washington University in St. Louis

04:35 – 05:30 **Networking**

Opening Remarks

Hui-Kuang "Tiffany" Yu, Ph.D. 游慧光博士
Director of Science and Technology Division
Taipei Economic & Cultural Office (TECO) in Houston

Dr. Tiffany Hui-Kuang Yu is the director of the Science and Technology Division at Taipei Economic and Cultural Office in Houston. She was a professor of public finance at Feng Chia University before she took a leave of absence for this international assignment by the Ministry of Science and Technology of Taiwan. Dr. Yu is an economist and has research interests spanning from Econometric, Health Economics, and Public Finance to Big Data Analysis. She has published more than 50 citation journal papers, served as journal co-editors and reviewers and been very active for international cooperation. She has served as the Dean of Office of International Affairs, Director of Chinese Language Center, Director of Overseas Youth Vocational Training School and the Chair of Department of Public Finance at Feng Chia University and the director of NISA (Network for International Students Advisors) project office and Director of Taiwan Education Center in Malaysia, for Ministry of Education. Dr. Yu received her B.A. in economics from National Taiwan University in 1986 and M.S. and Ph.D. in economics from Texas A&M University in 1989 and 1994 respectively.

Let me help you!

As a Director of Science and Technology Division, TECO-Houston Ministry of Science and Technology 駐休士頓臺北經濟文化辦事處科技組, my job is to help your needs. I will deliver what and how we can help students, postdoc fellows, scholars and faculty.

Beauty of Immunology 認識免疫學:

Keynote Address

Ta-Chiang Liu, MD, Ph.D. 劉大強博士
Assistant Professor, Pathology & Immunology
Washington University in St. Louis

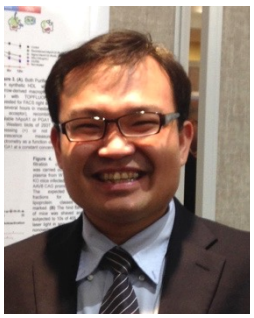
Dr. Liu received his education from National Yang Ming University in Taipei, Taiwan, and Imperial College London, UK. He did postdoctoral training at Massachusetts General Hospital, residency at Washington University in St. Louis, and clinical training at Johns Hopkins University. He became a faculty member in the Department of Pathology and Immunology at Washington University in 2012. Professor Liu's main research interest is in the role of Paneth cells in Crohn's Disease. He is particularly interested in the molecular mechanisms of how morphologic patterns of cytoplasmic antimicrobial granules are affected by genetics and environmental triggers, and their clinical relevance. Another research interest is to develop disease pathogenesis-relevant prognostic biomarkers.

Harnessing Big Data to Refine Translational Research of Complex Immunologic Diseases

Ta-Chiang Liu
Department of Pathology & Immunology, Washington University in St. Louis, USA

The pathogenesis of complex immunologic diseases (including systemic lupus erythematosus, rheumatoid arthritis, inflammatory bowel disease [IBD], among others) involve environmental triggers on genetically susceptible hosts. Genome-wide association studies have identified a wide spectrum of susceptibility genes for these diseases. For instance, over 170 susceptibility genes are associated with the development of IBD, including identification of ethnic-specific gene lists. Likewise, several potential environmental triggers have been shown to be associated with IBD development and/or prognosis (e.g., microbiome). The collection of the various genetic, transcriptomic, and microbiota big data sets provide us clues of how these factors can potentially interconnect. These big data pools also facilitate the development of next generation, rationally-designed translational research strategies for IBD and other complex immunologic diseases that can identify disease-relevant pathways and therapeutic targets.

Morning Scientific Session



Li-Hao Huang, Ph.D. 黃立豪博士
Post-doctoral Research Scholar in Gwendalyn Randolph Laboratory
Washington University in St. Louis

I graduated in Chemistry at National Taiwan University, where my passion for science started to be ignited. I studied the biochemical functions of a validated drug target for type II diabetes named DPP4. It was a very enlightening experience in the beginning of my scientific career. Based on the published crystal structure, which revealed active form of DPP4 existing as a dimer, we hypothesized that the monomeric form is inactive. I then tested the hypothesis and discovered that a conserved single mutation, located at the interface of two monomers, is required for DPP4 dimer stability and activity. The results implicated a potential therapeutic opportunity to inhibit DPP4 activity by targeting the interface. I really enjoyed the thought process that eventually would lead to answers. Inspired by this research experience, I decided to pursue my Ph.D. in Biochemistry at the Geisel School of Medicine at Dartmouth. My Ph.D. work was to study a puzzle that has existed for over a decade: global loss of ACAT1 enlarges atherosclerotic lesions, while ACAT inhibitors reduce those lesions in various animal models. I found that ACAT1 deficiency in stem/progenitor cells induces leukocytosis, potentially leading to atherosclerosis. I generated a conditional myeloid-specific ACAT1 knockout mouse and showed that it is protected from obesity and atherosclerosis. The results strongly suggested that ACAT1 in macrophages could be a potential target for various metabolic diseases. My graduate school training reinforced my passion for science and led me to expand my interests in immunology. I am currently a post-doctoral fellow in Dr. Gwendalyn Randolph's lab in the Pathology and Immunology Department at Washington University in St. Louis. I am keenly interested in understanding how tissue-localized HDL egresses from tissues and recirculates back to the blood compartment through lymphatic vasculature, and how this process is regulated through immune cells. This may answer a long unsolved question: why does a pharmacological increase in plasma HDL levels fails to provide clinical benefits? Ultimately, I wish to pursue a career as a principal investigator focusing on how the crosstalk between lipoproteins and immune cells impacts metabolic and inflammatory diseases.

Impaired Interstitial HDL Transit in Psoriasis: an Immunological Clue in Atherosclerosis

Li-Hao Huang[1], Bernd H. Zinselmeyer[1], Chih-Hao Chang[1], Brian T. Saunders[1], Brian S. Kim[2], Helge Wiig[3], Michael J. Thomas[4], Mary G. Sorci-Thomas[5], Gwendalyn J. Randolph[1]

[1]Department of Pathology & Immunology; [2]Division of Dermatology, Department of Medicine; Washington University School of Medicine, St Louis, MO, 63110 USA

[3]Department of Biomedicine, University of Bergen, Jonas Lies vei 91, N-5009 Bergen, Norway

Pharmacology and Toxicology, and Blood Research Institute; Medical College of Wisconsin, Milwaukee, Wisconsin 53226

High-density lipoprotein (HDL) is cardioprotective, but plasma HDL levels do not necessarily predict cardiovascular outcomes. The major HDL-associated protein apolipoprotein A-I (apoA-I) picks up its cholesterol from cells within extravascular compartments to return it to plasma and then bile. Yet, tools are lacking to quantify the important step of HDL transit through extravascular spaces. Here, we developed recombinant photoactivatable apoA-I to quantify endogenous HDL recirculation. Using the tool, we studied HDL passage through skin in healthy mice versus those with experimental psoriasis, wherein collagen density increased in the skin in a $CD4^+$ T cell-dependent manner. In control mice, photoactivated HDL mobilized to plasma within 2 h but was retained in collagen-enriched skin of mice with psoriasis. These data suggest that cardiovascular comorbidity in psoriasis might be linked to T cell-mediated structural changes in skin that impedes systemic recirculation of HDL. This new tool is likely to find wide application in HDL research.

Chin-Wen "RC" Lai 賴勁文
Graduate student in Thaddeus Stappenbeck Laboratory
Washington University in St. Louis

Born in Yilan, grew up in Pingtung, studied in Taipei. Now a 4th year immunology graduate student in Saint Louis, USA. Work between pathology and development biology, try to set the tone as an immunologist before graduation.

The Role of Mesothelin in Tissue Repair

Chin-wen "RC" Lai, Nicholas A. Manieri, and Thaddeus S. Stappenbeck
Department of Pathology & Immunology, Washington University School of Medicine, St. Louis, USA

Local tissue damage must be limited by host factors. Without such factors, minor damage to the inner lining of the intestine can't activate proper responses against the damage. Our hypothesis is that locally induced factors protect the tissue through macrophage activation. We performed an expression screen at the colonic wound site and found that mesothelin (Msln), a GPI-linked molecule linked to cancer metastasis, was highly induced. In situ hybridization showed its expression is limited to epithelial and mesothelial cells near the wound site. Mucosal injuries to *Msln*$^{-/-}$ mice have less macrophages in the muscularis propria by day 6 post injury. Through cytokine screening, we identified downregulation in interleukin (IL)-33 in *Msln*$^{-/-}$ mice's wounds. *Il33*$^{-/-}$ mice phenocopied *Msln*$^{-/-}$ mice in terms of macrophage in the muscularis propria. *In vitro* culture also showed that *Il33*$^{-/-}$ and *Msln*$^{-/-}$ mesothelial cells both had delayed transdifferentiation and proliferated less. The two other models, including intestinal cancer and dextran sulfate sodium (DSS)-induced colitis, showed the same defect in macrophage recruitment. Overall, these findings show novel mechanism of tissue repair by Msln, and suggest that Msln modulates the tissue repair in a macrophage and IL-33-dependent manner.

Yi-Chieh "EJ" Perng, Ph.D. 彭義傑博士
Post-doctoral Research Associate in Deborah Lenschow Laboratory
Washington University in St. Louis

EJ received his B.S. in Zoology from National Taiwan University in 2003. He received his master's degree of microbiology and immunology from Graduate Institute of Microbiology, National Taiwan University in 2005. He came to Washington University in St. Louis for Ph.D. in 2007 and majored in molecular microbiology and microbial pathogenesis. During his Ph.D. training, he worked on molecular virology of human cytomegalovirus. After completing his Ph.D., he started his post-doctoral research focusing on identification of novel antivirals and protein functions of interferon-stimulated genes during various viral infection. He received research supports from Wash U OTM (Bear Cub award, 2014-2015) and is a post-doctoral scholar of Children's Discovery Institute from 2015-2017.

Identification of BET Bromodomain Inhibitors as Novel Antivirals of Human Cytomegalovirus

Yi-Chieh Perng, Dong Yu, Deborah Lenschow
Department of Medicine, Washington University in St. Louis, USA

Human CMV is a widespread opportunistic pathogen and established lifelong infections in 50-80% of adults in United States. Even though human CMV infection is usually asymptomatic, it acts as an opportunistic pathogen and is a major cause of morbidity and mortality in immunocompromised individuals, including transplant recipients and AIDS/HIV patients. Most importantly, CMV is the most frequent congenital infection and the leading viral cause of birth defects in newborns. Following congenital infection, CMV pneumonitis may be severe, even lethal. Annually approximately 40,000 U.S. infants are born with congenital CMV infection, causing an estimated 400 deaths and leaving 8,000 with permanent disabilities such as hearing and vision loss, or mental retardation. However, current CMV therapeutics are problematic. A limited number of drugs are licensed for the treatment of CMV infection and no vaccine is available. Standard therapy for CMV relies on oral/intravenous Ganciclovir (GCV) or its oral prodrug, Valganciclovir. Although efficacious, GCV treatment suffers from dose-related toxicities. Forscarnet (FOS) and Cidofovir (CDV), the two commonly used second-line treatments for CMV, are also associated with significant toxicities, including renal

toxicity. Due to drug toxicities, standard therapies for CMV are restricted from neonatal usage. Moreover, during prolonged/repeated application, CMV can become resistant to GCV and lead to treatment failure. In recent years, GCV resistance in CMV infections has increased and cross-resistance to either or both second-line agents (FOS & CDV) is frequently encountered because all licensed drugs share a common target molecule, the viral DNA polymerase pUL54. Therefore, the development of new anti-CMV therapeutics focusing on a novel mechanism of action (MOA) beyond DNA polymerase inhibitors is of critical important.

To address the unmet need of CMV therapeutics, we explored the molecular mechanisms of CMV transcription to explore potential antiviral targets. Since CMV utilizes the host transcriptional machinery to transcribe its own transcripts, we adopted the drug-repurposing approach to identify host factors essential to viral transcription. Targeting a host protein instead of a viral one reduces the chance of a drug-resistant virus emerging. Through this approach, we identified BET bromodomain proteins, human epigenetic readers, as a potential novel target. BET bromodomain proteins, including Brd2, Brd3, Brd4, and BrdT, binds to acetylated lysines on histones to regulate gene transcription. Small molecule inhibitors of BET bromodomain proteins (iBETs) are the next-generation of epigenetic-based therapeutics being developed by pharmaceutical companies for cancers, cardiovascular diseases, inflammatory diseases, and male contraception. Preclinical research and preliminary results of clinical trials have shown that iBETs possess good bioavailability, low toxicity, and long-term usage capacities. However, the anti-viral potentials of iBETs against CMV infection have never been tested.

Using an *in vitro* cell culture system, we found that iBETs effectively inhibit viral infections of both human CMV and murine CMV, the mouse homolog of human CMV, at concentrations that have no known toxicities. Further characterization further revealed that iBETs could elicit anti-viral activities *in vivo*. Our preliminary studies identifying iBETs as an exciting new class of potential anti-CMV therapies.

Afternoon Scientific Session

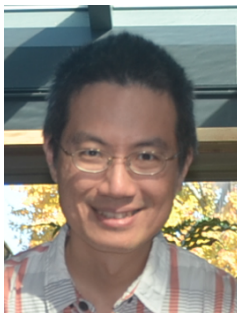
Ching-Cheng "Stanley" Huang, Ph.D. 黃景政博士
Post-doctoral Research Associate in Marco Colonna Laboratory
Washington University in St. Louis

Dr. Huang (黃景政) obtained his bachelor's degree in Chemistry, and master's degree in Microbiology from Chung-Yuan Christian University (中原大學) and Chang Gung University (長庚大學), Taiwan respectively. He received his Ph.D. degree in Molecular & Cellular Biology from Imperial College London, UK in 2010; then joined the laboratory of Professor Edward Pearce as an American Heart Association postdoctoral fellow. Currently, he is a postdoctoral research associate in the laboratory of Professor Marco Colonna at the Department of Pathology & Immunology at Washington University School of Medicine.

Metabolic Controls in Macrophage Innate Immunity

Stanley Ching-Cheng Huang[1], *Joel D. Schilling*[1], *Marco Colonna*[1] & *Edward J. Pearce*[2]
[1]Department of Pathology & Immunology, Washington University School of Medicine, St. Louis, USA. [2]Department of Immunometabolism, Max Planck Institute of Immunobiology and Epigenetics, Germany.

Macrophages are important effector cells that play pivotal roles in a broad range of immunologic and homeostatic processes in different tissues. The ability of these cells to serve multiple functions reflects their ability to express different genes in response to distinct extracellular signals including pathogenic insults and cytokines. Alternatively activated (M2) macrophages are induced by Th2 cytokines interleukin-4 (IL-4) and/or IL-13, and which are important in immunity to helminth infections, tissue remodeling, wound repair and also tumorigenesis. Recent work has revealed that macrophage activation is linked to changes of metabolic status. My research interests focus on understanding how metabolic pathways (intrinsically and/or extrinsically) control M2 macrophage innate immunity, and what the reciprocal interactions between M2 macrophages and diseases are.



Chih-Hao "Lucas" Chang, Ph.D. 張志豪博士
Instructor, Pathology & Immunology
Washington University in St. Louis

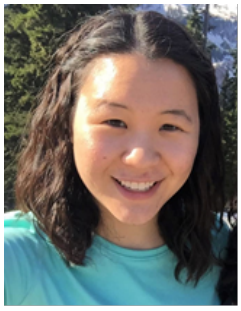
Chih-Hao earned his undergraduate degree in Agriculture Chemistry (a.k.a. Biochemical Science and Technology) from National Taiwan University, and later obtained a master's degree in Microbiology in 2001. He had served in the army as a second lieutenant to fulfill a compulsory 2-year military service in Taiwan. Afterwards he worked in academic/industrial settings as a researcher and developed shrimp vaccines against shrimp white spot syndrome virus. During the time, he decided to have advanced education abroad and pursue a career in scientific research. He went to England studying for his doctoral work under the mentorship of Prof. Andrew McMichael at Weatherall Institute of Molecular Medicine, University of Oxford. There his doctoral project focused on the impact of HIV subtype diversity and evolution in shaping host immune responses. He completed his doctoral degree in Clinical Medicine in 2010. He then joined in Dr. Erika Pearce's laboratory in 2011. He is currently investigating metabolic regulation in T cells and how cellular metabolism regulates the generation, maintenance and functions of these cells.

Tumor Microenvironment Sugar Fighting: Metabolic Competition Can Determine Cancer Progression

Chih-Hao Chang[1], *Jing Qiu*[1,2], *David O'Sullivan*[1,2], *Michael D. Buck*[1,2], *Takuro Noguchi*[1], *Jonathan D. Curtis*[1,2], *Mariel Gindin*[1], *Qiongyu Chen*[1], *Matthew M. Gubin*[1], *Elena Tonc*[1], *Gerritje J.W. van der Windt*[1,3], *Robert D. Schreiber*[1], *Edward J. Pearce*[1,2], *Erika L. Pearce*[1,2]
[1]Department of Pathology and Immunology, Washington University School of Medicine, St. Louis, MO, 63110, USA
[2]Present Address: Max Planck Institute of Immunobiology and Epigenetics, Freiburg 79108, Germany
[3]Present Address: Academic Medical Center, 1105 AZ Amsterdam, Netherlands

Properly functioning T cells are critical for effective tumor immunity. Upon activation, T cells engage aerobic glycolysis and this metabolic adaptation is required for the optimal production of effector cytokines that mediate tumor clearance. However, during cancer T cells often experience a progressive decline in effector functions, preventing tumor regression. Failure of T cells to protect against

cancer is thought to result from lack of antigen recognition, chronic activation, and/or suppression by other cells. Using a mouse sarcoma model, we showed that glucose consumption by tumors metabolically restricts T cells in the tumor microenvironment, which dampens their mTOR activity and glycolytic capacity, limits their IFN-γ production, and leads to tumor progression. We demonstrated that enhancing glycolysis in an antigenic 'regressor' tumor is sufficient to override the ability of T cells to respond to a major tumor rejection antigen, allowing progression of tumors that are normally rejected. Checkpoint blockade therapy is used clinically to promote immune rejection of progressing tumors. We found that checkpoint blockade (anti-CTLA-4, anti-PD-1, and anti-PD-L1) monoclonal antibodies restore glucose in the microenvironment of progressing tumors, permitting T cell glycolysis and IFN-γ production. Together our results show that metabolic competition in the tumor microenvironment dictates effector T cell function and that this influences cancer progression. Combining therapies that blunt tumor metabolism with those that promote glycolysis in T cells could provide new effective treatments for cancer.



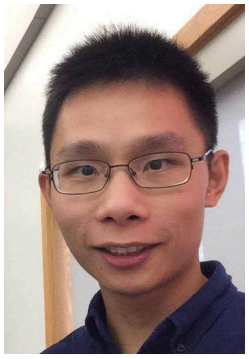
Rachel Wong 黃之妤
Graduate student in Deepta Bhattacharya Laboratory
Washington University in St. Louis

I graduated from the University of Wisconsin-Madison in 2013 with a Biochemistry Major. That same year, I enrolled in Washington University in St. Louis Immunology graduate school program. I'm currently a 4th year graduate student in the Bhattacharya lab. Ultimately, I would like to apply the skills I learn from graduate school to either an industry job or as a consultant.

B Cell Receptor Affinity-Based Signals and Fate Decisions into the B Cell Memory Compartment

Rachel Wong, Haiyan Zhao, Justin Richner, Michael S. Diamond, Daved H. Fremont, and Deepta Bhattacharya
Department of Pathology and Immunology, Washington University School of Medicine, St. Louis, MO, 63110, USA

Germinal center B cells are selected to enter either the memory B cell or the long-lived plasma cell compartment, yet the underlying mechanisms that control this decision are unknown. Our recent work utilizing a West Nile Virus (WNV) infection system demonstrated that the antigen specificity between the memory B cell and long-lived plasma cell compartments differed. Long-lived plasma cells primarily recognized the dominantly neutralizing epitope, the lateral ridge, of West Nile Virus envelope protein domain III (DIII). In contrast, memory B cells could recognize the lateral ridge epitope and non-lateral ridge epitopes. We hypothesized that non-lateral ridge-specific B cell receptors fail to reach an affinity threshold that promotes the long-lived plasma cell fate. To assess this, we utilized DIII-tetramers that are or are not mutated at the lateral ridge to identify antigen-specific memory B cells and long-lived plasma cells. V(D)J sequences from these cells were cloned into expression vectors for antibody production. The binding patterns of these monoclonal antibodies, at the affinity matured and germline-reverted states, were assessed by ELISA and Bio-Layer Interferometry (BLI). Preliminary data indicates that non-lateral ridge-specific antibodies bind DIII more poorly than lateral ridge-specific antibodies. This difference is more striking when germline forms of these antibodies are compared. Non-lateral ridge-specific antibodies are present during early germinal center reactions when memory B cells are formed, but are progressively lost at later timepoints when long-lived plasma cells emerge. Our preliminary data suggest the existence of an antibody affinity threshold that promotes germinal center B cell retention and long-lived plasma cell selection.



Chih-Chung "Jerry" Lin 林致中
Graduate student in Brian Edelson Laboratory
Washington University in St. Louis

Mr. Chih-Chung Lin (林致中) obtained his bachelor's degree in Biochemistry from National Taiwan University in 2009. After graduation from college, he served in the Taiwan army for 11 months. He came to Washington University in St. Louis for Ph.D. training in 2011, and he expects to receive his doctoral degree in immunology in May 2017. In the Edelson lab at Washington University, Mr. Lin reported the transcription factor, Bhlhe40, was absolutely required for the induction of experimental autoimmune encephalomyelitis (EAE), an animal model of multiple sclerosis. This factor promotes a pro-inflammatory cytokine, GM-CSF, production while repressing an anti-inflammatory, IL-10, expression by $CD4^+$ T cells, and is positively regulated by IL-1β. His results further illustrate an active "pertussis toxin-IL-1β-Bhlhe40" pathway in autoreactive T-cell activation during EAE.

Finding B cells: Characterization of a Mouse Strain Lacking B cells
Chih-Chung Lin, Nicholas N. Jarjour, Melissa E. Cook, Tara R. Bradstreet, Elizabeth A. Schwarzkopf, Chun Chou, Rachel Wong, Brian T. Edelson*
Department of Pathology and Immunology, Washington University School of Medicine, St. Louis, MO, 63110, USA
*Present Address: Memorial Sloan Kettering Cancer Center, New York, NY, 10065, USA

B cell (B lymphocyte) plays an important role in the adaptive as well as innate immunity by secreting antibodies, presenting antigens to T cells, and releasing cytokines. In rodents and humans, B cells originated from hematopoietic stem cells in the bone marrow. There, the developing B cells undergo immunoglobulin (Ig) gene loci arrangements and positive/negative selections. IgM^+ immature B cells exit the bone marrow and migrate to the spleen passing through two transitional stages, T1 and T2. Some T2 B cells later differentiate into mature IgM^+IgD^+ follicular B (FO B) cells, or marginal zone B (MZB) cells in the spleen.

We accidentally found that the CX_3CR1-GFP knock-in transgenic mice in our mouse colony lacked most of the B cell populations in multiple tissues, including blood, spleen, inguinal lymph node, bone marrow and peritoneal cavity, although regular CX_3CR1-GFP knock-in transgenic mice have normal lymphocyte populations. Not

surprisingly, the serum immunoglobulin (antibodies) levels in these affected transgenic mice were significantly reduced. The remaining tissue B cells were all IgD^-, indicating their immature statuses. Characterization of developing B cell subsets by flow cytometry revealed that B cell development was arrested at T1 stage in the spleen. In mixed bone marrow chimeric settings, CX_3CR1-GFP bone marrow showed a severe cell-intrinsic disadvantage to give rise to mature B cells. By crossing affected CX_3CR1-GFP mice with wild-type mice, we found that the B cell phenotype was autosomal recessive and unrelated with CX_3CR1-GFP allele. We hypothesized that these mice in our animal facility might have acquired spontaneous mutation(s) that was indispensable for B cell development. We would use whole exome sequencing (WES) to identify exosomal variants that might be responsible for the severe B cell phenotype.

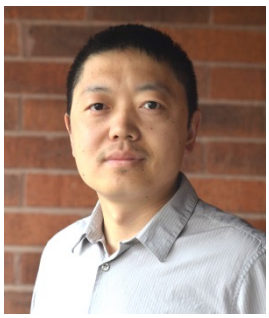
Opportunities in Biotech Industries 了解工業界:
Panel Discussion

Yie-Hwa Chang, Ph.D. 張義華博士
Founder and President
Mediomics, LLC

Dr. Yie-Hwa Chang received his BS in Chemistry from National Taiwan University, his Ph.D. in Biochemistry from California Institute of Technology and his postdoctoral training in Molecular Biology and Genetics from Harvard Medical School. He completed the Bio-Executive Program from the Business School of University of California at Berkeley and the Emerging Leader Program Organized by the Small Business Association of USA. Mediomics is focusing on developing rapid, cost-effective and innovative methods, biosensors and high-affinity binding reagents (ScFv and aptamers) for quantitative analysis of proteins, biomarkers and pathogens. Under his leadership, Mediomics has received more than $7 million US dollars' funding from the National Cancer Institute, the Department of Defense and the CDC and several key platform technologies have been successfully developed. He has more than 40 peered review publications and book chapters in the field of protein engineering, protein processing, angiogenesis, proteomics and bioassay development. He is also the inventor of more than 20 awarded or pending patent applications. His inventions have licensed to more than 11 Pharmaceutical Companies to develop anti-cancer and anti-obesity drugs. He is currently supervising two funded projects at Mediomics: 1. Developing novel tools to detect RNA modification at single nucleotide resolution; 2. Developing specific and cost-effective immunoassays for Zika infection.

Adekunle Onadipe, Ph.D.
Associate Research Fellow in Bioprocess R&D
Pfizer Inc.

Dr. Adekunle Onadipe is an Associate Research Fellow in Bioprocess R&D, Cell Line Development at Pfizer Inc. He leads a group of scientists responsible for the construction, development and characterization of mammalian and microbial cell lines for biotherapeutics and vaccines production. His group is also involved in the scale-up of bioprocesses from bench top to pilot scale bioreactors and process development for the establishment of cell banks to support the manufacture of biopharmaceutical products for early phase clinical trials. Kunle joined Pfizer Limited in the UK in 2005 in Discovery Biology with responsibility for optimizing cell culture processes for the production of cell-based assay reagents. Prior to this he worked for 15 years at Lonza Biologics plc., in Slough UK where, as a Principal Group Leader in cell culture process development, he was responsible for constructing and developing production mammalian cell lines and culture processes, subsequently transferring them to full-scale production for clinical trials. A microbiologist by training, Kunle has been involved in the production of biopharmaceuticals for more than 29 years and has a broad experience of microbial and mammalian cell culture methods. He obtained his PhD in Microbiology from the University of Surrey, Guildford, UK.

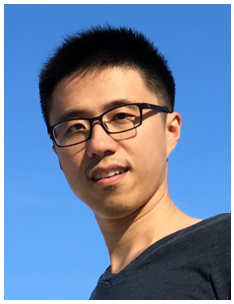


Min Zhang, M.S.
Scientist in Bioprocess R&D
Pfizer Inc.

Min Zhang received his B.S. of Chemistry in Nankai University and M.S. of Chemistry in University of Missouri-Columbia. When worked at Intercell Inc. and Vaccine Research Center in NIH, he was focusing on protein purification process development, technology transfer and process validation for ten years. He also had GMP manufacturing experience by working in pilot plants as an engineer for four years. Min joined Pfizer in 2015 as a Scientist in Bioprocess R&D group. His function is downstream process development and technology transfer of preclinical and clinical manufacturing process, especially for monoclonal antibodies. His current projects are continuous manufacturing platform development, gene therapy process development and technology transfer.

Kelly A. Robinson, Ph.D.
Molecular Biologist, Regulatory Sciences
Monsanto Company

Kelly received her B.S. in Biology from Marquette University in 1993 and Ph.D. from Loyola University of Chicago in 2000. After completing her Ph.D., she moved to Washington University Medical School where she conducted a Post-doctoral fellowship developing molecular and genetic tools to study the biology of the protozoan parasite *Leishmania*. In December of 2004, Kelly accepted a position at Monsanto in the Genetic Quality Laboratory within the Manufacturing Organization. In this role, Kelly was responsible for the development, implementation, and production support of DNA and protein detection assays in this high throughput genetic seed testing laboratory. In 2008, Kelly accepted a new role in the Regulatory Sciences Organization of Monsanto. During her time in Regulatory, Kelly has used her scientific expertise to develop scientific regulatory strategies and conduct regulatory studies for submission to US and global regulatory agencies resulting in global deregulation of multiple biotechnology products. In addition to these efforts, she has also led multiple compliance, process improvement, and electronic system implementation efforts within Regulatory Sciences.



Cheng-Hung Yeh, Ph.D. 葉澄鴻博士
Imaging Engineer, Breeding Division
Monsanto Company

Dr. Cheng-Hung Yeh studied for his Ph.D. degree at Washington University in St. Louis, Missouri under the supervision of Dr. Lihong V. Wang, Gene K. Beare Distinguished Professor. He is currently an imaging engineer of Breeding Division at Monsanto (Monsanto, top sustainable agriculture companies) in the United States. His interests are the development of novel imaging techniques including Light Detection and Ranging (LIDAR) and photoacoustic tomography. As a member at Monsanto research center, he helps design new hardware, implement cutting edge algorithms, and build sensors to help improve the famers from around the world.

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- Hsinchu Science Park, Hsinchu Science Park Bureau, Ministry of Science and Technology 新竹科學工業園區

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Company

Overview
Milestones
Management
Board of Directors
Corporate Responsibility
Locations

Overview

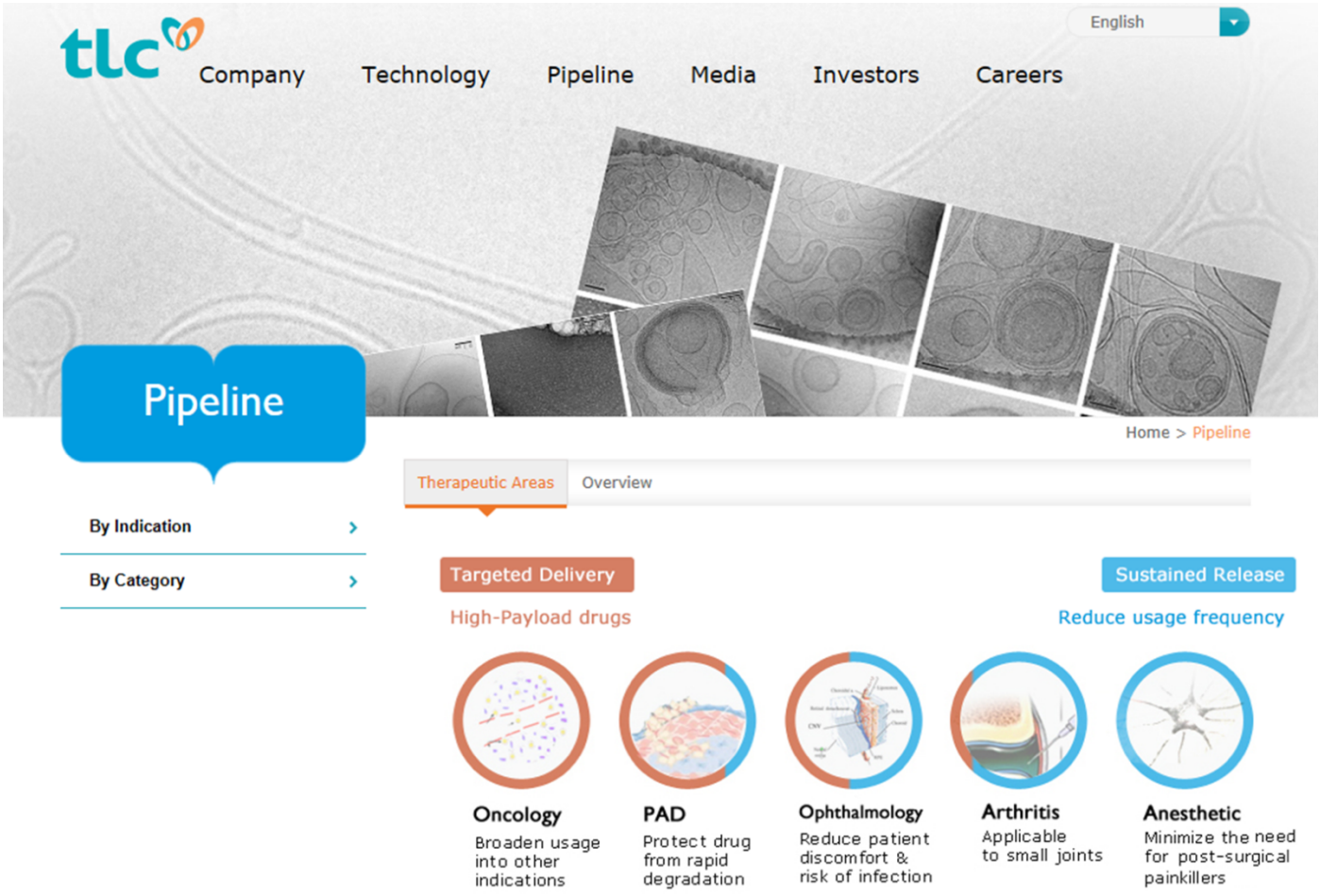
Taiwan Liposome Company (TLC) is a biopharmaceutical company focused on the research, development and commercialization of innovative pharmaceutical products based on its proprietary drug delivery technologies. Our strengths lie in lipid-based formulation and scale-up for parenteral drugs using micelles and nanoparticles to optimize the pharmacokinetics of drugs for better efficacy and lower toxicity, and thus prolong the product lifecycle of branded drugs.

Vision

TLC is dedicated to maximizing the benefits of medications for patients and improving their quality of life through constantly advancing our technology & know-how.

Mission

TLC strives to become a global leading biopharmaceutical company, to contribute more towards making a difference in the healthcare industry and make Taiwan biotechnology industry visible in the global arena.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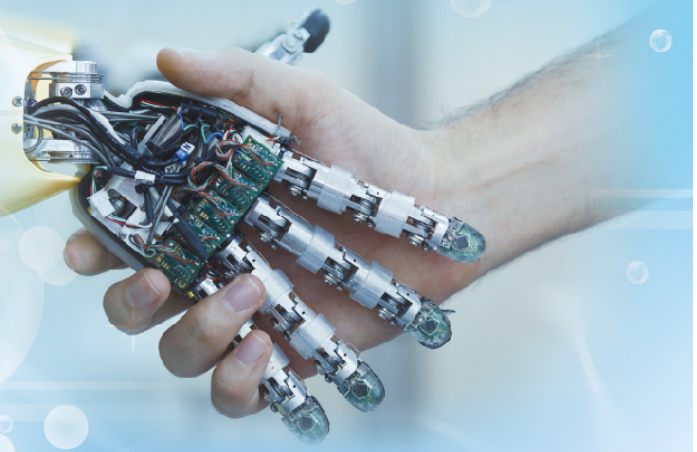

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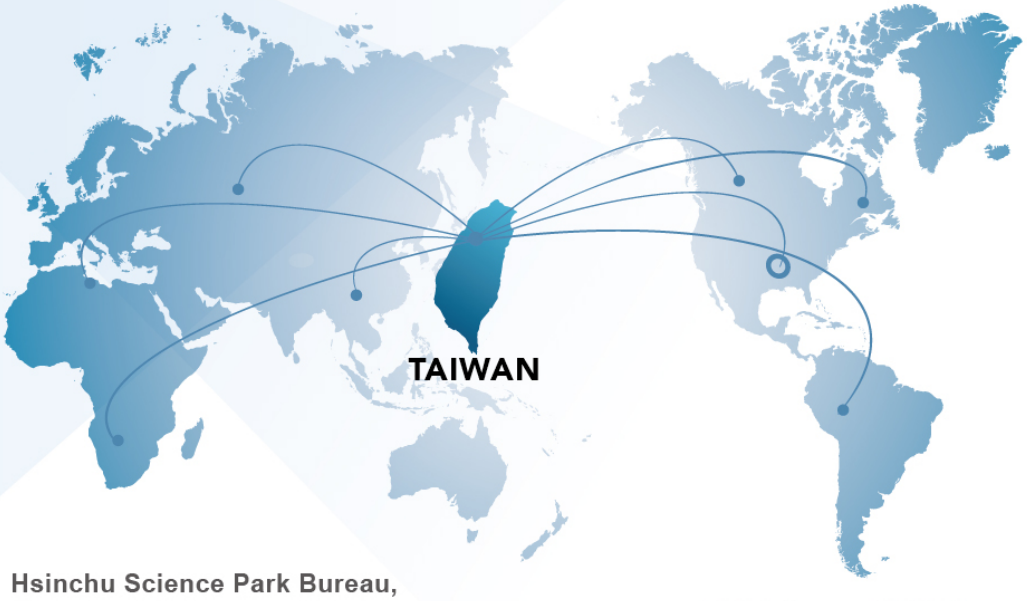

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