Stanford

Trung Hoang Minh Pham

Assistant Professor of Pediatrics (Infectious Diseases)

Pediatrics - Infectious Diseases

CLINICAL OFFICE (PRIMARY)

- **Pediatric Infectious Disease**

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Bio

CLINICAL FOCUS

- Pediatric Infectious Diseases

ACADEMIC APPOINTMENTS

- Assistant Professor, Pediatrics - Infectious Diseases
- Member, Maternal & Child Health Research Institute (MCHRI)

HONORS AND AWARDS

- Basic Science Research Faculty Award - Early Career, Stanford Department of Pediatrics (2022)
- K08 Mentored Career Development Award, NIAID (2019)
- Alpha-Omega-Alpha Postgraduate Fellowship, AOA Honor Medical Society (2017)
- PIDS Stanley A. Plotkin Sanofi Pasteur Fellowship, Pediatric Infectious Diseases Society (2016)
- Ernest and Amelia Gallo Endowed Postdoctoral Fellowship, Maternal and Child Health Research Institute (2015)
- Consulting Fellow Teaching Award, Stanford University Pediatrics Residency Program (2015)
- Letter of Teaching Distinction in Pediatrics Clerkship, Stanford University School of Medicine (2012)

PROFESSIONAL EDUCATION

- Board Certification, American Board of Pediatrics , Pediatric Infectious Diseases (2017)
- Board Certification, American Board of Pediatrics , General Pediatrics (2014)
- Fellowship: Lucile Packard Children's Hospital at Stanford University Medical Center (2017) CA
- Residency, Stanford University School of Medicine , Pediatrics (2014)
- M.D., University of California, San Francisco , Medicine
- Ph.D., University of California, San Francisco , Cell Biology/Immunology
- B.S., University of California, San Diego , Biochemistry and Cell Biology

LINKS

- Pham lab website: https://trungphamlab.stanford.edu

Research & Scholarship

CURRENT RESEARCH AND SCHOLARLY INTERESTS

Uncovering mechanisms of tissue immunity and immunophysiology during persistent infection

The immune system safeguards the health of complex organisms by rapidly eliminating invading pathogens, curbing infection-induced tissue disruptions, and maintaining tissue homeostasis. Many bacterial pathogens evade host antimicrobial mechanisms and persist in infected tissues at low levels for long periods of time even in the presence of innate and adaptive immune resistance. During persistent infection, the immune system simultaneously orchestrates antimicrobial responses to contain the pathogen, repairs damaged tissue, regulates nutrient resources, and maintains other tissue physiologic functions to ensure host survival. Failure of any of these tasks leads to uncontrolled infection, devastating disease, and even death. The goals of our research are to understand:

1) What are the innate and adaptive immune cellular mechanisms that contain pathogens during persistent infection?
2) How are tissue physiological functions, such as tissue repair and nutrient regulation, maintained during persistent infection?
3) How do pathogens survive innate and adaptive antimicrobial mechanisms in infected tissues?
4) How does persistent infection impact host immunity to secondary infections of a similar or different pathogen?

Through investigating these fundamental questions, we may be able to decode the underlying cellular and molecular mechanisms that can be harnessed to eradicate infections and help restore health after an infectious insult. We employ animal infection models and bring together immunology, tissue biology, microbiology, and genetics to uncover the mechanisms of tissue immunity and immunophysiology during persistent infection from the molecular to organismal level.

Current areas of research:
• Development, maintenance, and plasticity of macrophage functional diversity in infected tissue
• Tissue repair and nutrient regulation during persistent infection
• Cellular dynamics and bacterial persistence in lymphoid organs

We are looking for highly motivated team members who are passionate about making impactful scientific discoveries to join our group at all levels. For opportunities and positions available for pre-doctoral students and postdoctoral fellows, please contact tpham8@stanford.edu!

Teaching

GRADUATE AND FELLOWSHIP PROGRAM AFFILIATIONS

- Immunology (Phd Program)
- Pediatric Infectious Diseases (Fellowship Program)

Publications

PUBLICATIONS

- **Turning foes into permissive hosts: manipulation of macrophage polarization by intracellular bacteria.** *Current opinion in immunology*
  Pham, T. H., Monack, D. M.
  2023; 84: 102367

- **Single-cell profiling identifies ACE+ granuloma macrophages as a nonpermissive niche for intracellular bacteria during persistent Salmonella infection.** *Science advances*
  Pham, T. H., Xue, Y., Brewer, S. M., Bernstein, K. E., Quake, S. R., Monack, D. M.
  2023; 9 (1): eadd4333

- **Near-fatal Legionella pneumonia in a neonate linked to home humidifier by metagenomic next generation sequencing.** *Med (New York, N.Y.)*
  West, P. T., Brooks, E. F., Costales, C., Moreno, A., Jensen, T. D., Budvytiene, I., Khan, A., Pham, T. H., Schwenk, H. T., Bhatt, A. S., Banaei, N.
  2022

- **A Salmonella Typhi RNA thermosensor regulates virulence factors and innate immune evasion in response to host temperature.** *PLoS pathogens*
  Brewer, S. M., Twittenhoff, C., Kortmann, J., Brubaker, S. W., Honeycutt, J., Massis, L. M., Pham, T. H., Narberhaus, F., Monack, D. M.
  2021; 17 (3): e1009345

- **Salmonella-Driven Polarization of Granuloma Macrophages Antagonizes TNF-Mediated Pathogen Restriction during Persistent Infection.** *Cell host & microbe*
  Pham, T. H., Brewer, S. M., Thurston, T., Massis, L. M., Honeycutt, J., Lugo, K., Jacobson, A. R., Vilches-Moure, J. G., Hamblin, M., Helaine, S., Monack, D. M.
  2019

- **Salmonella Effector SteE Converts the Mammalian Serine/Threonine Kinase GSK3 into a Tyrosine Kinase to Direct Macrophage Polarization.** *Cell host & microbe*
  Panagi, I., Jennings, E., Zeng, J., Gunster, R. A., Stones, C. D., Mak, H., Jin, E., Stapels, D. A., Subari, N. Z., Pham, T. H., Brewer, S. M., Ong, S. Y., Monack, et al
  2019

- **A Gut Commensal-Produced Metabolite Mediates Colonization Resistance to Salmonella Infection** *CELL HOST & MICROBE*
  Jacobson, A., Lam, L., Rajendram, M., Tamburini, F., Honeycutt, J., Trung Pham, Van Treuren, W., Pruss, K., Stabler, S., Lugo, K., Bouley, D. M., Vilches-Moure, J. G., Smith, M., et al
  2018; 24 (2): 296-+

- **Well-Appearing Newborn With a Vesiculobullous Rash at Birth** *PEDIATRICS*
  Stewart, S. E., Lin, J. L., Everhart, J. L., Pham, T. H., Marqueling, A. L., Rieger, K. E., Hilgenberg, S. L.
  2018; 141 (3)

- **Pseudogenization of the Secreted Effector Gene sseI Confers Rapid Systemic Dissemination of S. Typhimurium ST313 within Migratory Dendritic Cells.** *Cell host & microbe*
  Carden, S. E., Walker, G. T., Honeycutt, J., Lugo, K., Pham, T., Jacobson, A., Bouley, D., Idoyaga, J., Tsolis, R. M., Monack, D.
  2017; 21 (2): 182-194

- **DOCK8 is essential for T-cell survival and the maintenance of CD8(+) T-cell memory** *EUROPEAN JOURNAL OF IMMUNOLOGY*
  Lambe, T., Crawford, G., Johnson, A. L., Crockford, T. L., Bouriez-Jones, T., Smyth, A. M., Pham, T. H., Zhang, Q., Freeman, A. F., Cyster, J. G., Su, H. C., Cornall, R. J.
  2011; 41 (12): 3423-3435

- **GRK2-Dependent S1PR1 Desensitization Is Required for Lymphocytes to Overcome Their Attraction to Blood** *SCIENCE*
  Arnon, T. I., Xu, Y., Lo, C., Trung Pham, T., An, J., Coughlin, S., Dorn, G. W., Cyster, J. G.
  2011; 333 (6051): 1898-1903

- **Lymphatic endothelial cell sphingosine kinase activity is required for lymphocyte egress and lymphatic patterning** *JOURNAL OF EXPERIMENTAL MEDICINE*
  Pham, T. H., Baluk, P., Xu, Y., Grigorova, I., Bankovich, A. J., Pappu, R., Coughlin, S. R., McDonald, D. M., Schwab, S. R., Cyster, J. G.
  2010; 207 (1): 17-27

- **Sphingosine-1-phosphate in the plasma compartment regulates basal and inflammation-induced vascular leak in mice** *JOURNAL OF CLINICAL INVESTIGATION*
  Camerer, E., Regard, J. B., Cornelissen, I., Srinivasan, Y., Duong, D. N., Palmer, D., Pham, T. H., Wong, J. S., Pappu, R., Coughlin, S. R.
  2009; 119 (7): 1871-1879

- **Cortical sinus probing, S1P(1)-dependent entry and flow-based capture of egressing T cells** *NATURE IMMUNOLOGY*
  Grigorova, I. L., Schwab, S. R., Phan, T. G., Pham, T. H., Okada, T., Cyster, J. G.
  2009; 10 (1): 58-65

- **S1P(1) receptor signaling overrides retention mediated by G alpha(i)-coupled receptors to promote T cell egress** *IMMUNITY*

Pham, T. H., Okada, T., Matioubian, M., Lo, C. G., Cyster, J. G.

2008; 28 (1): 122-133

- **Epistasis between mouse Klra and major histocompatibility complex class I loci is associated with a new mechanism of natural killer cell-mediated innate resistance to cytomegalovirus infection** *NATURE GENETICS*

  Desrosiers, M. P., Kielczewska, A., Loredo-Osti, J. C., Adam, S. G., Makrigiannis, A. P., Lemieux, S., Pham, T., Lodoen, M. B., Morgan, K., Lanier, L. L., Vidal, S. M.

  2005; 37 (6): 593-599