Stanford

(Jian Xiong)

Postdoctoral Scholar, Chemical Engineering

Bio

BIO

I thrive to understand the roles of lysosomes in physiological and pathological conditions. Lysosomes are both degradation compartment and metabolic controlling hub, and dysregulation of lysosomal functions are frequently implicated in a vast number of diseases including neurodegenerative diseases, however, the systematic knowledge of the molecular mechanism by which lysosomal contributes to these diseases is lacking. Ion channels are the primary mediators of neuronal activity, defects in neuronal ion channel activity are linked with many kinds of neurodegenerative diseases. Interestingly, besides typical ion channels that are involved in the neuronal activity, defects in lysosomal ion channels, such as TRPML1, CLN7 and CLC-7 are also implicated in neuropathy. My previous work as Ph.D student in University of Texas MD Anderson Cancer Center focused on regulation of lysosomal function by ion channels and metabolites. I discovered a mechanism of lysosomal Na+ channel regulate mTORC1 activation by regulating lysosomal amino acid accumulation. I also discovered role of glutamine in controlling lysosomal degradation capacity. In the meantime, I developed novel methods to isolate organelles. My ultimate research goal is to understand the key developmental pathways and how alterations in gene sequences and expression contribute to human disease, therefore, I am pursuing independent academic researcher as my career goal. Starting Feb 2022, I work with Dr. Monther Abu-Remaileh at Stanford University on role of lysosomes in neurodegenerative diseases. I use genetics, chemical biology and omics approaches to study lysosome function under various physiological and pathological conditions, especially age-associated neurodegenerative disorders, and monogenic neurodegenerative lysosome storage diseases. In Stanford, I aim to integrate ionic regulation, metabolomic regulation and functional proteomic regulation to systematically understand the biology of lysosome in physiological conditions and pathological conditions.

INSTITUTE AFFILIATIONS

- Member, Maternal & Child Health Research Institute (MCHRI)

PROFESSIONAL EDUCATION

- B.S., Wuhan University , Biological Science and Biotechnology (2010)
- M.S, University of Texas MD Anderson Cancer Center , Cell and Regulatory Biology (2014)
- Ph.D, University of Texas MD Anderson Cancer Center , Biochemistry and Cell Biology (2020)

STANFORD ADVISORS

- Monther Abu-Remaileh, Postdoctoral Research Mentor
- Monther Abu-Remaileh, Postdoctoral Faculty Sponsor

Publications

PUBLICATIONS

- **Granulin loss and TMEM106B risk converge on lysosomal C-terminal fragment pathology in frontotemporal dementia.** *bioRxiv : the preprint server for biology*
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- **Mucin-binding protein shuttles enable delivery of brain-targeted therapeutics.** *bioRxiv : the preprint server for biology*
  Shi, S. M., Tender, G. S., Xiong, J., Buff, J. K., Park, H. I., Mendiola, J. H., Wilson, E. N., Abu-Remaileh, M., Bertozzi, C. R., Wyss-Coray, T.
  2026

- **Hematopoietic Stem Cell Transplantation with Enhanced Brain Conditioning Corrects Progranulin Deficiency in mouse models**
  Colella, P., Suarez-Nieto, M., Tejeda-Polanco, E., Vera, L., Poletto, E., Basurto, J., Nyame, K., Xiong, J., Abu-Remaileh, M., Gomez-Ospina, N.
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- **CNS-wide repopulation by hematopoietic-derived microglia-like cells corrects progranulin deficiency in mice.** *Nature communications*
  Colella, P., Sayana, R., Suarez-Nieto, M. V., Sarno, J., Nyame, K., Xiong, J., Pimentel Vera, L. N., Arozqueta Basurto, J., Corbo, M., Limaye, A., Davis, K. L., Abu-Remaileh, M., Gomez-Ospina, et al
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- **Glycerophosphodiesters inhibit lysosomal phospholipid catabolism in Batten disease.** *Molecular cell*
  Nyame, K., Hims, A., Aburous, A., Laqtom, N. N., Dong, W., Medoh, U. N., Heiby, J. C., Xiong, J., Ori, A., Abu-Remaileh, M.
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- **CNS Repopulation by Hematopoietic-Derived Microglia-Like Cells Corrects Progranulin deficiency.** *Research square*
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- **A gain-of-function TPC2 variant R210C increases affinity to PI(3,5)P2 and causes lysosome acidification and hypopigmentation.** *Nature communications*
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