药学院
School of Pharmacy

中文 English

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师资队伍
李学义

李学义

包氏基金特别研究员/博士生导师/课题组长

研究方向主要是通过再循环内体（或胞吞再循环区室）膜动态学的分子机制研究，以基因工程小鼠和由病人皮肤细胞诱导产生的诱导性多能干细胞为模型，结合生物化学、细胞生物学、系统生物学和动物行为学的方法和技术探索再循环内体在神经系统疾病发病过程中的作用，鉴定药物干预的靶点分子，并开展药物筛选和鉴定。

Xueyi Li, PhD, Pao Endowed Chair. The focus of Dr. Li's research is centered on membrane dynamics of the recycling endosome also referred to as endocytic recycling compartments elsewhere, which processes internalized receptors, transporters and adhesion molecules recycling back to the cell surface for reuse. Dr. Li's research employs genetically modified animals and induced pluripotent stem cells (iPSCs) generated from patient skin fibroblasts to dissect the role for the recycling endosome in the pathogenesis of neurological diseases. Approaches include state-of-the-art biochemistry, cell biology, and systems biology techniques and animal behavior analysis. The overarching goal of Dr. Li's research is to translate basic biomedical discoveries in his research into new therapeutics and or disease biomarkers.

*□□□简历

2003 德国海德堡大学生物化学中心，分子细胞生物学专业，博士学位

2003－2004 耶鲁大学医学院细胞生物学系，博士后

2004－2011 哈佛大学医学院附属麻省总院神经病学系，博士后、讲师

2011－ 哈佛大学医学院附属麻省总院神经病学系，助理教授

2014－ 上海交通大学药学院，药理学，特别研究员

*□□□主要成果

主要从事遗传性神经退行性疾病亨廷顿氏病的发病机制研究，揭示了亨廷顿氏病致病突变通过干预单体三磷酸鸟苷酸酶Rab11的激活而影响膜受体及运载蛋白内吞后再循环到细胞表面再利用，并阐释内吞后再循环障碍是引起神经元氧化应激失调及葡萄糖摄取降低的重要原因。

*□□□研究领域

内体囊泡运输（侧重于胞吞后再循环）的分子机制

胞吞后再循环在神经发育及神经退行性疾病发病过程中的作用

干预胞吞后再循环的新药研发

Xueyi Li

Pao Endowed Chair/Principal Investigator

Resume

2003 Ph.D., Heidelberg University Biochemistry Center

2003-2004 Postdoc, Department of Cell Biology, Yale University School of Medicine

2004-2011 Postdoc & Instructor, Department of Neurology, Massachusetts General Hospital & Harvard Medical School

2011-Present Assistant Professor of Neurology, Massachusetts General Hospital & Harvard Medical School

2014-Present Principal Investigator, School of Pharmacy, SJTU

Major Research Fields

Mechanism of endocytic recycling

Role of endocytic recycling in neurodevelopmental & neurodegenerative diseases

Discovery & development of drugs targeting endocytic recycling

Selected publications

1. McCory H, Williams D, Sapp E, Gatune LW, Wang P, DiFiglia M, Li X*. Glucose transporter 3 is a rab11-dependent trafficking cargo and its transport to the cell surface is reduced in neurons of CAG140 Huntington's disease mice. *Acta Neuropathol Commun*, 2014, 2:178.

2. Li X*, Valencia A#, Sapp E, Masso N, Alexander J, Patrick R, Kegel KB, Aronin N, DiFiglia M. Aberrant Rab11-dependent trafficking of the neuronal glutamate transporter EAAC1 causes oxidative stress and cell death in Huntington's disease. *J Neurosci*, 2010, 30: 4552-4561.

3. Li X*, Sapp E, Chase K, Comer-Tierney LA, Masso N, Alexander J, Patrick R, Kegel KB, Valencia A, Esteves M, Aronin N, DiFiglia M. Disruption of Rab11 activity in a knock-in mouse model of Huntington's disease. *Neurobiol Dis,* 2009, 36: 374-383.

4. Li X, Standley C, Sapp E, Valencia A, Qin ZH, Kegel KB, Yoder J, Comer-Tierney LA, Esteves M, Chase K, Alexander J, Masso N, Sobin L, Zeitlin S, Bellve K, Tuft R, Lifshitz L, Fogarty K, Aronin N, DiFiglia M. Mutant huntingtin impairs vesicle formation from recycling endosomes by interfering with rab11 activity. *Mol Cell Biol,* 2009, 29: 6106-6116.

5. Li X, Kaloyanova D, van Eijk M, Eerland R, van der Goot G, Oorschot V, Klumperman J, Lottspeich F, Starkuviene V, Wieland FT & Helms JB. Involvement of a GPI-anchored protein in maintenance of the Golgi structure. *Mol Biol Cell*, 2007, 18:1261-1271.

联系方式:

Email: xueyili@sjtu.edu.cn