Mechanism of Hyperhomocysteinemia-Induced Podocyte Injury

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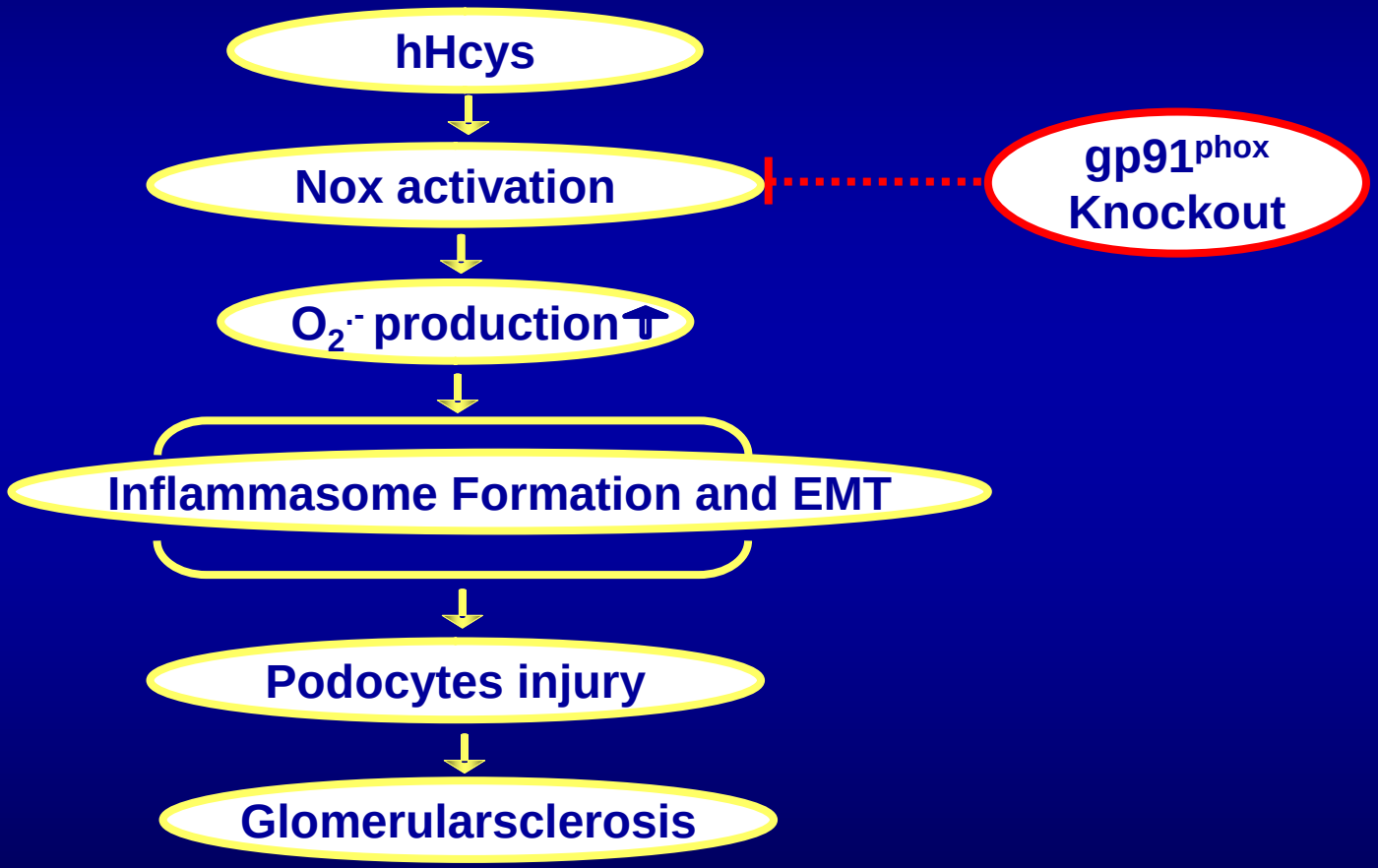
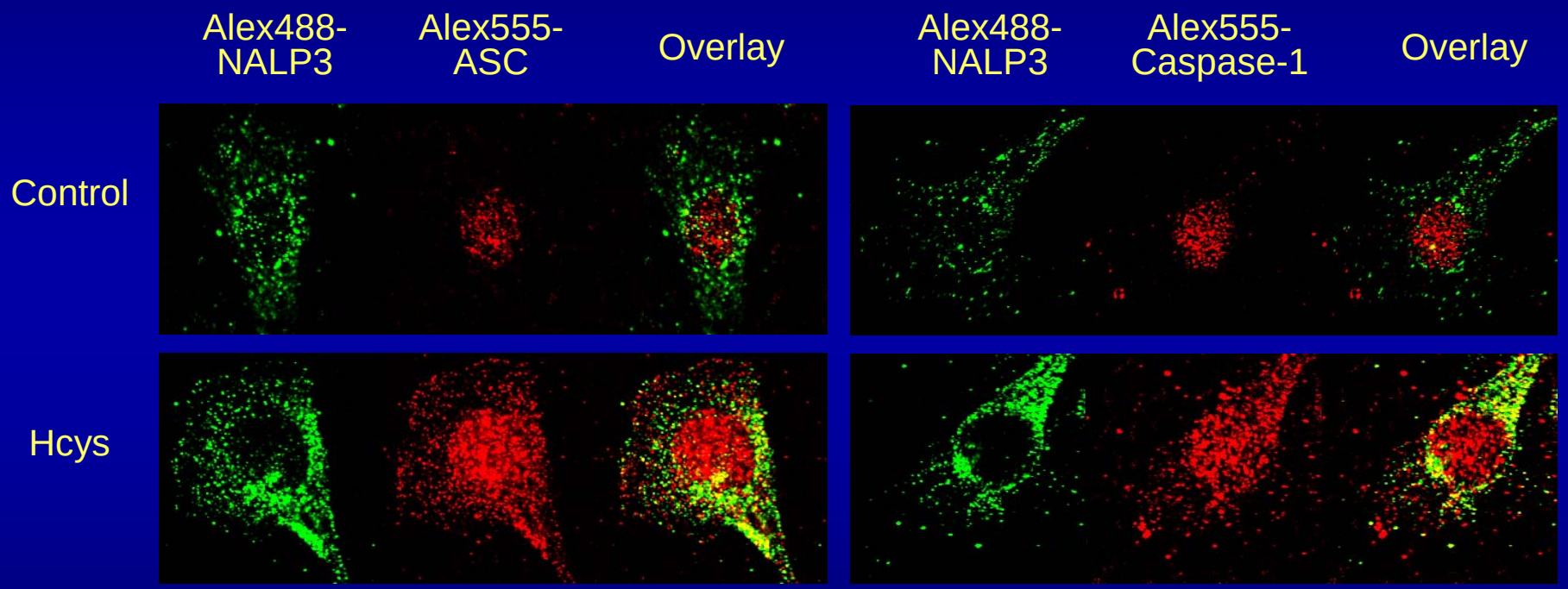
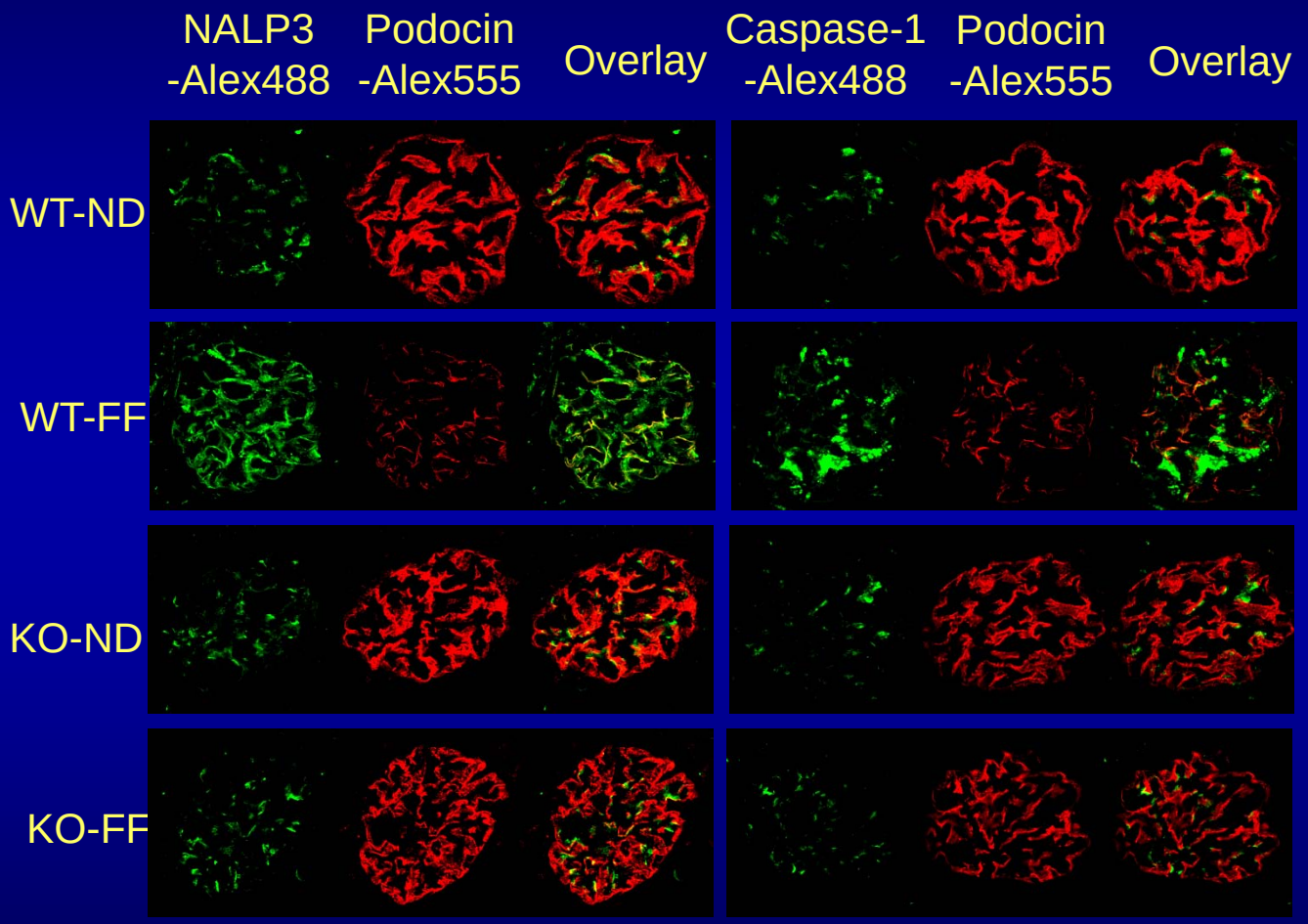
Hypothesis


hHcys
Nox activation
gp91phox
Knockout
O2·- production
Inflammasome Formation and EMT
Podocytes injury
Glomerularsclerosis

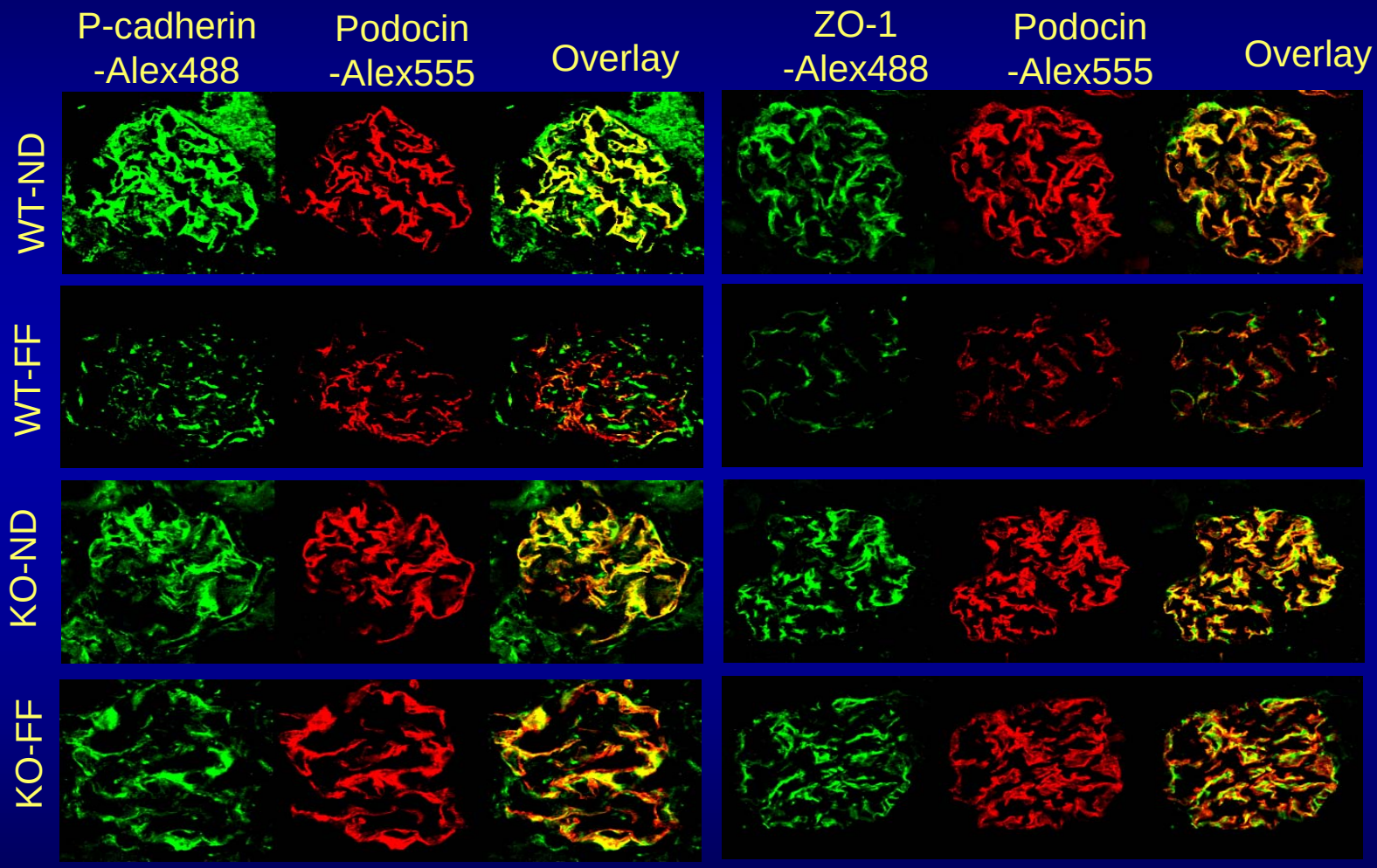
Immunofluorescent Staining-Inflammasome Formation in Podocytes



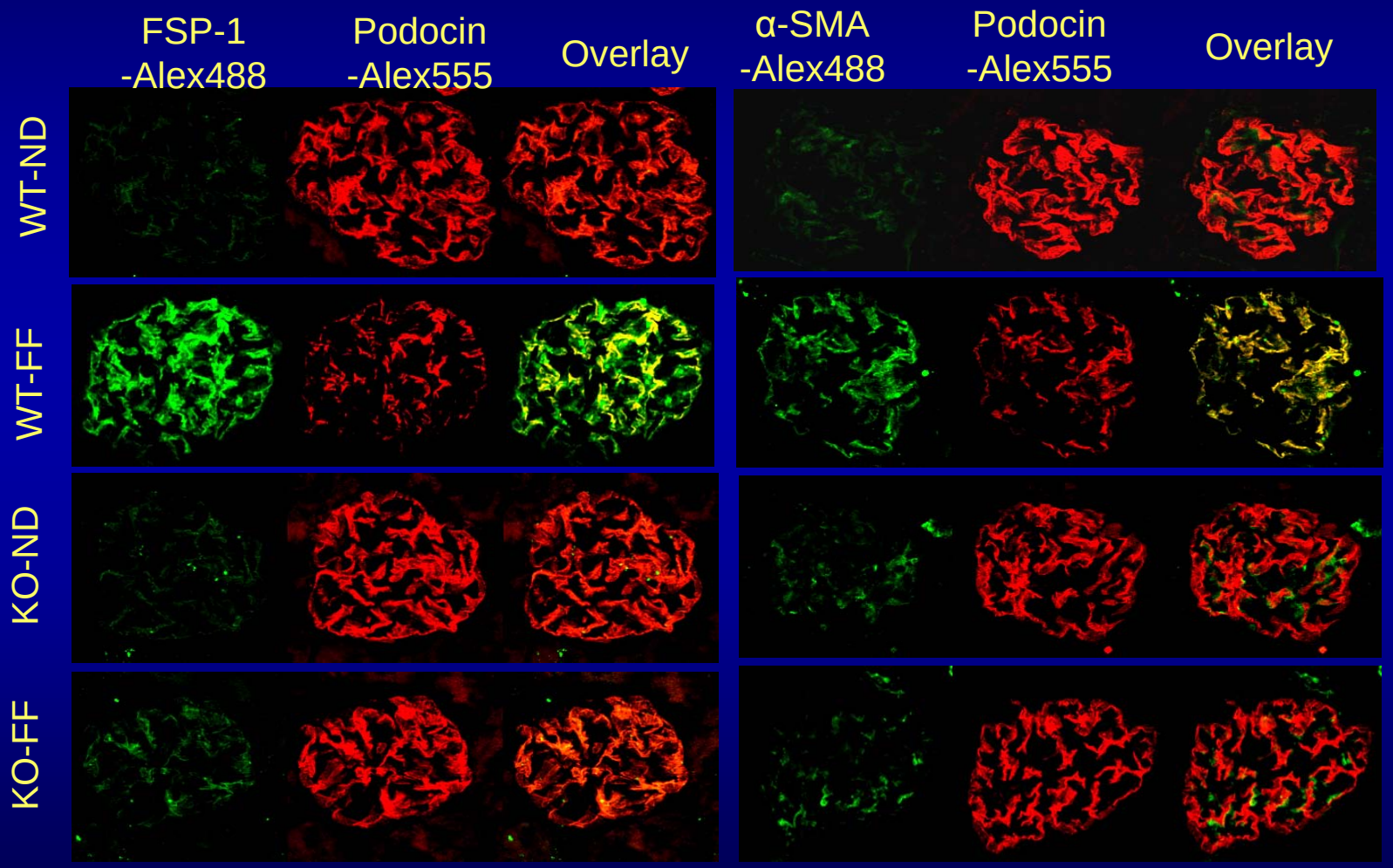
Immunofluorescent Staining-Inflammasome



Immunofluorescent Staining-EMT



Immunofluorescent Staining-EMT



Other Experiments

1. Protein preparation for ESR analysis, Western blot, and DHE experiments
2. RNA isolation and preparations
3. Immunohistochemistry staining
4. Animal sample collections including plasma, urine, and renal tissues

Publications

Articles:

1. Zhang C, Yi F, Xia M, Boini KM, Zhu Q, Laperle LA, Abais JM, Brimson CA, Li PL. NMDA Receptor-Mediated Activation of NADPH Oxidase and Glomerulosclerosis in Hyperhomocysteinemic Rats. Antioxid Redox Signal. 2010 Jun 30. [Epub ahead of print] PMID: 20406136.

2. Zhang C, Hu JJ, Xia M, Boini KM, Brimson CA, Laperle LA, Li PL. Protection of podocytes from hyperhomocysteinemia-induced injury by deletion of gp91phox gene. Free Radic Biol Med. 2010, 15;48(8):1109-17.

Meeting abstract:

Zhang C, Xia M, Boini KM, Hu JJ, Laperle LA, Li PL. Amelioration of glomerulosclerosis by NMDA receptor blockade in hyperhomocysteinemic rats. FASEB J. 2010 24:1059.6.

Lab Organization and Management

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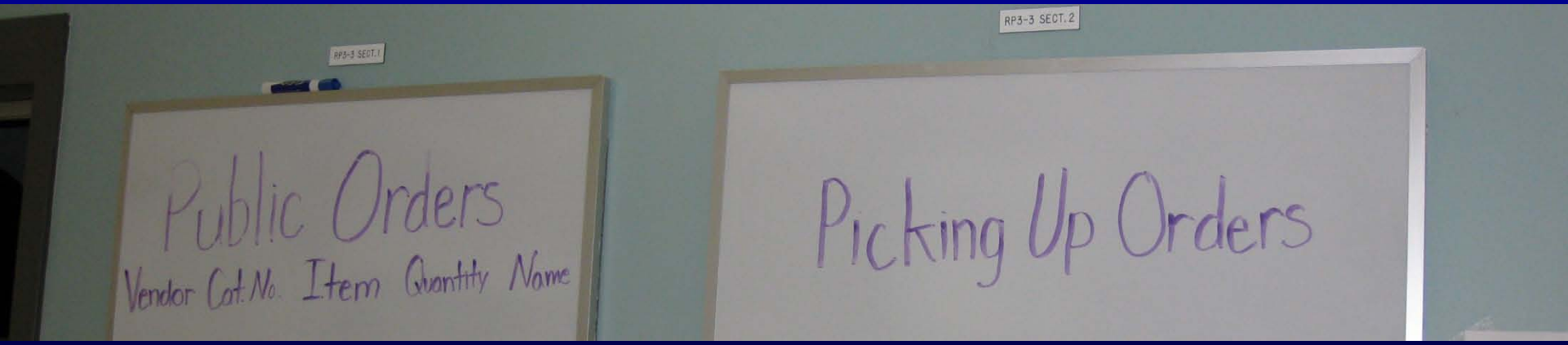

Acknowledgement

Dr. Li and all lab members

SPUR Program

Department of Pharmacology