BIOGRAPHICAL SKETCH

NAME: Xie, Xiaoqiao (Alison)

eRA COMMONS USERNAME (credential, e.g., agency login): XIAOQIAO_XIE

POSITION TITLE: Assistant Professor

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date	FIELD OF STUDY
University of Science and Technology of China (USTC), Hefei, Anhui, P.R. China	B. S.	07/2002	Biophysics
USTC, Hefei, Anhui, P.R. China	M. S.	07/2005	Neurobiology and Biophysics
University of California Riverside (UCR), Riverside, California, USA	Ph. D.	08/2011	Neuroscience
University of North Carolina at Chapel Hill (UNC-CH), Chapel Hill, North Carolina, USA	Postdoctoral training	09/2016	Pharmacology and glial biology

A. Personal Statement

It has come to light that astrocytes, the glial fibrillary acidic protein-expressing ($GFAP^+$) glial cells in the central nervous system participate in all aspects of neurobiology. However, little is known about the role of peripheral $GFAP^+$ glia in health and disease. My research goal is to investigate the role of $GFAP^+$ glial cells regulating micturition, with a special focus on the $GFAP^+$ satellite glial cells (SGCs) in the peripheral sensory and autonomic ganglia innervating bladder. As a member of *the Basic and Translational Urology Research Program* in the Department of Surgery and as a $GFAP^+$ glial biologist, I am excited to lead the research effort as the principal investigator.

My scientific training has a strong focus on glial modulation of neuronal activity and organ functions. I started studying $GFAP^+$ glia during my Ph.D. training. My thesis described for the first time homeostatic plasticity in astrocytic G-protein coupled receptors (GPCRs) signaling, which promoted my interest in studying $GFAP^+$ glial Gq-GPCR signaling *in vivo*. One challenge in studying glial function *in vivo* has always been our inability to selectively perturb glial signaling in intact animals. During my postdoctoral training, I developed and characterized the first transgenic mouse line that enabled remote glial activation in awake animals. Using these mice, I discovered that Gq-GPCR activation in sympathetic SGCs potentiates sympathetic output and cardiovascular function. My work was the first report describing the function of SGCs in autonomic ganglia.

In addition to studying sympathetic SGCs, I also investigated the role of SGC Gq-GPCR signaling in sensory ganglia. Unlike sympathetic SGCs, Gq-GPCR activation in sensory SGCs decreases sensory neuronal excitability, which leads to potent and long-lasting anesthesia. In addition, Gq-GPCR activation in sensory SGCs completely reversed the mechanical hypersensitivity in animal models of inflammatory pain. Again, my work on sensory SGCs was the first report on the analgesic effect of peripheral glia activation *in vivo*.

My long-term research goal has always been to target glial signaling for therapeutic interventions. Since becoming an Assistant Professor in the Division of Urology, Department of Surgery, I started probing the neuromodulatory role of sensory SGCs in bladder afferent signaling. With the support of my previous CCTSI pilot grant, I confirmed that Gq-GPCR activation in lumbosacral SGCs reversed the symptoms of bladder overactivity and visceral hypersensitivity in animal models of overactive bladder and chronic pelvic pain. I am currently investigating the molecular mechanism underlying SGC-neuron interaction in sensory ganglia. In addition, I am interested in studying the role of autonomic SGC signaling in major pelvic ganglia (MPGs), the autonomic ganglia innervating bladder and urethra. With a broad skillset in mouse genetics, animal disease models, electrophysiological, physiological, and behavioral recordings, I am looking forward to making valuable contributions to the neuro-urology field on and outside of CU-AMC.

B. Positions and Experience

Research Positions and Employment

2019-present Assistant Professor, Division of Urology, Department of Surgery, CU-AMC, Aurora, CO
2017-2019 Instructor, Division of Urology, Department of Surgery, CU-AMC, Aurora, CO
2016-2017 Research Assistant Professor, Department of Pharmacology, UNC-CH, Chapel Hill, NC
2011-2016 Postdoctoral research associate, Department of Pharmacology, UNC-CH, Chapel Hill, NC
2006-2011 Graduate research assistant, Department of Cell Biology and Neuroscience, UCR, Riverside,CA
2003-2005 Graduate research assistant, Dept. of Neurobiology and Biophysics, USTC, Hefei, Anhui, China
2001-2003 Undergraduate research assistant, Dept. of Neurobiology and Biophysics, USTC, Hefei, China

Teaching and Mentoring Experience

2014 Excellence in Mentoring Undergraduates, Office for Postdoctoral Affairs and Office of Undergraduate Research, UNC-CH
2011 University Teaching Certificate, Graduate Division, UCR
2008-2011 Graduate teaching assistant, Department of Cell Biology & Neuroscience, UCR

Professional Memberships and Service

Current South-Central Section (SCS) of the American Urological Association (AUA), member
Current Society for Basic Urologic Research (SBUR), faculty member
Current American Society of Neurochemistry (ASN), faculty member
Current Colorado Clinical Translational Science Institute (CCTSI), faculty member
Current Center for NeuroScience (CNS), CU-AMC, faculty member
Current Neuroscience Graduate Program, faculty member
2019-present CCTSI Pilot Grant Program Review Panel, reviewer
2018-present Denver Metro Regional Science and Engineering Fair (DMRSEF), Judge

Theses and Selected Scientific Presentations

2002 B. S. in Biophysics, “Effects of different stimulation modes on short-term synaptic plasticity of visual cortex in adult rats”
2005 M. S. in Biophysics, “The function of visual cortex neurons in aging rats”
2011 Ph. D. in Neuroscience, “Bidirectional scaling of astrocytic metabotropic glutamate receptor signaling following long term changes in neuronal synaptic transmission”
2013 Gordon Conference, *Glial Biology*, “Studying the role of glial Gq signaling in the regulation of the cardiovascular system”
2014 Podium Presentation, Cold Spring Harbor Laboratory Meeting, *Glia in Health & Disease*, “Ganglionic $GFAP^+$ glia regulate cardiovascular function via Gq-GPCR activation”
2015 Poster Presentation, Gordon Conference, *Functional Interactions among Glia & Neurons*, “Ganglionic $GFAP^+$ glia regulate cardiovascular function”
2016 Podium Presentation, American Society for Neurochemistry (ASN) annual meeting, “Chemogenetic activation of satellite glial Gq Signaling enhances cardiac function *in vivo*”
Invited talk, UNC-CH, “Gq-GPCR Signaling in Sympathetic Satellite Glial Cells Regulate Cardiovascular Functions *in vivo*”
Podium Presentation, Duke Neuroimmunology and Glia Group Conference, “Gq-GPCR Signaling in Sympathetic Satellite Glial Cells Regulate Cardiovascular Functions *in vivo*”
Podium Presentation, Hypertension 2016, “Chemogenetic Activation of Peripheral Glial Gq GPCR Signaling Enhances Cardiovascular Function in vivo”
Invited talk, UNC-CH, “Targeting sympathetic satellite glial cells for treating cardiovascular diseases”
2017 Invited talk, University of Colorado, Boulder, “DREADD the Glia: Pharmacogenetic Approaches for Studying the Role of $GFAP^+$ Glia in Physiology and Disease”
2018 ASN annual meeting, “Targeting Satellite Glial Signaling for the Treatment of Chronic Pain”
Podium Presentation, American Urology Association (AUA) annual meeting, “VEGF-induced bladder nerve remodeling and visceral hyperalgesia in bladder pain”
Poster Presentation, South Central Section (SCS) of the AUA annual meeting, “VEGF-induced bladder nerve remodeling and visceral hyperalgesia in bladder pain”
2019 Poster Presentation, SCS of the AUA annual meeting, “Pharmacogenetic inhibition of afferent excitability alleviates VEGF-induced visceral allodynia and hyperalgesia in a mouse model of urological chronic pelvic pain syndrome (UCPPS)”

Poster Presentation, Society for Basic Urologic Research (SBUR) annual meeting, "Pharmacogenetic inhibition of afferent excitability alleviates VEGF-induced visceral allodynia and hyperalgesia in a mouse model of UCPPS"

2020 Virtual Presentation, AUA annual meeting, "Pharmacogenetic inhibition of lumbosacral spinal and sensory neurons alleviates visceral pain and improves lower urinary tract symptoms in animal model of UCPPS"

Virtual Presentation, SCS of the AUA meeting, "Lumbar-sacral neuromodulation alleviates visceral pain and improves lower urinary tract symptoms in animal model of urological chronic pelvic pain syndrome"

Virtual Presentation, SBUR annual meeting, "Adeno-associated viral vector (AAV)-mediated pharmacogenetic inhibition of lumbosacral sensory neurons alleviates visceral hypersensitivity in a mouse model of UCPPS"

2021 Virtual Presentation, ASN annual meeting, "Pharmacogenetic Inhibition of Afferent Excitability Alleviates VEGF-induced Visceral Hypersensitivity in a Mouse Model of Urological Chronic Pelvic Pain Syndrome (UCPPS)"

Virtual Presentation, AUA annual meeting, "Sensory glial Gq-GPCR signaling alleviates visceral pain and improves micturition function in an animal model of urological chronic pelvic pain syndrome"

Invited talk, Department of Anesthesiology, University of Colorado, Anschutz Medical Campus, "The analgesic role of peripheral GFAP$^+$ glia *in vivo*"

2022 Invited talk, Neuroscience Graduate Program, University of Colorado, Anschutz Medical Campus, "Peripheral glia modulation of autonomic control: from heart to bladder"

C. Contributions to Science

1. Sensory Neuroplasticity in Developing and Aging Brain

My early research focused on neuroplasticity in the geniculo-cortical visual pathway. Using rat as a model, my work characterized the importance of intra-cortical inhibitory circuits to the appropriate signal-to-noise ratio in visual processing. My work also showed that age-related changes in cortical inhibitory synaptic transmission result in declined signal-to-noise ratio as well as quicker visual adaptation in cortical neurons, both of which are accounted for decreased visual acuity in aging visual cortex.

- Jia F, **Xie X**, Zhou Y. (2004) Short-term depression of synaptic transmission from rat lateral geniculate nucleus to primary visual cortex *in vivo*. *Brain Research*. 2004 Mar 26;1002(1-2):158-61. doi: 10.1016/j.brainres.2004.01.001
- Jia F, Wei H, Li X, **Xie X**, Zhou Y. (2006) Short-term synaptic plasticity in the rat geniculo-cortical pathway during development *in vivo*. *Neuroscience Letters*. 2006 May 1;398(1-2):73-7. doi: 10.1016/j.neulet.2005.12.054
- Wang H, **Xie X**, Li X, Chen B, Zhou Y. (2006) Functional degradation of visual cortical cells in aged rats. *Brain Research*. 2006 Nov 29;1122(1):93-8. Doi: 10.1016/j.brainres.2006.09.010

2. Neuronal Activity Induced Homeostatic Plasticity of Glia GPCRs

My Ph.D. study concerned how GPCR signaling in glial cells adapts to neuronal network activity. Astrocytes are resident glial cells in the CNS and play important roles in governing neuronal excitability and providing GPCR-mediated feedback to synaptic transmission. Prior to my work, little was known about how astrocytic signaling changes in response to neuronal network activity. Using murine brain slices as *in situ* model and combined with patch-clamping electrophysiology and intracellular Ca^{2+} recordings, I showed that astrocytic Gq-GPCRs exhibit homeostatic scaling in their signaling cascades. This was the first report on neuronal-induced astrocytic long-term plasticity, and the first demonstration of homeostatic scaling of glial receptors. In addition, my work established a novel method of translating intracellular Ca^{2+} signature to Gq-GPCR signaling, which was published separately on *JOVE*.

- **Xie AX**, Sun MY, Murphy T, Lauderdale K, Tiglao E, Fiacco TA. (2012) Bidirectional scaling of astrocytic metabotropic glutamate receptor signaling following long-term changes in neuronal firing rates. *PLoS One*. 2012; 7(11):e49637. doi: 10.1371/journal.pone.0049637
- **Xie AX**, Lauderdale K, Murphy T, Myers TL, Fiacco TA. (2014) Inducing plasticity of astrocytic receptors by manipulation of neuronal firing rates. *JoVE*. 2014 Mar 20;(85). doi: 10.3791/51458

- Sun MY, Devaraju P, **Xie AX**, Holman I, Samones E, Murphy TR, Fiacco TA. (2014) Astrocyte calcium microdomains are inhibited by bafilomycin A1 and cannot be replicated by low-level Schaffer collateral stimulation in situ. *Cell Calcium*. 2014 Jan;55(1):1-16. doi: 10.1016/j.ceca.2013.10.004

3. Glia Modulation of Autonomic Functions and *in vivo* Manipulation of Glia GPCR Signaling

The long-term goal of conducting glia research is to target glial signaling for disease treatments. But first, we need to understand the role of glia *in vivo* and in intact neural circuits. When I graduated with my Ph. D., there was no established animal model to selectively perturb signaling pathways in glia *in vivo*. During the first part of my postdoctoral research, I characterized the GFAP-Gq-DREADD transgenic mice, in which pharmacogenetic activation of Gq-GPCR signaling in $GFAP^+$ glia was made possible in conscious and free-moving animals for the first time. $GFAP^+$ glia consist of astrocytes in the brain and spinal cord, satellite glial cells in peripheral ganglia, and terminal Schwann cells in peripheral tissue. Using the GFAP-Gq-DREADD mice, I discovered that satellite glial activation in sympathetic ganglia significantly increases cardio output and blood pressure. Moreover, persist activation of sympathetic satellite glia results in changes in blood pressure in sex-dependent manner. These findings were the first report on the function of sympathetic satellite glial cells. As the primary researcher and project manager in these studies, I served as co-investigator and are corresponding authors on key publications.

- Agulhon C, Boyt KM, **Xie AX**, Friocourt F, Roth BL, McCarthy KD. (2013) Modulation of the autonomic nervous system and behaviour by acute glial cell Gq protein-coupled receptor activation *in vivo*. *J Physiol.* 2013 Nov 15;591(22):5599-609. doi: 10.1113/jphysiol.2013.261289.
- **Xie AX**, Petravicz, J, McCarthy KD. (2015) Molecular approaches for manipulating astrocytic signaling *in vivo. Front Cell Neurosci.* 2015 Apr 21;9:144. doi: 10.3389/fncel.2015.00144
- **Xie AX*,** Lee JJ, McCarthy KD. (2016) Ganglionic $GFAP^+$ Glial Gq-GPCR Signaling Enhances Heart Functions in vivo. *Corresponding author. *JCI Insight*. 2017;2(2):e90565. doi:10.1172/jci.insight.90565.
- **Xie AX*,** Chaia A, McCarthy KD. (2017) Targeting sympathetic glia for treating cardiovascular diseases. *Corresponding author. *Receptors and Clinical Investigation*. 2017;4:e1572. doi: 10.14800/rci.1572.

4. Inflammation-induced Neuroplasticity in Animal Models of Bladder Overactivity and Pelvic Pain

Chronic pelvic pain syndrome (CPPs) and bladder overactivity (OAB) affects millions in the US alone. Individuals with CPPs and OAB experience increased frequency and urgency of urination, which are closely associated with hypersensitivity of primary afferents innervating the urinary bladder. After joining the Urology Research Program in the Department of Surgery at CU-AMC, I worked on multiple visceral pain and OAB animal models to identify the molecular mechanisms underlying visceral hypersensitivity. In our recent publications, I described the epigenetic changes in sensory pathways associated with inflammatory pelvic pain. Most recently, our work using pharmacogenetic inhibition of sensory neurons to reverse visceral hypersensitivity in vascular endothelial growth factor (VEGF) induced UCPPS animal model has been submitted.

- **Xie AX,** Pan XQ, Meacham RB, Malykhina AP. (2018) The Expression of Transcription Factors MeCP2 and CREB Is Modulated in Inflammatory Pelvic Pain. *Front Syst Neurosci.* 2019 Jan 11;12:69. doi: 10.3389/fnsys.2018.00069. eCollection 2018.
- Iguchi N, Carrasco A Jr, **Xie AX**, Pineda RH, Malykhina AP, Wilcox DT. (2021) Functional constipation induces bladder overactivity associated with upregulations of Htr2 and Trpv2 pathways. *Sci Rep.* 2021 Jan 13;11(1):1149. doi: 10.1038/s41598-020-80794-0.
- **Xie AX,** Iguchi N, Clarkson TC, Malykhina AP. (2022) Pharmacogenetic inhibition of sensory afferent excitability alleviates bladder VEGF signaling-induced visceral hypersensitivity in an animal model of UCPPS. *PLOS ONE, in press.*

5. The Analgesic Role of Peripheral Sensory Glia and Its Implication in CPP and OAB

Using the GFAP-Gq-DREADD mice, in which sensory glia can be remotely activated *in vivo* and in awake animals, I have discovered that satellite glial activation in lumbosacral sensory ganglia alleviates inflammatory pain in mice. Following their Gq-GPCR activation, lumbosacral satellite glial cells inhibit sensory neuronal activity, likely via local adenosine signaling, and in turn produces strong analgesic effect *in vivo*. Pharmacogenetic activation of sensory satellite glia completely reversed inflammation-induced mechanical and thermal sensitivity in mouse hind paws. In addition, I have discovered that Gq-GPCR activation in terminal Swann cells also alleviates nerve injury-induced neuropathic pain (currently under review). These data suggested that the neuromodulatory effect of peripheral glia shows strong therapeutic potential in acute control of afferent

excitability. Based on these fundings, I recently received a R01 award by NIDDK to study the neuromodulatory role of sensory satellite glia in visceral sensitivity and bladder function.

- **Xie AX*,** Madayag A, Minton SK, McCarthy KD, Malykhina AP. (2020) Sensory satellite glial Gq-GPCR activation alleviates inflammatory pain via peripheral adenosine 1 receptor activation. *Corresponding author. *Sci Rep.* 2020 Aug 25;10(1):14181. doi: 10.1038/s41598-020-71073-z.
- **Xie AX†*,** Taves S†, McCarthy KD. (2021) Nuclear factor Kappa B-COX2 pathway activation in non-myelinating Schwann cells is necessary for the maintenance of neuropathic pain in mice. *Corresponding author. *Front. Cell. Neurosci.* 14 January 2022 doi: 10.3389/fncel.2021.782275

D. List of Published Work (in English)

- Jia F, **Xie X**, Zhou Y. (2004) Short-term depression of synaptic transmission from rat lateral geniculate nucleus to primary visual cortex *in vivo*. *Brain Research*. 2004 Mar 26;1002(1-2):158-61. doi: 10.1016/j.brainres.2004.01.001
- Jia F, Wei H, Li X, **Xie X**, Zhou Y. (2006) Short-term synaptic plasticity in the rat geniculo-cortical pathway during development *in vivo*. *Neuroscience Letters*. 2006 May 1;398(1-2):73-7. doi: 10.1016/j.neulet.2005.12.054
- Wang H, **Xie X**, Li X, Chen B, Zhou Y. (2006) Functional degradation of visual cortical cells in aged rats. *Brain Research*. 2006 Nov 29;1122(1):93-8. doi: 10.1016/j.brainres.2006.09.010
- Carson MJ, Crane J, **Xie AX**. (2008) Modeling CNS microglia: the quest to identify predictive models. Drug Discov Today Dis Models. 2008;5(1):19-25. doi: 10.1016/j.ddmod.2008.07.006.
- **Xie AX**, Sun MY, Murphy T, Lauderdale K, Tiglao E, Fiacco TA. (2012) Bidirectional scaling of astrocytic metabotropic glutamate receptor signaling following long-term changes in neuronal firing rates. *PLoS One*. 2012; 7(11):e49637. doi: 10.1371/journal.pone.0049637
- Agulhon C, Boyt KM, **Xie AX**, Friocourt F, Roth BL, McCarthy KD. (2013) Modulation of the autonomic nervous system and behaviour by acute glial cell Gq protein-coupled receptor activation *in vivo*. *J Physiol.* 2013 Nov 15;591(22):5599-609. doi: 10.1113/jphysiol.2013.261289.
- **Xie AX**, Lauderdale K, Murphy T, Myers TL, Fiacco TA. (2014) Inducing plasticity of astrocytic receptors by manipulation of neuronal firing rates. *JoVE*. 2014 Mar 20;(85). doi: 10.3791/51458
- Sun MY, Devaraju P, **Xie AX**, Holman I, Samones E, Murphy TR, Fiacco TA. (2014) Astrocyte calcium microdomains are inhibited by bafilomycin A1 and cannot be replicated by low-level Schaffer collateral stimulation in situ. *Cell Calcium*. 2014 Jan;55(1):1-16. doi: 10.1016/j.ceca.2013.10.004
- **Xie AX**, Petravicz, J, McCarthy KD. (2015) Molecular approaches for manipulating astrocytic signaling *in vivo. Front Cell Neurosci.* 2015 Apr 21;9:144. doi: 10.3389/fncel.2015.00144
- **Xie AX*,** Lee JJ, McCarthy KD. (2016) Ganglionic GFAP$^+$ Glial Gq-GPCR Signaling Enhances Heart Functions in vivo. *Corresponding author. *JCI Insight*. 2017;2(2):e90565. doi:10.1172/jci.insight.90565.
- Annis RP, Swahari V, Nakamura A, **Xie AX**, Hammond SM, Deshmukh M. (2016) Mature neurons dynamically restrict apoptosis via redundant premitochondrial brakes. FEBS J. 2016 Dec;283(24):4569-4582. doi: 10.1111/febs.13944. Epub 2016 Nov 18.
- **Xie AX*,** Chaia A, McCarthy KD. (2017) Targeting sympathetic glia for treating cardiovascular diseases. *Corresponding author. *Receptors and Clinical Investigation*. 2017;4:e1572. doi: 10.14800/rci.1572.
- **Xie AX,** Pan XQ, Meacham RB, Malykhina AP. (2019) The Expression of Transcription Factors MeCP2 and CREB Is Modulated in Inflammatory Pelvic Pain. *Front Syst Neurosci.* 2019 Jan 11;12:69. doi: 10.3389/fnsys.2018.00069. eCollection 2018.
- **Xie AX*,** Madayag A, Minton SK, McCarthy KD, Malykhina AP. (2020) Sensory satellite glial Gq-GPCR activation alleviates inflammatory pain via peripheral adenosine 1 receptor activation. *Corresponding author. *Sci Rep.* 2020 Aug 25;10(1):14181. doi: 10.1038/s41598-020-71073-z.
- Iguchi N, Carrasco A Jr, **Xie AX**, Pineda RH, Malykhina AP, Wilcox DT. (2021) Functional constipation induces bladder overactivity associated with upregulations of Htr2 and Trpv2 pathways. *Sci Rep.* 2021 Jan 13;11(1):1149. doi: 10.1038/s41598-020-80794-0.

- **Xie AX†*,** Taves S†, McCarthy KD. (2021) Nuclear factor Kappa B-COX2 pathway activation in non-myelinating Schwann cells is necessary for the maintenance of neuropathic pain in mice. *Corresponding author. *Front. Cell. Neurosci.* 14 January 2022 doi: 10.3389/fncel.2021.782275
- **Xie AX,** Iguchi N, Clarkson TC, Malykhina AP. (2022) Pharmacogenetic inhibition of sensory afferent excitability alleviates bladder VEGF signaling-induced visceral hypersensitivity in an animal model of UCPPS *PLoS ONE*, in Press.

E. Research Support

Ongoing Research Support

R01 DK129260-01 Xie (PI) 08/05/2021-08/04/2026
Activating Peripheral Glia to Relieve Visceral Pain in Animal Models of Urological Chronic Pelvic Pain Syndrome (UCPPS)
Role: PI
Direct cost: $220,000/year
This proposal is the first proposal on the roles of peripheral $GFAP^+$ glia in bladder function and disease.

R01 DK116648-01A1 Malykhina (PI) 09/08/2020-05/30/2023
Mechanisms of neurogenic voiding dysfunction in a viral murine model of multiple sclerosis
Role: Key Personnel
The project will investigate the neural mechanisms of lower urinary tract symptoms in multiple sclerosis.

R01 DK121506-01 Malykhina (PI) 08/01/2019-07/30/2022
Regulation of pelvic pain and micturition reflex by VEGF in urological chronic pelvic pain syndrome
Role: Key Personnel
This application evaluates the pathological role of VEGF signaling in the sensory neural plasticity innervating the lower urinary tract in urological chronic pelvic pain syndrome.

Recently Completed Research Support

Academic Enrichment Funds, Surgery, CU-AMC Xie (PI) 09/01/2020-08/30/2021
The role of mechanosensitive TREK-1 channels in detrusor overactivity and voiding dysfunction in patients with overactive bladder (OAB)
Role: PI
Direct cost: $40,000
This project investigates the cellular and molecular mechanisms of aberrant mechano-sensitivity in idiopathic detrusor overactivity.

Colorado Pilot Program Mentored Award, CCTSI Xie (PI) 03/01/2019-02/29/2020
Beyond the neurons: the role of peripheral glia in neurogenic bladder dysfunction
Role: PI
Direct cost: $30,000
This project tests if satellite glial Gq-GPCR activation modulates visceral sensitivity *in vivo*.