CURRICULUM VITAE

KRISHNAKUMAR KIZHATIL

Professional address: The Jackson Laboratory
600 Main Street
Bar Harbor, ME 04609

Phone: 207-288-6262 (w) / 919-699-1948 (c)

e-mail: krish.kizhatil@jax.org / kizhatil@gmail.com

Citizenship: United States of America

Education:

Bachelor of Pharmacy (Honors, June 1992)- Birla Institute of Technology and Science (BITS), Pilani, Rajasthan, India

Doctor of Philosophy (Ph.D., June 2000)- Department of Microbiology and Immunology, University of Tennessee Health Science Center, Memphis, TN, USA;
Thesis advisor: Lorraine M. Albritton, Ph.D.; Thesis title: Role of the cellular cytoskeleton in retrovirus entry
Postdoctoral Fellow (April 2000-May 2006)- HHMI and Department of Cell Biology, Duke University Medical Center, Durham, North Carolina.
Supervisor: Vann Bennett, M.D., Ph.D.

Professional Positions

January 1992 - June 1992: Research Assistant and Visiting Student, Practical Session Adviser: Dr. Brahm S. Srivastava, Cholera Vaccine Laboratory; Central Drug Research Institute, Lucknow, Uttar Pradesh, India

August 1993 - December 1999: Graduate Teaching Assistant, Department of Microbiology and Immunology, College of Medicine, The University of Tennessee, Memphis Tennessee.

January 2000 - March 2000: Postdoctoral Fellow, Department of Microbiology and Immunology, College of Medicine, The University of Tennessee, Memphis, Tennessee.

April 2000 - October 2004: Postdoctoral Fellow, HHMI and Department of Cell Biology, Duke University Medical Center, Durham, North Carolina.

November 2004 - May 2006: Postdoctoral Fellow, Department of Cell Biology, Duke University Medical Center, Durham, North Carolina.

May 2006 - May 2009: Senior Research fellow, Department of Cell Biology, Duke University Medical Center, Durham, North Carolina.

June 2009 – September 2017: Associate Research Scientist, The Jackson Laboratory [Laboratory of Dr. Simon John; Howard Hughes Medical Institute]

October 2017 –2019: Research Scientist, The Jackson Laboratory [Laboratory of Dr. Simon John; Howard Hughes Medical Institute]

November 2019 –Present: Research Scientist, The Jackson Laboratory [Laboratory of Dr. Gareth Howell]

Honors and Awards

Recipient - Barbara and Joseph Cohen Young Investigator Award for 2010, 2015, 2017

Lewis Rudin Glaucoma Prize of the New York Academy of Medicine for 2014 for the most outstanding scholarly article on glaucoma published in a peer-reviewed journal. (Awarded in 2015)

Grants/Research Support

Active:

09/30/2017 – 08/31/2022
Agency: NIH/NEI RO1 (R01 EY028175),
Role: PI
Title: Determining How Lymphatic Molecules Control Conventional Outflow.
Total Direct Costs: $250,000

09/30/2020 – 08/31/2021
Agency: NIH/NEI RO1 (Administrative Supplement, R01 EY028175-04S1),
Role: PI
Title: Determining How Lymphatic Molecules Control Conventional Outflow.
Total Direct Costs: $156,158

01/01/2021 – 11/30/2025
Agency: NIH/NEI (R01 EY032062)

Title: Does VECAD at Schlemm canal cell-junctions determine IOP and glaucoma risk?

Role: Co-PI with Dr. Simon John, Columbia University.

Total Direct Costs: $179,781 (Year 1)

03/31/2021 – 2/29/2024
Agency: Bright Focus Foundation (Special opportunity grant)

Title: Selective Targeting of Schlemm's Canal Inner Wall for Next-Generation Glaucoma Drugs.

Role: Co-PI with Drs. Simon John (Columbia University), Dan Stamer (Duke University), Ross Ethier (Georgia Tech), Darryl Overby (ICL, UK), Joseph Sherwood-van Batenburg (ICL, UK).

Total Direct Costs: $69,950

Completed Research Support:

07/01/2017 - 6/30/2019
Agency: Bright Focus Foundation G2017152
Role: PI
Title: Determining the Neuronal Control of Intraocular Pressure.
Total Direct Costs: $75,000

Scientific/ Professional activities

Peer reviewer: Glaucoma Foundation, Journal of Biological Chemistry, Proceeding of National Academy of Science, IOVS, Experimental Eye Research, PlosOne, ELife, Molecular Vision, Survey of Ophthalmology, npj Regenerative Medicine.

Session Moderator: Platform Session I: Neurodegeneration, ISER/BrightFocus Glaucoma Symposium, Atlanta, Georgia, October 24, 2019.

Rudin prize committee member 2018

Professional Affiliations

Association for Research in Vision and Ophthalmology (ARVO)
International Society for Eye Research (ISER)
Society for Neuroscience (SfN)
American Society for Cell Biology (ASCB)

Publications

1. **K. K. Kumar,** R. Srivastava, V. B. Sinha, J. Michalski, J. B. Kaper, and B. S. Srivastava. 1994. recA mutations reduce adherence and colonization by classical and El Tor strains of Vibrio cholerae. Microbiology 140 (Pt 5)**:**1217-1222. PubMed

2. **K. Kizhatil** and L. M. Albritton. 1997. Requirements for different components of the host cell cytoskeleton distinguish ecotropic murine leukemia virus entry via endocytosis from entry via surface fusion. Journal of Virology **71:**7145-7156. PubMed

3. M. Chung, **K. Kizhatil**, L. M. Albritton, and G. N. Gaulton. 1999. Induction of syncytia by neuropathogenic murine leukemia viruses depends on receptor density, host cell determinants, and the intrinsic fusion potential of envelope protein. Journal of Virology 73**:**9377-9385. PubMed

4. **K. Kizhatil,** A. Gromley, and L. M. Albritton. 2001. Two point mutations produce infectious retrovirus bearing a green fluorescent protein-SU fusion protein. Journal of Virology 75**:**11881-11885. PubMed

5. S. M. Jenkins, **K. Kizhatil**, N. R. Kramarcy, A. Sen, R. Sealock, and V. Bennett 2001. FIGQY phosphorylation defines discrete populations of L1 cell adhesion molecules at sites of cell-cell contact and in migrating neurons. Journal of Cell Science 114**:**3823-3835. PubMed

6. **K. Kizhatil** and L. M. Albritton. 2002. System y+ localizes to different membrane subdomains in the basolateral plasma membrane of epithelial cells. American Journal of Physiology Cell Physiology 283**:**C1784-1794. PubMed

7. **K. Kizhatil**, Y. X. Wu, A. Sen, and V. Bennett. 2002. A new activity of doublecortin in recognition of the phospho-FIGQY tyrosine in the cytoplasmic domain of neurofascin. Journal of Neuroscience 22**:**7948-7958. PubMed

8. **K. Kizhatil**, and V. Bennett. 2004. Lateral membrane biogenesis in human bronchial epithelial cells requires 190-kDa ankyrin-G. Journal of Biological Chemistry **279:**16706-16714. PubMed

9. **K. Kizhatil,** W. Yoon, P. J. Mohler, L. H. Davis, J. A. Hoffman, and V. Bennett. 2007. Ankyrin-G and beta2-spectrin collaborate in biogenesis of lateral membrane of human bronchial epithelial cells. Journal of Biological Chemistry 282**:**2029-2037. PubMed

10. **K. Kizhatil**, J. Q. Davis, L. Davis, J. Hoffman, B. L. Hogan, and V. Bennett. 2007. Ankyrin-G is a molecular partner of E-cadherin in epithelial cells and early embryos. Journal of Biological Chemistry 282**:**26552-26561. PubMed

11. **K. Kizhatil**, N. Sandhu, N. Peachey, and V. Bennett. 2009. Ankyrin-B coordinates assembly of beta-2 spectrin, the Na/K ATPase and the Na/Ca exchanger in the inner segment of rod photoreceptors. Experimental Eye Research 88**:**57-64. PubMed

12. **K. Kizhatil**, S. Baker, V. Arshavsky, and V. Bennett. 2009. Ankyrin-G promotes cyclic nucleotide-gated channel transport to rod photoreceptor sensory cilia. Science 323**:**1614-1617. PubMed

13. G. Ayalon, J. D. Hostettler, J. Hoffman, **K**. **Kizhatil,** J. Q. Davis, and V. Bennett. 2011. Ankyrin-B interactions with spectrin and dynactin-4 are required for dystrophin-based protection of skeletal muscle from injury. Journal of Biological Chemistry 286**:**7370-7378. PubMed

14. **K. Kizhatil**, M. Ryan, J. K. Marchant, S. Henrich, and S.W. M. John. 2014. Schlemm's canal is a unique vessel with a combination of blood vascular and lymphatic phenotypes that forms by a novel developmental process. PLoS Biology **1**2**:**e1001912. Pubmed

15. I. Martinez-Corral , M. H. Ulvmar, L. Stanczuk, F. Tatin, **K. Kizhatil**, S. W. John, K. Alitalo, S.Ortega and T. Makinen. 2015. Nonvenous origin of dermal lymphatic vasculature. Circulation Research 116:1649-1654. Pubmed

16. T. Souma, S. W. Tompson, B. R. Thomson, O. M. Stiggs, **K. Kizhatil**, S. Yamaguchi, L. Feng, V. Limivipuvadh, K. N. Whisenhunt, S. Maurer-Stroh, T. L. Yanovitch, L. Kalaydjieva, D. N. Azmanov, S. Finzi, L. Mauri, S. Javadiyan, E. Souzeau, T. Zhou, A. W. Hewitt, B. Kloss, K. P. Burdon, DA. Mackey, K. F. Allen, J. B. Ruddle, S-H. Lim, S. Rozen, K-N. Tran-Viet, X. Liu, S. John, J. L. Wiggs, F. Pasutto, J. E. Craig, J. Jin, S. E. Quaggin and T. L. Young. 2016. Mutations in the angiopoietin receptor TEK cause primary congenital glaucoma with variable expressivity. J Clin. Invest. 126:2575-2587. Pubmed

17. **K. Kizhatil**, A. Chlebowski, N. G. Tolman, N. F. Freeburg, M. M. Ryan, N. N. Shaw, A. D. M. Kokini, J. K. Marchant and S.W.M. John. 2016. An *in vitro* perfusion system to enhance outflow studies in mouse eyes**.** Inves. Ophthalmol and Vis Sci. 57:5207-5215. Pubmed

18. B. R. Thomson, T. Souma, S. W. Tompson, T. Onay, **K. Kizhatil**, O. M. Siggs, L. Feng, KN. Whisenhunt, T. L. Yanovitch, L. Kalaydjieva, D. N. Azmanov, S. Finzi, CE. Tanna, A. W. Hewitt, D. A. Mackey, Y. S. Bradfield, E. Souzeau, S. Javadiyan, J. L. Wiggs, F. Pasutto, X. Liu, S. W. John, J. E. Craig, J. Jin, T. L. Young and S. E. Quaggin. 2017. Angiopoietin-1 is required for Schlemm's canal development in mice and humans. J Clin. Invest**.**127:4421-4436. Pubmed

19. P. A. Williams, C. E Braine, **K Kizhatil**, N. E Foxworth, N. G Tolman, J. M Harder, R. A Scott, G. L Sousa, A. Pantich, G. R Howell, and S. W. M John. 2019. Inhibition of monocyte-like cell extravasation protects from neurodegeneration in DBA/2J glaucoma. Mol Degeneration. 14: 6. Pubmed

20. N. G. Tolman, D. .G Macalinao, A.L Kearney, K. H MacNicoll, W. N de Vries, I. J Jackson, S. H Cross, **K Kizhatil**, K. S Nair, S. W. M John. 2020. Genetic background modifies vulnerability to

glaucoma related phenotypes in *Lmx1b* mutant mice. Dis Model Mech. 14 (2): dmm046953. Pubmed

21. K. S. Nair, C. Srivastava, R. Brown, S. Koli, H. Choquet, H. S Kang, Y-M Kuo, S. Grimm, C. Sutherland, A. Badea, G. A Johnson, Y. Zhao, J. Yin, K. Okamoto, G. Clark, T. Borras , G. Zode, **K. Kizhatil,** S. Chakrabarti, S. John, E. Jorgenson and A. Jetten. 2020. GLIS1 regulates trabecular meshwork function and intraocular pressure and is associated with glaucoma in humans. (Accepted Nat. Comm.)

Selected Presentations and Abstracts

1. **K. Kizhatil.** "Polarized targeting of E-cadherin to sites of cell-cell contact in early embryos and epithelial cells requires ankyrin-G and beta-2-spectrin**." Invited speaker,** Minisymposium: Cytoskeleton, adhesion and disease, ASCB Annual Meeting. San Diego, CA, December 9-13, 2006.

2. **K. Kizhatil.** "Schlemm's canal is a unique vessel with a combination of blood vascular and lymphatic phenotypes that forms by a novel developmental process**." Invited speaker,** Rudin glaucoma prize lecture, NYU Ophthalmology Grand Rounds, Department of Ophthalmology. NYU Langone Medical Center, NC, December 15, 2015.

3. **K. Kizhatil.** "Schlemm's canal is a unique vessel with a combination of blood vascular and lymphatic phenotypes that forms by a novel developmental process**." Invited speaker**, Lymphangiogenesis, lymphatics and IOP session, ISER, September 29, 2016.Tokyo, Japan.

4. **K. Kizhatil,** H. Gim, G. Clark and SWM. John " Sympathetic innervation of developing aqueous humor drainage structures", IOVS 2018; 59(9):4700, ARVO, May 2, Honolulu, Hawaii.

5. M. Xuyi, D. Wu, **K. Kizhatil,** S.W.M John, C.L Cepko, W. Xiong. "AAV-mediated overexpression of nuclear SRBP1/2 induces buphthalmous and retinal degeneration in mice", IOVS 2018; 59(9):4497, ARVO, May 2, Honolulu, Hawaii.

6. **K. Kizhatil.** "Neuronal control of intraocular pressure through innervation of the Schlemm's canal**." Invited speaker**, Schlemm's canal and beyond, novel targets for glaucoma, ISER, September 10, 2018. Belfast, N. Ireland.

7. **K. Kizhatil** "Innervation of aqueous humor drainage structures and neuronal control of intraocular pressure**" Invited speaker**, Schlemm's canal and beyond, novel targets for glaucoma, TM CLUB MEETING, December 7, 2018. San Diego, USA.

8. N. G Tolman, S. Kneeland, K. MacNicoll, S. Cross, **K Kizhatil,** S. John. "Genetic studies towards determining disease mechanisms in *Lmx1b* mutant mice", IOVS 2019; 60(9):4251, , ARVO, May 1, 2019. Vancouver, Canada.

9. **K. Kizhatil,** D. Sunderland, G. Clark and S. John**. "**Permeability of cell junctions in the Schlemm's canal correlates with pressure-dependent phosphorylation of VE-CADHERIN", **Invited speaker**, IOVS 2019; 60(9):5211, ARVO, May 1, 2019. Vancouver, Canada.

10. **K. Kizhatil** "Neuronal control of IOP". **Invited speaker**, Molecular pathways involved in outflow changes in glaucoma, ISER, October 25-29, 2020. Buenos Aires, Argentina. (Cancelled due to COVID19 pandemic)

Contribution to Science

1. Discovery that Schlemm's canal is a unique vessel that forms by a new sequence of vascular development (Research Scientist-Simon John Lab). Schlemm's canal (SC) plays central roles in aqueous humor drainage (AQH) and ocular physiology. Improper function of the AQH drainage system results in elevated IOP a key risk factor for glaucoma. The Simon John lab had previously characterized the anatomy of mouse AQH drainage structures including SC, demonstrating that they are remarkably similar to those in humans using conventional two dimensional tissue sectioning methods. On this basis the mouse was exploited to identify key genes and pathways that affected the drainage structures in ocular developmental abnormalities and glaucoma. SC functions was proposed to depend on the molecular phenotypes of SC endothelial cells (SECs). However, the nature of SEC phenotype and also the details of SC development remained poorly defined when I joined the John lab. To allow a modern and extensive analysis of SC and its origins, I developed a new whole-mount procedure to visualize its development in the context of surrounding tissues and then applied genetic lineage tracing, specific-fluorescent reporter genes, immunofluorescence, high-resolution confocal microscopy, and three-dimensional (3D) rendering to study SC. Using these techniques, I and my colleagues showed that SECs had a unique phenotype that is a blend of both blood and lymphatic endothelial cell phenotypes. We showed that SC develops from blood vessels through a newly discovered process that we name "canalogenesis". Canalogenesis has features of vasculogenesis, angiogenesis and lymphangiogenesis and is thus a newly discovered blend of vascular developmental programs. These advances defined SC as a unique vessel with a combination of blood vascular and lymphatic phenotypes and will be important for dissecting its functions that are essential for ocular health and normal vision. This study also provided a new paradigm of studying lymphatic molecules as key in SC development, aqueous humor drainage and glaucoma treatment. In collaboration with Dr. Sue Quaggin we showed a critical role for TEK/ANGPT1 system in SC development in both mice and humans. We also developed a mouse eye perfusion device to measure outflow facility in the mouse. This device has a great potential for organ culture and live imaging of SC function.

K. Kizhatil, M. Ryan, J. K. Marchant, S. Henrich, and S.W. M. John. 2014. Schlemm's canal is a unique vessel with a combination of blood vascular and lymphatic phenotypes that forms by a novel developmental process. PLoS Biology **1**2**:**e1001912. Pubmed

T. Souma, S. W. Tompson, B. R. Thomson, O. M. Stiggs, **K. Kizhatil**, S. Yamaguchi, L. Feng, V. Limivipuvadh, K. N. Whisenhunt, S. Maurer-Stroh, T. L. Yanovitch, L. Kalaydjieva, D. N. Azmanov, S. Finzi, L. Mauri, S. Javadiyan, E. Souzeau, T. Zhou, A. W. Hewitt, B. Kloss, K. P. Burdon, DA. Mackey, K. F. Allen, J. B. Ruddle, S-H. Lim, S. Rozen, K-N. Tran-Viet, X. Liu, S. John, J. L. Wiggs, F. Pasutto, J. E. Craig, J. Jin, S. E. Quaggin and T. L. Young. 2016. Mutations in the angiopoietin receptor TEK cause primary congenital glaucoma with variable expressivity. J Clin. Invest. 126:2575-2587. Pubmed

K. Kizhatil, A. Chlebowski, N. G. Tolman, N. F. Freeburg, M. M. Ryan, N. N. Shaw, A. D. M. Kokini, J. K. Marchant and S.W.M. John. 2016. An *in vitro* perfusion system to enhance outflow studies in mouse eyes**.** Inves. Ophthalmol and Vis Sci. 57:5207-5215. Pubmed

B. R. Thomson, T. Souma, S. W. Tompson, T. Onay, **K. Kizhatil**, O. M. Siggs, L. Feng, KN. Whisenhunt, T. L. Yanovitch, L. Kalaydjieva, D. N. Azmanov, S. Finzi, CE. Tanna, A. W. Hewitt, D. A. Mackey, Y. S. Bradfield, E. Souzeau, S. Javadiyan, J. L. Wiggs, F. Pasutto, X. Liu, S. W. John, J. E. Craig, J. Jin, T. L. Young and S. E. Quaggin. 2017. Angiopoietin-1 is required for Schlemm's canal development in mice and humans. J Clin. Invest**.**127:4421-4436. Pubmed

2. Discovered a novel role for Ankyrin adaptor proteins in formation of specialized membrane domains in cells (Postdoctoral fellow- Vann Bennett Laboratory). Ankyrins are membrane skeleton associated adaptor proteins that help localizes channels, transporters and adhesion molecules to specialized membrane domains. Historically, these proteins were not thought to have any role besides linking membrane proteins to the spectrin-actin membrane skeleton. In a series of papers, we demonstrated a novel role for the ankyrin proteins in definition of entire membrane domains. I initiated and drove these projects at an intellectual and experimental level under the mentorship of Dr. Vann Bennett. I showed that in epithelial cells ankyrin-G was required for lateral membrane biogenesis Ankyrin-G collaborated with beta-2-spectrin in this process. We then went on to identify E-cadherin as a new molecular partner of ankyrin-G in epithelial cells and early embryos. We also showed ankyrin-G was required for post Golgi trafficking of E-cadherin. These initial findings have since been validated in mice by Vann Bennett's laboratory at Duke University Medical Center. I next showed that ankyrins were required for defining the inner and outer segments of rod photoreceptors. Ankyrin-B was required for coordinated expression of beta-2-spectrin, Na/K ATPase and Na/Ca exchanger in the inner segment of photoreceptors. Ankyrin-G was required for the transport of cyclic nucleotide gated channels to the outer segment and required for formation of the outer segment. Together these papers established a novel role for ankyrins in the morphogenesis of specialized membrane domains in vertebrate cells.

K. Kizhatil, and V. Bennett. 2004. Lateral membrane biogenesis in human bronchial epithelial cells requires 190-kDa ankyrin-G. Journal of Biological Chemistry **279:**16706-16714. PubMed

K. Kizhati, W. Yoon, P. J. Mohler, L. H. Davis, J. A. Hoffman, and V. Bennett. 2007. Ankyrin-G and beta2-spectrin collaborate in biogenesis of lateral membrane of human bronchial epithelial cells. Journal of Biological Chemistry 282**:**2029-2037. PubMed

K. Kizhatil, J. Q. Davis, L. Davis, J. Hoffman, B. L. Hogan, and V. Bennett. 2007. Ankyrin-G is a molecular partner of E-cadherin in epithelial cells and early embryos. Journal of Biological Chemistry 282**:**26552-26561. PubMed

K. Kizhatil, N. Sandhu, N. Peachey, and V. Bennett. 2009. Ankyrin-B coordinates assembly of beta-2 spectrin, the Na/K ATPase and the Na/Ca exchanger in the inner segment of rod photoreceptors. Experimental Eye Research 88**:**57-64. PubMed

K. Kizhatil, S. Baker, V. Arshavsky, and V. Bennett. 2009. Ankyrin-G promotes cyclic nucleotide-gated channel transport to rod photoreceptor sensory cilia. Science 323**:**1614-1617. PubMed

3. Discovered a role for the cellular cytoskeleton in retrovirus entry. At the initiation of the project, the host cell factors that mediated retrovirus entry were largely unknown. Using murine ecotropic retrovirus (MLV-E) as a model virus, under the supervision of Dr. Lorraine Albritton, I demonstrated a requirement for the cellular cytoskeleton in virus entry. At the beginning of the work it was known that the virus entered cells using a common receptor but site of virus fusion (cell surface or endosome) differed based on cell type. Clustering of the virus receptor on surface of cells suggested an interaction with the cytoskeleton. Based on this observation, we tested the requirement of the cytoskeleton in virus entry. An early critical requirement for the actin network prior to internalization of virus was discovered that was common to both modes of virus entry based on sites of virus fusion. Disruption of microtubules before and shortly after virus internalization markedly reduced entry in the case of endosomal virus fusion, while entry mediated by cell surface fusion remained efficient. These data suggested that intact microtubules are required in a post-penetration step unique to efficient virus entry via endocytosis. Following this

work, other groups showed a requirement of the cellular cytoskeleton for HIV entry and amphotrophic retrovirus entry. This work was part of my doctoral thesis.

K. Kizhatil and L. M. Albritton. 1997. Requirements for different components of the host cell cytoskeleton distinguish ecotropic murine leukemia virus entry via endocytosis from entry via surface fusion. Journal of Virology **71:**7145-7156. PubMed

M. Chung, **K. Kizhatil**, L. M. Albritton, and G. N. Gaulton. 1999. Induction of syncytia by neuropathogenic murine leukemia viruses depends on receptor density, host cell determinants, and the intrinsic fusion potential of envelope protein. Journal of Virology 73**:**9377-9385. PubMed

K. Kizhatil, A. Gromley, and L. M. Albritton. 2001. Two point mutations produce infectious retrovirus bearing a green fluorescent protein-SU fusion protein. Journal of Virology 75**:**11881-11885. PubMed

REFEREES

Simon John, Ph.D.
Robert Burch Professor of Ophthalmic Sciences, Columbia University
Member, Zuckerman Institute
Investigator, Howard Hughes Medical Institute
Phone: 212-8530-241/212-342-3756
sj2967@cumc.columbia.edu

W. Daniel Stamer, Ph.D., FARVO
Joseph A. C. Wadsworth Professor of Ophthalmology
Professor of Biomedical Engineering
Duke University
Phone: 919-684-3745
dan.stamer@duke.edu

C. Ross Ethier, Ph.D.
Lawrence L. Gellerstedt, Jr. Chair in Bioengineering
Georgia Research Alliance Eminent Scholar in Biomechanics and Mechanobiology
Professor of Biomedical Engineering
Wallace H. Coulter Department of Biomedical Engineering at Georgia Institute of Technology, Emory University School of Medicine
Phone/Fax: 404-385-0100/404-385-1397
ross.ethier@bme.gatech.edu