BIOGRAPHICAL SKETCH

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NAME	POSITION TITLE
Morrison, Brett, Michael	Assistant Professor
eRA COMMONS USER NAME (credential, e.g., agency login) BRETTMORRISONPI	

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	MM/YY	FIELD OF STUDY
Amherst College, Amherst, MA	B.A.	1988-1992	Neuroscience major
Mount Sinai Medical School, New York, NY	M.D.	1992-2001	Medicine
City University of New York, New York, NY	Ph.D.	1992-2001	Biomedical Sciences

A. Personal Statement

I completed an M.D. and Ph.D. at Mount Sinai Hospital/City University of New York in 2001. During my Ph.D., I detailed the pathology in one of the first transgenic models of neurodegenerative disease, the mutant superoxide dismutase model of amyotrophic lateral sclerosis (ALS), under the mentorship of Dr. John H. Morrison (no relation). Following my studies at Mount Sinai Hospital, I completed a neurology residency and a neuromuscular clinical fellowship at The Johns Hopkins Hospital. During this time, I developed skills in neurophysiology and neuromuscular pathology, confirmed my interest in neuromuscular diseases, and began to appreciate the unmet needs of patients with both ALS and peripheral neuropathy. During my fellowship, I worked with Dr. Kathryn Wagner on techniques for enhancing strength and reducing muscle atrophy in mouse models of ALS, learning techniques for the investigation of neuromuscular junction and muscle pathology. From this work, I successfully competed for grants from The Passano Foundation and the Muscular Dystrophy Association. Following completion of my fellowship, I began my faculty appointment in the department of neurology at The Johns Hopkins University. Clinically, I see primarily patients with peripheral neuropathy, but also patients with ALS and other neuromuscular diseases. During the last six years, my research, under the mentorship of Drs. Jeffrey Rothstein and Ahmet Hoke, has focused on the role of monocarboxylate transporters in the central and peripheral nervous system, specifically how they contribute to neurodegeneration in ALS.

B. Positions and Honors

Positions and Employment

2001-2002: Internship, University of Maryland, Department of Medicine
2002-2005: Residency and Chief Residency, The Johns Hopkins Hospital, Department of Neurology
2005-2006: Clinical Neurophysiology and Neuromuscular Fellow, The Johns Hopkins Hospital, Department of Neurology
2006-2007: Instructor, The Johns Hopkins University, Department of Neurology
2007-present: Assistant Professor, The Johns Hopkins University, Department of Neurology

Honors and Awards

- Inductee into Sigma Xi Honor Society, 1992
- James Olds Memorial Neuroscience Award, Amherst College, 1992
- Magna Cum Laude in Neuroscience, Amherst College, 1992
- Medical Scientist Training Program, The National Institutes for Health, 1992-2001
- Morris Bender Award in Clinical Neurology, 1999
- Mount Sinai School of Medicine Doctoral Dissertation Award, 1999
- Inductee into Alpha Omega Alpha Honor Society, 2000

- Jay Slotkin Research Award, Johns Hopkins Hospital, 2005
- Passano Foundation Physician Scientist Award, 2006-2008

Professional Certification and Memberships

2006	Diplomat of Neurology, American Board of Psychiatry and Neurology
2005	Maryland State Medical License
2001	United States Medical Licensing Exam, Parts I, II, III successfully completed
1993-1999	Society for Neuroscience
2001-present	American Academy of Neurology
2008-present	Society for Neuroscience
2011-present	Peripheral Nerve Society

C. Peer-reviewed Publications

1. **Morrison, B.M.**, Gordon, J.W., Ripps, M.E., and Morrison, J.H. (1996) Quantitative immunocytochemical analysis of the spinal cord in G86R superoxide dismutase transgenic mice: neurochemical correlates of selective vulnerability. J. Comp. Neurol. 373 (4): 619-631.
2. **Morrison, B.M.**, Hof, P.R., and Morrison, J.H. (1998) Determinants of neuronal vulnerability in neurodegenerative diseases. Ann. Neurol. 44 (Suppl. 1): S32-S44.
3. **Morrison, B.M.**, Janssen, W.G., Gordon, J.W., and Morrison, J.H. (1998) Time course of neuropathology in the spinal cord of G86R superoxide dismutase transgenic mice. J. Comp. Neurol. 391 (1): 64-77.
4. **Morrison, B.M.**, Janssen, W.G., Gordon, J.W., and Morrison, J.H. (1998) Light and electron microscopic distribution of the AMPA receptor subunit, GluR2, in the spinal cord of control and G86R mutant superoxide dismutase transgenic mice. J. Comp. Neurol. 395: 523-534.
5. **Morrison, B.M.**, and Morrison, J.H. (1999) Amyotrophic lateral sclerosis associated with mutations in superoxide dismutase: a putative mechanism of degeneration. Brain Res. Rev. 29: 121-135.
6. **Morrison, B.M.**, Shu, I-Wei, Wilcox, A.L., Gordon, J.W., and Morrison, J.H. (2000) Early and selective pathology of light chain neurofilament in the spinal cord and sciatic nerve of G86R mutant superoxide dismutase transgenic mice. Exp. Neurol. 165: 207-220.
7. Kiaei, M., Bush, A.I., **Morrison, B.M**., Morrison, J.H., Cherny, R.A., Volitakis, I., Beal, M.F., and Gordon, J.W. (2004) Genetically decreased spinal cord copper concentration prolongs life in a transgenic mouse model of amyotrophic lateral sclerosis. J. Neurosci. 24: 7945-7950.
8. Zhang J, Zhang G, **Morrison B.**, Mori S., Sheikh K.A. (2008) Magnetic resonance imaging of mouse skeletal muscle to measure denervation atrophy. Exp. Neurol. 212: 448-457.
9. **Morrison, B.M**., Lachey, J.L., Warsing, L.C., Ting, B.L., Pullen, A.E., Underwood, K.W., Kumar R., Sako, D., Grinberg A., Wong, V., Colantuoni, E., Seehra, J.S., Wagner K.R. (2009) A soluble activin type IIB receptor improves function in a mouse model of amyotrophic lateral sclerosis. Exp. Neurol. 217: 258-68.
10. Aggarwal, S.P.*, Zinman, L.*, Simpson E., McKinley J., Jackson K.E., Pinto H., Kaufmann P., Conwit R.A., Schoenfeld, D., Shefner J., Cudkowicz M. and the **Northeast ALS (NEALS) and Canadian ALS (CALS) Consortia** (2010) Safety and efficacy of lithium in combination with riluzole for treatment of amyotrophic lateral sclerosis: a randomized, double-blind, placebo-controlled trial. Lancet Neurology 9: 481-488.
11. Ostrow, L.W., Corse, A.M., **Morrison, B.M.,** Huff, C.A., Carrino, J.A., Hoke, A., Mammen, A.L. (2012) Expanding the spectrum of monoclonal light chain deposition disease in muscle. Muscle and Nerve: 45(5): 755-61.
12. **Morrison, B.M**. and Chaudhry, V. (2012) Medication, toxic, and vitamin-related neuropathies. Continuum 18 (1): 139-160.
13. Lee, Y.*, **Morrison, B.M.***, Li, Y., Lengacher, S., Farah, M.H., Hoffman, P.N., Liu, Y., Tsingalia, A., Jin, L., Zhang, P-W., Pellerin, L., Magistretti, P.J., Rothstein, J.D. (2012) Oligodendroglia metabolically support axons and contribute to neurodegeneration. Nature 487 (7408): 443-448.
    *Authors contributed equally to this work

14. Cudkowitz, M.E., van den Berg, L.H., Shefner, J.M., Mitusmoto, H., Mora, J.S., Ludolph, A., Hardiman, O., Bozik, M.E., Ingersoll, E.W., Archibald, D., Meyers, A.L., Dong, Y., Farwell, W.R., Kerr, D.A., **EMPOWER investigators**. Lancet Neurology 2013; 12(11): 1059-1067.
15. **Morrison, B.M.**, Lee, Y., Rothstein, J.D. Oligodendroglia metabolically support axons and maintain structural integrity. Trends Cell Biol. 2013; 23 (12): 644-651.

D. Research Support

Current:
1. Dates: July 1, 2014 to June 30, 2016
Title: Oligodendroglial MCT1 and Metabolic Support of Axons in Multiple Sclerosis
Sponsor: Department of Defense (DOD)
Total Direct Costs: $400,000
Principal Investigator: Jeffrey David Rothstein
Role: Co- Principal Investigator, 10% effort

Previous:
1. Dates: July 1, 2006 to June 30, 2008
Title: Role of Myostatin in Acute and Chronic Denervation
Sponsor: The Passano Foundation and Johns Hopkins University Clinician Scientist Award
Total Direct Costs: $160,000
Principal Investigator: Brett Michael Morrison
Role: Principal Investigator, 70% effort
The main goals of this project are:
1) To determine the mechanism(s) by which myostatin interacts with the denervation atrophy mechanism.
2) To determine if increasing muscle size and strength by genetic deletion or pharmacologic inhibition of myostatin is beneficial in mouse models of ALS.

2. Dates: July 1, 2007 to June 30, 2010
Title: Increasing Muscle Size and Strength by Reducing Myostatin in Mouse Models of ALS
Sponsor: Muscular Dystrophy Association Developmental Award
Total Direct Costs: $135,000
Principal Investigator: Brett Michael Morrison
Role: Principal Investigator, 15% effort
The main goals of this project are:
1) To determine if the complete absence of myostatin leads to significant alteration in the pathology, function, and disease course in two models of ALS.
2) To determine if pharmacological inhibition of myostatin results in significant improvement in function and disease course in same two models of ALS.

3. Dates: January 1, 2010 to December 31, 2012
Title: Astroglial Monocarboxylate Transporter (MCT) Pathway in ALS
Sponsor: Muscular Dystrophy Association Research Award
Total Direct Costs: $300,000
Principal Investigator: Brett Michael Morrison
Role: Principal Investigator, 8% effort
The main goals of the project are:
1) To determine if astroglial lactate export by MCT1 or MCT4 is necessary for neuronal survival in vitro.
2) To investigate whether MCT1 and MCT4 are critical for motor neuron survival in vivo.
3) To determine if MCTs are disrupted in mouse models of ALS and if repair of this pathway is neuroprotective.

4. Dates: March 15, 2010 to February 28, 2014
Title: MCT1 biology and neurodegeneration
Sponsor: National Institute of Neurologic Diseases and Stroke (NIH)

Total Direct Costs: $950,868
Principal Investigator: Jeffrey David Rothstein
Role: Co-investigator, 74% effort
The main goals of this project are:

1) To determine if astroglial lactate export is critical for neuronal survival in vitro.
2) To determine if astroglial lactate transporters, MCT1/MCT4 or basigin, participate in normal neuronal function/activity and are necessary for neuronal survival in vivo.
3) To determine if dysregulation of MCT expression contributes to the neurodegeneration in animal models of ALS.