BIOGRAPHICAL SKETCH

Provide the following information for the key personnel and other significant contributors in the order listed on Form Page 2.
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NAME	POSITION TITLE
Scott M Kulich	Assistant Professor
eRA COMMONS USER NAME kulichsm	

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

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	YEAR(s)	FIELD OF STUDY
University of Wisconsin – Stevens Point	B.S.	1984-1989	Biology
Medical College of Wisconsin	Ph.D.	1991-1994	Microbiology
Medical College of Wisconsin	M.D.	1989-1996	Medicine
University of Pittsburgh		1996-2001	Anatomic and Clinical Pathology
University of Pittsburgh		2001-2003	Neuropathology

A. Personal Statement

B. Positions and Honors

Positions and Employment

July 2003-present	University of Pittsburgh School of Medicine	Assistant Professor
July 2003-present	Veterans Affairs Pittsburgh Healthcare System	Staff Physician Pathology and Laboratory Service
September 2008-present	Geriatric research education and clinical center Veterans Affairs Pittsburgh Healthcare System	Member

Honors and Awards

1988	Sigma Xi Outstanding Undergraduate Research Award
1989	Phi Kappa Phi National Graduate Fellowship
1989	James H. Albertson Award
1989	Medical Scientist Training Program Fellowship, Medical College of Wisconsin
1992	Top Student Presentation, North Central Branch, American Society for Microbiology
1995	Alpha Omega Alpha Medical Honor Society
1996	Pasteur Award , Department of Microbiology, Medical College of Wisconsin
2001	Post-Doctoral Training Program Fellowship, Department of Pathology, University of Pittsburgh
2002	First Prize Poster presentation, 3rd International Conference on Oxygen/Nitrogen Radicals: Cell Injury and Disease
2005	Bronze award, Transfusion Medicine, Excellence in Government Award

Certification and Professional Societies

1. Certification

American Board of Pathology (Anatomic & Clinical Pathology)	Diplomat, 2001
American Board of Pathology (Neuropathology)	Diplomat, 2003

2. Professional societies

Association for Molecular Pathology	2010-present
American Society for Clinical Pathology	2007-present
College of American Pathologists	2007-present
American Association of Neuropathologists	2002-present
Society for Neuroscience	2002-present
American Association of Blood Banks	2003-present
United States and Canadian Academy of Pathology	2003-present
Society for Free Radical Biology and Medicine	2004-2007

B. Peer-reviewed publications (in chronological order)

1. **SM Kulich**, DW Frank, and JT Barbieri (1993) Purification and characterization of exoenzyme S from *Pseudomonas aeruginosa* 388. Infect. Immun. **61:**307-313. PMID: 8418052

2. **SM Kulich**, TL Yahr, LM Mende-Mueller, JT Barbieri, and DW Frank. (1994) Cloning the structural gene for the 49-kDa form of exoenzyme S from *Pseudomonas aeruginosa* strain 388. J. Biol. Chem. **269:**10431-10437. PMID: 8144626

3. TL Yahr, AK Hovey, **SM Kulich**, and DW Frank. (1995) Transcriptional analysis of the *Pseudomonas aeruginosa* exoenzyme S structural gene. J. Bacteriol. **177:**1169-1178. PMID: 7868588

4. DA Knight, V Finck-Barbancon, **SM Kulich**, and JT Barbieri (1995) Functional domains of *Pseudomonas aeruginosa* exoenzyme S. Infect. Immun. **63:** 3182-3186. PMID: 7622246

5. **SM Kulich**, DW Frank, and JT Barbieri (1995) Expression of recombinant exoenzyme S of *Pseudomonas aeruginosa*. Infect. Immun. **63:**1-8. PMID: 7806344

6. S Liu, **SM Kulich**, and JT Barbieri (1996) Identification of glutamic acid 381 as a candidate active site residue of *Pseudomonas aeruginosa* exoenzyme S. Biochem. **35:**2754-2758. PMID: 8611582

7. **SM Kulich** and CT Chu (2001) Sustained extracellular signal-regulated kinase activation by 6-hydroxydopamine: Implications for Parkinson's disease. J. Neurochem. **77:** 1058-1066. PMID: 11359871

8. CL Fattmann, CT Chu, **SM Kulich**, JJ Enghild, and TD Oury (2001) Altered expression of extracellular superoxide dismutase in mouse lung after bleomycin treatment. Free Rad. Biol. & Med. **31**:1198-1207. PMID: 11705698

9. AM Scarrow, EI Levy, **SM Kulich**, CT Chu, and PC Gerszten (2001) Epidermoid cyst of the thoracic spine: case history. Clin. Neurol. & Neurosurg.. **103**:220-222. PMID: 11714565

10. JH Zhu, **SM Kulich**, TD Oury & CT Chu (2002) Cytoplasmic aggregates of phosphorylated extracellular signal-regulated protein kinases in Lewy body diseases. Am. J. Pathol. **161**: 2087-2098. PMID: 12466125

11. **SM Kulich** & CT Chu **(**2003) Role of reactive oxygen species in ERK phosphorylation and 6-hydroxydopamine cytotoxicity. J. Biosci **28:**83-89. PMID: 12682429

12. **SM Kulich**, C Horbinski, M Patel, CT Chu (2007) 6-hydroxydopamine induces mitochondrial ERK activation. Free Radic. Biol. Med. **43**:372-383. PMID: 17602953

13. RK Dagda, JH Zhu, **SM Kulich**, & CT Chu (2008) Mitochondrially localized ERK2 regulates mitophagy and autophagic cell stress induced by 6-hydroxydopamine. Autophagy. **4**:770-82. PMID: 18594198

14. RK Dagda, SJ Cherra III, **SM Kulich**, A Tandon, D Park, & CT Chu (2009) Loss of PINK1 function promotes mitophagy through effects on oxidative stress and mitochondrial fission. J. Biol. Chem. 280:13843-55 PMID: 19279012

C. Research Support

Completed Research Support

1. CTMP in Parkinsonian neuronal autophagy

Source/Project # or type (P.I.): VA competitive pilot program fund
Period (Annual Direct/Indirect): 4/2009-3/2010 ($50,000/$0).
Role, % Effort: PI, 5%

The goal of this pilot grant is to assess whether or not CTMP, a recently identified PINK-1 interacting protein, affects autophagy in the context of genetic and neurotoxic Parkinsonian neuronal injury models.

2. Regulation of PTEN-induced kinase 1 (PINK1)

Source/Project # or type (P.I.): NIH R21 (Chu)
Period (Annual Direct/Indirect): 3/2006-2/2009 ($112,500/$54,563).
Role, % Effort: Co-Investigator, 20%

This proposal was designed to determine if PINK1 functions as a kinase, participating in death-regulatory signaling networks with other mitochondrially targeted kinases.

3. PINK1 depletion and Parkinsonian neurotoxin induced cytotoxicity

Source/Project # or type (P.I.): American Parkinson Disease Association
Period (Annual Direct/Indirect): 9/2005-8/2008 ($50,000/$0).
Role, % Effort: PI, 5%

The goal of this pilot grant is to investigate the effect of siRNA-mediated depletion of PTEN-induced kinase 1 (PINK-1), which has been recently linked to familial Parkinson's disease, on MPP+ and 6-hyroxydopamine mediated cytotoxicity on SH-SY-5Y cells.

4. Role of ERK phosphatases and isoforms in Parkinsonian neurotoxicity

Source/Project # or type (P.I.): VA/ARCD
Period (Annual Direct/Indirect): 10/2005-9/2008 ($264,300)
Role, % effort: PI, 40%

The major goals of this grant are to investigate the influence of the Parkinsonian neurotoxins MPP+ and 6-hyroxydopamine on ERK phosphatase activity both in vitro and in vivo as well as characterizing the role ERK and ERK isoforms on neurotoxicity in in vitro and in vivo models of Parkinson's disease. This grant is an extension of my recently completed PPRTP grant.

5. Identification of PINK1 interacting proteins

Source/Project # or type (P.I.): VA competitive pilot program fund
Period (Annual Direct/Indirect): 7/2005-6/2007 ($50,000/$0).

Role, % Effort: PI, 5%

The goal of this pilot grant is to identify PTEN-induced kinase 1 (PINK-1) interactions with BRAP2 and other neuronal proteins through the use of a variety of methodologies including two hybrid screens, photo-affinity crosslinking, and immunoprecipitation studies.

6. Sustained extracellular signal-regulated kinase phosphorylation: The role of cytosolic sequestration and mitogen activated protein kinase phosphatase-3 activity in mediating cytotoxicity.

Source/Project # or type (P.I.): Pathology Post-doctoral Research Training Program
Period (Annual Direct/Indirect): July 1, 2001-June 30,2004($10,000/ $0).
Role, % Effort: Principal Investigator, 10%

The purpose of this grant is to provide seed money to generate preliminary data on the investigation of the role of alterations of ERK and ERK phosphatases in neuronal cytotoxicity.