BIOGRAPHICAL SKETCH

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NAME: Julie K. Andersen, Ph.D.

eRA COMMONS USER NAME (credential, e.g., agency login): JULIEANDERSEN

POSITION TITLE: Professor of Research in Aging and Age-Related Neurodegenerative Disease

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

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Washington State University	B.S.	06/1983	Biochemistry
University of California, Los Angeles	Ph.D.	09/1989	Neurobiological Chemistry
Harvard School of Medicine/MGH	Postdoctoral	09/1993	Neurology

A. Personal Statement

I have 25 years of experience as a faculty member, first at the University of Southern California and currently at the Buck Institute. I have a broad background in neuroscience, with specific training and expertise in the study of age-related neurodegenerative disorders particularly in the use of *in vitro* cell models including iPSC-derived populations and *in vivo* mouse models.

B. Positions and Honors

Positions and employment

1981-1983	**Undergraduate technician**, W.S.U. Institute of Biological Chemistry, Pullman, WA (Clarence R. Ryan, Ph.D., NAS member).
1983-1984	**Intern**, University of Washington science intern program, Batelle Northwest Laboratories, Richland, WA (James Morris, Ph.D.)
1984-1989	**Research Assistant**, Department of Biological Chemistry, UCLA School of Medicine, Los Angeles, CA (Bruce D. Howard, Ph.D.)
1989-1993	**Postdoctoral fellow**, Molecular Neurogenetics Unit, Neurology Service, Massachusetts General Hospital/Harvard Medical School (Xandra O. Breakefield, Ph.D.)
1993-1999	**Assistant Professor**, Biogerontology Division, Andrus Gerontology Center, University of Southern California, Los Angeles, CA.
1999-2000	**Associate Professor**, Biogerontology Division, Andrus Gerontology Center, University of Southern California, Los Angeles, CA.
2000-2005	**Associate Professor**, Buck Institute for Age Research, Novato, CA.
2005-present	**Full Professor**, Buck Institute for Age Research, Novato, CA. Adjunct Professor, USC, Touro, and Dominican.

Other experience, professional membership, and honors

1979	Bausch and Lomb Honorary Science Award
1979-1983	W.S.U. Honor Roll
1979-1983	Member, W.S.U. Honor Society
1980	Phi Eta Sigma Freshman Honor Society

1982	Phi Lambda Upsilon National Honorary Chemistry Society
1983	Phi Beta Kappa
1984	U. Washington science internship program
1984-1985	Lucille Markey UCLA graduate fellowship
1986-1989	NIMH pre-doctoral NRSA fellowship # SF31MNO 9514
1990-1993	NIH post-doctoral NRSA fellowship # F32NS08810-01
1994	Brookdale National Fellow
1995	Recipient, Gerontological Society Nathan Shock New Investigator Award
1998-2000	Paul F. Glenn Chair in Molecular and Cellular Gerontology
2000-present	Editorial Review Board, Free Rad. Biol. Med.
2002-2007	Steering Committee, NIEHS Parkinson's Disease Environmental Research Consortium
2005	Invited NINDS Workshop on Brain Iron and Neurodegenerative Disease
2004	Chair, Gordon Research Conference on the Biology of Aging
2007	Invited Member, Brookdale Institute on Aging
2007	Glenn Award for Research in Biological Mechanisms in Aging Chair, Gordon Research Conference, Biology of Aging
2007-2011	Standing Member, NIH Review Committee, MDCN
2008-2010	Member, Buck Faculty Executive Committee (CEO equivalent)
2007-present	Council Member, Neurotoxicity Society
2010 -2013	Review Group member, Ellison New Scholar Program
2010-present	Editorial Review Board, Aging Cell
2010	Cade Scholar, University of Melbourne, Australia
2011	Award for scholarly contributions to research science, XIX World Congress on Parkinson's disease
2011	Chair, Gordon Research Conference on Oxidative Stress and Disease
2013-present	Buck-USC Graduate Committee
2013	Fellow of the Society for Free Radical Biology and Medicine
2015	Keynote address, Annual Basic Science of Aging meeting, U. Pitt
2015-present	SAB, U Pitt Medical Center Basic Science of Aging program
2015-present	Editorial Board, eNeuro (J. Neuroscience e-pub)
2015-2021	Standing Member, NIH study section, CNNT
2015	Parkinson Pioneer award, National Parkinson's Foundation
2016	Invited Participant, NIA Mitophagy and Mitochondrial Function Workshop
2017	Organizer, International Neurotoxicology Association meeting, Florianopolis, Brazil
2017	Invited Participant, NIEHS Workshop on Neurotoxicant Screening
2017	Invited Speaker, International Conference on Aging, Sao Paolo, Brazil
2018	Invited Speaker, Michael J. Fox Foundation Therapeutics Conference
2018	Invited Speaker, 2018 Gerontological Society of America Presidential Symposium
2018	Organizer, 2018 Gerontological Society of America Neurodegenerative Disease Symposium
2018	Invited Speaker, Annual Delaware Neuroscience Research Symposium
2018	Invited Participant, NIA Systems Biology Approaches in Neurodegeneration Meeting
2019	Invited Speaker, Mini-symposium on Neurodegeneration and Aging, Rostock
2019	Invited Speaker, Annual Society of Neuroimmune Pharmacology meeting, Portland, OR
2019	Invited Speaker, Annual Brown University Biology of Human Aging Colloquium
2019	Invited Speaker, Annual Undoing Aging meeting, Berlin
2019	Invited Speaker, Cell Symposia: Neuro-immune Axis, Long Beach, CA
2020	Invited Speaker, Gordon Research Conference on Oxygen Radicals, Ventura CA

C. Contribution to Science

My laboratory has a long-standing interest in understanding the basic mechanisms underlying neuronal cell death associated with brain aging and age-related neurodegenerative diseases using both cellular and *in vivo* models. Our publications have revealed many new insights into the mechanisms driving declines in brain function associated with these processes. Our observations have led to many cases of novel targets for therapeutic intervention.

Earlier publications from the laboratory were directed towards understanding how apoptosis contributes to neurodegenerative diseases, particularly the role of caspases in Parkinson's and related neurological disorders.

(1) Viswanath, V., Wu, Z., Wei, Q., Fonck, C., and Andersen, J.K. (2000). Transgenic mice neuronally expressing baculoviral p35 are resistant to diverse types of induced apoptosis including seizure-associated neurodegeneration. Proc Natl Acad Sci 97: 2270-227.
(2) Viswanath, V., Yantiri, F., Boonplueang, R., Yang, Y., Beal, M.F., and Andersen, J.K. (2001). Caspase-9 activation results in downstream caspase-8 mediated bid cleavage in toxin-induced Parkinson's disease. J Neurosci 21: 9519-9528.
(3) Peng, J., Wu, Z., Wu, Y., Hsu, M., Stevenson, F.F., Boonplueang, R., Roffler-Tarlov, S.K., and Andersen, J.K. (2002). Inhibition of caspases protects cerebellar granule cells of the weaver mouse from apoptosis and improves behavioral phenotype. J Biol Chem 277: 44285-44291.
(4) Peng, J., Stevenson, F.F., and Andersen, J.K. (2004). The herbicide paraquat induced dopaminergic nigral apoptosis through sustained activation of the JNK pathway. J Biol Chem 279: 32626-32632.

My laboratory has made important contributions towards understanding the role of losses in protein homeostasis in the neurodegenerative disease process, in particular the role of age-related autophagy.

(1) Kang I, Schilling B, Sorensen DJ, Sahu AK, Kapahi P, Andersen JK, Swoboda P, Killilea DW, Gibson BW, and Lithgow GJ (2014). Iron promotes protein insolubility and aging in *C. elegans*. Aging 6: 975-991. PMCID: PMC4276790.
(2) Angeli S, Barhydt T, Jacobs R, Killilea DW, Lithgow GJ, and Andersen JK (2014). Manganese disturbs metal and protein homeostasis in *C. elegans*. Metallomics 6: 1816-1823. PMCID: PMC4309368
(3) Siddiqui A, Bhaumik D, Chinta SJ, Rane A, Rajagopalan S, Lieu CA, Lithgow GJ, and Andersen JK (2015). Mitochondrial quality control via the PGC1alpha-TFEB signaling pathway is compromised by parkin mutation but independently restored by rapamycin. J Neurosci 35: 12833-12844. PMCID: PMC4571606
(4) Rajagopalan S, Rane A, Chinta SJ, and Andersen JK (2016). Regulation of ATP13A2 via PHD2-HIF1alpha signaling is critical for cellular iron homeostasis: implications for Parkinson's disease. J Neurosci 36: 1086-1095. PMCID: PMC6404282

My laboratory, along with others in the field, has provided importance evidence supporting a role for oxidative stress and mitochondrial dysfunction in age-related neurodegenerative diseases.

(1) Kaur, D., Yantiri, F., Rajagopalan, S., Kumar, J., Mo, J.Q., J., Boonplueang, R., Viswanath, V., Jacobs, R., Yang, L., Beal, M.F., DiMonte, D., Volitaskis, I., Ellerby, L., Cherney, R.A., Bush, A.I., and Andersen, J.K. (2003). Genetic or pharmacological iron chelation prevents MPTP-induced neurotoxicity in vivo: a novel therapy for Parkinson's disease. Neuron 37: 1-20.
(2) Chinta SJ, Kumar MJ, Hsu M, Rajagopalan S, Kaur D, Anand Rane A, Nicholls DG, Andersen JK (2007). Inducible alterations of glutathione levels in adult dopaminergic midbrain neurons results in nigrostriatal degeneration. J Neurosci 27: 13997-14006.
(3) Peng J, Peng L, Stevenson FF, Doctrow SR, Andersen JK (2007). Iron and paraquat as synergestic environmental risk factors in sporadic Parkinson's disease accelerate age-related neurodegeneration. J Neurosci 27:6914-6922.
(4) Siddiqui A, Bhaumik D, Chinta SJ, Rane A, Rajagopalan S, Lieu CA, Lithgow GJ, and Andersen JK (2015). Mitochondrial quality control via the PGC1alpha-TFEB signaling pathway is compromised by parkin mutation but independently restored by rapamycin. J Neurosci 35: 12833-12844. PMCID: PMC4571606

My laboratory has been involved in pre-clinical drug testing in animals of PD and AD in an effort to move them towards phase II clinical trials.

(1) Kim YH, Rane A, Lussier S, Andersen JK (2011). Lithium protects against oxidative stress-mediated cell death in alpha-synuclein overexpressing *in vitro* and *in vivo* models of Parkinson's disease. J. Neurosci. Res.89: 1666-1675. PMCID: PMC3154577

(2) Lieu CA, Dewey CM, Chinta SJ, Rane A, Rajagopalan S, Batir S, Kim Y-H, and Andersen JK (2014). Lithium prevents parkinsonian behavioral and striatal phenotypes in an aged parkin transgenic mouse model. Brain Res. 1591: 111-117. PMCID: PMC4254598
(3) Lazzara CA, Riley RR, Rane A, Andersen JK*, and Kim YH (2015). The combination of lithium and L-Dopa/Carbidopa reduces MPTP-induced abnormal involuntary movements (AIMs) via calpain inhibition in a mouse model: relevance for Parkinson's disease therapy. Brain Res. 1622: 127-136. *co-corresponding author. [Subject of review by Valera and Masliah in Mov. Disord. 2015 entitled 'Combinations therapies: the next logical step for the treatment of synucleinophathies?']. PMCID: PMC4562891

My laboratory has also maintained an ongoing interest in the role of neuroinflammation brain aging and neurodegenerative diseases. Recent studies (as part of a funded MJFF grant) involve the use of human post-mortem tissues, iPSC-derived astrocytes, and mouse PD models to study a process known as cellular senescence. This work interrogates the involvement of a here-to-fore unexplored mechanism by which brain aging may be driving neurodegenerative diseases.

(1) Peng J, Xie L, Stevenson FF, Melov S, Di Monte DA, Andersen, JK (2006). Nigrostriatal dopaminergic neurodegeneration in the weaver mouse is mediated via neuroinflammation and alleviated by minocycline administration. J Neurosci 26: 11644-11651.
(2) Mallajosyula JK, Kaur D, Chinta SJ, Rajagopalan S, Rane A, Nicholls DG, DiMonte D, Macarthur H, and Andersen JK (2008). MAO-B elevation in mouse brain astrocytes results in Parkinson's pathology. PLoS ONE 3: e1616. PMCID: PMC2229649.
(3) Chinta SJ, Woods G, DeMara M, Rane A, Zou Y, McQuade AK, Rajagopalan S, Limbad C, Madden D, Campisi J, Andersen JK (2018). Cellular senescence is induced by the environmental neurotoxin paraquat and contributes to neuropathy linked to Parkinson's disease. Cell Reports 2018 Jan 23rd. PMCID: PMC5806534
(4) Woods G, Andersen JK (2018). Screening method for identifying toxicants capable of inducing astrocyte senescence. Toxicol. Sci. doi: 10.1093/toxsci/kfy181. [Epub ahead of print].

Completed List of Published Work including PMCID Numbers in my Bibliography:
http://www.ncbi.nlm.nih.gov/sites/myncbi/1Pe18EGC6loAg/bibliography/43437537/public/?sort=date&direction=ascending

D. Additional Information: Research Support and/or Scholastic Performance

Ongoing Research Support

RF1 AG057358 (Andersen) 09/15/17 – 06/30/22
We propose to determine: (1) the impact of systemic administration of urolithin A (UA) on modulation of activity of bile acid FXR activity and downstream TFEB-mediated autophagy within brain neurons impacted in AD and whether this can protect against proteotoxic AD phenotypes *in vivo* and (2) whether age-related reductions in production of UA by the gut microbiota can be restored via introduction of a youthful microbiome and result in increased neuroprotection. Results from these studies, if successful, would further our understanding of how interactions between the brain and the gut impact on neurons affected in AD, how age-related changes in metabolite production in the gut affect this process, and if reinstating a more youthful microbiome restores gut-brain signaling and protects brain neurons.

RF1 AG057358 (MPI: Lithgow, Andersen) 09/15/17 – 06/30/22
NIA/NIH
A temporal bioenergetics, metabolomics, and proteomic map of Alzheimer's disease in invertebrate models. This project proposes to a new deep understanding of AD by undertaking a holistic systems biology-based approach to examine overall cellular functions and, in doing so, discover new therapeutic approaches for the disease.

Pittsburgh Foundation (Andersen) 06/01/19 – 05/31/20
The goal of these funds is to promote research towards the studying the potential role of cellular senescence within neurons in both Alzheimer's and Parkinson's disease.

Hillblom Foundation Research Center Grant (Lithgow) 02/01/19 – 01/31/20
A unifying theme across the Center is the development and use of genetic and pharmacological interventions to slow or prevent chronic disease.
Role: Co-Investigator

Completed Research Support (last 3 years)

SENS Foundation (Andersen) 01/25/17 – 04/30/19
Proprietary

R01 AG029631-09S1 (MPI: Lithgow, Andersen) 09/01/17 − 04/30/19
NIH/NIA supplement
Pharmacology of lifespan extension
Identify the mechanism of lifespan extension with focus on vitamin D – shown to maintain protein homeostasis and extend lifespan in *C. elegans*. This will uncover novel mechanisms for interventions in aging and age related disease.

R21 NS095758 (Andersen) 07/01/16 – 09/30/18
NINDS/NIH
Potential Role of Lysosomal ATP13A2 in Cellular Iron Homeostasis
Our goal in this proposal is to interrogate whether a redistribution of chelatable iron occurs as a consequence of ATP13A2 deficit, the mechanisms underlying this alteration, and whether this in turn predisposes neurons for increases in mitochondrial dysfunction and cell loss.

MJFF #12113 (Andersen) 10/15/16 – 10/14/18
Michael J. Fox Foundation
Alpha-synuclein aggregates as inducers of glial cell senescence: potential role in Parkinson's disease
We seek to explore whether induction of astrocytic senescence occurs in response to alpha-synuclein aggregation, if this in turn contributes to progression of PD, and, importantly, whether senescence cell ablation prevents neuropathological phenotypes associated with the disorder.