BIOGRAPHICAL SKETCH

Name	Position Title
Jung Hyuk Suh ERA COMMONS USER NAME JUNGHSUH	Associate Staff Scientist

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
Boston University	B.A.	1993	Biology
Boston University School of Public Health	M.P.H.	1996	Epidemiology & Biostatistics
Oregon State University	Ph.D.	2003	Biochemistry & Biophysics
Children's Hospital Oakland Research Institute	Post-Doc	2003-2007	Integrated Physiology & metabolism

A. Personal Statement

I am an Associate Staff Scientist at CHORI and Visiting Scientist in the Department of Molecular and Cellular Biology, University of California, Berkeley. I am currently in charge of the metabolomics facility in the Nutrition and Metabolism Center at CHORI. The mass spectrometry core at CHORI is equipped with all of the necessary analytical tools needed to support this study. Oxidative and inflammatory stresses are generally recognized to be major contributors to cell damage associated with disease initiation and progression, as well as aging. Maintenance of redox balance in healthy tissues is essential for many critical metabolic pathways whose rate-limiting steps are regulated by redox mechanisms (e.g., insulin receptor signaling). My core research roadmap involves two major stages. The first stage involves the development and application of redox metabolomics as a sensitive exploratory tool to identify unique redox patterns involved in different disease and nutritional conditions. The second stage aims to establish the clinical significance of these changes to particular disease processes. My research focuses on establishing how different disease processes and other sources of metabolic stress (e.g., bad diets) differentially modulates the relative concentrations of redox metabolites (e.g. NADPH, sulfur amino acids, and peptides) and if these shifts provides specific clues to disease mechanisms that may lead to better diagnostics and therapeutic options.

B. Positions and Honors

Professional Experience

1993 – 1994 Research Assistant, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts
2003 – 2005 Post-doctoral Fellow, Children Hospital Oakland Research Institute, Oakland, California
2005 – 2009 Assistant Staff Scientist, Children Hospital Oakland Research Institute, Oakland, California
2009 – Present Associate Staff Scientist, Children Hospital Oakland Research Institute, Oakland, California

Other Experience / Professional Memberships / Honors

1999 Invited Speaker on 29th Annual Meeting of American Aging Association
1999 – 2001 Linus Pauling Institute Graduate Student Fellowship
1999 – 2001 American Heart Pre-Doctoral Fellowship (9910089Z)
1999 – Present Member of Society of Free Radicals and Biology and Medicine
2003 Member of Sigma Xi, The scientific research society
2003 Invited Speaker on 2nd Asia Pacific Conference and Exhibition on Anti-Ageing Medicine – National University of Singapore
2004 Oxygen Club of California young Investigators award

C. Selected Peer-reviewed Publication

1. **Suh JH**, Shigeno ET, Morrow JD, Cox B, Rocha AE, Frei B, and Hagen TM (2001) Oxidative stress in the aging rat heart is reversed by dietary supplementation with (*R*)-(α)-lipoic acid. *FASEB J*. 15:700-6. PMID: 11259388
2. **Suh JH**, Heath D, and Hagen T (2003) Two subpopulations of mitochondria in the aging rat heart display heterogeneous levels of oxidative stress. *Free Radical Biol Med* 35:1064-72. PMID: 14572609

3. **Suh JH**, Zhu B, and Frei B (2003) Ascorbate does not act as a pro-oxidant towards lipids and proteins in human plasma exposed to redox-active transition metal ions and hydrogen peroxide. *Free Radical Biol Med* 34:1306-14. PMID: 12726918
4. **Suh JH**, Wang , Liu RM, Liu JK, and Hagen TM (2004) (*R*)-α-Lipoic Acid Reverses the Age-related Loss in GSH Redox Status in Post-Mitotic Tissues: Evidence for Increased Cysteine Requirement for GSH Synthesis. *Arch Biochem Biophys* 423:126-35. PMID: 14871476
5. **Suh JH**, Zhu BZ, deSzoeke E, Frei B, and Hagen TM (2004) Dihydrolipoic acid lowers the redox activity of transition metal ions but does not remove them from the active site of enzymes. *Redox Rep* 9:57-61. PMID: 15035828
6. **Suh JH**, Shenvi SV, Wang H, Jaiswal AK, Liu RM, and Hagen TM (2004) Decline in transcriptional activity of Nrf2 causes age-related loss of glutathione synthesis, which is reversible with lipoic acid. *Proc Natl Acad Sci USA* 101:3381-6. PMC373470
7. **Suh JH**, Moreau R, Heath SH, and Hagen TM (2005) Dietary supplementation with (*R*)- α-lipoic acid reverses the age-related accumulation of iron and depletion of antioxidants in the rat cerebral cortex. *Redox Rep* 10:52-60. PMID: 15829111
8. Serikov VB, Leutenegger C, Krutilina R, Kropotov A, Pleskach N, **Suh JH**, and Tomilin NV (2006) Cigarette smoke extract inhibits expression of peroxiredoxin V and increases airway epithelial permeability. *Inhal Toxicol* 18:79-92.
9. Schwarzer C, Illek B, **Suh JH**, Remington SJ, Fischer H, and Machen TE (2007) Organelle redox of CF and CFTR-corrected airway epithelia. *Free Radic Biol Med* 43:300-16. PMID: 15829111
10. Morris CR, **Suh JH**, Hagar W, Larkin S, Bland DA, Steinberg MH, Vichinsky EP, Shigenaga M, Ames B, Kuypers FA, and Klings E (2008) Erythrocyte glutamine depletion, altered redox environment, and pulmonary hypertension in sickle cell disease. *Blood* 111:402-10. PMC2200820
11. **Suh JH**, Walsh WJ, McGinnis WR, Lewis A, and Ames BN (2008) Altered Sulfur Amino Acid Metabolism in Immune Cells of Children Diagnosed with Autism. *Am J Biochem Biotech* 4:105-13.
12. Lal A, Atamna W, Killilea D, **Suh JH**, and Ames BN (2008) Lipoic Acid and Acetyl-Carnitine Reverse Iron-Induced Oxidative Stress in Human Fibroblasts. *Redox Rep* 13:2-10. PMID: 18284845
13. Dixon BM, Heath SHD, Kim RY, **Suh JH**, and Hagen TM (2008) Assessment of Endoplasmic Reticulum Glutathione Redox Status Is Confounded by Extensive Ex Vivo Oxidation. *Antioxid Redox Signal* 10:963-72. PMID: 18205546
14. Schwarzer C, Fischer H, Kim EJ, Barber KJ, Mills AD, Kurth MJ, Gruenert DC, **Suh JH**, Machen TE, and Illek B (2008) Oxidative Stress Caused by Pyocyanin Impairs CFTR Cl(-) Transport in Human Bronchial Epithelial Cells. *Free Radic Biol Med* 45:1653-62. PMC2628806
15. **Suh JH**, Kim RY, Yavuz B, Lee D, Lal A, Ames BN, and Shigenaga MK (2009) Clinical Assay of Four Thiol Amino Acid Redox Couples by LC-MS/MS: Utility in Thalassemia. *J Chromatogr B Analyt Technol Biomed Life Sci*. 877:3418-27. PMID: 19616487
16. Mietus-Snyder ML, Shigenaga, MK, **Suh JH**, Shenvi SV, Lal A, McHugh T, Olson D, Lilienstein J, Krauss RM, Gildengoren G, McCann JC, and Ames BN (2012) A micronutrient-dense, high fiber, fruit-based supplement bar increases HDL, particularly large HDL, lowers homocysteine, and raises glutathione in a 2-week trial. *FASEB J*, 26(8):3515-27. doi:10.1096/fj.11-201558. PMID:22549511

C. Research Support

Ongoing Research Support

1 RC1 AG036203-01 (Ames, BN) 09/30/09 – 08/31/12 0.96 calendar PM
NIH/NIA $112,500 currently on a no-cost extension

mtDNA mutation/heteroplasmy: a sensitive functional biomarker of oxidative stress

We propose that mutation is a disease-relevant target endpoint because of its known association with aging and causal relationship to cancer. We also propose that mitochondrial DNA (mtDNA) mutation leading to heteroplasmy is likely to be an ultra sensitive sentinel biomarker of oxidative stress. We will evaluate the sensitivity of this assay to oxidative stress, the specificity of mutational spectra resulting from different causes of oxidative stress, and the responsiveness of the assay to modulatory effects of antioxidants.
Role: Co-Investigator

CHRCO Institutional Award (Lal, A) 09/01/11 – 08/31/12 0.24 calendar PM
Functional Significance of Mitochondrial DNA $40,000 Role: Assistant Staff Scientist

Abnormalities in β -Thalassemia Major
Our goal is to study the association between mtDNA injury, iron burden and oxidative stress, and investigate the consequences of mtDNA abnormalities on the expression of mitochondrial proteins and the proliferative capacity of lymphocytes. Understanding the significance of insufficient or dysfunctional mitochondria would provide a sensitive biomarker to monitor the toxicity of iron. This would aid in the development of functional assessment of iron chelation therapy, which is currently based entirely on the measurement of tissue iron burden.

CHRCO Institutional Award (Kanathezhath, B) 09/01/11 – 08/31/12 0.60 calendar PM
Significant decline in thiol redox status in $60,000
allogeneic transplantation is predictive of graft versus host disease
This research proposal has the potential to discover novel biomarkers in the diagnosis of graft versus host disease, a dreaded complication of allogeneic transplantation. The goal of this study is to identify patients at higher risk of development of graft versus host using the novel method of amino acid metabolomic analysis.

Completed Research Support

PO1 AT002620 09/30/04 – 06/30/09 0.60 calendar PM, in kind
NIH/NCCAM (Peden, DB) $79,412 Role: Assistant Staff Scientist
Translational Research Center for CAM Therapy of Asthma
Examine the effect of g-tocopherol on inflammatory and lung function response of asthmatic volunteers to ozone challenge to determine if ozone challenge can be employed as a method to screen potential CAM therapies for asthma for large-scale clinical trials.

T32 DK078514-06A2 04/01/08 – 01/31/10 9.60 calendar PM
NIH/NIDDKD (Lubin, B) $45,048 Role: Trainee
Training: Hematology, Immunology & Stem Cell Biology
The major goal of this study is to foster and encourage interest in research programs in the areas of hematology, immunology and stem cell biology for postdoctoral training.

Department of Defense (DOD) (Herbert M) 09/01/08 – 08/31/10 0.60 calendar PM, in kind
A Prospective Multi-System Evaluation of $27,900 Role: Collaborator
Infants At Risk for Autism
Determine the longitudinal patterns of metabolomic alterations in infants sibs of autistic probands to establish the potential temporal relationship between altered oxidative stress and neurobehavioral abnormalities in autism.

R21 AT004493 (Ames, BN) 02/01/09 – 01/31/12 0.60 calendar PM
NCCAM $275,204 currently on a no-cost extension
Antioxidant Therapy to Reduce Inflammation in Sickle Cell Disease
We hypothesize that Lipoic Acid / Acetyl-L-Carnitine will lower systemic inflammation in patients with Sickle Cell Disease by reducing oxidative stress, which will result in a decrease in the frequency of vaso-occlusive pain episodes and improve their quality of life. This study is a clinical trail. ClinicalTrials.gov Identifier: NCT01054768.
Role: Collaborator, Assistant Staff Scientist

R01FD003531-01 (Morris, C) 04/01/09 – 03/31/12 1.20 calendar PM, in kind
FDA $60,000
Orphan Drug Development
Glutamine Therapy for Hemolysis-Associated Pulmonary Hypertension.
Role: Collaborator

JC Hempel Foundation (Suh, J) 05/26/09 – 03/31/12 0.60 calendar PM, in kind
Metabolomic profile of $30,000
Niemann Pick Type C – disease
NPC is an inherited metabolic disorder that involves defective lipid metabolism. Consequently, excess cholesterol accumulation cause severe abnormal development of brain and a gradual decline in physical and mental functions. Most children with NPC die between the ages of 5 and 15 years. This study aims to define the broad secondary metabolic complications that arise due to NPC1 mutation.

Stephen Bechtel Fund (Suh, J) 05/29/09 – 05/28/12 7.20 calendar PM
Redox Metabolism and Its Impact on Nutritional Requirements for Optimum Health $150,000

Nutritional requirements for optimal health are quite likely to vary between individuals and also within a person under different disease conditions. Nutritional sciences have also been traditionally narrowly focused with a concentration on understanding the relationships that exists between single nutrients and overt disease outcomes. Our goal is to Refine techniques for assessing metabolic health, open new frontiers in nutrition management, monitor and determine the metabolic basis of human diseases.