BIOGRAPHICAL SKETCH

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NAME: **Peter J. Gianaros**

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

POSITION TITLE: **Professor of Psychology**

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
University of Florida, Gainesville, FL	**B.S.**	**1995**	**Psychology**
The Pennsylvania State University, University Park, PA	**Ph.D.**	**2000**	**Psychology**

A. Personal Statement

My work focuses on the neurobiology of psychological stress, emotion, and socioeconomic health disparities. This focus has encompassed studies of how the brain (*i*) functionally regulates and represents autonomic, immune, and cardiovascular stress responses, (*ii*) how the brain influences and is influenced by biological and behavioral risk factors for chronic illnesses, including atherosclerotic cardiovascular disease (CVD), and (*iii*) links socioeconomic inequalities to health over the lifespan. These studies have resulted in 100+ publications, and they have used a wide range of approaches, including the *integrated* use of functional and structural brain imaging, psychophysiological, epidemiological, behavioral, and basic laboratory approaches.

I've been principal investigator or co-principal investigator of 4 NIH RO1 grants, an NIH K01 grant, and a research grant from the Pennsylvania Department of Health. I also have longstanding experience in graduate and post-doctoral mentorship. Hence, in addition to serving as a Co-I on 7 prior NIH grants, I was the Director of 2 NIH sponsored graduate and professional training programs in multimodal brain imaging.

B. Positions and Honors

Positions and Employment

1993-1995 Research Associate, National Institute of Mental Health Center for the Study of Emotion and Attention, University of Florida
1995-2000 Graduate Research and Teaching Assistant, Department of Psychology, The Pennsylvania State University
2000-2003 Postdoctoral Fellow, Department of Psychiatry, University of Pittsburgh
2004-2013 Assistant to Associate Professor, Departments of Psychiatry and Psychology, University of Pittsburgh
2005-pres. Faculty, Center for the Neural Basis of Cognition, University of Pittsburgh
2006-pres. Faculty, Center for Neuroscience, University of Pittsburgh
2013-pres. Director, Multimodal Neuroimaging Training Program, University of Pittsburgh
2014-pres. Professor, Department of Psychology, University of Pittsburgh, Pittsburgh, PA

Editorial Positions

2009-2012 Editorial Board, Neuropsychology
2011-2013 Editor, Special Series on Neuroscience in Health and Disease, Psychosomatic Medicine
2012-pres. Associate Editor, Psychosomatic Medicine
2016-2019 Consulting Editor, Health Psychology

Selected Honors

1999 NIMH Individual National Research Service Award (NRSA Predoctoral Fellowship)
2002 NHLBI Individual National Research Service Award (NRSA Postdoctoral Fellowship)
2008 The Herbert Weiner Early Career Award, American Psychosomatic Society
2010 The Distinguished Scientific Award for Early Career Contribution to Health Psychology, American Psychological Association

C. Contributions to Science

1) Neurobiology of psychological stress, emotion, and physical health. How does the brain regulate our cardiovascular and autonomic nervous systems during stressful and emotional experiences? How do these experiences impact the brain? For the past 16 years, I have conducted neuroimaging research addressing these questions. My earliest research focused on mapping the human brain systems that control cardiovascular and autonomic nervous system (e.g., blood pressure, heart rate, heart rate variability) changes are linked to risk for poor physical health (e.g., hypertension, CVD events). As part of this work, my lab developed the first standardized battery of psychological stress tasks for use in brain imaging studies of stress reactivity (http://bnl.pitt.edu/resources.html). Our work bearing on these questions above has shown that individuals who show 'exaggerated' or presumably metabolically-excessive cardiovascular rises during acute stressful experiences also show: (*i*) heightened neural activity at rest and during stressor task performance, (*ii*) reduced gray matter volume, and (*iii*) increased functional coupling (cross-correlated activity) within a network of cortical and limbic brain areas that appear to jointly regulate peripheral physiology and process emotional and stress-related information. These regions encompass the anterior cingulate cortex, ventromedial prefrontal cortex, insula, amygdala, periaqueductal gray, and pons. Moreover, we have shown that heightened activity in several of these same areas during the processing of emotional stimuli and regulation of emotional experiences relates to a greater severity of preclinical atherosclerosis, particularly via systemic inflammatory pathways. This work has led to a conceptual model in which we posit that the functional interplay between cortical and limbic brain areas play a role in calibrating short-term peripheral physiological changes to meet the metabolic and contextual demands of stimuli and events that are appraised as stressful or demanding by the individual. Moreover, our work has provided additional support for the view that individuals who are prone to showing elevated or dysregulated patterns of neural activity during stressful experiences may be at heighted risk for chronic illnesses, including CVD. In related work, we have shown that the same regions involved in the stress- and emotion-related regulation of peripheral physiology also exhibit plasticity (e.g., volumetric reductions) in the context of chronic psychological stress, suggesting a basis for interactions between acute and chronic stress and emotion processes at the level of the brain.

a) Gianaros PJ, Matthews KA, Jennings JR, Sheu LK, Manuck SB, Hariri AR (2008). Individual differences in stressor-evoked blood pressure reactivity vary activation, volume, and functional connectivity of the amygdala. J Neurosci, 28, 990-999. PMCID: PMC2526972

b) McEwen BS & Gianaros PJ (2011). Stress- and allostasis-induced brain plasticity. Annu Rev Med, 62, 431-445. PMCID: PMC4251716

c) Gianaros PJ, Marsland AL, Kuan D C-H, Jennings JR, Sheu LK, Hariri AR, Gross JJ, Manuck SB (2014). An inflammatory pathway links atherosclerotic cardiovascular disease risk to neural activity evoked by the cognitive regulation of emotion. Biol Psychiatry, 75, 738-45. PMCID: PMC3989430

d) Gianaros PJ & Wager TD (2015). Brain-body pathways linking psychological stress and physical health. Curr Dir Psychol Sci. PMCID: pending (NIHMS674625)

2) Neurobiology of socioeconomic health disparities. In conducting research on the neurobiology of stress and emotion, I have also developed a line of research on the neurobiology of social health disparities, with a focus on disparities patterned by socioeconomic status. This work is motivated by prominent socioeconomic disparities in chronic disease morbidity and mortality that appear to be widening. More precisely, my research on the neurobiology of health disparities has characterized the neural correlates of socioeconomic status to clarify how different dimensions of socioeconomic status might, over the lifespan and through particular brain systems, affect aspects of behavior, cognition, emotion, and physiology that proximally influence physical health. Thus far, this research has shown that (*i*) individuals who perceive

themselves to be of low socioeconomic status show reduced gray matter volume in the cingulate cortex, which may relate to this region's regulation of emotions and peripheral physiology; (*ii*) individuals who report that their parents were of low socioeconomic status show heightened activation to threatening social stimuli in the amgydala, which may also relate to this region's regulation of emotions and peripheral physiology; (*iii*) individuals whose parents attained a lesser education than others show altered patterns of neural activity in cortical and striatal brain areas that are important for self-regulation and impulse control; and (*iv*) individuals from disadvantaged backgrounds and residing in disadvantaged neighborhoods show increased levels of systemic inflammation, which in turn is associated with a reduced integrity of white matter fiber tracts that facilitate neural transmission. In aggregate, findings from this research has led to conceptual models of health disparities that propose that particular brain networks link socioeconomic disadvantage to cognitive, behavioral, psychological, and physiological factors that promote physical disease risk.

a) Gianaros PJ & Manuck SB (2010). Neurobiological pathways linking socioeconomic position and health. Psychosom Med, 72, 450-461.PMCID: PMC2903752

b) McEwen BS & Gianaros PJ (2010). Central role of the brain in stress and adaptation: Links to socioeconomic status, health, and disease. Ann N Y Acad Sci, 1186, 190-222. PMCID: PMC2864527

c) Gianaros PJ, Marsland AL, Sheu LK, Erickson KI, Verstynen TD (2013). Inflammatory pathways link socioeconomic inequalities to white matter architecture. Cereb Cortex, 23, 2058-2071. PMCID: PMC3729196

d) Gianaros PJ, Kuan D C-H, Marsland AL, Sheu LK, Hackman DA, Miller KG, Manuck SB (in press). Community socioeconomic disadvantage in midlife relates to cortical morphology via neuroendocrine and cardiometabolic pathways. Cerebral Cortex

3) Impact of chronic disease risk on the brain. Longstanding epidemiological evidence has linked biological and behavioral risk factors for chronic illnesses to signs of premature cognitive aging. A focus of our work in this area has been on characterizing aspects of brain morphology that might (*i*) be adversely affected by biobehavioral risk factors in the context of aging and (*ii*) mediate aging related changes in cognitive function. In particular, this work has maintained a focus on studies of brain morphology changes as potential sequelae of risk factors for chronic illness and premature aging, including hypertension, inflammation, and obesity. We have provided evidence, for example, that elevated blood pressure relates to reduced gray matter volume in the supplementary motor area, which in turn accounts for poorer executive function performance. We have also found that heightened systemic inflammation associates with reductions in cortical and subcortical gray matter volume and cortical surface area, which in turn accounts for poorer executive function and memory performance in midlife. Finally, we have shown abdominal adiposity relates to reduced white matter integrity, and that this relationship is accounted for by elevated levels of systemic inflammation and other components of the metabolic syndrome.

a) Gianaros PJ, Greer PJ, Ryan CM, Jennings JR (2006). Higher blood pressure predicts lower regional grey matter volume: Consequences on short-term information processing. NeuroImage, 31, 754-765. PMCID: PMC2254305

b) Marsland A, Gianaros PJ, Brown SM, Manuck SB, Hariri AR (2008). Interleukin-6 covaries inversely with hippocampal grey matter volume in middle-aged adults. Biol Psychiatry, 64, 484-490. PMCID: PMC2562462

c) Verstynen T, Weinstein AW, Erickson KI, Sheu LK, Marsland AL, Gianaros PJ (2013). Competing physiological pathways link individual differences in weight and abdominal adiposity to white matter microstructure. NeuroImage, 79, 129-37. PMCID: PMC3752776

d) Marsland AL, Gianaros PJ, Kuan DCH, Sheu LK, Krajina K, Manuck SB (in press). Brain morphology links systemic inflammation to cognitive function in midlife adults. Brain, Behavior, and Immunity. PMCID: pending

URL for NCBI Bibliography:

http://www.ncbi.nlm.nih.gov/myncbi/browse/collection/41154743/?sort=date&direction=ascending

D. Research Support

Ongoing Research Support

2 NHLBI R01 089850 Gianaros (PI) 2/1/2012 - 1/31/2018
Neurobiological Pathways Linking Stress and Emotion to Atherosclerosis
Tests the organizing hypothesis that stressor-evoked functional activity in the cingulate cortex, insula, and amygdala predicts the 3-year longitudinal progression of preclinical atherosclerosis.

1 NIDDK R01 110041 Gianaros (Co-PI) Marsland (Co-PI)
Metabolic and Inflammatory Pathways of Midlife Neurocognitive Disparities
Examines neurobiological, metabolic, and inflammatory pathways linking socioeconomic disadvantage with neurocognitive aging.

Completed Research Support (last 5 years)

R90 DA023420 Gianaros (PI) 9/30/2006 - 7/13/2017
Multimodal NeuroimagingTraining Program
To provide basic neuroscience training for integrative neuroimaging research, including training in the underlying principles, modeling, and applications of structural magnetic resonance imaging (MRI), functional MRI (fMRI), positron emission tomography (PET), magnetoencephalography (MEG), electroencephalography (EEG), and optical imaging. Students supported by this program integrate multiple methods and carry out multimodal neuroimaging projects over a 6-week period every summer.

T90 DA022761 Gianaros (PI) 9/30/2006 - 7/13/2017
Multimodal NeuroimagingTraining Program
To provide basic neuroscience training for integrative neuroimaging research, including training in the underlying principles, modeling, and applications of structural magnetic resonance imaging (MRI), functional MRI (fMRI), positron emission tomography (PET), magnetoencephalography (MEG), electroencephalography (EEG), and optical imaging. Graduate students in this program receive stipends and research support on an annual basis to integrate multiple methods and carry out multimodal neuroimaging projects for their graduate research.

NIDDK R01 DK095172 Gianaros (Co-I) 7/15/12 - 6/30/17
Influence of physical activity and weight loss on brain plasticity (PI, Erickson)
This application proposes to add a neuroimaging arm to an NIH funded 12-month diet and physical activity intervention.

NSF DMS 1557482 Gianaros (Co-PI) 9/10/2015 – 9/10/2016
QuBBD: Collaborative Research: Personalized Predictive Neuromarkers for Stress-Related Health Risks
Uses machine learning to predict individual differences in stressor-evoked blood pressure reactivity and longitudinal progression of cardiovascular risk.

NHLBI P01 HL040962 Gianaros (Co-I, Director Neuroimaging Statistics Core) 9/01/07 - 1/31/2015
Biobehavioral studies of cardiovascular disease (PI, Stephen B. Manuck)
This project examines genetic and neurobehavioral risk factors for coronary heart disease in adults.

NHLBI R01 HL101959 Gianaros (Co-I) 4/1/2010 - 3/31/2015
The brain as a target for pre- and essential hypertension (PI, J. Richard Jennings)
This project examines pre-hypertensive individuals to establish that the brain sequelae of the disease are present prior to diagnostic levels of high blood pressure.

NIA R01 AG037451 Gianaros (Co-I) 5/1/2011 - 4/30/2015
Resilience to mobility impairment: Neural correlates and protective factor (PI, Caterina Rosano)
This project examines functional and structural neural correlates of cognitive aging among late life adults.

NIDDK R01 DK089028 Gianaros (Co-I) 4/1/2010-3/31/2014
Characterization and validation of imaging biomarkers of accelerated brain aging in Type 1 diabetes (PI, Caterina Rosano)
This project uses functional and structural neuroimaging to characterize and establish signatures of brain aging and cognitive impairments among patients with insulin dependent diabetes.

NHLBI R01 HL101421 Gianaros (PI) 02/09/10 – 01/31/2013
Central Mechanisms for Cardioprotective Behavioral Effects of W-3 Fatty Acids
This project examines the effects of a 4-month fish oil intervention on pre- to post-intervention patterns of brain morphology and brain activation implicated in depressive and impulsive symptomatology, as well as cardiovascular disease risk.

NHLBI R01 089850 Gianaros (PI) 5/01/08 – 01/31/2012
Neural reactivity to stress and atherosclerosis
This project examines the brain systems that regulate stressor-evoked cardiovascular and autonomic reactions and patterns of stressor-evoked neural reactivity associated with preclinical atherosclerosis in adults.