those that are modifiable by their lifestyles. By extending the variation in aging rate among different individuals, and predict their regulatory networks and their contributions to cators. We further profiled blood cell mRNA and lncRNA fast-agers that are significantly supported by health indi after age 40. Using this predictor we identified slow- and by +/-6 years in facial age, with the deviations increasing that on average people of the same chronological age differ phenome. We constructed a robust age predictor and found the first comprehensive mapping of the aging human facial

**SESSION 1245 (SYMPOSIUM)**

**PRESIDENTIAL SYMPOSIUM: EXPANDING THE GEROSCIENCE NETWORK**
Chair: Matt Kaeberlein, University of Washington, Seattle, Washington, United States

In keeping with the 2019 GSA Annual Meeting theme of “Strength in Age—Harnessing the Power of Networks”, the Biological Sciences Presidential Symposium focuses on cutting edge approaches to understand the biology of aging using network and systems approaches.

**OMICS IN AGING RESEARCH: FROM BIOMARKERS TO SYSTEMS BIOLOGY**
Daniel Promislow, 1. University of Washington, Seattle, Washington, United States

Advances in whole genome sequencing have dramatically increased our potential to understand what shapes variation in rates of aging and age-related disease in natural populations, but we are still far from realizing this potential. Researchers have identified thousands of genetic markers associated with complex human traits. However, these markers typically explain a very small fraction of the observed variance, leaving an enormous explanatory gap between genotype and phenotype. I will present data from diverse species to illustrate the power of so-called endophenotypes—the epigenome, transcriptome, proteome, and metabolome—to bridge the genotype-phenotype gap. Using multivariate and network models that integrate genetic information with other endophenotype variation, we are closer than ever to understanding the mechanisms that account for natural variation in aging and age-related disease, and the evolutionary forces that have shaped that variation.

**HETEROGENEITY OF AGING IN HUMAN POPULATIONS**
Jing-Dong Jackie Han, 1. CAS-MPG Partner Institute for Computational Biology, Shanghai, China, China

Recently by analyzing the 3D facial images, we generated the first comprehensive mapping of the aging human facial phenome. We constructed a robust age predictor and found that on average people of the same chronological age differ by +/-6 years in facial age, with the deviations increasing after age 40. Using this predictor we identified slow- and fast-agers that are significantly supported by health indicators. We further profiled blood cell mRNA and lncRNA expression by RNA-seq of this cohort and computationally predict their regulatory networks and their contributions to the variation in aging rate among different individuals, and those that are modifiable by their lifestyles. By extending the study to a large Northern Chinese cohort of 10,000 people we can now use deep learning AI approaches to precisely estimate aging status based on 3D facial images and their associations with individuals’ health and medical history.

**EPIGENETIC AND METABOLIC REGULATION OF AGING**
Anne Brunet, 1. Stanford University, Stanford, California, United States

Aging is accompanied by a decline in the regenerative potential of most tissues. The mammalian brain contains regenerative neurogenic niches composed of neural stem cells (NSCs), neural progenitors, and other cells, including microglia, and endothelial cells. Neurogenic niches become less functional with increasing age. This deterioration could underlie cognitive and sensory restriction with age, although the exact age at which it occurs is still debated in humans. How the neurogenic niche changes during aging, and whether new cell types arise in older individuals, is not known. Our lab has embarked on a global characterization of the neurogenic niche during aging. This work provides a global understanding of the old neurogenic niche and suggests possible cause for NSC decline during aging. Results from these studies could open new avenues to counter age-related decline in the neurogenic niche and brain aging.

**SESSION 1250 (SYMPOSIUM)**

**PSYCHOLOGICAL AND HEALTH CONSEQUENCES OF HELPING OTHERS: INNOVATIVE METHODS TO UNDERSTAND STRAINS AND GAINS**
Chair: William E. Haley, University of South Florida, Tampa, Florida, United States
Discussant: Karl Pillemer, Cornell University, Ithaca, New York, United States

Older adults are often involved in prosocial behaviors including volunteering, informal assistance to family members, or extensive caregiving for family with chronic disease or disability. Many studies find that volunteering and providing informal support can enhance health and well-being, but family caregiving has generally been characterized as being highly stressful and harmful to health and well-being. Recent research has suggested that involvement in prosocial activities, including caregiving, can actually build resilience and buffer the impacts of stress, and that the commonalities across different types of prosocial behaviors in older adults deserve greater attention. This symposium brings together researchers who are using innovative methods to study prosocial behaviors, including measuring daily experiences and their linkages with affect, epidemiological methods, and use of health outcomes including serum biomarkers of inflammation and immunity, activity tracking, and mortality. Results across the presentations show that the effects of helping others can be considered as mixed blessings, with potentially harmful and helpful effects depending on contextual factors. Factors including a history of adverse childhood experiences, and dementia caregiving, can create particular challenges. The Discussant, Dr. Karl Pillemer, will discuss implications for future research on volunteering, informal assistance to family, and family caregiving. He will also address ways that