the literature with strong evidence supporting the hypothesis between two factors. Using the data from Taiwan's National Health Insurance Database this study is the first one comparing medical expenditure between vegetarian and non-vegetarian groups among older adults in Taiwan. In the National Health Insurance Database there were 2127 vegetarians matched with 4254 non-vegetarians based on age and gender from 2005 to 2014. Vegetarian diet is associated with approximately 10 – 15% lower total and outpatient medical expenditure. Vegetarians had lower medical expenditure incurred by diabetes and metabolic diseases, mental diseases, and genitourinary diseases. Implication for future research will be discussed at the presentation.

SESSION 3405 (SYMPOSIUM)

EPIGENETICS OF STRESS AND ADAPTATION ACROSS HEALTHSPAN AND LIFESPAN

Chair: Luigi Ferrucci, *Translational Gerontology Branch, Intramural Research Program, National Institute on Aging, National Institutes of Health, Baltimore, Maryland, United States*
Co-Chair: Shabnam Salimi, *University of Maryland Baltimore School of Medicine, Baltimore, Maryland, United States*
Discussant: Luigi Ferrucci

Any stimulus that endangers body integrity (stressor) results in an adaptive response to resolve stressful state and determine adaptive or and maladaptive responses. Both chronic extrinsic and intrinsic stressors can produce long-lasting, epigenetic changes in various organs that can eventually result in accelerated changes in bio-physio-pathology. There is initial evidence that stress response involves mechanisms of the epigenetic basis of adaptation and stress response to biological aging and chronic diseases. With aging, homeostasis stability declines causing augmented vulnerability to the external and internal stress. Individuals in whom vulnerability trespass a certain threshold experience accelerated aging and deterioration of health and/ or "secondary aging" phenomena such as premature mortality. Because of substantial heterogeneity of the rate of decline in homeostatic stability, there is inter-individual variability in the age of appearance of chronic diseases and the increased risk of disability and mortality. Thus, tools for the quantification of a stress response would be clinically valuable. Therefore, this symposium suggests approaches to study the epigenetic basis of molecular adaptations across various age, organs' health-span, and life-span.

DNA METHYLATION AT THE CROSSROADS OF PHYSIOLOGICAL RESPONSE TO STRESS AND PATHOLOGICAL BIOLOGY

Shabnam Salimi, *University of Maryland Baltimore School of Medicine, Baltimore, Maryland, United States*

It is likely that the dimension and the scope of stress perception are different in different body systems conditioning their specific rates of aging. Moreover, in addition to between-individual variabilities in body response to stress, there is inter-organs variability in adaptations resulting in pleiotropy of phenotypes evolved with aging. Epigenetic measures may be considered possible readouts of the extent and proxy of the adaptive response to intrinsic and/or extrinsic stress in body organs. It is thus important to disentangle the epigenetic basis of stress, adaptation, and pathology. In this symposium we discuss a combination of the specific DNA methylation patterns relate to the diseases as an intrinsic stressor and adaptation responses imposed to bodily systems, and also accelerated body organ morbidities. We explain the static and dynamic DNA methylation differentiation over time as the "epigenetic code" of accelerated pathology across various age using longitudinal data.

EPIGENETICS MARKERS OF AGING AND EXPOSURES

Joanne Murabito[1], *1. Boston University, Boston, Massachusetts, United States*

Widespread changes to the epigenome occur with aging. DNA methylation is one of the most commonly studied epigenetic mechanisms, reflects lifetime exposures that impact aging, and is associated with age-related disease risk. Many longitudinal cohort studies have existing cross-sectional or longitudinal DNA methylation data along with genotype and expression data permitting investigation of relationships between DNA methylation markers, exposures, and disease. The data can be leveraged to conduct large epigenome-wide association studies (EWAS) of aging and age-related disease to identify DNA methylation biomarkers and lead to insights into novel biologic pathways for development of interventions to delay aging. DNA methylation age measures robustly predict chronologic age and associate with both healthspan and lifespan. During the workshop, examples from cohort studies and the CHARGE consortium will be presented.

ENVIRONMENTAL EPIGENETICS AND AGING

Andrea Baccarelli[1], *1. Columbia, Mailman School of Public Health, New York, New York, United States*

The human epigenome is a flexible, environmental sensitive component of human biology that changes over time. Multiple studies have identified prospective changes in epigenetic marks that indicate that the epigenome ages as we grow older. These changes have been leveraged to create multiple indicators of age that may also predict mortality and age-related disease. There is ongoing research to determine the extent to which age-related epigenomics changes are inherent to cell biology and/or driven by lifestyle and environmental factors. In this presentation, I will review the current evidence derived from human aging studies and potential contributions to human health and disease. I will discuss the source of data, methodological challenges for large human studies, limitations and possible future directions.

EPIGENETIC CLOCKS OF COMPUTED TOMOGRAPHY MEASURES OF FATTY ORGANS

Ake T. Lu,[1] and Steve Horvath[2], *1. Department of Human Genetics, University of California, Los Angeles, Los Angeles, California, United States, 2. UCLA, Los Angeles, California, United States*

DNA methylation (DNAm) based biomarkers collectively known as "epigenetic clock" yielding accurate measure of age in any tissue across the entire life course have associated with a large host of age-related conditions. To study their implications in metabolic syndromes,