and functional declines in immune and inflammatory processes. All share a common connection in metabolic dysfunction. Furthermore, aging itself is associated with changes in metabolism, although the underlying drivers for these changes are unknown. Here we introduce speakers working at the cutting edge in metabolism research, and whose studies are of direct relevance to aging. Dr. Chandel will focus on mitochondrial biology, describing recent advances in understanding the mechanisms of the beneficial effects of metformin. Dr. Haigis takes the mitochondrial theme to cancer biology, the area of research that revived metabolic perspectives in biomedical research. Dr. Najt's talk describes a less well studied organelle, the lipid droplet, and its role in a rapidly expanding area of research on lipid metabolic regulation specifically in the context of aging. Dr. Brown-Borg will present data on nutritional and genetic modulation of metabolism and how pathways converge to influence chromatin and epigenetic regulation of gene expression. Together our speakers explore new concepts in metabolism research that are of particular relevance to aging. This session aligns with the concept of GeroScience, the more we know of aging biology the better we understand diseases and disorders of aging. This session will demonstrate that metabolism, its regulation, and its influence on key processes linked to health and longevity, place it in a central position as we seek to discover targets and interventions to improve human aging.

THE ROLE OF MITOCHONDRIA IN AGING AND CANCER

Marcia Haigis, *Harvard Medical School, Boston, Massachusetts, United States*

METHIONINE METABOLISM IN AGING REGULATION

Holly Brown-Borg, *University of North Dakota School of Medicine & Health Sciences, Grand Forks, North Dakota, United States*

Aging is the major risk factor for many diseases but the mechanisms are poorly understood. The risk of developing hepatic steatosis increases with age and the health impact of this disease is negative and high. When challenged with high fat diets, long living Ames mice withstand the detrimental metabolic effects that occur in normal mice. We examined transcriptomic and epigenomic profiles of Ames and wild type hepatocytes in the presence or absence of fat to demonstrate that the epigenomic profile drives transcription factor and downstream gene expression resulting in susceptibility or resistance to fatty liver disease. We found that markers of steatosis are related to gene expression in wild type and Ames mice, and dwarf mice retain fewer lipid droplets compared to wild type mice. These studies will provide data to guide our understanding of mechanisms leading to hepatic disease and define factors that provide protection from age-related metabolic disorders.

METFORMIN INHIBITS MITOCHONDRIAL COMPLEX I TO PROMOTE HEALTH

Navdeep Chandel, *Northwestern University, Illinois, United States*

The major function of mitochondria in cellular homeostasis has been the generation of ATP through oxidative phosphorylation. However, we have previously demonstrated that mitochondria can serve as signaling organelles by releasing low levels of reactive oxygen species (ROS) and TCA cycle metabolites that are essential for hypoxic activation of HIF, antigen activation of T cells, cellular differentiation and proliferation of cancer cells. The anti-diabetic drug metformin has been proposed to inhibit mitochondrial complex I. We will present data indicating that metformin inhibits mitochondrial complex I to exert it's biological effects through controlling ROS, ATP, and NAD+.

LIPID DROPLET SIGNALING IN METABOLIC HEALTH AND AGING

Charles Najt,[1] and Douglas Mashek,[2] *1. University of Minnesota, 2. University of Minnesota, Minneapolis, Minnesota, United States*

Lipid droplets (LDs) are neutral lipid rich organelles involved in lipid storage, fatty acid trafficking, and signaling. Emerging evidence from our laboratory and others suggests that the specific LD resident proteins couple/uncouple cells and tissues from inflammation and metabolic dysfunction. However, the mechanism by which LD proteins influences these critical pathways remains unknown. We will present data delving into the role of LD proteins Perilipin (PLIN) 2 and 5 in balancing cellular energy metabolism, mitochondrial function, and inflammation. Data will be presented defining novel mechanisms through which PLIN2 orchestrates eicosanoid production as a means to promote inflammation. We will contrast these findings to PLIN5, which uncouples LD accumulation from metabolic dysfunction and inflammation, in part due to its promotion of SIRT1 signaling. Overall, these studies will highlight a crucial role of LD metabolism and signaling in regulating cellular energy homeostatic processes known to be key players in governing healthspan.

Session 4110 (Paper)

Physical Activity and Well-Being

DELAYING HEALTH CARE DUE TO THE COVID-19 PANDEMIC: ASSOCIATIONS WITH PHYSICAL AND MENTAL HEALTH AND PREVENTIVE CARE

Felicia Wheaton,[1] Terika Scatliffe,[2] and Matilda Johnson,[2] *1. Xavier University of Louisiana, New Orleans, Louisiana, United States, 2. Bethune-Cookman University, Daytona Beach, Florida, United States*

Health care is important for maintaining optimal physical and mental health. However, due to the COVID-19 pandemic, many older adults have delayed or postponed care. Data from the special midterm release of the 2020 Health and Retirement Study (HRS) were used to examine the relationship between chronic conditions and delayed care, as well as between delayed care and mental health outcomes and preventative care among Americans aged 50+ (N=3,266). Approximately 30% of respondents said yes when asked "Since March 2020, was there any time when you needed medical or dental care, but delayed getting or did not get it at all?" Of those, 55% said their provider cancelled, closed or suggested rescheduling, 28.5% decided it could wait, and 20.8% were afraid to go. Results from OLS and