illnesses. Alzheimer’s disease and related dementias (ADRD) are terminal, debilitating conditions, yet little is known about the use of palliative care in this population. Using Pennsylvania Medicaid claims for dual-eligible beneficiaries over age 65 merged with Medicare enrollment data and restricted to beneficiaries who were dually-eligible and not in a Medicare managed care plan, we identified individuals who: died in 2014 or 2015 and were diagnosed with ADRD at least 12 months prior to death. We used multivariate logistic regression to examine the association between having a diagnosis of ADRD and use of palliative care, controlling for age, gender, race, use of long-term services and supports, region, and comorbidities. We found that people with ADRD were less likely to use any palliative care than people without a diagnosis (OR .74; CI .60 to .91).

SESSION 1320 (SYMPOSIUM)

M. POWELL LAWTON AWARD LECTURE
Chair: X. Dong, Rush University Medical Center, Chicago, Illinois

The Lawton Award is presented annually to an individual who has made outstanding contributions from applied research that has benefited older people and their care. The lecture will be given by the 2017 recipient, XinQi Dong, MD, MPH of Rush Medical College at Rush University. The Lawton Award is generously funded by the Polisher Research Institute of the Madlyn and Leonard Abramson Center for Jewish Life.

CONFUCIUS VS. EINSTEIN: WHO WOULD BE A BETTER GERONTOLOGIST?
X. Dong, Rush University Medical Center, Chicago, Illinois, United States

Confucius and Albert Einstein are two of the most widely celebrated scholars throughout history. While known for philosophy and physics, respectively, Confucius and Einstein were great thinkers whose work and musings can be applied to many other disciplines. Dr. XinQi Dong, the 2017 recipient of the M. Powell Lawton Award, will discuss the life and work of both Confucius and Einstein in the context of how they might approach the field of gerontology and the care of older adults and which approach would align with what we know about best practices today.

SESSION 1325 (SYMPOSIUM)

OXIDATIVE STRESS AND AGE-RELATED DISEASES
Chair: H. Van Remmen, Oklahoma Medical Research Foundation, Oklahoma City, Oklahoma

Oxidative stress has long been implicated in the underlying mechanisms contributing to aging. Perhaps the most evident role of oxidative stress is in the initiation and progression of a number of age related diseases. Here we focus on a range of pathologies related to oxidative stress in animal and cell culture models. Work from the Van Remmen laboratory describes a promising new intervention for age related sarcopenia, a compound that activates the SERCA pump and helps to control calcium homeostasis. Data suggests that this compound can reduce muscle atrophy and weakness in the Sod1-/- mouse, a model of oxidative stress and sarcopenia. Dr. Rabinovitch will discuss a mitochondrial targeted treatment to reverse age-related functional declines. His team finds that that old mice that receive 8 weeks SS-31 (elamipretide) have improved muscle energetics and enhanced skeletal muscle and heart function, suggesting that short-term treatments can enhance mitochondrial function and reverse muscle aging phenotypes. Using primary cultures of retinal pigment epithelium (RPE) cells Dr. Ferrington has shown that RPE from AMD donors are more resistant to oxidative inactivation of two energy producing pathways (glycolysis and oxidative phosphorylation) and less susceptible to oxidation-induced cell death. Finally, data from Dr. Kasinath support a clear protective role for hydrogen sulfide generation in the aging kidney, reducing the aging-induced increase in urinary albumin excretion and inhibited matrix accumulation and reducing oxidative stress through via Nrf2 activation and decrease in NOX4 expression. These studies further support an important role for oxidative stress in age related pathologies.

SERCA ACTIVATION AS AN INTERVENTION TO REDUCE MUSCLE ATROPHY AND WEAKNESS
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We tested a pharmacological intervention to modulate sarcopenia, i.e., activation of the sarcoendoplasmic reticulum (SR) calcium ATPase (SERCA), that returns Ca2+ back into the SR following muscle contraction. We asked whether CDN1163, an allosteric activator of SERCA, could modulate muscle mass and function in a mouse model of sarcopenia (Sod1-/- mice). Two month old Sod1-/- mice were treated for 7 weeks with CDN1163 (50 mg/kg, i.p., 3x/week). After treatment, gastrocnemius mass of CDN1163-treated Sod1-/- mice was 23% greater than untreated Sod1-/- mice and the 22% reduction in specific force in untreated Sod1-/- versus wild type mice was reversed. CDN1163 also prevented the increase in muscle mitochondrial ROS generation in mitochondria from Sod1-/- mice and reduced muscle oxidative damage measured as F2-isoprostanes by 50% compared to untreated Sod1-/- mice. These findings suggest that pharmacological stabilization of SERCA with CDN1163 may be a powerful tool to counter age- and oxidative stress-associated muscle impairment.