40%) and were younger than their non-LGB counterparts. These findings highlight dementia risk and related problems among LGBTQ older adults. Future studies are needed to better understand dementia risk and recruiting, screening and improving dementia-related outcomes in LGBTQ older adults.

GENDER DIFFERENCES IN HOW THE AMERICAN PUBLIC REACTS TO A PERSON WITH MILD-STAGE DEMENTIA

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Many studies show that caregivers for those with Alzheimer's Disease (AD) are disproportionately female, but few studies have investigated how public attitudes influence this gender disparity. We analyzed secondary data from an experimental study of public reactions to AD dementia. Analysis included 944 respondents who read a vignette about a man with mild stage dementia and completed a modified Family Stigma in Alzheimer's Disease Scale (FS-ADS), which assesses 7 domains of stigma. Multivariable ordered logistic regression compared men and women on FS-ADS ratings. Women were less likely than men to endorse stronger negative aesthetic attributions (OR=0.75) and negative feelings (OR=0.76) and more likely to endorse stronger feelings of pity (OR=1.33; all $p<0.05$). No other differences were observed in FS-ADS domains (all $p>0.05$). The findings offer insights into relationships between gender and AD stigma, which may influence who is willing to become a caregiver for persons with AD and related dementias.

SESSION 7020 (SYMPOSIUM)

BREATHING WELL ACROSS THE LIFESPAN: PULMONARY AGING AND GEROSCIENCE-TARGETED THERAPIES

Chair: Jason Sanders

Excellent pulmonary function is one of the strongest predictors of longevity across animal models and human populations. Unfortunately, none of the major age-associated pulmonary diseases – obstructive lung disease, pulmonary fibrosis, and increased susceptibility to pneumonia – have strongly effective disease modifying therapies. There is growing evidence that normal age-associated decline in pulmonary function and major age-associated pulmonary diseases are linked to the hallmarks of aging including senescence, nutrient signaling dysregulation, mitochondrial dysfunction, and telomere disorders. This presents opportunities for collaboration between gerontologists and pulmonologists to unravel age-associated developmental mechanisms and design novel treatments. In this symposium, leaders in pulmonary aging research will present novel data on links between aging and pulmonary health and geroscience-based interventions under study. Dr. Sanders will provide an overview of the scientific and clinical space and present epidemiologic associations between aging biomarkers, early pulmonary fibrosis, and mortality. Dr. Le Saux will discuss senescence and specifically how eicosanoid biology may explain organ-specific patterns of senescence-associated fibrosis. Dr. Thannickal will discuss age-associated perturbations in metabolism and mitochondrial function and targeting these pathways to improve lung function and treat pulmonary diseases. Dr. Newton will discuss mechanisms and clinical applications of telomere biology to pulmonary aging. Symposium attendees will (1) be poised to generate collaborations between gerontologists and pulmonologists to address existing knowledge gaps in mechanisms of pulmonary aging, and (2) develop a better understanding of translational opportunities to design geroscience-based diagnostics and therapeutics to improve pulmonary health with aging.

ASSOCIATIONS BETWEEN AGING-RELATED BIOMARKERS, INTERSTITIAL LUNG ABNORMALITIES, AND MORTALITY

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Interstitial lung abnormalities (ILA) exist in ~10% of adults >50 and associate with increased morbidity/mortality. Their pathobiology is poorly understood; age is the strongest risk factor. In the Framingham Heart Study, we determined associations between ILA and 10 blood biomarkers previously robustly associated with aging and mortality. Odds of ILA increased directly with ln-transformed GDF15 (OR [95% CI] = 3.20 [1.74-5.91], p=0.0002), TNF-αRII (2.41 [1.34-4.34], p=0.003), IL6 (1.76 [1.39-2.22], p<0.0001), insulin (1.56 [1.11-2.20], p=0.01), and CRP (1.53 [1.27-1.84], p<0.0001). Causal analysis showed GDF15 (p=0.008), TNF-αRII (p=0.004), and IL6 (p<0.0001) mediate the age effect on ILA. In adjusted survival models, only higher ln(GDF15) and ln(TNF-αRII) were associated with mortality (HR [95% CI] = 4.3 [2.3-8.1], p<0.0001 and 2.9 [1.5-5.8], p=0.002). GDF15 results were replicated in the COPDGene Study. These results suggest aging biomarkers may help risk stratify adults with ILA, and unmeasured ILA may confound prior associations between biomarkers and mortality.

SENESCENCE AND ITS ROLE IN FIBROSIS

Claude Le Saux, *University of California San Francisco, San Francisco, California, United States*

The presence of senescent cells (epithelial and mesenchymal) in fibrotic organs has been well established. Removal of senescent cells in animal models of fibrosis indicate an overall beneficial effect. The general consensus is that the senescent cells contribute to the fibrotic phenotype by the secretion of factors, mainly cytokines and chemokines. We recently demonstrated that senescent cells can also secreted eicosanoids. These lipids are implicated in the pathogenesis of fibrosis in multiple organs. Prostaglandins, especially PGE2, are generally regarded as anti-fibrotic, whereas leukotrienes are thought to be pro-fibrotic. Recent studies indicate that the senescence-associated secretory profile is a dynamic process and its composition is cell, tissue, and time-dependent. In this session I will discuss how senescent cells from specific origin have the potential to regulate fibro-genesis and its resolution by switching their eicosanoid profile expression over time. These findings have important implications for