(1586.1 vs. 252.0 per 100,000/year; adjusted HR=3.09; 95% CI, 2.23-4.30). Those reentering the community in later life after prison are at higher risk of experiencing SUD-related hospitalizations or ED visits. Prevention and intervention efforts targeting later-life prison-to-community care transitions are needed. Part of a symposium sponsored by the Aging, Alcohol and Addictions Interest Group.

SESSION 7015 (SYMPOSIUM)

BEYOND SEX: GENDER, LGBTQ, AND ALZHEIMER'S DISEASE AND RELATED DEMENTIAS

Chair: C. Elizabeth Shaaban
Discussant: Michelle Mielke

Sex and gender are important sources of variation in Alzheimer's disease and related dementias (ADRD) and associated caregiving. Women comprise 2/3 of ADRD cases and the majority of ADRD caregivers. Sex encompasses biological differences due to sex chromosomes, reproductive tract, and hormones, while gender constitutes socioculturally constructed psychosocial aspects of sex. Several lines of research have begun to interrogate sex differences, but less is known about the relation of gender and lesbian, gay, bisexual, transgender, and/or queer (LGBTQ) status with ADRD. In this symposium featuring both trainees and faculty we highlight novel research addressing these factors from multiple perspectives. Two presentations address how psychosocial characteristics and their strengths of association with brain health may vary by gender. C. Elizabeth Shaaban presents analyses testing whether gendered psychosocial factors explain sex differences in white matter hyperintensities, a neuroimaging marker of cerebral small vessel disease and risk factor for ADRD. Justina Avila-Rieger presents results testing region of birth-based spatial patterning of dementia risk among Black men and women. Next, Jason Flatt presents prevalence estimates of subjective memory problems and dementia and describe factors associated with dementia among LGBTQ older adults. Finally, gender may also impact perceptions of individuals with dementia. Shana Stites explores gender differences in AD stigma and discuss implications for who is willing to be an AD caregiver. Michelle Mielke, an expert in sex and gender differences in neurodegenerative and age-associated diseases will facilitate conversation about these results and place them in the context of current sex and gender-based ADRD research.

DO GENDERED PSYCHOSOCIAL FACTORS EXPLAIN SEX DIFFERENCES IN WHITE MATTER HYPERINTENSITIES?

C. Elizabeth Shaaban,[1] Caterina Rosano,[1] Heather Shattuck-Heidorn,[2] Sara Godina,[3] Minjie Wu,[1] and Andrea Rosso,[4] 1. *University of Pittsburgh, Pittsburgh, Pennsylvania, United States, 2. Women and Gender Studies Program, University of Southern Maine, Portland, Maine, United States, 3. University of Pittsburgh Graduate School of Public Health, Pittsburgh, Pennsylvania, United States, 4. School of Public Health, University of Pittsburgh, Pittsburgh, Pennsylvania, United States*

Women have a greater burden than men of white matter hyperintensities (WMH), a marker of cerebral small vessel disease (cSVD). Psychosocial factors including education, household income, neighborhood socioeconomic status (nSES), happiness, and depression may differ by gender and could explain women's higher burden of WMH. In a cohort of older adults (N=250, median age=82, 58% women, 39% Black), we found that women had lower education, household income, nSES and were less happy and more depressed. Race stratified Spearman correlations showed women had greater whole brain WMH volume in white participants only (white: rho=0.23, p=0.004; Black: rho=-0.05, p=0.64). In partial Spearman correlations, education, happiness, and depression attenuated but did not fully explain the relationship when added individually or all together to the model for whites (fully adjusted rho=0.19, p=0.03). Gendered psychosocial factors may partially explain sex differences in WMH; interventions targeting these factors may reduce cSVD burden, particularly in white women.

DIFFERENTIAL ASSOCIATION OF GEOGRAPHICAL REGION OF BIRTH WITH DEMENTIA RISK ACROSS BLACK WOMEN AND MEN

Justina Avila-Rieger,[1] Audrey Murchland,[2] Nika Seblova,[3] Maria Glymour,[4] Adam Brickman,[5] Nicole Schupf,[3] Richard Mayeux,[5] and Jennifer Manly,[5] 1. *University of New Mexico, New York, New York, United States, 2. University of California, San Francisco, San Francisco, California, United States, 3. Columbia University Medical Center, New York, New York, United States, 4. University of California, San Francisco, California, United States, 5. Columbia University, New York, New York, United States*

Risk of dementia is both racially and spatially patterned. Less is known about sex/gender differences in pathways linking birth place to late-life cognitive outcomes in older non-Latino Blacks. The 1464 Black men and women included in these analyses were Northern Manhattan residents. Cox regressions revealed that Stroke-Belt South (SB) and Non-Stroke-Belt South (NSB) birth was associated with a higher dementia risk, adjusted for birth year, childhood SES, and risk of death. Compared to Northern-born (NB) men, SB men had the highest risk, followed by NSB women and SB women, while NSB men and NB women had a similar risk to NB men. The higher risk for SB men and NSB women remained after adjusting for education, adult income, and CVD burden. Future work should identify why birth in the SB is uniquely detrimental for cognitive health among Black men, while birth in NSB has the strongest impact on Black women.

THE EPIDEMIOLOGY OF DEMENTIA IN LGBTQ OLDER ADULTS

Jason Flatt, *University of Nevada, Las Vegas, School of Public Health, Las Vegas, California, United States*

Over 3 million or more adults aged 60 + live in the US who identify as lesbian, gay, bisexual, transgender, and/or queer (LGBTQ). Less is known about dementia risk in LGBTQ older adults. We will discuss dementia risk and related risk factors among LGBTQ adults from multiple population-based and cohort studies. We found higher rates of subjective memory problems among lesbian, bisexual and transgender adults compared to both gay men and heterosexual men and women. Using medical record data, 8% (343) of LGB adults aged 60+ were diagnosed with dementia. They were more likely to identify as male (63% vs. 44%), had a higher education level (college degree+ 63% vs.

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

Shana Stites,[1] Emily Largent,[1] Jeanine Gill,[1] Rebecca Johnson,[2] Kristin Harkins,[1] and Jason Karlawish,[1] *1. University of Pennsylvania, Philadelphia, Pennsylvania, United States, 2. Princeton University, Princeton, New Jersey, United States*

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

Jason Sanders, *Brigham And Women's Hospital, Wellesley, Massachusetts, United States*

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