There is increasing interest in examining aggression in older adults and persons living with dementia (PLWD) in nursing homes. Relational or indirect aggression between tenants is also of concern in assisted living (A/L). However, there is a dearth of knowledge about how people interpret the meaning of aggression in older adults in A/L. Such interpretations inform responses within interactions as well as in management, practice and policy. This study explored interpretations of aggression in older tenants of A/L through thematic and narrative analyses of qualitative interview data from 32 participants: 13 tenants and 19 staff. Tenants downplayed the existence of 'bullies' but spoke of (or themselves enacted, in the interview) relational aggression in tenant conflicts with 'troublemakers,' complainers, slackers and PLWD. Dementia was not universally interpreted as excusing inappropriate or aggressive behaviours, and tenants expressed discomfort interacting with PLWD. The narratives of both groups (tenants and workers) drew on and reproduced stigmatizing and/or patronizing views of dementia and aging. Workers commonly positioned dementia (or other conditions such as physical illness, changes in routine, isolation, and loss of independence) as mitigating culpability for aggression. They did so in part to avoid taking aggression personally; some were also concerned about protecting tenants from disciplinary measures. Narratives about aggression reflect and further reinforce relations of power within A/L, with implications for everyday interactions of life and work in these settings. A/L facilities can address relational aggression in part through addressing talk and actions that perpetuate ageism and dementiaism.

SESSION 2230 (PAPER)

ESPO/ BIOLOGICAL SCIENCES SECTION SYMPOSIUM: EMERGING INSIGHTS IN INTERACTIONS AND NETWORKS IN THE BIOLOGY OF AGING

DEVELOPING C. ELEGANS TO STUDY AGE-RELATED EXTRACELLULAR VESICLE SIGNALS

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All cells release vesicles into their extracellular environment. These extracellular vesicles (EVs) contain multiple classes of molecules, including nucleic acids, proteins, and lipids. EV-signaling has been shown to be impacted by many age-related physiological processes such as inflammation, mitochondrial stress, and autophagy as well as directly mediate critical functions in cellular senescence and aging. The isolation and analysis of EV cargos from mammalian cell culture and liquid biopsy samples has become a powerful approach for uncovering the messages that are packaged into these organelles. Caenorhabditis elegans is a premier model for dissecting the genetics of aging however, EV analysis has not been tenable in invertebrate model systems due to lack of methods for obtaining sufficient amounts of pure EVs. We developed a method for isolating pure EVs from C. elegans with yields sufficient for mass spectrometry and RNAseq. Here we present the analysis of the genetic and protein cargos of EVs collected from wild type and long-lived mutants collected at different time points across their lifespans. As the first investigation of age-related EV signals in an invertebrate model system we believe these results will provide insights into cell non-autonomous mechanisms of aging.

EPIGENETIC PROFILES OF BIOLOGICAL AGING HALLMARKS

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Investigation into the hallmarks of aging point to the existence of shared mechanisms that underlie the biological aging process. While there is a general consensus that hallmarks of aging rarely occur in isolation, little is known in regards to their overlapping networks or how interactions contribute to manifestations at the clinical level. Here, we examine whether shared epigenetic alterations—one of the proposed hallmark of aging—underlies diverse conditions characterized by other hallmarks, including cellular senescence, loss of proteostasis, genomic instability, mitochondrial dysfunction, and inflammation. Using weighted network analysis, we identified consistent overlaps in the methylation profiles across the different traits. For instance, epigenetic modules that were distinct in senescence were also affected in progeroid syndromes (Hutchinson-Gilford Progeria Syndrome and Werner's Syndrome) and smokers. These CpGs tended to be located in CpG islands, which are notable for their strong association with transcriptional regulation. Overall, our results suggest that epigenetic alterations intersect with various hallmarks of aging. In moving forward, incorporation of this understanding may lead to the development of biomarkers that better capture the biological (rather than chronological) aging process.

THE EFFECTS OF AGE AND SIZE ON THE COMPANION DOG METABOLOME: ROLES OF TRYPTOPHAN AND FATTY ACID METABOLISM

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Recently, the companion dog has been promoted as an ideal animal model for human aging. Dogs show an interesting phenomenon where smaller individuals are longer lived than their larger counterparts. However, many of the underlying molecular mechanisms that influence aging and longevity in the dog are unknown. To begin to uncover these physiological changes, we completed the largest metabolomics study to date in the companion dog. Here, we collected blood plasma samples from companion dogs in three in the United States for metabolomics analysis. We then looked at the effects of age and size on the metabolome to develop new hypotheses about healthy canine aging. Our most striking differences were found with regards to geographic location in the canine metabolome, in which metabolic profiles were more similar between dogs in the same city than across cities. After controlling for this location effect, we found a strong signal of amino acid metabolism, specifically tryptophan metabolism, associated with weight in the dog where metabolites in the tryptophan metabolism pathway were always higher in small, long-lived dogs. Future studies