mTBI participants experienced significantly greater myelin loss across ~96% of the cortex ($p < 0.05$), suggesting that mTBI has dramatic impact upon cortical myelin content. Myelin loss magnitude was comparable across mTBI and AD, particularly within temporal lobes. Future research should study whether post-traumatic demyelination increases the AD risk.

Session 9100 (Poster)

Biology of Aging: Immunity and Aging

ANALYSIS OF SINGLE CELL DATA AS IT RELATES TO AGING AND LONGEVITY

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Age-related disability and diseases are known to be delayed in people living to 100 years or more. Changes in the immune system with age are known, including in cell type composition and gene expression differences. To further explore changes in extreme longevity subjects, we investigated peripheral blood immune system cell subpopulations across age and extreme longevity at a single cell resolution. We performed an integrative analysis of public scRNA-seq datasets to define consensus cell types of longevity and age, and classified cell types in our novel New England Centenarian Study dataset. We integrated these datasets together to investigate cell type specific differences at a composition and gene expression level. Our findings identified higher cell type diversity in extreme longevity subjects compared to younger age groups, but no significant difference among younger age groups demonstrating that overall composition differences are unique to longevity. We identified novel differences in myeloid and lymphocyte populations; Extreme longevity subjects have higher composition of CD14+ Monocytes, Natural Killer cells, and T gamma delta populations and lower composition of CD16+ Monocytes and dendritic populations. We characterized gene expression differences between extreme longevity and younger age groups and differences in aging across younger age groups. We found that extreme longevity cell type specific signatures overlapped with the aging signatures by at least 50%. We identified unique genes to extreme longevity that are enriched for pathways specific to immune activation and inflammation, suggesting a protective mechanism for centenarians through efficient activation and regulation of immune subpopulations in peripheral blood.

ASSOCIATION BETWEEN SERUM 25-HYDROXYVITAMIN D LEVELS AND INCIDENCES OF INFECTION IN LONG-TERM CARE

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Insufficient serum 25-hydroxyvitamin D (25(OH)D) concentrations are associated with increased respiratory tract infections, influenza, and other infectious diseases. As the world deals with the COVID-19 pandemic, the interest of adequate serum levels to reduce the risk of infection has surfaced. This study determined if the number of infections per year are associated with serum 25(OH)D concentrations in long-term care (LTC). Participants (≥ 65 years) in a cross-sectional study were recruited across five LTC communities in Texas. Medical records were used to collect a one-year medical history using double-blind protocols. Blood draws were collected to measure serum 25(OH)D concentrations. Medical records were used to classify infections based upon documentation of signs and symptoms of infection concurrent with either a physician's note or antibiotic/antiviral medication prescription. Race, BMI, sex, age, and liver and renal disease diagnoses were used as confounders. Of the 177 participants (89% Caucasian, 63% female, mean age 83 years) 69% had ≥1 infection over year and 55% had insufficient serum 25(OH)D concentrations <30 mg/mL (mean 32.6 ng/mL). Linear regression did not show a significant association between serum 25(OH)D concentrations and number of infections (β 0.003; 95% CI -0.014, 0.018; p=0.760). Additionally, insufficient serum concentration did not increase the odds of having an infection (OR 1.02; 95% CI 0.05, -19.34; p=0.987). This study did not show a significant association between infection rates and serum 25(OH)D concentrations. However, further research is needed to determine if vitamin D supplementation could be an effective therapeutic intervention to reduce infection rates, including COVID-19.

COMBATING AGE-ASSOCIATED IMMUNE DECLINE USING KYNURENINE PATHWAY INTERVENTIONS

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Select kynurenine pathway interventions extend lifespan in invertebrate models and are of interest in treating age-associated diseases. Kynurenine pathway activity is responsive to inflammatory signaling, and we are evaluating the potential for these interventions to increase pathogen resistance and curtail age-associated immune decline in Caenorhabditis elegans and mammals. The kynurenine pathway facilitates the catabolism of tryptophan to nicotinamide adenine dinucleotide (NAD). Our lab has found that supplementing the kynurenine metabolite 3-hydroxyanthranilic acid (3HAA) or inhibiting the enzyme 3HAA dioxygenase (HAAO) extends lifespan in C. elegans. 3HAA has demonstrated pro/anti-inflammatory properties in mammals, suggesting a potential role in immune function. C. elegans have a primitive immune system that lacks an adaptive element, but it recapitulates aspects of innate immune signaling and pathogen response. I hypothesize kynurenine pathway interventions that impact C. elegans' lifespan similarly improve pathogen resistance and immunity. Interventions within the kynurenine pathway are capable of differentially impacting pathogenesis and lifespan of C. elegans challenged with Psuedomonas