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"Effect of Alcohol on HIV-associated Frailty Trajectories"

Background: Frailty is a geriatric syndrome of accelerated aging characterized by loss of multi-system physiologic reserve and increased vulnerability to stressors. While the paths to frailty are multiple, HIV infection is associated with increased frailty. With the advent of HAART/cART and effective means of viral suppression, people with HIV (PWH) can achieve nearly normal lifespans, but, notably, the increased prevalence of frailty persists despite viral suppression. Alcohol use disorders (AUD) and other forms of hazardous alcohol consumption represent a common comorbidity within this population. A prior 2020 cross-sectional analysis of the New Orleans Alcohol use in HIV (NOAH) study cohort[1] reported the association of lifetime alcohol exposure (LAE) with frailty in PWH. Further, this earlier analysis identified a seemingly paradoxical relationship between simultaneous higher LAE and decreasing timeline follow back (TLFB), a measure of recent alcohol use, with higher baseline frailty. Of particular note is that maximal frailty was observed in presently alcohol-abstinent participants with highest LAE. This finding is postulated to be the result of a "sick quitting" effect, whereby historic higher alcohol consumption and the resulting physiologic impacts produce an attenuation of later alcohol consumption. In this follow up longitudinal study, we hypothesize that active alcohol use accelerates unfavorable change in frailty trajectories and, further, that baseline frailty status and LAE moderate the effects of current alcohol consumption on frailty trajectories.
Methods: Utilizing follow up data from a window of 15-45 months since baseline testing, phenotypic frailty and deficit index scores have been generated and, after preliminary analysis at the cohort level, will be used to cluster NOAH participants into discrete trajectories representing favorable, unfavorable, or unchanged frailty status. Correlational analysis of these trajectories with alcohol use metrics will be performed to characterize these groups in terms of LAE, TLFB and PEth quantitation.
Results: A preliminary analysis of the longitudinal data has been completed at the cohort level comparing baseline data and first testing follow up within a window of 15-45 months post intake. The portion of the cohort that completed follow up during this time is n=237, with the majority tested at 30 months. At the cohort level, an average 0.2 increase in phenotypic frailty (PFI) and 0.2 decrease (i.e. a raw decrease of 1 tested criterion) in deficit index (DI-58) scores was identified. Similarly, a decrease in both PEth and the AUDIT and AUDIT-C scores was seen in the cohort average. Finally, while HIV viral load counts are still in progress, an average increase of 60.2 cells/mm3 in CD4+ lymphocyte cell counts was observed within the study population.
Discussion: Taken together, the above is suggestive of reduced alcohol consumption as co-occurring with improved HIV-associated immune measures and the broader DI-58 frailty measure. The apparent divergence of the PFI and DI-58 measures accords with the broader frailty literature, which has suggested phenotypic frailty as a less modifiable measure of frailty that belies potentially consequential change in health status. Next steps will seek to generate frailty trajectories based on alcohol consumption patterns, as well as stratification by baseline frailty, HIV viral load, and CD4 counts to elucidate potential differences in frailty progression based on baseline frailty and measures of effective viral suppression.

Funding: T35AA21097

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[1] Maffei VJ, Ferguson TF, Brashear MM, et al. Lifetime alcohol use among persons living with HIV is associated with frailty. AIDS. 2020;34(2):245-254.