

**Introduction:** Anhedonia is a key depression symptom but there is significant heterogeneity in its severity across individuals with Major Depressive Disorder. Anhedonia likely reflects altered functioning of key brain circuits involved in the reward process but it is unclear whether increased anhedonia is associated with distinct clinical and cognitive symptoms. The purpose of this study is to examine demographic, clinical and cognitive characteristics associated with increased levels of anhedonia in older depressed adults.

**Methods:** We examined 65 currently depressed older adults aged 60 years or older. Participants completed a standardized diagnostic interview and clinical assessments, including self-report of depressive symptoms. These assessments included the Snaith-Hamilton Pleasure Scale (SHAPS) to quantify severity of anhedonia. Participants additionally completed a neuropsychological battery, including tests focused on processing speed and executive function. We examined participant characteristics associated with SHAPS score in a staged approach, first examining demographics, then clinical measures, followed by cognitive profile. Analyses controlled for overall depression severity using a modified MADRS score with the anhedonia item removed. In secondary analyses, we dichotomized sample into those with high or low SHAPS scores determined by a median split and re-analyzed the clinical and cognitive variables.

**Results:** Total SHAPS score was not associated with demographic variables, but did exhibit a positive association with modified MADRS score (Pearson correlation coefficient = 0.32,  $p = 0.0094$ ). After controlling for modified MADRS score, SHAPS score was not associated with clinical measures of perceived stress, apathy, rumination, fatigue, worry, or insomnia. However, after controlling for age and medical morbidity, it was positively associated with disability ( $F = 5.03$ ,  $p = 0.0334$ ). After controlling for modified MADRS, age, and education, higher SHAPS scores were also positively associated with better performance on the Stroop test measures of processing speed (color naming;  $F = 9.51$ ,  $p = 0.0034$ ) and executive functioning (color-word interference;  $F = 9.43$ ,  $p = 0.0035$ ). When we dichotomized the sample into high and low anhedonia groups based on the median, we replicated the above findings. We additionally observed that the high anhedonia sample was younger ( $t = 2.76$ ,  $p = 0.0083$ ) with an earlier age of initial onset of depression ( $t = 2.18$ ,  $p = 0.0332$ ).

**Conclusions:** Greater anhedonia is associated with greater overall depression severity and with greater disability. However, it is also associated with better processing speed and executive function performance as measured by the Stroop test. This apparent discrepancy may be explained by analyses of high versus low anhedonic subjects, where individuals with higher anhedonia were more likely to be younger patients and report an earlier age of first episode. We propose greater anhedonia occurs in younger, earlier onset depressed elders with perhaps a greater lifetime duration of depression or higher number of recurrent depressive episodes. This may contribute to the greater reported disability, but their age may be related to the superior cognitive test performance.

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Poster Number: EI 16

### Exploring Ketamine Use in Geriatric Patients Suffering From Treatment-Resistant Depression

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**Introduction:** Ketamine is a glutamate NMDA receptor antagonist and is commonly used as an anesthetic. Low-dose subanesthetic intravenous ketamine is fairly new and an increasingly popular treatment for treatment-resistant depression (TRD) in the adult population; however, there is a scarcity of evidence of ketamine's use among geriatric patients. Previously, psychotropics and electroconvulsive therapy (ECT) have been used in the geriatric TRD population. Ketamine provides a possible new treatment modality for those patients concerned with ECT-induced cognitive effects and may also allow for use in patients with significant cardiovascular co-morbidities, who would likely not qualify for ECT.

**Methods:** We provide a literature review on the use of ketamine for TRD in the geriatric population.

**Results:** Studies and case series have shown the use of ketamine as a monotherapy and augmented therapy with electroconvulsive therapy in the adult and geriatric population. Literature supports efficacy with monotherapy and questionable benefit from augmentative therapy. Dosing ranges from 0.2 mg/kg to 0.5 mg/kg, with evidence showing remittance with ketamine dosing less than 0.5 mg/kg. Some studies have shown cognitive protection as compared to other TRD treatment modalities, while the majority of studies have not thoroughly analyzed systemic adverse risk profiles including cognitive and cardiovascular effects.

**Conclusions:** There is evidence in the literature for the use of intravenous ketamine in the TRD geriatric population. Larger randomized control trials are needed to provide guidance regarding dosing, cognitive and systemic effects, and treatment response.

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### **Amyotrophic Lateral Sclerosis and Late Life Depression**

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**Introduction:** Amyotrophic lateral sclerosis (ALS), commonly known as Lou Gehrig's disease, is a progressive, idiopathic, and chronic neurodegenerative disease. Upper and lower motor neurons responsible for voluntary muscle control are typically affected. This neural degeneration causes spastic paralysis and muscle dystrophy which results in disability and ultimately death, typically within 2–5 years after diagnosis. The national prevalence of ALS in 2011 was 3.9 per 100,000.

White males, non-Hispanics and those between the ages of 60–69 years are the most frequently affected populations. ALS is a progressive disease associated with multiple complications including respiratory insufficiency, sialorrhea, pseudobulbar affect, sleep disruption, spasticity, fatigue, laryngospasm, autonomic symptoms, pain, and depression. Depression is a common neuropsychiatric complication of ALS. Estimated prevalences range from 6% for severe depression and 16.2–29% for mild depression. Depression may be difficult to diagnose and treat given confounding factors related to the progressive nature of the primary disease. For example, mood symptoms may be difficult to separate from disease specific physical symptoms such as fatigue and weakness. Additionally, given the progressive nature of ALS, ongoing decline in physical functioning may make mood symptoms refractory to treatment. Several new disease specific rating scales, including the ALS Depression-Inventory, exist to aid clinicians with appropriate diagnosis and help guide treatment.

**Methods:** We present a case of an elderly man diagnosed with ALS who subsequently developed depressive symptoms in the context of his neurodegenerative disease and transition to a nursing home after experiencing a fall at home rendering him unable to independently care for himself.

**Results:** As with many ALS patients, his physical decline and debilitation contributed significantly to the development of depressive symptoms. He also had difficulty accepting his need for a higher level of care.

**Conclusions:** Our patient ultimately experienced remittance of his depressive symptoms after treatment with an SSRI. In addition to highlighting pertinent aspects of this case, our poster will present a literature review discussing the evaluation, diagnosis, and treatment of patients with ALS and co-morbid major depressive disorder.

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### **Functional Activation during Emotion Processing in Late-Life Depression: Early Markers of Treatment Response**

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**Introduction:** Treatment of major depression (MDD) often requires multiple trials of medications before an effective therapy can be identified, this poses a serious issue as it is associated with an increased risk of suicide and can contribute to worsening co-morbidities. In late-life depression (LLD), as the time required to respond to a single medication is on average longer (6 weeks compared to 4) – these risks may be worsened. Several studies have shown changes in functional activation/connectivity following acute doses as measured by functional magnetic resonance imaging (fMRI). In this study, we aimed to investigate early changes in functional brain activation (during emotion reactivity) that occur during a treatment trial.

**Methods:** Late-life LLD patients (N=52) were enrolled into a 12-week Venlafaxine treatment trial where an fMRI scan was collected at baseline, 12 hours following a placebo, 12 hours following their first dose, a week after beginning treatment, and at