

178. Cognitive Profile of Obese Adults with and without a Mood Disorder

Maria R. Restivo¹, Margaret C. McKinnon¹, Benicio N. Frey¹, Geoff Hall², Valerie Taylor³

¹Neuroscience, McMaster University, Hamilton, ON, Canada, ²Psychology, McMaster University, Hamilton, ON, Canada, ³Psychiatry, University of Toronto, Toronto, ON, Canada

Background: Individuals with mood disorders (Bipolar Disorder [BD] or Major Depressive Disorder [MDD]) have a greater prevalence of risk factors for cardiovascular disease, including diabetes and hypertension. This may be in part explained by the higher proportion of individuals with obesity in mood disorder populations. While many medical co-morbidities have been shown individually to adversely affect cognition, recent research suggest that adiposity itself may have a negative impact. This study examines the impact of weight on cognition by comparing cognitive performance in obese individuals, with or without a mood disorder.

Methods: Cognitive functioning in 22 obese individuals (BMI > 35 kg/m²) with MDD, 10 with BD, and 22 obese & 20 non-obese age-matched controls were compared. Cognitive performance across various domains was assessed via a standardized battery of neuropsychological tests. Anthropomorphic measurements and self-reports assessing nutritional intake, sleep quality and mood symptomatology were also collected.

Results: One-way ANOVA between group analyses reached significance across various tests of declarative memory, attention and executive function, but failed to show group differences in measures of visual-spatial skill or intellectual functioning. A consistent trend emerged across measures that reached significance: performance means were highest for healthy controls, followed by obese controls, obese MDD patients, and finally obese BD patients.

Conclusions: Obesity may be responsible for a portion of the impaired cognitive performance often seen in mood disorder patients. It may be that the cognitive improvement expected in medicated patients is lost over time, and may actually be seen as a cognitive decline, as a consequence of the associated weight gain of medication treatment.

Keywords: Major Depressive Disorder, Bipolar Disorder, Obesity, Memory, Executive Function

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179. Investigating the Effect of Medication Load on Autobiographical Memory Specificity in Individuals Diagnosed with Major Depressive Disorder

Olivia R. Allega^{1,2}, Melissa R. Parlar^{3,4}, Geoffrey B. Hall⁵, Carolina Oremus^{2,3}, Matthew King⁶, Glenda M. MacQueen^{3,7}, Brian Levine^{8,9}, Margaret C. McKinnon^{2,3}

¹Science, McMaster University, Hamilton, ON, Canada, ²Mood Disorders Program, St. Joseph's Healthcare Hamilton, Hamilton, ON, Canada, ³Psychiatry and Behavioural Neurosciences, McMaster University, Hamilton, ON, Canada, ⁴Ingerated Neuroscience and Discovery, McMaster University, Hamilton, ON, Canada, ⁵Psychology, Neuroscience and Behaviour, McMaster University, Hamilton, ON, Canada, ⁶Clinical Psychology, Ryerson University, Toronto, ON, Canada, ⁷Psychiatry, University of Calgary, Calgary, AB, Canada, ⁸Rotman Research Institute, Baycrest Centre, Toronto, ON, Canada, ⁹Psychology and Medicine (Neurology), University of Toronto, Toronto, ON, Canada

Background: A significant body of evidence points towards autobiographical memory (AM) impairment in patients with major depressive disorder (MDD) yet the mechanisms underlying this effect remain largely unknown. The objective of the current study was to examine the association between one potential contributing factor, medication load (ML), and autobiographical memory specificity in a sample of patients with MDD.

Methods: To date, ten patients with MDD underwent an Autobiographical Interview (AI; Levine et al., 2002) evaluating retrieval of positive, negative and neutral AMs. Autobiographical details were subsequently quantified as episodic (indexing details specific in time and in place) or non-episodic (factual details unrelated to the recollected events, extraneous meta-cognitive details and repetitions). Following Sackeim (2001), a composite index of ML was calculated by summing the codes of low-dose (1) or high-dose (2) medication categories for each participant. Correlation analyses were performed on AI scores and ML.

Results: Elevated medication load was associated with increased production of extraneous non-episodic details surrounding neutral and positive AMS (Neutral AM: $\rho=0.725$, $p=0.018$; Positive AM: $\rho=0.766$, $p=0.010$). No significant associations emerged, however, between ML and episodic details associated with event re-experiencing.

Conclusions: Increases in medication load were associated with recollection of non-episodic details tangential to positive and neutral AMs surveyed. Production of non-episodic information has been associated with poor AM specificity in patients with MDD. The results of the present study point towards medication as a potential factor underlying this effect.

Keywords: autobiographical memory, depression, medication load, autobiographical interview, memory specificity

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180. Impulsivity, Anger and Poor Self-Control in Intermittent Explosive Disorder Associated with Reduced Thalamo-Cortical Structural Integrity

Rebecca N. Preston-Campbell¹, Muhammad A. Parvaz¹, Farnam Kazi², Vish Parwani², Anna B. Konova¹, Pias Malaker¹, Kristin E. Schneider¹, Rita Z. Goldstein¹, Nelly Alia-Klein¹

¹Psychiatry, Icahn School of Medicine at Mount Sinai, New York, NY,

²Undergraduate Biology, Stony Brook University, Stony Brook, NY

Background: Reactive aggression, reflected on a continuum of severity culminating with recurrent bouts of anger in intermittent explosive disorder (IED), is a risk factor for multiple public health problems. Data on potential executive dysfunction and underlying neural morphological integrity in IED are limited. We explored the interplay between trait anger, impulsivity, self-control, and gray matter volume (GMV) of associated regions.

Methods: 30 male volunteers [high-aggression (HA) = 15, comprised of IED and subthreshold-IED; matched low-aggression (LA) = 15] completed trait anger, impulsivity, and self-control questionnaires. Of these, 21 (11 HA; 10 LA) were MRI scanned to assess GMV integrity.

Results: Compared to LA, HA showed 1. elevated scores on impulsivity and poor self-control driven by IED ($p<.001$; Figure 1); 2. reduced GMV in the right inferior frontal gyrus ($t(19) = 5.568$, $p<.001$) negatively correlated with attentional impulsivity ($r=-.448$, $p=.048$); and 3. reduced left thalamus GMV ($t(19)=4.157$, $p<.001$) negatively correlated with trait anger reactivity ($r=-.540$, $p=.011$) (Figure 2).

Conclusions: Individuals with IED reported elevated anger reactivity, motor and attentional impulsivity, and poorer trait self-control, separating them from subthreshold-IED. These trait differences, coupled with inferior frontal gyrus and thalamic structural abnormalities provide neurobiological correlates of IED and the higher-order typology of reactive aggression.

Figure 1. IED show higher anger, impulsivity and lower self-control as compared to subthreshold IED and controls.

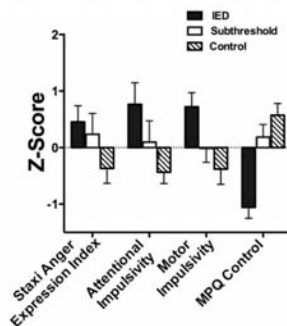
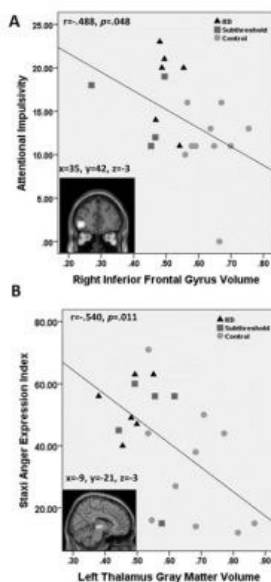


Figure 2. Correlations between A) Right IFG GMV and attentional impulsivity and B) left thalamus GMV and trait anger reactivity



Keywords: Aggression, MRI, intermittent explosive disorder, anger

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181. Brain and Behavior Response to Provocation in Intermittent Explosive Disorder

Rebecca N. Preston-Campbell¹, Joel L. Steinberg², Scott Lane³, Thomas Maloney¹, Muhammad A. Parvaz¹, Scott J. Moeller¹, Rita Z. Goldstein¹, Nelly Alia-Klein¹

¹Psychiatry, Icahn School of Medicine at Mount Sinai, New York, NY ²Psychiatry, Virginia Commonwealth University, Richmond, VA ³Psychiatry and Behavioral Sciences, University of Texas Health Science Center at Houston, Houston, TX

Background: In the typology of aggression, intermittent explosive disorder (IED) is marked by disproportionate reactivity to provocation. We explored the behavioral and fMRI response to the Point Subtraction Aggression Paradigm (PSAP), which automatically and objectively measures aggressive response to provocation.

Methods: 30 males completed aggression questionnaires and behavioral PSAP [high-aggression (HA)=15, comprised of IED=9 and subthreshold-IED=6; matched low-aggression (LA)=15]. Eighteen males [HA=9 (IED=5; subthreshold-IED=4) and LA=9] performed the functional MRI-adapted PSAP.

Results: Compared to the LA, the HA group trended towards more aggressive presses on the behavioral PSAP [$F(1, 28)=3.963, p=.056$]. Importantly, a linear contrast analysis between the three groups revealed a stepwise increase ($p=.029$), with the IED group exhibiting the highest level of initial aggression. These behavioral results were correlated with physical aggression (Figure 1; $r=.417, p=.022$). During fMRI, HA showed greater activation (whole-brain false-discovery-rate corrected two-tail cluster $p < 0.05$) in portions of the bilateral precuneus, (Figure 2) during aggressive responding (relative to non-aggressive monetary responding) compared to controls.

Conclusions: PSAP-measured aggressive responding was elevated in individuals with IED. Hyper-reactivity of the precuneus during aggressive responding could provide insight into the neural underpinnings of reactive aggression.

Figure 1: A) Change in behavioral PSAP aggressive response relative to the number of provocations as a function of group shown over three trials within a single session. B) Correlations shown by group between physical aggression and aggressive responding on the PSAP.

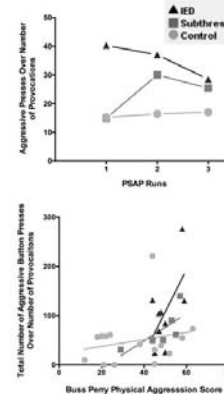
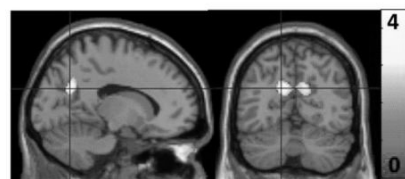


Figure 2. HA compared to LA during fMRI PSAP in aggressive, retaliatory responding, relative to non-aggressive responding (whole-brain false-discovery-rate [FDR] corrected two-tail cluster $p < 0.05$), maximal voxel $t(df 16) = 4.46$ at MNI $x=-14, y=-64, z=26$



Keywords: aggression, fMRI, intermittent explosive disorder, anger

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