Original Research Article

# Assessment of cognition in obese young adults

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## Abstract

**Background:** Obesity is abnormal or excessive accumulation of fat that has adverse effects on health. Obese individuals are at risk of several life threatening diseases and complications such as Type-2 Diabetes Mellitus, Hypertension, Metabolic syndrome, Cardiovascular diseases, Stroke, Cancers, and Psychosocial disorders. Apart from these, it also leads to neurological disorders like impairment in cognition, motor skills and higher executive functions. **Aim:** To assess the cognition in young adult obese males. **Materials and methods**: 80 male participants of which 40 obese males and 40 normal weight healthy subjects in the age group of 18 to 35 years were recruited from the Non Communicable Disease Outpatient Department, Body Mass Index(BMI),Waist Circumference(WC) and Waist Hip Ratio(WHR) were the obesity indices used to assess the obesity. Montréal Cognitive Assessment Score (MoCA score) was used to assess the cognition. **Results**: The data was analysed using Statistical Package for Social Sciences(SPSS) version 20. The study group were with the mean age of 34.02 $\pm$ 3.20 yrs, mean BMI of 32.06 $\pm$3.47,mean WHR of 0.86 $\pm$ 0.08, mean WC of 98.37 $\pm$5.35 and mean MoCA score of 26.09$\pm$0.49.Among the study group 28% of the participants had cognitive impairment with the score between 23-25. Negative correlation was observed between the obesity indices and MoCA score, among which BMI had better negative correlation with the MoCA score. **Conclusion**: Cognition was impaired in obese individuals and it was inversely related to the obesity indices. This shows that they are at the risk for early onset of dementia. Hence, early diagnosis and appropriate interventions may prevent severe impairment in cognition in obese individuals.

**Key words:** obesity, cognition, dementia, metabolic syndrome

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## Introduction

Obesity and overweight are defined as abnormal or excessive fat accumulation that may impair the health of the individual.Overweight and obesity result from an energy imbalance that occurs due to over eating of too much of calories and inadequate physical activity to use up those calories.

Obesity is associated with various comorbidities such as metabolic syndrome, glucose intolerance,

hyper-lipidemia, hypertension and hence it can lead to the development of Type-2 diabetes mellitus, coronary artery disease, heart failure,respiratory diseases such as obstructive sleep apnoea, gastrointestinal, and musculoskeletal disorders, thromboembolism, stroke and cancer.[1]

In addition, many studies have shown that obesity is associated with cognition impairment and early onset of dementia. Neuroinflammation,vascular inflammation, altered metabolites are the causal mechanism for cognitive decline in obesity. The ongoing inflammatory process in obesity couldhave been triggered by multiple factors such as insulin resistance, leptin dysregulation, altered gut brain axis and enhanced systemic mediators of inflammation.[2]

Recent evidences show that consumption of high fat diet and unbalanced diet alters the gut brain axis,leading to impaired cognition. The bacterial lipopolysaccharide (LPS) is a pro-infammatory neurotoxin released from the gut microbiome. It crosses the physiological barrier to access the hippocampus, leading to cognitive impairments,such as memory disturbances.[3]

The incidence of obesity is increasing among the younger age group. Mild cognition impairment that occurs at the early stage in obese individuals remain unnoticed and it may lead to severe cognitive decline as well as early onset of dementia. Most of the studies showed cognitive impairment in obese individuals aged above 45yrs.

Hence, inthis study we have taken younger age group to provide an insight into the earlier onset of impairment in cognitionin obese individuals. This shall pave way for early diagnosis and prompt interventions in order to prevent further impairment in cognition and a better quality of life.

**Aim**

To evaluate cognition in obese males in comparison with the control group.

**Objectives**

1. To assess cognition by using Montreal Cognitive Assessment (MoCA) Score in all the participants.

2. To measure the waist circumference, hip circumference and to calculate Body Mass Index andWaist Hip Ratio in them.

3. To find out the association between obesity indices and MoCA score in obese males.

**Materials and methods**

This is an analytical cross-sectionalstudy. Institutional ethical committee clearance was obtained. 40 obese males were recruited, based on the revised consensus guidelines for India (Normal weight - BMI 18 - 22.9, Overweight - BMI 23 - 24.9, Obese - BMI > 25)[4] from Non communicable disease outpatient department. Inclusion criteria were obese males (BMI > 25) of age 18 -35 years with minimum educational qualification$^{8th}$ standard.

Exclusion criteria included subjects with established diagnosis of dementia, co-morbid conditions that affect cognitive function such as neurological disorders, psychiatric disorders, history of acute / chronic illness, patients on any other chronic medications for and those with history of smoking, tobacco and alcohol abuse. 40 apparently healthy, age matched and gender matched subjects were included in the control group. All the subjects were explained about the study and written informed consent was obtained from each of them. A brief history was taken and clinical examination was done. The height was measured in metres (m) on barefoot using stadiometer and weight inkilograms by

using a standardized weighing machine for all the participants.Body Mass Index was calculated using Quetelet's Index (Weight / Height$^2$). Their waist circumference was measured in centimetres at the level of umbilicus or midway between the lower ribcage and pubic symphysis. Hip circumference was measured as widest circumference at the level of greater trochanter using inch tape. Then Waist-Hip Ratio was calculated using waist and hip circumference.

Cognitive function of all the participants was assessed by Montreal Cognitive Assessment Score (MoCA score). It includes several questions grouped into seven categories, representing different cognitive domains such as Orientation, Delayed Recall, Language, Abstraction, Attention, Naming and Visuospatial. Duration of the test was 10 minutes. The subjects were made comfortable and scoring for each question was done immediately on asking it in an orderly listed way.

**Table -1 Comparison of age, obesity indices and MoCA scores between obese and control group.**

| S.No. | Variable | Control group (n=40) Mean±S.D | Obese group (n=40) mean ±S.D | p value |
|---|---|---|---|---|
| 1. | Age (years) | 33.04±2.43 | 34.02±3.20 | 0.72 |
| 2. | Obesity Indices BMI (Kg/m$^2$)<br>WHR<br>WC (cm) | 22.33 ± 1.07<br>0.83 ± 0.43<br>78.34 ± 3.52 | 32.06 ±3.47<br>0.86 ± 0.08<br>98.37 ± 5.35 | 0.04*<br>0.53<br>0.02* |
| 3. | MoCA score<br>1. Orientation (6)<br>2. Naming (3)<br>3. Attention (6)<br>4. Delayed recall (5)<br>5. Language (3)<br>6. Visuospatial (5)<br>7. Abstraction (2)<br>Total (30) | <br>5.91 ± 0.12<br>2.88 ± 0.23<br>5.7 ± 0.61<br>4.85 ± 0.47<br>2.76 ± 0.32<br>4.98 ± 0.41<br>1.83 ± 0.39<br>28.91 ± 0.36 | <br>5.62 ± 0.49<br>2.84 ± 0.37<br>5.30 ± 0.76<br>3.88 ± 0.85<br>2.60 ± 0.49<br>4.76 ± 0.43<br>1.72 ± 0.39<br>26.09 ± 0.49 | <br>0.46<br>0.82<br>0.75<br>0.3<br>0.86<br>0.5<br>0.64<br>0.8 |

*p value < 0.05 significant, MoCA score - Montreal Cognitive Assessment Score.

The maximum total score is 30 and the score of 26 or above is considered as normal. The data obtained were analyzed using SPSS, version 20.An Independent Student's t-test was used to compare the means of variables. Pearson's test was used to find out the correlation between the variables,p value < 0.05 was considered significant.Correlation between obesity indices and MoCA score is given in Table-2. A significant

negative correlation was observed between BMI, WC and MoCA score. Though negative correlation was observed between WHR and MoCA score, it was not statistically significant. Among the obesity indices, BMI was found to have greater negative correlation with the MoCA score than WHR and WC.

Among the 40 obese males, 11 persons (28%) were found to have MOCA score between 23-25. Though 29 persons had normal MoCA scoring, there was a significant reduction in the scores of delayed recall domain and attention domain among them.

The distribution of cognitive impairment among the study population is shown in Fig-1

**Figure -1 Distribution of cognitive impairment among the obese group.**

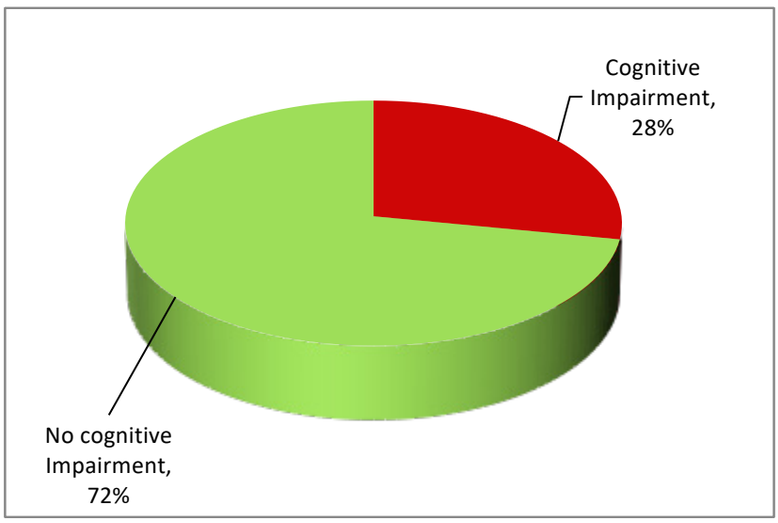


**Table-2 Correlation between obesity indices and MoCA score in the obese group.**

| Obesity indices | Correlation co-efficient ( r value) | p value |
|---|---|---|
| 1.Body Mass Index (BMI) (Kg/$m^2$) | -0.69 | 0.03* |
| 2.Waist Hip Ratio (WHR) | -0.03 | 0.49 |
| 3.Waist Circumference (WC) ( Cm) | -0.63 | 0.05* |

*p value < 0.05 significant

**Results**

Out of the total 80 participants, 40 were obese and 40 were normal weight subjects. Data are represented as mean±standard deviation. Table-1 shows the comparison of age, obesity indices and MoCA scores between obese and control groups. It shows that the mean age of the obese and control groups were 34.02 yearsand 33.04 years. Both groups were age matched. There was a significant difference(p value <0.05) in the mean levels of BMI and Waist Circumference between obese and control groups.WHR was higher in obese subjects when compared to controls which were not statistically significant. There was a slight decline in the mean MoCA score in the obese groupin comparison with control group which was not statistically significant.While considering the scores of individual domains, there was decline in scores of attention and delayed recall among the obese group but not statistically significant.

**Discussion**

In our study,80 participants were recruited. Obesity indices such as waist circumference, hip circumference were measured, then BMI and Waist-Hip ratio (WHR) were calculated. Cognition assessment was done using MoCA score. Kannaiyiram et al reported that MoCA appears to be a better screening test than MMSE for screening and diagnosing mild cognitive impairment.[5]

The mean age of our study group was 34.02 years.Most of the studies demonstrated cognitive decline among obese and overweight individuals in the age group of 40 to 55 years.[6]This age group was selected in our study to demonstrate early onset of cognitive impairment in obese subjects. Also only obese males without any co morbidities were recruited in order to prove that obesity is an independent risk factor for cognitive dysfunction.

The mean MoCA score was reduced in obese males when compared tothat of normal subjects which was not statistically significant. The overall prevalence of cognitive impairment among obese subjects was 28%. The lower prevalence observed in our study could be due to smaller sample size**.**We observed agreater decline in the scores of attention and delayed recall which were consistent with the results obtained by John Gunstad et al. John Gunstad et al showed that obesity is linked to reduced cognitive function,with more impact on attention and executive function.[7]Studies also have shown diminished performance on various measures of cognition such as executive function, motor speed, short term memory among the obese individuals.[8,9]

In our study, we obtained a negative correlation between BMI, WC, WHR and MoCA Score. Among the obesity indices, BMI was found to have greater negative correlation with the MoCA score than WHR and WC. Many Studies have shown that both BMI and WC were inversely related to cognitive decline.[10] Few studies have shown similar findings like that of our study.[11] On the contrary, WC was found to have better negative correlation than BMI in other studies.[12]

The probable cause for cognitive decline noted in obese individuals could be the following. The leptin receptor mRNA are highly expressed in brain regions including hippocampus, amygdala, brain stem, cerebellum and substantia nigra. It regulates the higher functions in those regions. Leptin modulates synaptic functions, neuronal excitability in hippocampus and enhances learning and memory.[13]Chen et al revealed

elevated levels of leptin and C-reactive protein (CRP) in obesity.Elevated CRP binds to leptin and prevents it spending to its receptor attributing to leptin resistance, resulting in cognitive decline in obese individuals.[14]

The inflammatory mediators such as C-reactive protein, IL-6 and visceral adipocyte derived factors are elevated in obesity. They may be neurotoxic causing slow deterioration of the higher functions of CNS leading to dementia later in life.[15]In another study, Raji *et al* reported that BMI>30kg/$m^2$ was associated with atrophy in frontal lobes, anterior cingulate gyrus, hippocampus and thalamus as compared to normal weight individuals, leading to deterioration of higher functions.[16]In addition to the above factors, deficiency of micronutrients such as , zinc, biotin, iodine , magnesium, vitamin B-12 , vitamin D and antioxidant vitamin C, vitamin E were observed in obese and were found to be associated with cognitive decline.[17,18]Since obesity is associated with cognitive decline which remains unnoticed, our study mainly focused on early identification of cognitive decline in young obese adults.Moreover,cognitive impairment is reversible with early intervention like life-style modifications, health education, increased physical activity, meditation.[19] Increasing physical activity is simple, beneficial, which can reverse the mild cognitive decline as well as other neurological disturbances. It is the best treatment until the development of therapeutic options to treat cognitive deficits or to prevent cognitive decline in obesity are available.[20]

**Conclusion**

Our study concludes that the young obese individuals do have mild impairment in cognition which was assessed by MoCA scoring even at the early stages. Obesity in later life may lead to severe impairment in cognition and progressive dementia. Hence early interventions such as life style modifications, increased physical activity along with micronutrient supplementation can reverse this impairment in cognition.

**Limitations**

Our present study was conducted in smaller sample size and only among males. Significant cognition impairment could have been demonstrated if women were included in the study. Age group included in our study was between 18 to 35 years, wide range in selection of age group including the middle age group could be done in future studies for assessing the impairment of cognition.

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**Conflict of interest:** Nil

**References**

1.Whitmar R.A, Gustafan D.R, Barret Connar, Haan M.N, (2008) Central obesity and increased risk of dementia more than three decades later.*Neurology*71(14),1057-1064.

2.Solas M, Milagro FI, Ramírez MJ, Martínez JA. Inflammation and gut-brain axis link obesity to cognitive dysfunction:plausible pharmacological interventions. CurrOpinPharmacol. 2017 Dec;37:87-92.

3. Zhao Y, Jaber V, Lukiw WJ. Secretory products of the human GI tract microbiome and their potential impact on Alzheimer's disease (AD): detection of lipopolysaccharide (LPS) in AD hippocampus. Frontiers in cellular and infection microbiology. 2017 Jul 11;7:318.

4. Misra A, Chowbey P, Makkar BM, Vikram NK, Wasir JS, Chadha D, et al. Consensus Statement for Diagnosis of Obesity, Abdominal Obesity and the Metabolic Syndrome for Asian Indians and Recommendations for Physical Activity, Medical and Surgical Management. J Assoc Physicians India. 2009;57:163–70.

5.Alagiakrishnan K, Zhao N, Mereu L, Senior P, Senthilselvan A. Montreal Cognitive Assessment is superior to Standardized Mini-Mental Status Exam in detecting mild cognitive impairment in the middle-aged and elderly patients with type 2 diabetes mellitus. BioMed research international. 2013 Oct;2013.

6.Hassing LB, Dahl AK, Thorvaldsson V, Berg S, Gatz M, Pedersen NL, Johansson B. Overweight in midlife and risk of dementia: a 40-year follow-up study. International journal of obesity. 2009 Aug;33(8):893-8.

7.Gunstad J, Paul RH, Cohen RA, Tate DF, Spitznagel MB, Gordon E. Elevated body mass index is associated with executive dysfunction in otherwise healthy adults. Comprehensive psychiatry. 2007 Jan 1;48(1):57-61.

8.Waldstein SR, Katzel LI. Interactive relations of central versus total obesity and blood pressure to cognitive function. International journal of obesity. 2006 Jan;30(1):201-7.

9.Cournot M, Marquié JC, Ansiau D, Martinaud C, Fonds H, Ferrières J, Ruidavets JB. Relation between body mass index and cognitive function in healthy middle-aged men and women. Neurology. 2006 Oct 10;67(7):1208-14.

10.Hermsdorff HH, Zulet MA, Puchau B, Martínez JA. Central adiposity rather than total adiposity measurements are specifically involved in the inflammatory status from healthy young adults. Inflammation. 2011 Jun;34(3):161-70.

11.Jeong SK, Nam HS, Son MH, Son EJ, Cho KH. Interactive effect of obesity indexes on cognition. Dementia and geriatric cognitive disorders. 2005;19(2-3):91-6.

12.West RK, Ravona-Springer R, Heymann A, Schmeidler J, Leroith D, Koifman K, D'Arcy RC, Song X, Guerrero-Berroa E, Preiss R, Hoffman H. Waist circumference is correlated with poorer cognition in elderly type 2 diabetes women. Alzheimer's & Dementia. 2016 Aug;12(8):925-9.

13.Mercer J. G., Hoggard N., Williams L. M., Lawrence C. B., Hannah L. T. and Trayhurn P. (1996) Localization of leptin receptor mRNA and the long form splice variant (Ob-Rb) in mouse hypothalamus and adjacent brain regions by in situ hybridization. FEBS Lett. 387, 113–116.

14.Chen K, Li F, Li J, et al. Induction of leptin resistance through direct interaction of C-reactive protein with leptin. Nat Med 2006;12:425–432.

15.Wardle J, Cooke L. The impact of obesity on psychological well-being. Best Pract Res Clin Endocrinol Metab. 2005 Sep;19(3):421-40.

16. Raji CA, Ho AJ, Parikshak NN, Becker JT, Lopez OL, Kuller LH, et al. Brain structure and obesity. Hum Brain Mapp. 2010;31:353–64.

17.Wasantwisut E. Nutrition and development: other micronutrients' effect on growth and cognition. The Southeast Asian journal of tropical medicine and public health. 1997 Jan 1;28:78-82.

18.Kaidar-Person O, Person B, Szomstein S, Rosenthal RJ. Nutritional deficiencies in morbidly obese patients: a new form of malnutrition?. Obesity surgery. 2008 Jul 1;18(7):870-6.

19.Lucke J, Partridge B. Towards a smart population: a public health framework for cognitive enhancement. Neuroethics. 2013 Aug 1;6(2):419-27.

20.Loprinzi PD, Herod SM, Cardinal BJ, Noakes TD. Physical activity and the brain: a review of this dynamic, bi-directional relationship. Brain Research. 2013 Nov 20;1539:95-104