UiT The Arctic University of Norway

# Body mass index and cognitive function in community-living elderly men and women: The Tromsø Study.

Ida J. E. Ylvisaker[1*]; Jan-Magnus Kvamme[1,2], MD, PhD; and Torgeir A. Engstad[1], MD, PhD.

[1]Department of Clinical Medicine, UiT – The Arctic University of Norway
[2]University Hospital of North Norway (UNN), Tromsø
*Corresponding author: Ida J. E. Ylvisaker, ijy022@post.uit.no

MED-3910: Prosjektoppgave forskerlinja, april 2020.

**Body mass index and cognitive function in community-living elderly men and women: The Tromsø Study.**


## Abstract

**Background:** Previous studies have reported conflicting results regarding the relationship between cognitive function and nutritional status in the elderly. The primary objective of this study was to investigate the associations between body mass index (BMI) and cognitive function in a sample of community-living elderly men and women.

**Methods:** This population-based study comprised data from 1148 men and 1344 women aged 65 years or older from the municipality of Tromsø, Norway. The data was collected from the 5th survey of the Tromsø Study. A cross-sectional design was used to examine the associations between nutritional status and cognitive function. BMI was categorized into six groups (≤19.99, 20.00-22.49, 22.50-24.99, 25.00-27.49, 27.50-29.99 and ≥30 kg/m2). Cognitive function was measured by use of the 12-word test, part one (immediate recall) and part two (short-term delayed recall), the digit symbol coding test, and the finger tapping test with dominant and non-dominant hand. The statistical associations between BMI and cognitive function were assessed using multiple logistic regression models. The data was adjusted for age, sex, educational level, co-morbidity, alcohol use and smoking habits, factors known to be associated with both BMI and cognitive function.

**Results:** A BMI ≥30 kg/m2 was associated with a reduced score on the finger tapping test in the non-dominant hand (OR: 1.53, 95% CI: 1.11-2.10, p= 0.009). A BMI of ≤22.49 kg/m2 was associated with a reduced score on the short-term delayed recall test (OR: 1.54, 95% CI: 1.07-2.23, p=0.020). For the other BMI categories and cognitive test scores there were no significant associations.

**Conclusion:** Overall, there were no statistically significant associations between underweight, overweight or obesity and cognitive function in this study, also when adjusting for age, sex and confounding factors. An obese subgroup showed reduced performance on the finger tapping test, whereas an underweight subgroup performed poorer on the short-term delayed recall test.



**Keywords:** elderly, body mass index, BMI, nutrition, nutritional status, cognitive function, cognition, cognitive decline, cognitive tests**.**

**Background**

The association between nutritional status and risk of cognitive decline in the elderly has been investigated in a number of epidemiological studies with conflicting results (1-4). In a systematic review and meta-analysis from 2016 (1), 16 studies reported on late-life overweight/obesity and incident dementia. Of these, seven reported on overweight; three of them found that being overweight lowered the risk of incident dementia, while no significant associations were reported by the remaining four studies. Six studies reported on obesity; one study found that being obese increased the risk of incident dementia, two studies reported that obesity reduced the risk, while three of the six reported a non-significant association. Four studies reporting on obesity were suitable for inclusion in the meta-analyses and yielded a statistically significant relative risk between BMI and cognitive decline (RR: 0.83, 95% CI: 0.74-0.94). The corresponding risk estimate for cognitive impairment among subjects in the overweight group, based on the five studies included, was almost significant (RR: 0.88, 95% CI: 0.76-1.02).

Studies reporting on underweight and risk of cognitive decline are fewer in number than those reporting on overweight or obesity. A review by *Emmerzaal et al.* (2) briefly discusses this problem. They presented five studies on underweight and risk of cognitive decline among the elderly. Three of the included studies reported that the underweight group had the highest risk of incident dementia, while two studies reported no difference in risk of dementia between the underweight and the normal weight participants.

The inconsistent results from previous studies may be explained by differences in age, inclusion and exclusion criteria, study design and duration of follow-up time (2, 5). Another source of discrepancy between results could be the way BMI has been modelled. Some studies treat BMI as a continuous variable, while others report BMI as a categorical variable with varying definitions of subgroups. How BMI is defined (quantiles or classical categories) and how BMI is measured (self-reported versus weighing) may also contribute to the variation in results (2, 6). Cognitive function has been evaluated by different cognitive tests and this makes comparison between studies difficult. Some studies measure cognition only by screening with the mini-mental state examination (or a modified version of the MMSE), while others use a

combination of different cognitive assessment tools (1). Numerous variables and confounders may interact with, and influence, the association between BMI and cognition. Inadequate standardization and weaknesses in study design may also contribute to the heterogeneous results reported on the relation between nutrition and cognition.

The present study is based on the 5th survey of the Tromsø study which has a number of unique characteristics including a high attendance rate, information regarding a wide range of medical conditions and lifestyle variables and the use of three validated cognitive tests. This makes the database suitable to explore the association between nutritional status and cognitive decline.

## Methods

### Study population and design

Participants were recruited from the 5th wave of the Tromsø Study, a single center, population-based longitudinal study with repeated health surveys conducted in the municipality of Tromsø, Norway. The 5th survey of the population was carried out in 2001-2002 and had a cross-sectional design (7, 8)

Our study sample is restricted to participants aged 65 years and older. A total of 4022 men and women in this age group were invited, and 3479 completed the survey, providing an overall attendance rate of 86.5 %, women 85 % and men 88 %, respectively. Participants with missing values for weight and/or height, and who were not willing to participate in research were excluded. In the analysis only participants who completed all cognitive tests were included. The final study sample is described in table 1 and included 1344 women (53.9 %) and 1148 men (46.1 %), with a mean age of 72.0 (4.9) and 71.6 (4.7) years, respectively.

The Tromsø Study has received recommendations from the Regional Committee for Medical Research Ethics and has been approved by the Data Inspectorate and the Norwegian Directorate of Health. All subjects gave written consent to participate, with the option to withdraw at any point (9).

**Measurements**

Information on baseline characteristics was collected by self-administered questionnaires and physical examination at a single time-point.

**Body Mass Index**

At the research centers height (cm) and weight (kg) were measured, to the nearest decimal, with participants wearing light clothing and no shoes. BMI was calculated as weight (kg) divided by height (m) squared. Based on BMI, the participants were categorized into six groups (≤19.99, 20.00-22.49, 22.50-24.99, 25.00-27.49, 27.50-29.99 and ≥30 kg/m$^2$), thereby including the definitions of overweight (25.00-29.99 kg/m$^2$) and obesity (≥30 kg/m$^2$) as given by the World Health Organization (WHO) (10). The subdivision of the lower and higher WHO categories made it possible to discriminate further between subgroups of participants.

**Cognitive measures**

Cognitive function was assessed by use of three different tests: the 12-word test, the digit symbol coding test and the finger tapping test.

The 12-word test, part one and two, measures verbal episodic memory, and is a modified version of the California Verbal Learning Test. Part one tests short-term verbal memory with immediate free recall of 12 nouns, presented verbally and visually. One point is given for each correctly recalled noun. In part two, after a five-minute delay, the participants are encouraged to identify the 12 words among a total of 24. This test is referred to as short-term delayed recall. A total score ($\delta$-value) is estimated by use of an algorithm based on correct or incorrect answers (11).

The digit symbol coding test is a part of the Wechsler Adult Intelligence Scale (WAIS) and measures cognitive domains such as attention, motor speed, visuo-perceptual functions and executive functions like planning, strategizing, performing and controlling. The test consists of rows of small, blank squares, each paired with a randomly assigned number from one to nine, and with a printed key above each row that paired each number with a different nonsense symbol. After a practice trail with the first seven numbers, the subjects were asked to consecutively fill in as many as possible of the blank spaces with the corresponding symbol, as fast and precise as

possible. The number of correctly coded symbols within a given time limit constitutes the score (12).

The computerized finger tapping test measures motor speed and motor control and is sensitive for brain damage, including subtle ones (13). Also, the finger tapping performance is impaired in populations with Alzheimer´s disease (14). The participant taps as many times as possible for 10 seconds with the index finger alternating between the dominant and the non-dominant hand. The number of taps are registered by a computer, and repeated four times for each hand, calculating the mean of the last three trials for the final result (15).

The three cognitive tests represent validated tools widely used in the assessment of cognitive function (16). However, there are no established cut-off values indicating cognitive decline in any of these tests. We defined the lower 25$^{th}$ percentile of the various cognitive test scores as the cut-off indicating lower cognitive function.

**Other covariates**

Information concerning education, marital status, smoking, alcohol, physical activity and medical conditions were obtained from self-administered questionnaires.

Marital status was dichotomized into single living (single, widow/widower, divorced, separated) and married (married, registered partnership).

Education was categorized into five groups: primary school (1-6 years), lower secondary school (7-9 years), upper secondary school (10-12 years), college/university less than 4 years (13-15 years) and college/university for 4 or more years (15 or more years).

Smoking was divided into three groups; never, previously and current.
Alcohol consumption during the last year was divided into five categories. Teetotalers and participants not drinking alcohol during the last year were defined as never. Alcohol intake a few times a year and once per month were defined as rarely and 2-3 times per month and once per week were defined as monthly. The last two categories being 2-3 times and 4-7 times per week (table 2). This five-category variable was then

dichotomized into weekly (2-3 times per week and 4-7 times per week) and seldom (never, rarely, monthly) for the regression analyses.

A history of antihypertensive medication was dichotomized into currently or previously/never. A previous myocardial infarction and/or prevalent angina were combined into a new variable called coronary heart disease (CHD) for the multivariate regression analyses but kept as separate entities in table 2 (baseline characteristics) as it gives a fuller description of the medical conditions of the participants. A history of diabetes or stroke were also included as cofactors.

**Statistical analyses**

The differences in baseline characteristics between men and women were assessed by the use of the chi-square test and independent sample t-test. Continuous variables are presented as means (standard deviations (SD)) and categorical variables are presented as numbers of observations (percentages of observations).

The relationship between nutritional status and cognition was assessed in a logistic regression model. Data from men and women were pooled due to the low numbers in some BMI groups in sex-stratified analyses. The different cognitive tests were treated as dependent variables and the BMI categories as independent variables. The BMI category 25.00-27.49 kg/m$^2$ was chosen as reference group. The estimates were adjusted for age, sex, educational level, smoking habits, alcohol consumption, hypertension, diabetes, coronary heart disease (CHD) and stroke. These potential confounders were entered into the model giving an adjusted odds ratio (OR) with corresponding 95 % confidence interval (CI) for performing under the 25$^{th}$ percentile across BMI categories.

The analyses were performed using SPSS statistical software version 26 (SPSS Inc., Chicago, Illinois, USA).

**Results**

Baseline characteristics of the study population including 1148 men and 1344 women are described in table 2. The mean age was 71.6 years for men and 72.0 for women. Men had a significantly higher educational level than women. There was a higher

proportion of women both in the underweight and the obese BMI group. Approximately two thirds of the study population were overweight or obese. There were statistically significant gender differences in most variables with the exception of diabetes, stroke and hypertension, with p-values of 0.28, 0.39 and 0.64 respectively.

The relationship between BMI categories and cognitive tests are described in table 3 and table 4. Overall, there were no significant associations between the various BMI categories and lower cognitive test scores. However, a BMI ≥30 kg/m2 was significantly associated with a reduced score on the finger tapping test for the non-dominant hand in model 1 (OR: 1.67, 95% CI: 1.23-2.27, p=0.001), and in model 2 after adjusting for all potential cofounders (OR: 1.53, 95% CI: 1.11-2.10, p=0.009).

The two lowest BMI groups, pooled into one (BMI ≤22.49 kg/m2), were significantly associated with a reduced score on the short-term delayed recall test in model 1 (OR: 1.55, 95% CI: 1.09-2.21, p=0.015), and also in the multivariate adjusted model 2 (OR: 1.54, 95% CI: 1.07-2.23, p=0.020).

As separate groups, a BMI of 20-22.49 kg/m2 was significantly associated with a reduced score on the short-term delayed recall test in model 1 (OR: 1.59, 95% CI 1.08-2.34, p=0.019) and remained so in the multivariate model 2 (OR: 1.59, 95% CI: 1.07-2.37, p=0.022). However, in the lowest underweight group (BMI ≤19.99) this association turned insignificant, probably due to the small number of participants in this group (table 3).

The finger tapping test for the dominant hand was associated with a statistically significant reduced score in the obese group (BMI ≥30 kg/m2) in model 1 (OR: 1.38, 95% CI: 1.09-1.88, p=0.038), but turned insignificant in the multivariate adjusted model (table 4).

Besides this, there were no significant associations between BMI and lower cognitive function (table 4).

**Discussion**

In the present study, there was no overall statistically significant association between BMI and lower cognitive test scores indicating cognitive decline.

One could argue that BMI as a single measure of nutritional status is inadequate. BMI is gender, race and age specific, and does not give information on body fat distribution and body composition (17, 18). Aging is associated with decline in stature, decreased bone mineral density, loss of muscle mass and altered fat distribution (19, 20). All of these are known risk factors for age-related diseases (19). Due to physical changes related to ageing and illness, malnutrition and reduced muscle mass may be present in the elderly although their BMI falls into the overweight categories (21). The use of BMI as the only anthropometric measure may, in part, contribute to the discrepancy in results reported in the earlier studies.

In the present study only cross-sectional data on BMI and cognition were used. It is possible that prospective data may give more reliable results. This is supported by recent studies which have investigated the possibility of a longitudinal link between BMI and cognition in late-life (17). This has led to the theory of the obesity paradox, where obesity in mid-life is associated with a higher risk of incident dementia, whereas late-life obesity reduces the risk (1). What is considered a risk factor at one point may very well change during a life-course, i.e. *the obesity paradox*.
BMI as a risk factor for cognitive decline should be considered over time, preferably in a life-time perspective (22).

The different profiles of cognitive deficits in the underweight and overweight group may reflect the cognitive domains that are affected in these two groups. To speculate, underweight and overweight patients could have different neuroanatomical localization of their brain dysfunction. The different cognitive profiles between the weight groups may be a reflection of this.

Interestingly, the performance on the finger tapping test was significantly reduced in the obese BMI group (BMI $\geq$30 kg/m$_2$) and remained so when adjusted for possible confounders. This finding is consistent with previous studies claiming that obesity is associated with cognitive deterioration in the elderly (23, 24). A possible mechanism

for reduced performance on finger tapping could be a default brain network in obese individuals. *Tregellas et al.* (25) argue that the function of this brain network, which is the main contributor to neuronal activity, is altered in obese individuals. *Christianson et al.* (26) argues in a study from 2004 that although motor functioning is controlled by many areas of the brain, the motor strip rostral to the central sulcus is most important. Furthermore, *Hugdahl et al.* (27) has reported on a corresponding extrinsic mode network interacting with the internal one, making this sophisticated interplay even more vulnerable to minor brain damage. It is possible that motor speed and function, as measured by finger tapping, could be most sensitive for minor brain dysfunction related to obesity. On the other hand, it is hard to explain why the performance on digit symbol coding test, which is mapping attention, motor speed and visuo-perceptual function, was unchanged in the obese BMI group (table 4). It is reasonable to assume that this test, reflecting speed and quality of the interplay between various brain regions, also should be sensitive to minor brain damage. It is well known that obesity is related to brain damage due to a history of diabetes, hypertension and coronary heart disease (28) (see table 2). Previous studies have found that the finger tapping test is very sensitive for cognitive dysfunctions following a history of stroke (29, 30).

When the two lowest BMI groups were pooled into one (BMI $\leq$22.49 kg/m$_2$), this group performed significantly lower on the short-term delayed recall test compared to the reference group (BMI 22.50-24.99 kg/m$_2$) in both models. Although the medical literature reporting on the association between underweight and delayed recall in the elderly is sparse, a study from Shanghai claims that delayed recall, long term as well as short term, is the most sensitive measure for the early diagnosis of Alzheimer´s disease (AD), and a predictor of conversion from mild cognitive impairment to AD (31). Another study from Seoul reports that underweight is a marker for identifying individuals in increased risk of AD in mild cognitive impairment (32). In the Canadian population-based study on elderly above 65 years (n=13176) the cognitive profile among those with underweight was characterized by low scores on semantic fluency (recalling as many names as possible from a specified category in one minute) and processing speed, both indicators of executive dysfunction (33). Deficits in cued delayed recall are suggestive of temporo-limbic amnesia and is an indicator of preclinical AD (34). Although there is no data providing evidence for an underlying

mild cognitive impairment or early AD in our underweight sub-group, such a hypothesis should not be rejected.

The causes for underweight in our lowest BMI groups may include malnutrition, starvation, sarcopenia, malignancies and even cachexia. Among these possible causes, sarcopenia is of particular interest. *Huang et al.* (35) reported that sarcopenia is significantly associated with impaired verbal fluency.

**Strengths and weaknesses**

This study has several limitations. First, the cross-sectional study design, from which one can only make assumptions based on measurements from a single point in time. Thus, no cause-effect conclusions can be drawn. A longitudinal design would have made it possible to say something about change in BMI and cognition over a time-period. Secondly, although widely used in epidemiological research, there is always a possibility of recall bias and social desirability bias when relying on self-reported data (36). Information regarding medical conditions and medication use were also self-reported and not validated through review of medical records. Third, to attend the Tromsø study, the participants had to be healthy and mobile enough to physically go to the study center. Due to this, institutionalized people, although invited, may not have been able to participate. Attendance rate among people with cognitive decline/dementia was probably low. Hence the study population reflects this selection bias, and the results are not necessarily a true representation of the population of elderly individuals. There were few participants in the underweight group and not enough participants to do sex and age-group stratified analyses across the six BMI categories.

Despite these limitations, there are several strengths to this study. Most of all, the large sample size and relatively low number of missing data in each variable included in the analyses. Furthermore, the cognitive tests used are validated tools and are all sensitive for measuring cognitive decline. The study population was relatively homogenous in terms of ethnicity being mainly Caucasians. In addition, the mean age was high, as was the attendance rate even among the oldest old. Finally, the results were adjusted for a wide range of potential confounders.

## Conclusion

In this study, no overall significant association was found between cognitive function and underweight, overweight or obesity compared to normal BMI. This conclusion remained unchanged when multiadjusting for potential confounding factors. This may be a reflection of the many interactions between known and unknown confounding factors which could influence cognitive test performance.

## Acknowledgements

This study was supported by the University of Tromsø – the Arctic University of Norway, through The Medical Student Research Program and funded by a Scholarship from Tom Wilhelmsen's Foundation. We would like to thank the foundation for making this study possible, as well as professor emeritus Rolf Seljelid who encouraged us to submit an application for funding and was available for advice.

## Disclosure statement

The authors have nothing to disclose.

## References


1. Pedditzi E, Peters R, Beckett N. The risk of overweight/obesity in mid-life and late life for the development of dementia: a systematic review and meta-analysis of longitudinal studies. Age Ageing. 2016;Jan;45(1):14-21.
2. Emmerzaal TL, Kiliaan AJ, Gustafson DR. 2003-2013: a decade of body mass index, Alzheimer's disease, and dementia. J Alzheimers Dis 2015;43(3):739-55.
3. García-Ptacek S, Faxén-Irving G, Cermáková P, Eriksdotter M, Religa D. Body mass index in dementia. Eur J Clin Nutr 2014 Nov;68(11):1204-9.
4. Elias MF, Goodell AL, Waldstein SR. Obesity, cognitive functioning and dementia: back to the future. J Alzheimers Dis 2012;30 Suppl 2:S113-25.
5. Nilsson LG, Nilsson E. Overweight and cognition. Scand J Psychol. 2009;50(6):660-7.
6. Anstey KJ, Cherbuin N, Budge M, Young J. Body mass index in midlife and late-life as a risk factor for dementia: a meta-analysis of prospective studies. Obes Rev 2011 May;12(5):e426-37.
7. Jacobsen BK, Eggen AE, Mathiesen EB, Wilsgaard T, Njølstad I. Cohort profile: The Tromsø Study. Int J Epidemiol 2012 Aug; 41(4): 961–7.
8. Iversen A. The Fifth Tromsø Study [web document]. Tromsø: UiT The Arctic University of Norway [updated 2016-02-25; cited 2020-03-26]. Available from: https://en.uit.no/prosjekter/prosjekt?p_document_id=91824 [
9. Consent for storing blood samples and connecting information with other registers [web document]. Tromsø: UiT The Arctic University of Norway [cited 2020-03-26]. Available from: https://en.uit.no/forskning/forskningsgrupper/sub?p_document_id=453582&sub_id=669747.
10. Obesity: preventing and managing the global epidemic. Geneva: World Health Organization; 2000. WHO Technical Report Series 894. Available from: https://www.who.int/nutrition/publications/obesity/WHO_TRS_894/en/.
11. Bäckman L, Forsell Y. Episodic memory functioning in a community-based sample of old adults with major depression: utilization of cognitive support. J Abnorm Psychol 1994 May;103(2):361-70.
12. Wechsler D. Wechsler Adult Intelligence Scale - Revised: Manual. San Antonio, Psychological Corp., 1981.
13. Christianson M, Leathem J. Development and standardisation of the computerised finger tapping test: Comparison with other finger tapping instruments. NZ J Psychol. 2004;33(2):44-9.
14. Wefel JS, Hoyt BD, Massman PJ. Neuropsychological functioning in depressed versus nondepressed participants with Alzheimer's disease. Clin Neuropsychol. 1999;13(3):249-57.
15. Gamberale G, Iregren A, Kjellberg A. The Swedish Performance Evaluation System, SPES Version 5.0: Depressive Symptoms, Anxiety or Educational Level. Solna/SE, National Institute of Occupational Health. 1993.
16. Lezak MD, Howieson DB, Loring DW. Neuropsychological Assessment, ed 4. Oxford, Oxford University Press, 2004.

17. Cereda E, Sansone V, Meola G, Malavazos AE. Increased visceral adipose tissue rather than BMI as a risk factor for dementia. Age Ageing. 2007;36(5):488-91.
18. Bergman RN, Stefanovski D, Buchanan TA, Sumner AE, Reynolds JC, Sebring NG, et al. A better index of body adiposity. Obesity (Silver Spring). 2011;19(5):1083-9.
19. Ponti F, Santoro A, Mercatelli D, Gasperini C, Conte M, Martucci M, et al. Aging and Imaging Assessment of Body Composition: From Fat to Facts. Front Endocrinol (Lausanne) 2019; 10: 861.
20. Santoro A, Bazzocchi A, Guidarelli G, Ostan R, Giampieri E, Mercatelli D, et al. A Cross-Sectional Analysis of Body Composition Among Healthy Elderly From the European NU-AGE Study: Sex and Country Specific Features. Front Physiol 2018 Nov 30; 9: 1693.
21. Bahat G, Tufan F, Saka B, Akin S, Ozkaya H, Yucel N, et al. Which body mass index (BMI) is better in the elderly for functional status? Arch Gerontol Geriatr 2012;54(1):78-81.
22. Tolppanen AM, Ngandu T, Kåreholt I, Laatikainen T, Rusanen M, Soininen H, et al. Midlife and late-life body mass index and late-life dementia: results from a prospective population-based cohort. J Alzheimers Dis 2014;38(1):201-9.
23. Wang C, Chan JS, Ren L, Yan JH. Obesity Reduces Cognitive and Motor Functions across the Lifespan. Neural Plast 2016;2016:2473081.
24. Bischof GN, Park DC. Obesity and Aging: Consequences for Cognition, Brain Structure, and Brain Function. Psychosom Med 2015 Jul-Aug;77(6):697-709.
25. Tregellas JR, Wylie KP, Rojas DC, Tanabe J, Martin J, Kronberg E, et al. Altered Default Network Activity in Obesity. Obesity (Silver Spring) 2011 Dec;19(12):2316-21.
26. Christianson MK, Leathem JM. Development and standardisation of the computerised finger tapping test: Comparison with other finger tapping instruments. NZ J Psychol 2004 Jul;33(2):44-9.
27. Hugdahl K, Raichle ME, Mitra A, Specht K.. On the existence of a generalized non-specific task-dependent network. Front Hum Neurosci. 2015 Aug 6;9:430.
28. Bray GA. Medical consequences of obesity. J Clin Endocrinol Metab. 2004 Jun;89(6):2583-9.
29. de Groot-Driessen D, van de Sande P, van Heugten C. Speed of finger tapping as a predictor of functional outcome after unilateral stroke. Arch Phys Med Rehabil. 2006 Jan;87(1):40-4.
30. Engstad T, Almkvist O, Viitanen M, Arnesen E. Impaired motor speed, visuospatial episodic memory and verbal fluency characterize cognition in long-term stroke survivors: the Tromsø Study. Neuroepidemiology. 2003 Nov-Dec;22(6):326-31.
31. Zhao Q, Lu Y, Zhou Y, Hong Z, Guo Q. Short-term delayed recall of auditory verbal learning test is equivalent to long-term delayed recall for identifying amnestic mild cognitive impairment. PLoS One 2012;7(12):e51157.

32. Joo SH, Yun SH, Kang DW, Hahn CT, Lim HK, Lee CU.. Body Mass Index in Mild Cognitive Impairment According to Age, Sex, Cognitive Intervention, and Hypertension and Risk of Progression to Alzheimer's Disease. Front Psychiatry 2018 Apr 17;9:142
33. Gilmour H. Cognitive performance of Canadian seniors. Statistics Canada, Catalogue no 82-003-XPE. Health Reports, 2011 Jun 22(2).
34. Papp KV, Rentz DM, Mormino EC, Schultz AP, Amariglio RE, Quiroz Y, et al. Cued memory decline in biomarker-defined preclinical Alzheimer disease. Neurology 2017 Apr 11;88(15):1431-8.
35. Huang CY, Hwang AC, Liu LK, Lee WJ, Chen LY, Peng LN, et al. Association of Dynapenia, Sarcopenia, and Cognitive Impairment Among Community-Dwelling Older Taiwanese. Rejuvenation Res 2016 Feb;19(1):71-8
36. Althubaiti A. Information bias in health research: definition, pitfalls, and adjustment methods. J Multidiscip Healthc 2016;9:211–7.

**Table 1.** Participation in different age groups of elderly men and women. The Tromsø study.

| Age (years) | No. invited | | No. attended | | Participation rate (%) | |
|---|---|---|---|---|---|---|
| | Men | Women | Men | Women | Men | Women |
| 65-69 | 637 | 687 | 591 | 636 | 92.8 | 92.6 |
| 70-74 | 542 | 683 | 495 | 604 | 91.3 | 88.4 |
| 75-79 | 470 | 627 | 390 | 495 | 83.0 | 78.9 |
| ≥80 | 162 | 214 | 118 | 150 | 72.8 | 70.1 |
| Total | 1811 | 2211 | 1594 | 1885 | 88.0 | 85.3 |

**Table 2.** Baseline characteristics of participants. The Tromsø Study.

| Total (n=2492) | Men (n=1148) | Women (n=1344) | P-value[a] |
|---|---|---|---|
| Age, years. Mean (SD) | 71.6 (4.7) | 72.0 (4.9) | 0.025[b] |
| Single living | 264 (23.0%) | 626 (46.6%) | <0.001[c] |
| BMI kg/m2, Mean (SD) | 26.4 (3.5) | 27.0 (4.5) | <0.001[b] |
| BMI categories | | | |
| ≤19.99 kg/m2 | 24 (2.1%) | 63 (4.7%) | |
| 20.00-22.49 kg/m2 | 113 (9.8%) | 132 (9.8%) | |
| 22.50-24.99 kg/m2 | 256 (23.3%) | 260 (19.3) | <0.001[c] |
| 25.00-27.49 kg/m2 | 354 (30.8%) | 316 (23.5%) | |
| 27.50-29.99 kg/m2 | 236 (20.6%) | 257 (19.1%) | |
| ≥30.00 kg/m2 | 165 (14.4%) | 316 (23.5%) | |
| Education | | | |
| Primary school | 84 (7.8%) | 63 (5.1%) | |
| Lower secondary | 556 (51.6%) | 834 (67.1%) | |
| Upper secondary | 275 (25.5%) | 235 (18.9%) | <0.001[c] |
| College/university < 4 years | 94 (8.7%) | 65 (5.2%) | |
| College/university ≥4 years | 68 (6.3%) | 45 (3.6%) | |
| Smoking | | | |
| Currently | 256 (22.4%) | 277 (20.9%) | |
| Previously | 686 (60.1%) | 397 (29.9%) | <0.001[c] |
| Never | 199 (17.4%) | 654 (49.2%) | |
| Alcohol | | | |
| Never | 193 (17.2%) | 398 (31.5%) | |
| Rarely | 448 (39.9%) | 552 (43.7%) | |
| Monthly | 311 (27.7%) | 207 (16.4%) | <0.001[c] |
| 2-3 times/week | 122 (10.9%) | 83 (6.6%) | |
| 4-7 times/week | 49 (4.4%) | 22 (1.7%) | |
| Medical conditions | | | |
| Diabetes | 68 (6.0%) | 64 (4.9%) | 0.276[c] |
| Heart attack | 179 (15.8%) | 80 (6.1%) | <0.001[c] |
| Angina | 216 (19.2%) | 156 (12.0%) | <0.001[c] |
| Stroke | 71 (6.3%) | 70 (5.4%) | 0.391[c] |
| Hypertension | 340 (30.5%) | 415 (31.9%) | 0.634[c] |

Values are mean (SD) for continuous variables and n (%) for categorical variables.

[a]The p-values for the difference between men and women were estimated by using the independent sample t-test[b] and the chi-square test[c].

**Table 3.** Regression models for the association between cognitive test performance and BMI categories.

| BMI groups (kg/m2) | Finger tapping (non-dominant hand) | | | | Short-term delayed recall* (12-word test, part two) | | | |
|---|---|---|---|---|---|---|---|---|
| | Model 1a | | Model 2b | | Model 1a | | Model 2b | |
| | OR (95% CI) | p | OR (95% CI) | p | OR (95% CI) | p | OR (95% CI) | p |
| ≤19.99 | 1.70 (0.97-2.99) | 0.063 | 1.68 (0.93-3.03) | 0.085 | 1.44 (0.78-2.64) | 0.242 | 1.41 (0.76-2.64) | 0.279 |
| 20.00-22.49 | 1.00 (0.66-1.51) | 0.987 | 1.03 (0.67-1.57) | 0.907 | **1.59 (1.08-2.34)** | **0.019** | **1.59 (1.07-2.37)** | **0.022** |
| 22.50-24.99 | 1.17 (0.82-1.55) | 0.465 | 1.21 (0.87-1.68) | 0.255 | 0.80 (0.57-1.13) | 0.199 | 0.83 (0.59-1.18) | 0.300 |
| 25.00-27.49 | 1.00 Reference | | | | 1.00 Reference | | | |
| 27.50-29.99 | 1.04 (0.75-1.44) | 0.828 | 1.01 (0.73-1.41) | 0.947 | 0.90 (0.64-1.27) | 0.540 | 0.90 (0.64-1.27) | 0.550 |
| ≥30.00 | **1.67 (1.23-2.27)** | **0.001** | **1.53 (1.11-2.10)** | **0.009** | 0.93 (0.66-1.32) | 0.696 | 0.96 (0.67-1.36) | 0.805 |
| Nc | 22.90% (486/2122) | | | | 17.06% (360/2110) | | | |

Significant test results are printed in bold.
aAdjusted for age and sex.
bAdusted for age, sex, educational level, smoking, alcohol consummation, diabetes, hypertension, stroke, coronary heart disease.
cSample size (with proportion of participants performing under the 25th percentile in %).

*Short-term delayed recall:
When the two lowest BMI categories were collapsed;
BMI ≤22.49 kg/m2:
Model 1a; **OR: 1.55, 95% CI: 1.09-2.21, p=0.015**
Model 2b; **OR: 1.54, 95% CI: 1.07-2.23, p=0.020**

**Table 4.** Regression models for the association between cognitive test performance and BMI categories.

| BMI groups ($kg/m^2$) | Finger tapping (dominant hand) | | | | Immediate recall (12-word test, part one) | | | | Digit symbol coding test | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Model 1[a] | | Model 2[b] | | Model 1[a] | | Model 2[b] | | Model 1[a] | | Model 2[b] | |
| | OR (95% CI) | p | OR (95% CI) | p | OR (95% CI) | p | OR (95% CI) | p | OR (95% CI) | p | OR (95% CI) | p |
| ≤19.99 | 1.36 (0.77-2.39) | 0.292 | 1.38 (0.76-2.56) | 0.288 | 1.14 (0.62-2.10) | 0.665 | 1.10 (0.59-2.05) | 0.777 | 1.31 (0.72-2.39) | 0.383 | 1.35 (0.72-2.53) | 0.358 |
| 20.00-22.49 | 0.82 (0.54-1.25) | 0.352 | 0.84 (0.54-1.29) | 0.414 | 1.14 (0.78-1.69) | 0.497 | 1.13 (0.76-1.69) | 0.536 | 1.09 (0.73-1.62) | 0.676 | 1.15 (0.75-1.75) | 0.523 |
| 22.50-24.99 | 0.91 (0.66-1.24) | 0.536 | 0.97 (0.70-1.35) | 0.875 | 0.91 (0.67-1.25) | 0.570 | 0.98 (0.71-1.34) | 0.894 | 0.88 (0.64-1.21) | 0.442 | 1.02 (0.73-1.42) | 0.920 |
| 25.00-27.49 | 1.00 Reference | | | | 1.00 Reference | | | | 1.00 Reference | | | |
| 27.50-29.99 | 1.08 (0.79-1.48) | 0.633 | 1.07 (0.77-1.47) | 0.713 | 1.18 (0.87-1.61) | 0.281 | 1.18 (0.87-1.62) | 0.288 | 0.92 (0.67-1.27) | 0.611 | 0.90 (0.64-1.26) | 0.540 |
| ≥30.00 | **1.38 (1.09-1.88)** | **0.038** | 1.26 (0.92-1.73) | 0.150 | 1.19 (0.87-1.62) | 0.287 | 1.14 (0.82-1.57) | 0.436 | 1.23 (0.89-1.68) | 0.206 | 1.08 (0.77-1.51) | 0.670 |
| N[c] | 22.76% (483/2122) | | | | 22.20% (470/2117) | | | | 21.39% (454/2122) | | | |

Significant test results are printed in bold.
[a]Adjusted for age and sex.
[b]Adusted for age, sex, educational level, smoking, alcohol consummation, diabetes, hypertension, stroke, coronary heart disease.
[c]Proportion of participants performing under the 25th percentile in % (sample size).