Does Elevated Alcohol Consumption Delay the Diagnostic Assessment of Cognitive Impairment among Older Adults?

Ben Kamsvaag^{a, b} Kjerstin Tevik^b Jūratė Šaltytė Benth^{a, c, d} Bei Wu^e
Sverre Bergh^{a, f} Geir Selbaek^{f, g, h} Anne-Sofie Helvik^{b, f}

^aResearch Centre for Age-Related Functional Decline and Disease, Innlandet Hospital Trust, Ottestad, Norway;

^bDepartment of Public Health and Nursing, Faculty of Medicine and Health Sciences, Norwegian University of Science and Technology (NTNU), Trondheim, Norway; ^cInstitute of Clinical Medicine, Campus Ahus, University of Oslo, Oslo, Norway; ^dHealth Services Research Unit, Akershus University Hospital, Lørenskog, Norway; ^eRory Meyers College of Nursing, New York University, New York, NY, USA; ^fNorwegian National Advisory Unit on Ageing and Health, Vestfold Hospital Trust, Tønsberg, Norway; ^gDepartment of Geriatric Medicine, Oslo University Hospital, Oslo, Norway; ^hInstitute of Clinical Medicine, Faculty of Medicine, University of Oslo, Oslo, Norway

Keywords

Cognitive impairment · Dementia · Alcohol consumption · Aged · Delayed diagnosis

Abstract

Introduction: The time from symptom debut to assessment of cognitive impairment (TSA) is usually substantial, and many factors can influence the length of this interval. Our objective was to discern whether elevated alcohol consumption is associated with TSA. **Methods:** Alcohol consumption was measured among 3,236 older Norwegians assessed for cognitive impairment. Elevated consumption was defined as drinking 4–7 times a week. TSA was defined as the number of months between symptom debut and assessment. The association between alcohol consumption and TSA was examined with a multiple regression analysis controlled for sociodemographic and clinical covariates. **Results:** Mean (SD) and median TSA were 34.8 (35.8) and 24.0 months, respectively. Elevated alcohol consumption was not associated with TSA. Longer TSA was associated with being male, having a high education level, being retired or unemployed, being single, having low scores on the Mini-Mental State Ex-

amination (MMSE) or Personal Activities of Daily Living (PADL), having high subsyndrome scores of depression or agitation on The Neuropsychiatric Inventory – Questionnaire (NPI-Q), or having a spouse/cohabitant as the designated next of kin. **Conclusion:** This study indicates that elevated alcohol consumption does not influence TSA. Possible explanations are discussed, but further research is needed to determine the effect of alcohol definitively. We did identify other novel characteristics associated with TSA which may be important in minimizing the risk of delayed cognitive assessments and should be kept in mind when considering assessment.

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Introduction

Dementia affects approximately 50 million people worldwide, and this number will quickly rise as the population of older adults will grow more rapidly in the coming decades [1]. It has an insidious onset and is characterized by cognitive, functional, and psychological impairment. It causes significant suffering to afflicted

individuals and imposes an enormous economic cost on society at large [2].

Timely assessment of cognitive symptoms is important for many reasons, not least ruling out other treatable causes of cognitive impairment. Both neuropsychiatric and cognitive symptoms of dementia may be misinterpreted as other mental disorders [3, 4] or signs of normal aging [5–8]. If dementia is the underlying cause of cognitive impairment, however, timely assessment is crucial. More specifically, a timely assessment can make it easier to plan for the future [9–11], rule out preventable and reversible causes of symptoms [12–14], improve quality of life by treating cognitive and neuropsychiatric symptoms [15–17], ensure maximum effect of medical treatment [18–20], reduce the burden of care and delay nursing-home admissions [16, 21, 22], and increase cost-effectiveness [16, 22, 23].

Despite the importance of a timely assessment of cognitive symptoms, more than 50% of people with dementia may be undiagnosed [16, 24]. Accordingly, documenting the amount of time either between symptom debut and assessment (TSA) or between symptom debut and diagnosis has been important. Point estimates of TSA are typically between 1 and 2 years [25–28], although the variability is large, and some people wait much longer. Demographic variables such as sex, age, and education seem to be associated with the time until assessment or diagnosis, but the evidence is mixed [29–34]. In addition, young-onset dementia typically takes longer to diagnose than late-onset dementia [33, 35], and frontotemporal dementia takes longer than Alzheimer's disease or vascular dementia [3, 33]. There are also various personal barriers preventing people from seeking assessment, such as denial, stigma, or unawareness of symptoms [36].

To our knowledge, no studies have examined whether elevated alcohol consumption influences TSA, nor the time until assessment or diagnosis of other health concerns. The focus of current research has been on how elevated alcohol consumption, across all ages, can delay or preclude assessment or treatment for alcohol use [37–40]. Importantly, however, this may also hold true for older adults with cognitive impairment [41]. One reason may be that general practitioners (GP) report being less willing to refer a person with cognitive symptoms to specialist care if this person also has an alcohol use disorder [41]. A possible consequence of this is that older adults with elevated alcohol consumption and cognitive impairment are less likely to receive a timely assessment of either problem. Furthermore, people with elevated alcohol consumption are less likely to have a stable informal network [42–46] and are at greater risk of developing a variety of

Fig. 1. Flowchart of the selection process. TSA, time from symptom debut to clinical assessment. *Two cases were excluded due to difficulties quantifying TSA accurately, and 1 case was excluded due to data suggesting the unlikely scenario that the case developed cognitive symptoms before the age of 20 years.

neuropsychological impairments [47–51], some of which might lead to misattribution or reduced awareness of dementia symptoms. Importantly, the lack of network support, misattribution of dementia symptoms, and unawareness of dementia symptoms have all consistently been identified as barriers to receiving an assessment of cognitive impairment [36]. Taken together, these findings suggest that elevated alcohol consumption could affect TSA. If so, documenting such an effect would be important in ensuring that these older adults are not neglected and that they receive adequate medical attention. Thus, this study examined whether elevated alcohol consumption has an effect on TSA among older adults with symptoms of cognitive impairment.

Table 1. NorCog measurements included in study analyses

Measurement	Description
Time from symptom debut to assessment of cognitive impairment (TSA) [52]	Single question from the NorCog assessing the number of months between awareness of the first symptom of cognitive impairment and clinical assessment within the specialist health care system. Based on information from next of kin. The question was, "How long ago did the symptoms in question start"? In Norway, the initial step in seeking medical attention is typically undertaken at a GP office. If deemed necessary, a person is then referred to the specialist health care system [53]. Thus, TSA as operationalized in the current study pertains to the time interval between symptom debut and assessment within the specialist health care system
Alcohol consumption [54]	Single question assessing alcohol consumption frequency on an 8-point scale. The next of kin's response was selected for analysis, as previous work has indicated that the next of kin may be the most valid source of this information [55]. The question, "About how often in the last 12 months did you drink alcohol," had the following response alternatives: "Never," "Not at all the last year," "A few times a year," "Once a month," "2–3 times a month," "Once a week," "2–3 times a week," to "4–7 times a week" Drinking 4–7 times a week was defined as "elevated alcohol consumption," based on definitions used by the US National Institute on Alcohol Abuse and Alcoholism [56] and the American Geriatrics Society [57]
Mini-Mental State Examination – Norwegian Revised Versions 2 and 3 (MMSE-NR2 and MMSE-NR3) [58, 59]	Screening instrument for brief assessment of cognitive impairment. Norwegian revisions were used to ensure the highest validity. Twenty items, score min–max: 0–30. Higher scores indicate better cognitive function. Conventionally, a cutoff score of 24 has been used to indicate cognitive impairment
The Neuropsychiatric Inventory – Questionnaire (NPI-Q) [60–62]	Structured interview of the next of kin assessing the participant's neuropsychiatric symptoms. Twelve symptoms are graded on a 0–3-point scale. Symptoms were categorized into three subsyndromes based on a previous factor analysis on NorCog data [62]: Depression, score min–max: 0–18 (items: depression, anxiety, disturbances in appetite, apathy, motor disturbances, and night-time disturbances); agitation, score min–max: 0–12 (items: euphoria, disinhibition, irritability, and agitation); psychosis, score min–max: 0–6 (items: hallucinations and delusions). Higher scores indicate higher subsyndrome severity
Personal Activities of Daily Living (PADL) [63]	Structured interview of the next of kin assessing the participant's ability to perform personal activities of daily living. Six symptoms are assessed and graded on a 1–5-point scale. The scales were re-coded into a dichotomous outcome of "loss of function" (0) or "normal function" (1). Score min–max: 0–6. Higher scores indicate better function of daily living activities
NorCog, Norwegian Registry of Persons Assessed for Cognitive Symptoms.	

Materials and Methods

Participants

This study included older adults (≥ 60 years) registered in the Norwegian Registry of Persons Assessed for Cognitive Symptoms (NorCog) between 2014 and 2018. NorCog aims to standardize the assessment of cognitive impairment at specialist, outpatient clinics in Norway. The selection process is illustrated in Figure 1. A total of 2,683 cases were excluded due to ambiguous, incompatible, or missing (alcohol or TSA) data. Our final sample consisted of 3,236 cases.

Measures

NorCog provides a comprehensive, standardized test battery including physical, psychological, and cognitive examinations, as well as standardized interviews with both the patient and a next of kin. When all necessary examinations have been completed, a medical professional forms a diagnostic conclusion with regard to cognitive function. Table 1 outlines and describes the assessment scales used in our analyses, while Table 2 lists the entire selection of variables used. The outcome variable was the number of months between cognitive symptom debut and assessment (TSA), while the primary independent variable was alcohol consumption.

Statistical Analysis

The data were analyzed with SPSS version 26, STATA version 16, and Excel. A multiple linear regression analysis examined the relationship between variables of interest and the outcome variable, TSA, which was ln-transformed due to a skewed distribution. The relative effect of each variable in the multiple regression model was assessed by a dominance analysis. Cluster effect at the level of health institution was assessed with the intraclass correlation coefficient (ICC). As ICC was close to zero, no adjustment was necessary. Tukey's method identified 178 outliers at the level of TSA. These outliers were not omitted as the 5% trimmed mean was comparable to the overall sample mean, and because further investigation revealed that these cases reflect true numbers observed in Norwegian clinical practice; as such, they provide valuable information about the study population.

Missing values of the PADL and NPI-Q measures were imputed for cases with $<50\%$ missing item values. For each item, a random number drawn from an empirical distribution was used to replace missing values. Missing values of marital status were logically imputed. Because another 947 cases were excluded from the regression due to missing values, a weighted linear regression weighting cases more demographically similar to the excluded cas-

Table 2. Demographic and clinical characteristics of the study sample ($n = 3,236$)

Participant characteristics	Statistic
Mean number of months from symptom to assessment (TSA) (SD), $n = 3,236$	34.8 (35.8)
Alcohol use, as reported by next of kin, $n = 3,236$	
Never	263 (8.1)
Not at all the last year	497 (15.4)
A few times a year	726 (22.4)
Once a month	238 (7.4)
2–3 times a month	268 (8.3)
Once a week	328 (10.1)
2–3 times a week	494 (15.3)
4–7 times a week	422 (13.0)
Sex, $n = 3,236$	
Female	1,693 (52.3)
Male	1,543 (47.7)
Mean age (SD), $n = 3,236$	75.8 (7.2)
Mean years of education (SD), $n = 3,089$	11.1 (3.6)
Employment status, $n = 3,081$	
Not currently working	1,001 (32.5)
Working 10% or more	127 (4.1)
Sick leave/disability benefits	168 (5.5)
Retired	1,785 (57.9)
Marital status, $n = 3,158$	
Partner (cohabited or married)	2,111 (66.8)
Single	1,047 (33.2)
Proportion receiving domiciliary care, $n = 3,216$	1,044 (32.5)
Dementia diagnosis, $n = 2,650^*$	
Subjective cognitive impairment	86 (3.2)
Mild cognitive impairment	846 (31.9)
Dementia	1,486 (56.1)
“Others” (including nondementia cases)	232 (8.8)
Mean number of chronic diseases (SD), $n = 3,039$	2.3 (1.7)
Mean number of registered medications (SD), $n = 3,087$	4.3 (3.1)
Tobacco smoking habits, as reported by next of kin, $n = 3,200$	
Never smoked	1,292 (40.4)
Smoked previously but no longer smokes	1,474 (46.1)
Currently smoking	434 (13.6)
Mean MMSE-NR2/3 score (SD), $n = 3,198$	22.9 (4.6)
Mean NPI-Q subsyndrome scores (SD)	
Depression (0–18 points), $n = 2,974$	3.6 (3.3)
Agitation (0–12 points), $n = 3,047$	1.4 (1.9)
Psychosis (0–6 points), $n = 3,098$	0.6 (1.2)
Mean PADL score (SD), $n = 3,087$	4.9 (1.4)
Next of kin characteristics	
Next of kin’s sex, $n = 1,563^*$	
Female	1,101 (65.8)
Male	572 (34.2)
Next of kin’s mean age (SD), $n = 2,906^*$	63.0 (13.1)
Next of kin’s relationship to participant, $n = 3,155$	
Spouse/cohabitant	1,701 (53.9)
Child/child-in-law	1,216 (38.5)
Others (neighbor, friend, sibling, etc.)	238 (7.5)

Numbers are n (%) if not otherwise specified. Percentages may not total 100 due to rounding. SD, standard deviation; MMSE-NR2/3, Mini-Mental State Examination – Norwegian Revised Version 2/3; NPI-Q, The Neuropsychiatric Inventory – Questionnaire; PADL, Personal Activities of Daily Living. * Not included in further analyses due to high number of missing values.

Table 3. Test of differences between included and excluded cases ($n = 4,671$)

Independent variables	Included cases ($n = 3,236$)	Excluded cases ($n = 1,435$)	p value
Sex, proportion female ($n = 3,236/1,435$), n (%)	1,693 (52.3)	751 (52.3)	0.992 ¹
Mean age (SD) ($n = 3,236/1,435$)	75.8 (7.2)	75.3 (8.2)	0.045 ²
Mean years of education (SD) ($n = 3,089/1,294$)	11.1 (3.6)	11.5 (3.9)	0.002 ²
Mean MMSE-NR2/3 score (SD) ($n = 3,198/1,400$)	22.9 (4.6)	24.1 (4.7)	<0.001 ²
Mean NPI-Q subsyndrome scores (SD)			
Depression (0–18 points) ($n = 2,974/984$)	3.6 (3.3)	3.4 (3.5)	0.049 ²
Agitation (0–12 points) ($n = 3,047/1,010$)	1.4 (1.9)	1.3 (2.0)	0.061 ²
Psychosis (0–6 points) ($n = 3,098/1,043$)	0.6 (1.2)	0.5 (1.2)	0.344 ²
Proportion receiving domiciliary care ($n = 3,216/1,339$), n (%)	1,044 (32.5)	538 (40.2)	<0.001 ¹

SD, standard deviation; MMSE-NR2/3, Mini-Mental State Examination – Norwegian Revised Version 2/3; NPI-Q, The Neuropsychiatric Inventory – Questionnaire. Significant p values in bold. ¹ χ^2 test. ²Independent samples t test.

es was performed post hoc. Ideally, dementia diagnosis would also have been included as a covariate in the regression analysis to account for diagnostic subtypes (see Table 2). However, it had a high amount of missing values, and there was no appropriate way to conduct imputation. Therefore, dementia diagnosis was excluded from further analysis, and MMSE scores were used to measure cognitive impairment.

Results

A total of 3,236 participants (52.3% female) with a mean (SD) age of 75.8 (7.2) years were included (Table 2). Approximately 56.1% were registered with a dementia diagnosis, 31.9% were registered with mild cognitive impairment (MCI), while the rest (12%) were registered with subjective cognitive impairment (SCI) or “others.” Mean (SD) MMSE score among the sample was 22.9 (4.6). Mean (SD) and median TSA were 34.8 (35.8) months and 24.0 months, respectively (min–max = 0–576 months). Figure 2 displays the cumulative distribution of TSA. A comparison between included cases ($n = 3,236$) and excluded cases ($n = 1,435$) (Table 3) revealed that included cases were slightly older, had fewer years of education, received domiciliary care less often, had lower mean MMSE scores, and had higher mean scores on the depression subsyndrome of the NPI-Q.

The results of the regression analysis are presented in Table 4. Elevated alcohol consumption was not associated with TSA. Longer TSA was associated with being male, having more years of education, being retired or not working, and being single. Scoring higher on the

Fig. 2. Cumulative distribution of time from symptom debut to assessment (TSA).

depression and agitation subsyndrome scales of the NPI-Q was also associated with longer TSA. On the other hand, working $\geq 10\%$, having a more distant designated next of kin such as a neighbor or friend (compared to a partner), and scoring higher on the MMSE or PADL were associated with shorter TSA. The results of the weighted regression analysis (not reported) were equivalent to the results of the primary regression analysis.

Table 4. Results of regression analysis of covariates related to TSA (*n* = 2,289)

Independent variables	Unadjusted model		Adjusted model	
	RC (95% CI)	<i>p</i> value	RC (95% CI)	<i>p</i> value
Participant characteristics				
Participant's alcohol use, as reported by next of kin				
Never	−0.10 (−0.24; 0.04)	0.178	−0.07 (−0.21; 0.07)	0.341 ⁷
Not at all the last year	−0.04 (−0.16; 0.08)	0.507	−0.04 (−0.15; 0.08)	0.495 ⁷
A few times a year	Reference		Reference	
Once a month	−0.004 (−0.16; 0.15)	0.956	0.007 (−0.14; 0.16)	0.924 ⁷
2–3 times a month	0.09 (−0.06; 0.23)	0.235	0.12 (−0.02; 0.26)	0.084 ⁷
Once a week	0.03 (−0.10; 0.16)	0.664	0.06 (−0.07; 0.19)	0.365 ⁷
2–3 times a week	−0.002 (−0.12; 0.12)	0.975	0.05 (−0.07; 0.17)	0.404 ⁷
4–7 times a week	0.03 (−0.09; 0.15)	0.627	0.005 (−0.12; 0.13)	0.934 ⁷
Sex, male	0.10 (0.03; 0.17)	0.004	0.11 (0.03; 0.19)	0.005 ⁶
Age	0.0003 (−0.005; 0.005)	0.893	−0.004 (−0.01; 0.003)	0.258 ¹³
Education (years, <i>n</i>)	0.01 (0.002; 0.02)	0.021	0.02 (0.01; 0.03)	0.001 ⁵
Employment status				
Not currently working	Reference		Reference	
Working 10% or more	−0.24 (−0.43; −0.04)	0.019	−0.21 (−0.41; −0.01)	0.037 ⁴
Sick leave/disability benefits	0.07 (−0.09; 0.24)	0.379	0.06 (−0.11; 0.23)	0.476 ⁴
Retired	0.08 (−0.0003; 0.15)	0.051	0.08 (0.003; 0.16)	0.041 ⁴
Marital status, single	0.05 (−0.02; 0.13)	0.182	0.15 (0.03; 0.27)	0.015 ¹¹
Receives domiciliary care	0.09 (0.02; 0.17)	0.016	0.02 (−0.07; 0.12)	0.611 ¹²
Chronic diseases, <i>n</i>	0.007 (−0.01; 0.03)	0.516	−0.006 (−0.03; 0.02)	0.610 ¹⁶
Medications, <i>n</i>	0.006 (−0.006; 0.02)	0.306	0.004 (−0.01; 0.02)	0.540 ¹⁵
Tobacco smoking habits, as reported by next of kin				
Never smoked	Reference		Reference	
Smoked previously but no longer smokes	0.005 (−0.07; 0.08)	0.894	−0.03 (−0.11; 0.05)	0.434 ¹⁴
Currently smoking	0.04 (−0.07; 0.15)	0.515	−0.004 (−0.12; 0.11)	0.951 ¹⁴
MMSE sum score	−0.01 (−0.02; −0.003)	0.006	−0.009 (−0.02; −0.001)	0.033 ⁹
NPI-Q subsyndrome scores				
Depression score	0.04 (0.03; 0.05)	<0.001	0.02 (0.004; 0.03)	0.011 ²
Agitation score	0.07 (0.05; 0.09)	<0.001	0.05 (0.03; 0.07)	<0.001 ¹
Psychosis score	0.07 (0.04; 0.10)	<0.001	0.005 (−0.03; 0.04)	0.795 ⁸
PADL sum score	−0.08 (−0.10; −0.05)	<0.001	−0.05 (−0.08; −0.02)	0.001 ³
Next of kin characteristics				
Relationship to participant				
Spouse/cohabitant	Reference		Reference	
Child/child-in-law	0.008 (−0.07; 0.08)	0.826	−0.08 (−0.20; 0.04)	0.210 ¹⁰
Others (neighbor, friend, sibling, etc.)	−0.10 (−0.24; 0.04)	0.175	−0.21 (−0.38; −0.03)	0.024 ¹⁰

Outcome values were ln-transformed before the analysis and back-transformed for presentation in the table. TSA, time from symptom debut to clinical assessment; RC, regression coefficient; CI, confidence interval; MMSE-NR2/3, Mini-Mental State Examination – Norwegian Revised Version 2/3; NPI-Q, The Neuropsychiatric Inventory – Questionnaire; PADL, Personal Activities of Daily Living. Significant *p* values in bold. ^{1–16} Dominance analysis rank.

Discussion

This study aimed to discern whether elevated alcohol consumption has an effect on TSA among older Norwegians seeking assessment of cognitive impairment. Mean and median TSA were 34.8 and 24.0 months, respectively, comparable to what is typically reported. There was con-

siderable variability, a consistent tendency in other studies, so it is evident that some people wait for many years before getting an assessment or receiving a correct diagnosis of cognitive impairment. This is disconcerting given that a delay of even a few years can be detrimental [11, 16, 64], so any factor that can reduce the wait is worth investigating. Elevated alcohol consumption was not as-

sociated with TSA. Longer TSA was associated with being male, having a high education level, being retired or not currently employed, being single, having low scores on the MMSE and PADL, having high NPI-Q subsyndrome scores of depression or agitation, or having a partner as the designated next of kin.

Unexpectedly, the results indicate that elevated alcohol consumption does not affect TSA. One reason for this may be the increased attention alcohol consumption among older adults has received in recent years. In the past, alcohol consumption among this group was frequently overlooked [65–67]. Recently, however, awareness of the importance of screening for alcohol consumption has been rising [68, 69]. This might translate to an increased willingness to refer older patients with cognitive symptoms to specialist assessment, to the point where elevated alcohol consumption may no longer cause significant delays in assessment. Alternatively, certain limitations of our study design might have made it challenging to uncover any effect of alcohol on TSA. For instance, there could be heterogeneity in alcohol consumption even among participants picking the same responses, as two participants drinking at the same frequency may nevertheless drink unequal amounts. We also excluded participants without a next of kin, who may drink more than people with an established next of kin. In addition, as explained in Table 1, our participants were initially examined by their GP, and NorCog does not document this visit. Thus, we cannot rule out the possibility that elevated alcohol consumption does indeed affect the length of time between symptom debut and the initial assessment performed by the GP, and that this effect is attenuated when the specialist health care assessment is used as the end point. Taken together, therefore, it is premature to conclude that elevated alcohol consumption does not affect TSA.

Surprisingly, having a closer designated next of kin resulted in longer TSA. Close next of kin are often the first to notice symptoms [25], which is likely one reason they are often so involved in help-seeking [70–73]. However, they do not always register the subtler changes related to cognitive impairment or may even deny their existence [5, 27, 74]. Two other demographic variables, marital and employment status, also yielded some novel results. Yet, it is unsurprising that being single could increase TSA because, as mentioned, close acquaintances are often instrumental in the help-seeking process. Therefore, it makes sense that our single participants, 92.0% of whom lived alone, had longer TSA. Furthermore, while low economic status could be a barrier to accessing health services [75], being employed could reduce TSA by virtue of

taking place in a social arena where other people may notice cognitive symptoms.

The clinical measures also deserve mention. While higher scores of the depression subsyndrome of the NPI-Q could conceivably prolong TSA directly, this association could instead be due to cognitive impairment sometimes being misdiagnosed as depression, thereby delaying assessment or diagnosis of cognitive impairment [3, 33]. The agitation subsyndrome was also associated with longer TSA, replicating previous research [32]. Agitation can negatively affect the relationship between the person with cognitive impairment and their next of kin [76–81], and it is not unlikely that a strained relationship could interfere with help-seeking. Conversely, longer TSA could contribute to more severe neuropsychiatric symptoms, as these sometimes intensify as cognitive impairment progresses [82]. Furthermore, counterintuitive as it may seem, we are not the first to find that MMSE and PADL scores are inversely associated with time until assessment or diagnosis [29, 32]. One possible explanation is that people with mild cognitive symptoms have more insight than those with more severe symptoms and are more likely to recognize such symptoms and seek help [32, 83]. If so, given the intertwined relationship between daily function and cognition [84], this might explain why higher scores of both MMSE and PADL were associated with shorter TSA.

Strengths and Limitations

One limitation of our study is the use of retrospective reports about symptom debut, which are prone to memory errors. That said, all reports about symptom debut were systematically collected at the first specialist visit, reducing the risk of such errors to some degree. Another is the cross-sectional study design, which precludes conclusive causal explanations. Furthermore, 2,683 cases were excluded due to missing values, which introduces the risk of selection bias. However, between-group analyses (Table 3) found only minor differences between included and excluded cases, indicating minimal loss of representativity due to exclusion. An additional consequence of these exclusions, however, was that the regression analysis did not delineate between cases of SCI, MCI, and dementia. While MMSE scores should give an appropriate indication of cognitive impairment [85, 86], they do not adequately account for the heterogeneity in cognitive diagnoses. Also, as explained in the discussion, the null result regarding elevated alcohol consumption and TSA may be due to design limitations. Finally, there were outliers with high TSA that indicated that some participants waited several de-

ades after symptom debut before being assessed. As mentioned previously, however, such numbers do occur in Norwegian clinical practice (e.g., due to misdiagnosis), and the inclusion of these outliers did not seem to affect our results considerably. This study also has notable strengths. Our large sample comprised individuals assessed at many different institutions from all health regions in Norway, and all assessments were standardized and consisted of internationally recognized tools.

Conclusion

This study found that elevated alcohol consumption was not associated with TSA, although more research is needed before conclusions can be drawn. Several other characteristics were associated with longer TSA, namely: being male, single, retired, or unemployed, having a high education level, low scores on the MMSE and PADL, high scores of depression or agitation, or having a partner as the designated next of kin. The presence of any one of these characteristics can increase the risk of undue delays of important assessments and should be kept in mind when deciding whether to pursue an assessment of cognitive impairment.

Statement of Ethics

Collection and storage of NorCog data were approved by the Norwegian Data Inspectorate. All NorCog participants signed an informed consent form that grants future use of their data. The

present study was approved by the South-Eastern Regional Committee for Medical Research Ethics (REK sør-øst B: 21490) and the NorCog publication board.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

Anne-Sofie Helvik, Kjerstin Tevik, and Sverre Bergh conceptualized the study. Ben Kamsvaag contributed core design ideas, wrote the manuscript, performed data analysis, and performed literature searches. Kjerstin Tevik also performed literature searches. Jūratė Šaltytė Benth took lead in data analysis and performed the majority of the analyses. All authors, including Bei Wu and Geir Selbaek, were involved with determining which variables to include for statistical analysis, proofreading, and critical revision.

Data Availability Statement

The data in this study are not publicly available but may be requested from the Norwegian National Advisory Unit on Ageing and Health.

References

- 1 Patterson C. [World alzheimer report 2018. The state of the art of dementia research: new frontiers](#). Alzheimer's Disease International; 2018.
- 2 Cantarero-Prieto D, Leon PL, Blazquez-Fernandez C, Juan PS, Cobo CS. The economic cost of dementia: a systematic review. [Dementia](#). 2020;19(8):2637–57.
- 3 Rosness TA, Haugen PK, Passant U, Engedal K. Frontotemporal dementia: a clinically complex diagnosis. [Int J Geriatr Psychiatry](#). 2008 Aug;23(8):837–42.
- 4 Johannessen A, Helvik AS, Engedal K, Thorsen K. Experiences and needs of spouses of persons with young-onset frontotemporal lobe dementia during the progression of the disease. [Scand J Caring Sci](#). 2017;31(4):779–88.
- 5 Boise L, Morgan DL, Kaye J, Camicioli R. Delays in the diagnosis of dementia: perspectives of family caregivers. [Am J Alzheimers Dis](#). 1999;14(1):20–6.
- 6 Prohaska TR, Keller ML, Leventhal EA, Leventhal H. Impact of symptoms and aging attribution on emotions and coping. [Health Psychol](#). 1987;6(6):495.
- 7 Mahoney DF, Cloutterbuck J, Neary S, Zhan L. African American, Chinese, and Latino family caregivers' impressions of the onset and diagnosis of dementia: cross-cultural similarities and differences. [Gerontologist](#). 2005;45(6):783–92.
- 8 Livingston G, Baio G, Sommerlad A, de Lusignan S, Poulimenos S, Morris S, et al. Effectiveness of an intervention to facilitate prompt referral to memory clinics in the United Kingdom: cluster randomised controlled trial. [PLoS Med](#). 2017 Mar;14(3):e1002252.
- 9 Robinson L, Tang E, Taylor JP. Dementia: timely diagnosis and early intervention. [BMJ](#). 2015 Jun 16;350:h3029.
- 10 Dubois B, Padovani A, Scheltens P, Rossi A, Dell'Agnello G. Timely diagnosis for Alzheimer's disease: a literature review on benefits and challenges. [J Alzheimers Dis](#). 2016; 49(3):617–31.
- 11 Rasmussen J, Langerman H. Alzheimer's disease: why we need early diagnosis. [Degener Neurol Neuromuscul Dis](#). 2019;9:123–30.
- 12 Connell CM, Gallant MP. Spouse caregivers' attitudes toward obtaining a diagnosis of a dementing illness. [J Am Geriatr Soc](#). 1996;44(8): 1003–9.
- 13 Larson EB, Reifler BV, Featherstone HJ, English DR. Dementia in elderly outpatients: a prospective study. [Ann Intern Med](#). 1984; 100(3):417–23.
- 14 Bridges-Webb C, Wolk J, Pond D, Britt H. [Care of patients with dementia in general practice guidelines](#). North Sydney: NSW Department of Health; 2003.

- 15 Howard R, McShane R, Lindesay J, Ritchie C, Baldwin A, Barber R, et al. Donepezil and memantine for moderate-to-severe Alzheimer's disease. *New Engl J Med*. 2012;366(10):893–903.
- 16 Prince M, Bryce R, Ferri C. [World Alzheimer report 2011: the benefits of early diagnosis and intervention](#). Alzheimer's Disease International. 2011.
- 17 Olazarán J, Reisberg B, Clare L, Cruz I, Peña-Casanova J, Del Ser T, et al. Nonpharmacological therapies in Alzheimer's disease: a systematic review of efficacy. *Dement Geriatr Cogn Disord*. 2010;30(2):161–78.
- 18 Leifer BP. Early diagnosis of Alzheimer's disease: clinical and economic benefits. *J Am Geriatr Soc*. 2003;51(5 Suppl Dementia): S281–8.
- 19 Geldmacher DS. Treatment guidelines for Alzheimer's disease: redefining perceptions in primary care. *Prim Care Companion J Clin Psychiatry*. 2007;9(2):113–21.
- 20 Wattmo C, Minthon L, Wallin ÅK. Mild versus moderate stages of Alzheimer's disease: three-year outcomes in a routine clinical setting of cholinesterase inhibitor therapy. *Alzheimers Res Ther*. 2016;8(1):7–15.
- 21 Banerjee S, Wittenberg R. Clinical and cost effectiveness of services for early diagnosis and intervention in dementia. *Int J Geriatr Psychiatry*. 2009;24(7):748–54.
- 22 Weimer DL, Sager MA. Early identification and treatment of Alzheimer's disease: social and fiscal outcomes. *Alzheimers Dement*. 2009;5(3):215–26.
- 23 Getsios D, Blume S, Ishak KJ, MacLaine G, Hernández L. An economic evaluation of early assessment for Alzheimer's disease in the United Kingdom. *Alzheimers Dement*. 2012;8(1):22–30.
- 24 Iliffe S, Robinson L, Brayne C, Goodman C, Rait G, Manthorpe J, et al. Primary care and dementia: 1. diagnosis, screening and disclosure. *Int J Geriatr Psychiatry*. 2009;24(9):895–901.
- 25 Carpentier N, Bernard P, Grenier A, Guberman N. Using the life course perspective to study the entry into the illness trajectory: the perspective of caregivers of people with Alzheimer's disease. *Soc Sci Med*. 2010;70(10): 1501–8.
- 26 Chrisp TA, Tabberer S, Thomas BD, Goddard WA. Dementia early diagnosis: triggers, supports and constraints affecting the decision to engage with the health care system. *Aging Ment Health*. 2012;16(5):559–65.
- 27 Knopman D, Donohue JA, Guterman EM. Patterns of care in the early stages of Alzheimer's disease: impediments to timely diagnosis. *J Am Geriatr Soc*. 2000;48(3):300–4.
- 28 Speechly CM, Bridges-Webb C, Passmore E. The pathway to dementia diagnosis. *Med J Aust*. 2008;189(9):487–9.
- 29 Cattel C, Gambassi G, Sgadari A, Zuccalà G, Carbonin P, Bernabei R. Correlates of delayed referral for the diagnosis of dementia in an outpatient population. *J Gerontol A Biol Sci Med Sci*. 2000;55(2):M98–102.
- 30 Wolff F, Dietzel N, Karrer L, Kratzer A, Hess M, Grassel E, et al. [Timely diagnosis of dementia: results of the bavarian dementia survey (BayDem)]. *Gesundheitswesen*. 2020;82(1):23–9.
- 31 Woods B, Arosio F, Diaz A, Gove D, Holmerová I, Kinnaird L, et al. Timely diagnosis of dementia? Family carers' experiences in 5 European countries. *Int J Geriatr Psychiatry*. 2019;34(1):114–21. German.
- 32 Helvik AS, Engedal K, Šaltytė Benth J, Selbæk G. Time from symptom debut to dementia assessment by the specialist healthcare service in Norway. *Dement Geriatr Cogn Dis Extra*. 2018 Jan–Apr;8(1):117–27.
- 33 van Vliet D, de Vugt ME, Bakker C, Pijnenburg YA, Vernooij-Dassen MJ, Koopmans RT, et al. Time to diagnosis in young-onset dementia as compared with late-onset dementia. *Psychol Med*. 2013 Feb;43(2):423–32.
- 34 Zhao M, Lv X, Tuerxun M, He J, Luo B, Chen W, et al. Delayed help seeking behavior in dementia care: preliminary findings from the clinical pathway for Alzheimer's disease in China (CPAD) study. *Int Psychogeriatr*. 2016 Feb;28(2):211–9.
- 35 Loi SM, Goh AMY, Mocellin R, Malpas CB, Parker S, Eratne D, et al. Time to diagnosis in younger-onset dementia and the impact of a specialist diagnostic service. *Int Psychogeriatr*. 2020 Aug 28;1–9. Epub ahead of print.
- 36 Parker M, Barlow S, Hoe J, Aitken L. Persistent barriers and facilitators to seeking help for a dementia diagnosis: a systematic review of 30 years of the perspectives of carers and people with dementia. *Int Psychogeriatr*. 2020:1–24. Epub ahead of print.
- 37 Torvik FA, Ystrom E, Gustavson K, Rosenström TH, Bramness JG, Gillespie N, et al. Diagnostic and genetic overlap of three common mental disorders in structured interviews and health registries. *Acta Psychiatr Scand*. 2018;137(1):54–64.
- 38 Cunningham JA, Sobell LC, Sobell MB, Agrawal S, Toneatto T. Barriers to treatment: why alcohol and drug abusers delay or never seek treatment. *Addict Behav*. 1993;18(3): 347–53.
- 39 DiBartolo MC, Jarosinski JM. Alcohol use disorder in older adults: challenges in assessment and treatment. *Issues Ment Health Nurs*. 2017;38(1):25–32.
- 40 Johannessen A, Helvik AS, Engedal K, Sørli VM. Older peoples' narratives of use and misuse of alcohol and psychotropic drugs. *Scand J Caring Sci*. 2016 Sep;30(3):586–93.
- 41 Jensen CJ, Lukow HR, Heck AL. Identifying barriers to care for older adults with substance use disorders and cognitive impairments. *Alcohol Treat Q*. 2012;30(2):211–23.
- 42 Peirce RS, Frone MR, Russell M, Cooper ML, Mudar P. A longitudinal model of social contact, social support, depression, and alcohol use. *Health Psychol*. 2000;19(1):28.
- 43 Hanson BS. Social network, social support and heavy drinking in elderly men: a population study of men born in 1914, Malmö, Sweden. *Addiction*. 1994;89(6):725–32.
- 44 Kelly JF, Magill M, Stout RL. How do people recover from alcohol dependence? A systematic review of the research on mechanisms of behavior change in alcoholics anonymous. *Addict Res Theory*. 2009;17(3):236–59.
- 45 Ammon L, Bond J, Matzger H, Weisner C. Gender differences in the relationship of community services and informal support to seven-year drinking trajectories of alcohol-dependent and problem drinkers. *J Stud Alcohol Drugs*. 2008;69(1):140–50.
- 46 Weisner C, Delucchi K, Matzger H, Schmidt L. The role of community services and informal support on five-year drinking trajectories of alcohol dependent and problem drinkers. *J Stud Alcohol*. 2003;64(6):862–73.
- 47 Moselhy HF, Georgiou G, Kahn A. Frontal lobe changes in alcoholism: a review of the literature. *Alcohol Alcohol*. 2001;36(5):357–68.
- 48 Hayes V, Demirkol A, Ridley N, Withall A, Draper B. Alcohol-related cognitive impairment: current trends and future perspectives. *Neurodegener Dis Manag*. 2016;6(6):509–23.
- 49 Bates ME, Bowden SC, Barry D. Neurocognitive impairment associated with alcohol use disorders: implications for treatment. *Exp Clin Psychopharmacol*. 2002;10(3):193–212.
- 50 Bernardin F, Maheut-Bosser A, Paille F. Cognitive impairments in alcohol-dependent subjects. *Front Psychiatry*. 2014;5:78.
- 51 Oscar-Berman M, Shagrin B, Evert DL, Epstein C. Impairments of brain and behavior: the neurological effects of alcohol. *Alcohol Health Res World*. 1997;21(1):65–75.
- 52 Norwegian National Advisory Unit on Ageing and Health. Brukermanual 2019. Praktisk gjennomføring, testinstruksjoner, normer og diagnoser. 2019.
- 53 Norwegian Directorate of Health. Nasjonal veileder for henvisninger til spesialisthelsetjenesten [nettdokument]. 2015.
- 54 HUNT Research Centre. Alcohol frequency last 12 months. 2006–2008.
- 55 Kamsvaag B, Bergh S, Šaltytė Benth J, Selbæk G, Tevik K, Helvik AS. Alcohol consumption among older adults with symptoms of cognitive decline consulting specialist health care. *Aging Ment Health*. 2021:1–9. Epub ahead of print.
- 56 Dufour MC, Archer L, Gordis E. Alcohol and the elderly. *Clin Geriatr Med*. 1992;8(1): 127–42.
- 57 Lang I, Guralnik J, Wallace RB, Melzer D. What level of alcohol consumption is hazardous for older people? Functioning and mortality in U.S. and English national cohorts. *J Am Geriatr Soc*. 2007 Jan;55(1):49–57.
- 58 Norwegian National Advisory Unit on Ageing and Health. Basal demensutredning. Anbefalte tester og skjemaer, samlet. 2019.

- 59 Folstein MF, Folstein SE, McHugh PR. "Minimal state": a practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res*. 1975;12(3):189-98.
- 60 Cummings JL, Mega M, Gray K, Rosenberg-Thompson S, Carusi DA, Gornbein J. The neuropsychiatric inventory: comprehensive assessment of psychopathology in dementia. *Neurology*. 1994;44(12):2308-14.
- 61 Kaufer DI, Cummings JL, Ketchel P, Smith V, MacMillan A, Shelley T, et al. Validation of the NPI-Q, a brief clinical form of the neuropsychiatric inventory. *J Neuropsychiatry Clin Neurosci*. 2000;12(2):233-39.
- 62 Siafrikas N, Selbaek G, Fladby T, Saltyte Benth J, Auning E, Aarsland D. Frequency and subgroups of neuropsychiatric symptoms in mild cognitive impairment and different stages of dementia in Alzheimer's disease. *Int Psychogeriatr*. 2018 Jan;30(1):103-13.
- 63 Lawton MP, Brody EM. Assessment of older people: self-maintaining and instrumental activities of daily living. *Gerontologist*. 1969; 9(3):179-86.
- 64 de Vugt ME, Verhey FR. The impact of early dementia diagnosis and intervention on informal caregivers. *Prog Neurobiol*. 2013;110: 54-62.
- 65 Johannesen A, Helvik AS, Engedal K, Ulstein I, Sørli V. Prescribers-of-psychotropic-drugs-experiences-and-reflections-on-use-and-misuse-of-alcohol-and-psychotropic-drugs-among-older-people. *Qual Prim Care*. 2015;23(3):134-40.
- 66 Lid TG, Nesvåg S, Meland E. When general practitioners talk about alcohol: exploring facilitating and hampering factors for pragmatic case finding. *Scand J Public Health*. 2015; 43(2):153-8.
- 67 Mules T, Taylor J, Price R, Walker L, Singh B, Newsam P, et al. Addressing patient alcohol use: a view from general practice. *J Prim Health Care*. 2012;4(3):217-22.
- 68 Johannesen A, Engedal K, Helvik AS. Assessment of alcohol and psychotropic drug use among old-age psychiatric patients in Norway: experiences of health professionals. *Nordisk Alkohol Nark*. 2017;34(3):243-54.
- 69 Ministry of Health and Care Services. Se meg! En helhetlig rusmiddelpolitikk (Meld. St. 30). 2011-2012.
- 70 Hodgson LG, Cutler SJ. Help seeking for personal concerns about developing Alzheimer's disease. *J Appl Gerontol*. 2004;23(4):385-410.
- 71 Podhorna J, Winter N, Zobelein H, Perkins T, Walda S. Alzheimer's diagnosis: real-world physician behavior across countries. *Adv Ther*. 2020 Feb;37(2):883-93.
- 72 Cameron L, Leventhal EA, Leventhal H. Symptom representations and affect as determinants of care seeking in a community-dwelling, adult sample population. *Health Psychol*. 1993;12(3):171-9.
- 73 Croyle R, Barger S. Illness cognition. *Int Rev Health Psychol*. 1993;2:29-49.
- 74 Teel CS. Rural practitioners' experiences in dementia diagnosis and treatment. *Aging Ment Health*. 2004;8(5):422-9.
- 75 Ortiz F, Fitten LJ. Barriers to healthcare access for cognitively impaired older Hispanics. *Alzheimer Dis Assoc Disord*. 2000;14(3):141-50.
- 76 Kaufer DI, Cummings JL, Christine D, Bray T, Castellon S, Masterman D, et al. Assessing the impact of neuropsychiatric symptoms in Alzheimer's disease: the neuropsychiatric inventory caregiver distress scale. *J Am Geriatr Soc*. 1998;46(2):210-5.
- 77 de Vugt ME, Stevens F, Aalten P, Lousberg R, Jaspers N, Winkens I, et al. Do caregiver management strategies influence patient behaviour in dementia? *Int J Geriatr Psychiatry*. 2004 Jan;19(1):85-92.
- 78 Tan LL, Wong HB, Allen H. The impact of neuropsychiatric symptoms of dementia on distress in family and professional caregivers in Singapore. *Int Psychogeriatr*. 2005;17(2): 253-63.
- 79 Matsumoto N, Ikeda M, Fukuhara R, Shinagawa S, Ishikawa T, Mori T, et al. Caregiver burden associated with behavioral and psychological symptoms of dementia in elderly people in the local community. *Dement Geriatr Cogn Disord*. 2007;23(4):219-24.
- 80 Petrovic M, Hurt C, Collins D, Burns A, Camus V, Liperoti R, et al. Clustering of behavioural and psychological symptoms in dementia (BPSD): a European Alzheimer's disease consortium (EADC) study. *Acta Clin Belg*. 2007;62(6):426-32.
- 81 Gitlin LN, Marx KA, Stanley IH, Hansen BR, Van Haitsma KS. Assessing neuropsychiatric symptoms in people with dementia: a systematic review of measures. *Int Psychogeriatr*. 2014 Nov;26(11):1805-48.
- 82 Stella F, Laks J, Govone JS, De Medeiros K, Forlenza OV. Association of neuropsychiatric syndromes with global clinical deterioration in Alzheimer's disease patients. *Int Psychogeriatr*. 2016;28(5):779-86.
- 83 Thorsen O, Hartveit M, Baerheim A. General practitioners' reflections on referring: an asymmetric or non-dialogical process? *Scand J Prim Health Care*. 2012;30(4):241-6.
- 84 Agüero-Torres H, Fratiglioni L, Guo Z, Viitanen M, von Strauss E, Winblad B. Dementia is the major cause of functional dependence in the elderly: 3-year follow-up data from a population-based study. *Am J Public Health*. 1998;88(10):1452-6.
- 85 Tombaugh TN, McIntyre NJ. The mini-mental state examination: a comprehensive review. *J Am Geriatr Soc*. 1992;40(9):922-35.
- 86 Lancu I, Olmer A. [The minimal state examination: an up-to-date review]. *Harefuah*. 2006;145(9):687-701. Hebrew.