Research Article

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

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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 Examination (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

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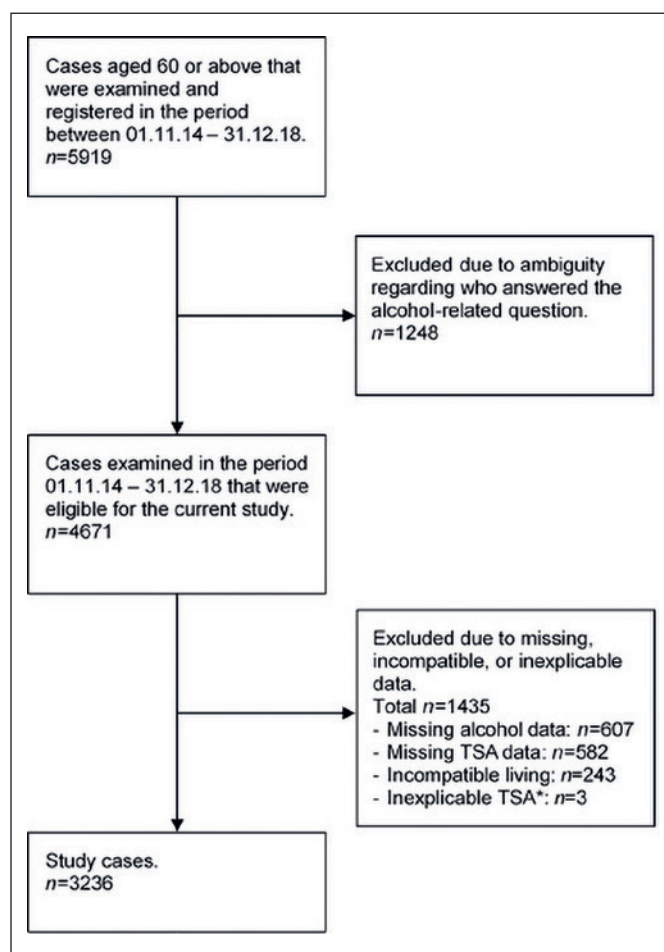
Fig. 1. Flowchart of the selection process. TSA, time from symptom debut to clinical assessment. *Two cases were excluded due to difficulties quantifying TSA accurately, and 1 case was excluded due to data suggesting the unlikely scenario that the case developed cognitive symptoms before the age of 20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

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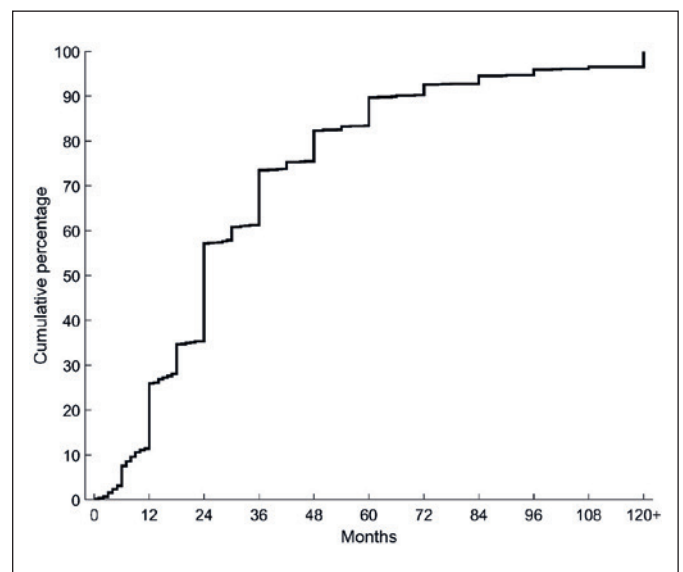
Fig. 2. Cumulative distribution of time from symptom debut to assessment (TSA).

depression and agitation subsyndrome scales of the NPI-Q was also associated with longer TSA. On the other hand, working ≥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.

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

Independent variables	Unadjusted model		Adjusted model	
	RC (95% CI)	*p* value	RC (95% CI)	*p* 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, *n*)	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, *n*	0.007 (−0.01; 0.03)	0.516	−0.006 (−0.03; 0.02)	0.610[16]
Medications, *n*	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-

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cades 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.

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.

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