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STUDIES IN HUMANS



Selenium intake is an effective strategy for the improvement of cognitive decline in low cognition older Americans

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ABSTRACT

Age-related cognitive decline is a prominent concern in older adults and selenium (Se) deficiency has been found to be associated with cognitive deficits. For the first time, the present study explored the association between Se intake and cognitive performance in older people with/without cognitive impairment using the data from the National Health and Nutrition Examination Survey 2011–2014. Weighted linear regression models were conducted to evaluate the association between dietary Se/total Se intakes and cognitive assessments. A total of 2387 participants were included. The significant positive association between dietary Se/total Se intakes and total scores of cognitive functioning tests existed only in the older people with low cognitive performance ($p < 0.001$), not in those with normal cognitive performance. In conclusion, Se intake was beneficial for cognitive decline only in the low cognition older people but failed in normal cognition older people.

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KEYWORDS

Se intake; cognition; national health and nutrition examination survey; older adults

Introduction

Cognitive decline, a prominent concern among the older population, might develop into mild cognitive impairment (MCI) and even dementia. Dementia, a dominant cause of disability and dependency for older people, affected approximately 55 million people and caused substantially financial burden worldwide (Zhang and Bai 2023). Unfortunately, to date, there is no effective therapy for dementia. Consequently, it is of great significance to prevent the occurrence of cognitive decline and delay its progression.

Selenium (Se), an important trace element in the synthesis of selenoproteins, plays critical roles in the maintenance of brain function and its deficiency has been found to be associated with cognitive deficits (Bai and Zhang 2023; Li et al. 2023). Extensive animal studies demonstrated cognitive deficits caused by Se deficiency (Castex et al. 2016) and cognitive impairments restored by Se supplementation (Ahmed et al. 2020; Zhang et al. 2021). To date, only three reported studies from the National Health and Nutrition Examination Survey (NHANES) have disclosed a positive association between Se intake and cognitive function (Ferdous et al. 2023; Li et al. 2019; Wang et al.

2022), in which the participants were not grouped by cognition status. Furthermore, Se supplementation did not prevent dementia among asymptomatic older men in the Prevention of Alzheimer's Disease (AD) by Vitamin E and Selenium primary prevention trial (Kryscio et al. 2017), suggesting that it was necessary to analyse an association between Se intake and cognitive function by grouping the cognition-based subjects. Therefore, for the first time, the present study explored the association between dietary Se/total Se intake and cognitive performance in older people with/without cognitive impairment using the data from the NHANES 2011–2014. Our work provides valuable hints for the prevention and improvement of cognitive decline among the elderly.

Materials and methods

Study design and participants

The NHANES was a program for the assessments of the health and nutritional status of children and adults in the USA and used a multistage probabilistic sampling to obtain a nationally representative sample. The survey was approved by the National Centre of Health

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Statistics Research Ethics Review Board and all the participants signed the informed consent. The present study combined the data from two consecutive cycles (NHANES 2011–2012 and 2013–2014) with a total of 19931 individuals and finally included 2387 participants for analysis following the exclusion criteria shown in Figure 1. No additional ethics approval was needed because the present study was based on a secondary analysis.

Se intake assessment

Se intakes from foods and supplements were obtained *via* two non-consecutive 24-h dietary recalls. The first dietary recall interview was collected in-person in the Mobile Examination Centre followed by the second interview *via* telephone 3 to 10 days later. The total Se intake was calculated by summing the average intakes of dietary Se and supplemental Se in two recall interviews. For each participant, estimates of nutrient intakes from each food or beverage, including Se intake, were calculated using the Food and Nutrient Database for Dietary Studies by the US Department of Agriculture that developed a computer-assisted food coding and data management system. According to the recommended dietary allowance and the tolerable upper intake level for Se, inadequate, adequate and excessive Se intakes were defined as dietary Se intake less than 55 µg/day, 55–400 µg/day and more than 400 µg/day, respectively (Institute of Medicine (US) Panel on Dietary Antioxidants and Related Compounds 2000).

Cognitive performance assessment

Cognitive performance was measured among the participants aged 60 years and older using the consortium to establish a registry for Alzheimer's disease (CERAD) word learning subtest, the animal fluency test (AFT) and the digit symbol substitution test (DSST). The

CERAD test consisted of three consecutive learning trials and a delayed recall. The delayed word recall occurred after AFT and DSST. In each learning test, the participants were required to read 10 unrelated words aloud once and recall as many words as possible after the order of these 10 words shown on a computer monitor was randomly changed. The full score of each test was 10 points, with one point for each correct word. The total score of three tests and one delayed recall was the total score of CERAD test. The AFT required participants to name animals as many as possible in one minute. A point was given for each named animal. The DSST exercise was conducted using a paper form containing 9 numbers paired with symbols. The participants needed to copy the corresponding symbols in the 133 boxes that adjoin the numbers within 2 min. The total score was the total number of correct matches with a maximum score of 133 points. The total score of cognitive assessments was calculated by summing the scores of each assessment. Due to the lack of gold standard for low cognitive performance, according to the reported method (Chen et al. 2017), the lowest quartile of the total score was considered low cognitive performance.

Study covariates

Factors that may affect cognition were used as covariates based on previous reports (Qin et al. 2020; Rexroth et al. 2013), including gender, age (60–69, 70–79 and ≥80), race (Mexican American, Other Hispanic, Non-Hispanic White, Non-Hispanic Black and Other Race-Including Multi-Racial), marital status (married/living with a partner, widow/divorced/separated and never married), educational status (<high school, high school and >high school), poverty income ratio (PIR, <1.3, 1.3–4.0 and ≥4.0), body mass index (BMI, <18.5, 18.5–25.0, 25.0–30.0 and ≥30.0), smoking status (every day, some days and not at all), alcohol use (yes or no), diabetes history (yes or no), stroke history (yes or no) and hypertension history (yes or no).

Statistical analysis

Considering the combination of two cycles of the NHANES data, the new sample weight (the original 2-year sample weight divided by 2) was constructed according to the analytical guidelines of the NHANES. Demographic variables were compared between those with and without cognitive impairment using

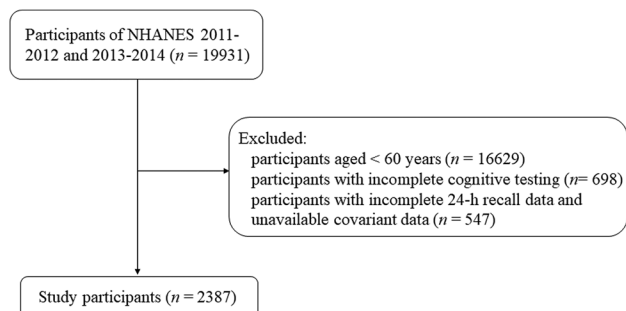


Figure 1. The Selection process of study participants.

Rao–Scott χ^2 test. Weighted linear regression models were conducted to evaluate the association between dietary Se/total Se intakes and cognitive assessments in the participants with low cognitive performance and normal cognitive performance. The first model (Model 1) was a crude model without adjusting any covariates. Model 2 was adjusted for gender, age, race, marital status, educational status, PIR, BMI, smoking status and alcohol use. Model 3 was a fully adjusted model for all the covariates. A $p < 0.05$ was considered statistically significant. All statistical analyses were conducted by SAS 9.4 version (SAS Institute Inc., Cary, NC, USA).

Results

Population characteristics

The demographic characteristics of the participants by the status of cognitive performance are presented in Table 1. The weighted mean total scores of the low cognition group and normal cognition group were 57.6 and 103.9, respectively. The weighted average values of dietary Se intake and total Se intake in the low cognition group were 88.5 and 106.4 $\mu\text{g}/\text{day}$, respectively, and those in the normal cognition group were 106.7 and 132.5 $\mu\text{g}/\text{day}$, respectively. The weighted percentages of participants with inadequate, adequate and excessive Se intakes were 9.0%, 90.9% and 0.1%, respectively, suggesting adequate Se intake in most participants. The weighted percentage of participants taking supplements was 38.3% and weighted average supplement doses were 64.3 $\mu\text{g}/\text{day}$. Significant differences were found in age, race, marital status, educational status, PIR, alcohol use, diabetes history, stroke history, hypertension history, dietary Se intake and total Se intake ($p < 0.001$) but not in gender, BMI and smoking status ($p > 0.05$) between two groups. The participants with cognitive impairment consumed less Se intake ($p < 0.001$).

Association between Se intake and cognitive performance

Table 2 shows the association between Se intake and cognitive performance. Among the population with cognitive impairment, there was a statistically significant positive association between dietary Se/total Se intakes and total score of cognitive performance with or without relevant adjustments of covariates ($p < 0.001$, Supplementary Tables 1 and 2). However, among those without cognitive impairment, no association was found between dietary Se intake and cognition with

or without adjustments of covariates, and the association between total Se intake and cognition only existed prior to adjustments of covariates ($p < 0.05$) not after adjustments of covariates (Supplementary Tables 3 and 4). Additionally, a statistically significant positive association only existed between Se intake and AFT among the low cognition participants ($p < 0.05$, Table 3). For CERAD and DSST, a significant positive association was not found after adjustments of all the covariates among the population with/without cognitive impairment (Tables 3 and 4).

Discussion

For the first time, the cognition-based grouping method was employed to evaluate the association between Se intake and cognitive performance in American adults. On the whole, the participants with cognitive decline had significantly lower Se intake compared to those without cognitive decline, which was in line with previous results (Cardoso et al. 2010; S. Li et al. 2019; Wang et al. 2022). The association between blood Se and cognitive impairment was inconsistent in previous studies (Cardoso et al. 2017; Cardoso et al. 2010; Ceballos-Picot et al. 1996; Rita Cardoso et al. 2014; Yan et al. 2020). People with lower cognitive scores had lower blood Se concentrations (Yan et al. 2020) and AD patients exhibited decreased blood Se levels (Cardoso et al. 2010; Rita Cardoso et al. 2014), however, there was no difference in serum Se concentration between AD patients and healthy subjects (Cardoso et al. 2017) and higher plasma Se concentration was observed in AD patients compared to healthy subjects (Ceballos-Picot et al. 1996). The inconsistent findings might be attributed to different Se exposure besides diets for the participants.

In the present study, a positive association between dietary Se/total Se intakes and cognitive performance was observed only in the low-cognition older population, which was in accordance with the previous result of the positive effects of 6-month Se-enriched Brazil nut consumption on cognitive functions such as verbal fluency and constructional praxis among older adults with MCI through the enhancement of blood Se concentration and glutathione peroxidase activity (Godos et al. 2022; Rita Cardoso et al. 2016). Increased oxidative stress and chronic inflammation with increasing age were closely related to age-associated cognitive decline (Cornejo and von Bernhardt 2016). Se exhibited antioxidant and anti-inflammation roles in neurons and produced positive cognitive effects (S. Zhang et al. 2010).

Table 1. Sociodemographic characteristics of the participants categorised by cognition status, NHANES 2011–2014.

Characteristic	Overall (n=2387)		Cognitive performance				P
	N	Weighted% (95%CI)	Low		Normal		
			n	Weighted% (95%CI)	n	Weighted% (95%CI)	
Gender							0.39
Male	1151	45.5 (43.6, 47.3)	326	48.0 (42.4, 53.7)	825	45.1 (42.7, 47.5)	
Female	1236	54.5 (52.7, 56.3)	253	52.0 (46.3, 57.6)	983	54.9 (52.5, 57.3)	
Age (y)							<0. 001
60–69	1286	56.6 (53.7, 59.4)	224	27.9 (20.5, 35.4)	1062	61.2 (58.0, 64.4)	
70–79	712	29.1 (26.6, 31.7)	199	36.1 (30.7, 41.5)	513	28.0 (25.1, 31.0)	
≥80	389	14.3 (12.5, 16.1)	156	36.0 (27.7, 44.2)	233	10.8 (9.3, 12.3)	
Race							<0.001
Mexican American	199	3.1 (1.6, 4.6)	71	7.8 (3.7, 12.0)	128	2.3 (1.2, 3.4)	
Other Hispanic	226	3.3 (2.0, 4.6)	100	10.9 (6.5, 15.3)	126	2.1 (1.3, 2.8)	
Non-Hispanic White	1212	81.6 (77.8, 85.4)	194	60.0 (50.4, 69.7)	1018	85.1 (82.1, 88.0)	
Non-Hispanic Black	551	7.7 (5.4, 10.1)	192	18.8 (12.3, 25.4)	359	5.9 (4.1, 7.8)	
Other Race-Including Multi-Racial	199	4.3 (3.3, 5.4)	22	2.4 (1.3, 3.5)	177	4.6 (3.4, 5.9)	
Marital status							<0.001
Married/living with partner	1399	65.8 (63.5, 68.1)	299	53.5 (47.3, 59.7)	1100	67.8 (65.5, 70.1)	
Widow/divorced/ separated	850	30.0 (28.2, 31.9)	247	41.4 (34.9, 47.8)	603	28.2 (26.3, 30.2)	
Never married	138	4.2 (3.2, 5.2)	33	5.1 (2.5, 7.7)	105	4.0 (2.9, 5.2)	
Educational status							<0.001
<High school	566	14.9 (11.8, 18.1)	310	45.0 (37.5, 52.5)	256	10.1 (7.5, 12.7)	
High school	573	22.4 (19.2, 25.5)	140	28.2 (22.3, 33.9)	433	21.4 (18.2, 24.6)	
>High school	1248	62.7 (59.0, 66.4)	129	26.8 (21.0, 32.5)	1119	68.5 (64.8, 72.1)	
PIR							<0.001
<1.3	619	16.0 (13.3, 18.8)	253	40.0 (33.2, 46.8)	366	12.3 (9.6, 14.9)	
1.3–4.0	977	45.1 (40.8, 49.5)	207	46.7 (41.8, 51.7)	770	44.9 (40.3, 49.4)	
≥4.0	610	38.9 (33.9, 43.7)	60	13.3 (9.4, 17.3)	550	42.9 (37.3, 48.4)	
BMI (kg/m²)							0.10
<18.5	34	1.4 (0.8, 2.0)	15	2.8 (1.7, 3.9)	19	1.2 (0.5, 1.9)	
18.5–25.0	598	25.3 (22.4, 28.2)	146	29.0 (23.2, 34.7)	452	24.7 (21.3, 28.2)	
25.0–30.0	829	35.9 (33.6, 38.3)	192	33.5 (28.9, 38.1)	637	36.3 (33.7, 38.9)	
≥30.0	896	37.4 (34.7, 40.0)	208	34.7 (26.9, 42.6)	688	37.8 (34.8, 40.8)	
Smoking status							0.45
Every day	238	17.5 (14.5, 20.5)	64	18.4 (12.8, 24.1)	174	17.4 (14.4, 20.4)	
Some days	44	2.3 (1.4, 3.2)	15	3.4 (1.6, 5.0)	29	2.1 (1.1, 3.2)	
Not at all	932	80.2 (77.2, 83.2)	216	78.2 (72.6, 83.9)	716	80.5 (77.5, 83.4)	
Alcohol use							<0.001
Yes	1645	73.6 (70.8, 76.4)	359	60.4 (55.5, 65.2)	1286	75.7 (72.8, 78.6)	
No	729	26.4 (23.6, 29.2)	212	39.6 (34.8, 44.5)	517	24.3 (21.4, 27.2)	
Diabetes history							<0.001
Yes	589	20.0 (17.9, 22.1)	201	32.8 (26.9, 38.8)	388	18.0 (15.5, 20.4)	
No	1798	80.0 (77.9, 82.1)	378	67.2 (61.2, 73.1)	1420	82.0 (79.6, 84.5)	
Stroke history							<0.001
Yes	156	6.0 (5.0, 6.9)	66	11.4 (7.6, 15.3)	90	5.1 (4.1, 6.1)	
No	2231	94.0 (93.1, 95.0)	513	88.6 (84.7, 92.4)	1718	94.9 (93.9, 95.9)	
Hypertension history							<0.001
Yes	1476	58.0 (55.0, 60.9)	395	70.5 (65.8, 75.2)	1081	55.9 (52.6, 59.2)	
No	911	42.0 (39.1, 45.0)	184	29.5 (24.8, 34.2)	727	44.1 (40.8, 47.4)	
Dietary Se intake (µg/d)							<0.001
10.6–70.8	595	22.4 (20.0, 24.8)	210	36.6 (31.9, 41.3)	385	20.1 (17.8, 22.5)	
70.9–95.0	597	24.3 (21.4, 27.2)	146	27.0 (22.1, 31.8)	451	23.9 (20.9, 26.8)	
95.1–122.9	597	26.4 (24.0, 28.7)	125	23.0 (18.9, 27.1)	472	26.9 (24.2, 29.6)	
123.0–839.7	598	26.9 (24.0, 29.8)	98	13.5 (10.1, 16.9)	500	29.1 (26.0, 32.1)	
Total Se intake (µg/d)							<0.001
10.6–79.6	594	22.0 (19.9, 24.0)	205	35.8 (30.7, 40.8)	389	19.7 (17.7, 21.8)	
79.7–110.9	599	23.9 (21.6, 26.2)	159	28.1 (24.3, 31.9)	440	23.2 (20.6, 25.9)	
111.0–152.6	597	25.9 (23.8, 28.1)	122	20.6 (16.2, 25.0)	475	26.8 (24.4, 29.2)	
152.7–894.7	597	28.2 (25.6, 30.8)	93	15.5 (11.8, 19.3)	504	30.2 (27.3, 33.2)	

Se: selenium.

BMI: Body mass index, it was calculated as weight in kilograms divided by height in metres squared and then rounded to one decimal place.

PIR: Poverty income ratio, it was calculated by dividing total annual family (or individual) income by the poverty guidelines specific to the survey year.

Table 2. Association between Se intakes and cognition assessed by weighted linear regression models, NHANES 2011–2014.

	Se intake (µg/day)	Model 1		Model 2		Model 3	
		β (95% CI)	<i>P</i>	β (95% CI)	<i>P</i>	β (95% CI)	<i>P</i>
Low cognitive performance	Dietary Se	0.03 (0.02, 0.05)	<0.001	0.04 (0.02, 0.06)	<0.001	0.04 (0.02, 0.06)	<0.001
	Total Se	0.03 (0.02, 0.05)	<0.001	0.04 (0.02, 0.05)	<0.001	0.03 (0.02, 0.05)	<0.001
Normal cognitive performance	Dietary Se	0.01 (-0.01, 0.03)	0.18	0.00 (-0.02, 0.02)	0.92	0.00 (-0.02, 0.02)	0.91
	Total Se	0.02 (0.00, 0.04)	<0.05	0.01 (-0.00, 0.03)	0.07	0.01 (0.00, 0.03)	0.12

Se: selenium.

Model 1: Unadjusted any covariates.

Model 2: Adjusted for gender, age, race, marital status, educational status, PIR, BMI, smoking status and alcohol use.

Model 3: Adjusted for gender, age, race, marital status, educational status, PIR, BMI, smoking status, alcohol use, diabetes history, stroke history and hypertension history.

Table 3. Association between Se intakes and CERAD score/AFT score/DSST score in the participants with low cognitive performance, NHANES 2011–2014.

	Se intake (µg/day)	Model 1		Model 2		Model 3	
		β (95% CI)	<i>P</i>	β (95% CI)	<i>P</i>	β (95% CI)	<i>P</i>
Low cognitive performance				CERAD test			
	Dietary Se	0.00 (0.00, 0.01)	0.69	0.01 (0.00, 0.02)	0.14	0.01 (0.00, 0.02)	0.15
	Total Se	0.00 (0.00, 0.00)	0.84	0.01 (0.00, 0.01)	0.18	0.01 (0.00, 0.01)	0.14
				Animal fluency test			
	Dietary Se	0.02 (0.01, 0.02)	<0.001	0.02 (0.01, 0.03)	<0.05	0.02 (0.01, 0.03)	<0.001
	Total Se	0.02 (0.01, 0.02)	<0.05	0.01 (0.00, 0.02)	<0.05	0.01 (0.00, 0.02)	<0.05
				DSST test			
	Dietary Se	0.01 (-0.01, 0.04)	0.19	0.01 (-0.01, 0.03)	0.21	0.02 (0.00, 0.04)	0.12
	Total Se	0.02 (0.01, 0.04)	<0.05	0.02 (0.00, 0.03)	<0.05	0.02 (0.00, 0.03)	0.05

Se: selenium.

Model 1: Unadjusted any covariates.

Model 2: Adjusted for gender, age, race, marital status, educational status, PIR, BMI, smoking status and alcohol use.

Model 3: Adjusted for gender, age, race, marital status, educational status, PIR, BMI, smoking status, alcohol use, diabetes history, stroke history and hypertension history.

CERAD: consortium to establish a registry for Alzheimer's disease.

DSST: digit symbol substitution test.

Table 4. Association between Se intakes and CERAD score/AFT score/DSST score in the participants with normal cognitive performance, NHANES 2011–2014.

	Se intake (µg/day)	Model 1		Model 2		Model 3	
		β (95% CI)	<i>P</i>	β (95% CI)	<i>P</i>	β (95% CI)	<i>P</i>
Normal cognitive performance				CERAD test			
	Dietary Se	0.00 (-0.01, 0.00)	0.73	0.00 (0.00, 0.01)	0.40	0.00 (0.00, 0.01)	0.50
	Total Se	0.00 (0.00, 0.00)	0.73	0.00 (0.00, 0.00)	0.46	0.00 (0.00, 0.00)	0.58
				Animal fluency test			
	Dietary Se	0.01 (0.01, 0.02)	0.003	0.00 (0.00, 0.01)	0.73	0.00 (-0.01, 0.01)	0.77
	Total Se	0.01 (0.00, 0.01)	<0.05	0.00 (0.00, 0.01)	0.15	0.00 (0.00, 0.01)	0.17
				DSST test			
	Dietary Se	0.00 (-0.01, 0.02)	0.60	0.00 (-0.02, 0.01)	0.70	0.00 (-0.02, 0.01)	0.59
	Total Se	0.01 (0.00, 0.02)	<0.05	0.01 (0.00, 0.02)	0.09	0.01 (0.00, 0.02)	0.14

Se: selenium.

Model 1: Unadjusted any covariates.

Model 2: Adjusted for gender, age, race, marital status, educational status, PIR, BMI, smoking status and alcohol use.

Model 3: Adjusted for gender, age, race, marital status, educational status, PIR, BMI, smoking status, alcohol use, diabetes history, stroke history and hypertension history.

CERAD: consortium to establish a registry for Alzheimer's disease.

DSST: digit symbol substitution test.

Mechanisms of Se on cognition also included regulation of levels of metals including zinc and copper (Cu), inhibition of ferroptosis, modulation of *de novo* synthesis of L-serine and PI3K/AKT/mTOR/GSK3 and

improvement of synaptic plasticity (Bai et al. 2024; Shehata et al. 2024).

There was no association between dietary Se/total Se intakes and cognitive performance in the normal

cognition older people in our study. A previous study on the association between Se status and cognitive performance in Australia showed that plasma Se was not related to cognitive outcomes in healthy older adults (Cardoso et al. 2018), partly because the maximisation of selenoprotein synthesis associated with adequate Se resulted in the plateaued expression of selenoproteins (Cardoso et al. 2018; Thomson 2004). Our regression analysis indicated that gender, age, race, education, PIR, smoking, alcohol use and diabetes history affected cognition among older adults with normal cognitive performance (Supplementary Tables 3 and 4). In terms of gender and race, the females and white race older population had better cognitive performance, which was in line with previous results (Rexroth et al. 2013; Van der Elst et al. 2005). As for age, the participants with normal cognitive performance were significantly younger than those with cognitive impairment, which was in agreement with previous results (Rexroth et al. 2013). Results of no relationship between marital status and cognition (Supplementary Tables 3 and 4) were consistent with previous reports (Qin et al. 2020; Scherr et al. 1988). Regarding BMI, it was not related to cognition in the present study (Supplementary Tables 3 and 4), however, some previous studies reported contradictory results with obesity being positively or negatively associated with cognition in the older population, partly because BMI was not an accurate marker of body composition with increasing age (Smith et al. 2011). Smoking was not beneficial to cognition in our study probably due to the nervous system damage caused by cigarettes, but current smokers had slightly better immediate memory performance and there was no relation between smoking and delayed memory, attention and orientation (Scherr et al. 1988). Different from the previous findings that alcohol consumption was not significantly related to cognitive performance (Scherr et al. 1988), our results demonstrated that alcohol consumption was correlated to better cognitive performance. Similarly, low dose of ethanol even significantly facilitated cognition under some certain task conditions (Rossetti et al. 2002). Additionally, the effects of alcohol on nutrition were negligible for the well-nourished non-alcoholic elderly and age did not affect the rate of absorption and elimination of alcohol (Dufour et al. 1992). The participants with higher levels of income and education perhaps had more cognitive reserve, *i.e.* income-education-cognition, which was supported by the previous study (Cagney and Lauderdale 2002). Similar to the previous findings

(Rexroth et al. 2013), diabetes was found to be negatively associated with greater cognition in the present study. This study showed no effect of stroke on cognition (Supplementary Tables 3 and 4), in contrast to the result that those with stroke history performed more poorly on the memory test, which might be because the stroke pathology involved the brain (Scherr et al. 1988). Hypertension did not affect cognition in this study (Supplementary Tables 3 and 4) possibly because hypertension did not directly affect the brain. We speculated that these factors (gender, age, race, education, PIR, smoking, alcohol use, diabetes history) affected the association between Se intake and cognition in the normal cognition older population.

Strengths and limitations

The present study had some strengths. A large national representative sample of older adults and the high-quality NHANES datasets were used. The weighted linear regression models were conducted considering relevant covariates. The cognition-based grouping method was first used for the assessment of the dietary Se/total Se intakes and cognitive performance. Several limitations exist as follows. First, the NHANES was a cross-sectional study that failed to evaluate the causal association between Se intake and cognition. Second, the inevitable recall bias appeared in 24-h dietary recalls which only reflected short-term Se intake. Forms of Se compounds were not available in the NHANES. Some of the low cognitive performance-associated metals (Balachandran et al. 2020; Santa Maria et al. 2019) were not assessed in the diets of the NHANES participants. Third, CREAD, AFT and DSST used in the NHANES did not comprehensively represent cognitive ability. Finally, a classification standard for cognition status was not clearly defined in the NHANES.

Conclusion

The positive effects of Se intake on cognition depended on the status of cognitive performance in the older adults, that is to say, Se intake was beneficial for cognitive decline only in the low cognition older people, but failed in normal cognition older people. For older adults, developing more healthy lifestyles and avoiding risk factors may be helpful in the prevention of cognitive decline. More prospective studies are required to explore the effects of long-term Se intake on cognition in the future.

Authors' contributions

Ya-Zhi Bai: preparation of original draft, generation, collection, assembly, analysis and interpretation of data. Shuang-Qing Zhang: conceptualisation, supervision, review and editing. All authors read and approved the final manuscript.

Disclosure statement

The authors report there are no competing interests to declare.

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Data availability statement

Datasets are publicly available at <https://www.cdc.gov/nchs/nhanes/index.htm>.

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