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Dietary sodium/potassium intake and cognitive impairment in older patients with hypertension: Data from NHANES 2011–2014

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Abstract

This study aimed to assess the relationship between dietary sodium/potassium intake and cognition in elderly individuals with hypertension. We designed a cross-sectional study based on the 2011–2014 National Health and Nutrition Examination Survey (NHANES) 2011–2014. A multivariable-logistic regression analysis was performed to analyze the relationship between sodium/potassium intake and cognitive impairment. Restricted cubic spline (RCS) based on regression analysis to assess the nonlinear dose-response relationship between dietary sodium intake and cognitive performance. Out of the 2276 participants included in this study, 1670 patients had hypertension. Compared with the lowest quartile of dietary sodium intake, the lowest weighted odds ratio of cognitive impairment in DSST was observed in Q4 (OR = 0.45, 0.29–0.70), and a similar trend was observed in AFT (OR = 0.34, 0.18–0.65). After adjusting the covariates, the lowest weighted multivariable-adjusted OR of cognitive impairment in DSST were also observed in Q4 (OR = 0.47, 0.26-0.84) compared with the lowest quartile of dietary sodium intake. The RCS results showed that dietary sodium intake was U-shaped and associated with the risk of cognitive impairment in the DSST ($P_{non-linearity}$ = 0.0067). In addition, no significant association was observed between dietary potassium intake and different dimensions of cognitive performance. In conclusion, excessively high and low low dietary sodium were associated with impairment of specific processing speed, sustained attention, and working memory for elderly patients with hypertension in the United States. However, no association was observed between dietary potassium intake and cognition.

KEYWORDS
cognition, dietary sodium, hypertension

[#]Chengkun Kou and Xu Zhao contributed equally to this study.

1 | INTRODUCTION

Salt is a widely used condiment worldwide, and sodium chloride is its main component. Sodium is an essential macronutrient that maintains fluid balance and cellular homeostasis[1] and is involved in nerve conduction[2] in the human body. However, a high level of dietary sodium intake is considered to be associated with hypertension, a major risk factor for cardiovascular disease (CVD).[3] A high level of dietary sodium intake is also a primary dietary risk for death and disability-adjusted life-years in China, Japan, and Thailand.[4] In 2017, the estimated global mean salt intake was 6 g/day, greatly exceeding optimal levels.[4]

Properly managing sodium intake is a highly effective way to significantly reduce the risk of cardiovascular disease, and dietary sodium intake has been considered a common modifiable risk factor for hypertension.[5–7] Additionally, potassium-containing salts have been used as substitutes for sodium.[8–10] Many studies have shown that high potassium intake may lower blood pressure and CVD risk partly,[11–13] and high potassium intake may mitigate the negative impacts of high sodium intake.[14]

Cognitive impairment is linked to cardiovascular risk factors in the general population,[15] and diet is considered an important factor in cognitive function.[16–18] It is suggested that dietary sodium may influence cognition through cerebrovascular and cerebral blood flow.[19,20] Animal experiments have shown that tau hyperphosphorylation can be induced by high levels of dietary sodium, followed by cognitive impairment[21]; nonetheless, their findings have remained controversial. A prospective cohort study has reported that sodium and potassium intake were not associated with cognitive impairment.[22] Another cross-sectional study has shown that lower sodium intake was associated with an increased risk of cognitive impairment in older adults.[23]

We have hypothesized that both excessively high and low levels of dietary sodium intake may increase the risk of cognitive impairment. Accordingly, this study was performed to evaluate the association between sodium intake and cognitive impairment in older individuals with hypertension. In this study, we aimed to establish a continuous dose-response relationship between sodium and cognitive impairment and assess the relationship between dietary potassium intake and cognition.

2 | METHODS

2.1 | Study design

The National Health and Nutrition Examination Surveys (NHANES) is a program of studies designed to assess the health and nutritional status of adults and children in the United States. The program aims to select a representative sample of approximately 10,000 individuals every 2 years on a national level. The survey includes information about demographic, socioeconomic, dietary, and health-related questions, as well as physical examinations comprising medical, dental, and physiological measurements and laboratory tests.

2.2 | Study population

Data were extracted from the 2011–2012 and 2013–2014 NHANES database. The participants with complete demographic data, smoking and alcohol use information, Hemal biochemistry data, and cognitive data were included in this study. All the participants included in this study were aged 60 years or older.

The protocols of NHANES were approved by the National Center for Health Statistics Research Ethics Review Board. Written informed consent was obtained from all participants. According to the policy of our local Research Ethics Committee, the published available data does not need a secondary review.

2.3 | Diagnosis of hypertension

Participants were considered hypertensive if they met the following criteria: (1) they had been told by a doctor or other health professional that they had hypertension (also called high blood pressure), (2) self-reported antihypertensive drug use, and (3) had a high blood pressure measurement (SBP $\geq$ 140 mmHg or DBP $\geq$ 90 mmHg). The NHANES consists of three consecutive blood pressure measurements and an additional measurement if required.

2.4 | Cognitive tests

Cognition was assessed using a questionnaire that included (1) word learning and recall modules from the Consortium to Establish a Registry for Alzheimer's Disease (CERAD),[24] (2) the Animal Fluency test (AFT),[25] and (3) the Digit Symbol Substitution Test (DSST)[26] in NHANES. The CERAD test assesses the immediate and delayed learning abilities for new verbal information. The test consisted of three consecutive learning trials and a delayed-recall trial. We calculated the sum of the three learning and recall trial scores as the final score of the CERAD test. The AFT was used to examine the executive function, and the DSST was used to assess processing speed, sustained attention, and working memory. We also extracted the scores of the AFT and DSST in the NHANES as indices of cognition.

However, there is no acknowledged threshold for the DSST, CERAD, and AFT to distinguish cognitive impairment. Age was considered a significant confounding factor for performance on cognitive tests. Therefore, the participants were stratified according to age: 60–65 years, 66–70 years, 71–75 years, and $\geq$76 years. We used the 25th percentile of the score according to age stratification as the threshold for identifying cognitive impairment.

2.5 | Dietary sodium intake

It is considered that 24-h urine sodium excretion is the gold standard for monitoring sodium intake. However, this method is complicated.

Previous studies have confirmed that dietary recall has a significant dose-response association with estimated 24-h urine sodium excretion,[27,28] and it is another main method for accessing salt intake.[29] In this study, dietary sodium intake was extracted from 24-h dietary recall interviews in the NHANES database and expressed in milligrams. The U.S. Department of Agriculture Food and Nutrient Database for Dietary Studies was used to process the total daily nutrient intake of food and beverages.

2.6 | Covariates

We also included other variables of interest as covariates consisting of age, sex (male and female), race (Non-Hispanic White, Mexican American, Non-Hispanic Black, Other Hispanic, and Other Race), education (less than 9th grade, 9–11th grade, high school graduate; some college or AA degree, college graduate or above), body mass index (BMI), SBP, DBP, total energy intake, dietary potassium intake, serum sodium, creatinine, smoking, alcohol use, diabetes, stroke, and use of antihypertensive drugs.

2.7 | Statistical analysis

All statistical analyses in this study were performed using R version 4.1.3 (R Foundation for Statistical Computing, Vienna, Austria https://www.r-project.org/). Survey package version 4.1-1 and NHANES R package version 0.9.3.8 were used to analyze complex survey samples. The raw data are shown in Supplementary file 1. Continuous variables in this study are expressed as mean (standard error, SE), while categorical variables are expressed as frequencies (percentages). Dietary sodium intake was grouped into quantiles from lowest (first quantile, Q1) to highest (fifth quantile, Q5). We compared the covariates of participants with and without cognitive impairment. Student's *t*-test was used to compare continuous variables, while the Rao-Scott chi-square test was used to compare categorical variables. Logistic regression models were used to analyze the relationship between sodium intake and cognitive impairment. Model 1 was established using a binary logistic regression. Model 2 was adjusted for age and sex. Model 3 was adjusted for age, sex, race, education, BMI, SBP, DBP, total energy intake, dietary potassium intake, serum sodium, creatinine, smoking, alcohol use, diabetes, stroke, and antihypertensive drug use. Additionally, we performed a restricted cubic spline (RCS) to assess the nonlinear dose-response relationship between dietary sodium intake and cognitive performance after adjustment for all covariates.

3 | RESULTS

3.1 | Characteristics of study population

In this study, 19,931 participants were included in the NHANES from 2011 to 2014. After excluding participants with incomplete data, 2276 participants were included in this study, of which 1670 patients had hypertension (Figure 1).

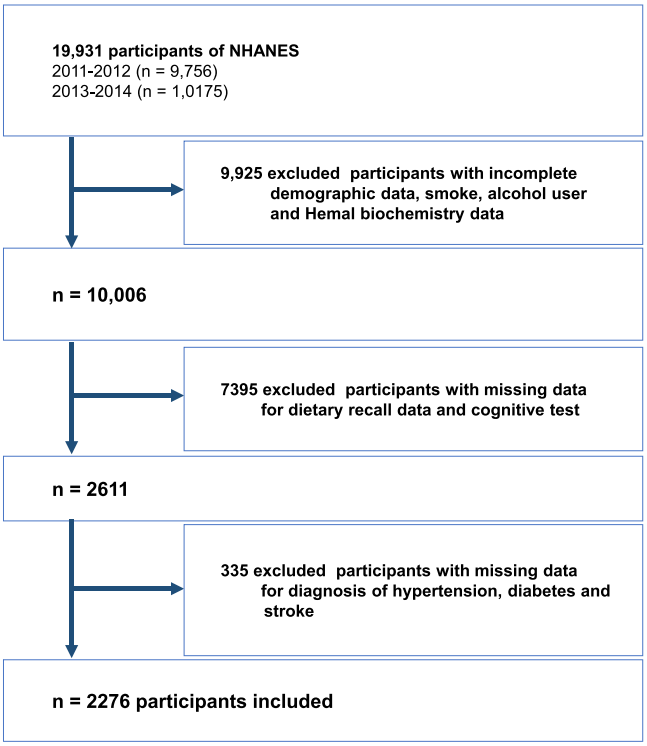

FIGURE 1 Flow diagram of study identification, screening, eligibility assessment, and inclusion.

The mean dietary sodium intake of all participants was 3183.31 mg/day, which was similar to that of participants with hypertension (3159.13 mg/day). Dietary sodium intake was grouped into quantiles from lowest (first quantile, Q1) to highest (fifth quantile, Q5). Table 1 shows the clinical characteristics of the participants with hypertension according to dietary sodium intake quintiles. The results indicate that there were significant differences between different dietary sodium intake levels in the distribution of age, sex, race, education, dietary energy, dietary potassium, creatinine, alcohol use, diabetes, and stroke among patients with hypertension. Moreover, there were significant differences between different dietary potassium intake levels in the distribution of sex, race, education, digit symbol score test, animal fluency score, dietary energy, dietary sodium, serum potassium, serum sodium, alcohol use, and smoking among patients with hypertension (Table S1). Tables S2–S3 show the clinical characteristics of all participants.

3.2 | Dietary sodium intake and cognition

A logistic regression model was used to assess the association between dietary sodium intake and cognitive performance in all participants (Table 2). Compared with the lowest quartile of dietary sodium intake, the lowest weighted odds ratios of cognitive impairment in DSST were observed in Q4 (OR = 0.42, 0.31–0.57), and a similar trend

TABLE 1 Characteristics of the study population with hypertension.

	Total daily dietary sodium (mg/day)						
	Total $N = 1610$	Q1 (286–2076) $N = 333$	Q2 (2076–2604) $N = 334$	Q3 (2604–3226) $N = 328$	Q4 (3226–3944.5) $N = 316$	Q5 (3944.5–8765) $N = 299$	*p*-value
Age (years)[a]	69.79 (0.33)	71.68 (0.72)	70.39 (0.68)	69.85 (0.61)	69.98 (0.48)	67.74 (0.59)	**<0.001**
Sex, *n* (%)[b]							**<0.0001**
Female	845 (52.48)	241 (79.71)	220 (74.11)	165 (51.78)	134 (47.02)	85 (23.05)	
Male	765 (47.52)	92 (20.29)	114 (25.89)	163 (48.22)	182 (52.98)	214 (76.95)	
Race/ethnicity, *n* (%)[b]							**0.01**
Non-Hispanic White	789 (49.01)	136 (68.44)	167 (79.79)	168 (76.79)	169 (78.86)	149 (83.50)	
Mexican American	128 (7.95)	32 (5.80)	22 (2.14)	27 (3.20)	21 (3.56)	26 (2.90)	
Non-Hispanic Black	419 (26.02)	93 (13.40)	92 (10.69)	81 (9.50)	73 (8.84)	80 (7.96)	
Other Hispanic	153 (9.5)	49 (6.83)	39 (5.00)	24 (3.82)	25 (2.85)	16 (1.35)	
Other Race	121 (7.52)	23 (5.53)	14 (2.39)	28 (6.69)	28 (5.89)	28 (4.29)	
Education, n (%)[b]							**0.02**
Less than 9th grade	160 (9.94)	53 (11.22)	29 (5.32)	32 (5.34)	25 (4.79)	21 (4.48)	
9–11th grade	234 (14.53)	61 (11.39)	50 (11.50)	52 (11.56)	34 (8.04)	37 (9.47)	
High school graduate/GED or equivalent	411 (25.53)	86 (34.12)	92 (22.69)	82 (24.58)	76 (20.76)	75 (23.19)	
Some college or AA degree	468 (29.07)	88 (28.59)	110 (37.04)	83 (24.80)	105 (38.13)	82 (25.77)	
College graduate or above	337 (20.93)	45 (14.68)	53 (23.45)	79 (33.72)	76 (28.27)	84 (37.09)	
BMI ($kg \cdot m^2$)[a]	29.93 (0.32)	29.27 (0.55)	29.35 (0.61)	30.04 (0.67)	29.45 (0.38)	31.18 (0.77)	0.2
Digit symbol score test[a]	51.08 (0.86)	46.30 (1.53)	50.44 (1.40)	50.96 (1.35)	53.64 (1.69)	52.94 (1.11)	**0.01**
Animal fluency score total[a]	17.54 (0.25)	16.13 (0.45)	17.22 (0.51)	17.45 (0.51)	18.35 (0.46)	18.18 (0.66)	**0.04**
CERAD score[a]	25.57 (0.41)	24.59 (0.56)	25.20 (0.80)	25.42 (0.38)	25.83 (0.57)	26.47 (0.62)	0.07
SBP (mmHg)[a]	135.35 (0.71)	137.53 (1.36)	135.11 (2.12)	135.95 (1.32)	136.44 (1.41)	132.56 (1.32)	0.05
DBP (mmHg)[a]	68.73 (0.54)	68.15 (1.13)	68.95 (0.72)	68.86 (1.09)	68.07 (1.34)	69.42 (0.98)	0.92
Dietary energy (kcal)[a]	1867.68 (28.62)	1125.52 (16.23)	1532.68 (29.67)	1810.88 (19.35)	2043.87 (30.00)	2567.95 (60.07)	**<0.0001**
Dietary sodium (mg)[a]	3159.13 (63.96)	1635.79 (27.00)	2343.73 (11.08)	2929.88 (15.52)	3531.09 (17.82)	4787.03 (80.07)	**<0.0001**
Dietary potassium (mg)[a]	2628.31 (36.71)	1719.78 (45.64)	2317.90 (69.58)	2591.03 (49.39)	2844.93 (65.21)	3375.95 (87.91)	**<0.0001**
Creatinine (umol/L)[a]	88.73 (0.99)	88.47 (3.62)	84.23 (1.78)	89.01 (1.74)	88.42 (2.79)	92.65 (2.38)	0.14

(Continues)

TABLE 1 (Continued)

	Total daily dietary sodium (mg/day)						
	Total $N = 1610$	Q1 (286–2076) $N = 333$	Q2 (2076–2604) $N = 334$	Q3 (2604–3226) $N = 328$	Q4 (3226–3944.5) $N = 316$	Q5 (3944.5–8765) $N = 299$	*p*-value
Serum potassium (mmol/L)[a]	4.07 (0.02)	4.01 (0.05)	4.04 (0.03)	4.08 (0.02)	4.07 (0.03)	4.11 (0.06)	0.36
Serum sodium (mmol/L)[a]	139.40 (0.14)	139.28 (0.25)	139.37 (0.25)	139.24 (0.17)	139.57 (0.21)	139.51 (0.30)	0.66
Alcohol user, n (%)[b]							**0.01**
Yes	913 (56.71)	155 (49.19)	189 (63.30)	193 (67.99)	176 (63.65)	200 (70.52)	
No	697 (43.29)	178 (50.81)	145 (36.70)	135 (32.01)	140 (36.35)	99 (29.48)	
Smoke, *n* (%)[b]							0.45
Yes	184 (11.43)	40 (11.00)	37 (7.73)	36 (6.53)	27 (7.96)	44 (7.76)	
No	1426 (88.57)	293 (89.00)	297 (92.27)	292 (93.47)	289 (92.04)	255 (92.24)	
Diabetes, *n* (%)[b]							**0.04**
Yes	597 (37.08)	145 (35.92)	117 (23.10)	129 (38.58)	108 (28.29)	98 (28.55)	
No	1013 (62.92)	188 (64.08)	217 (76.90)	199 (61.42)	208 (71.71)	201 (71.45)	
Stroke, *n* (%)[b]							0.21
Yes	130 (8.07)	39 (10.71)	18 (4.32)	25 (8.67)	27 (8.74)	21 (5.29)	
No	1480 (91.93)	294 (89.29)	316 (95.68)	303 (91.33)	289 (91.26)	278 (94.71)	
Antihypertensive drug user, *n* (%)[b]							0.73
Yes	311 (19.32)	68 (20.89)	66 (19.97)	65 (18.34)	53 (15.99)	59 (16.74)	
No	1299 (80.68)	265 (79.11)	268 (80.03)	263 (81.66)	263 (84.01)	240 (83.26)	

Abbreviations: BMI, body mass index; CERAD, Consortium to Establish a Registry for Alzheimer's Disease; DBP, diastolic blood pressure; SBP, systolic blood pressure.

[a]Values shown are mean (standard error).

[b]Values shown are numbers (weighted percentage).

TABLE 2 Weighted odds ratios (95% confidence interval) of cognitive impairment by quartiles of sodium intake in all participants.

	DSST						
Dietary sodium (mg/day)	**Case, *n* (%)**	**Model 1***	***P*-value**	**Model 2***	***P*-value**	**Model 3***	***P*-value**
Q1 (286–2076)	224 (32.77)	1.00 (Ref.)	–	1.00 (Ref.)	–	1.00 (Ref.)	–
Q2 (2076–2604)	171 (21.35)	**0.56 (0.37–0.84)**	**0.01**	**0.51 (0.34–0.77)**	**0.002**	0.60 (0.30–1.23)	0.13
Q3 (2604–3226)	146 (21.00)	**0.55 (0.39–0.77)**	**0.001**	**0.46 (0.32–0.68)**	**<0.001**	0.68 (0.33–1.39)	0.24
Q4 (3226–3944.5)	144 (17.01)	**0.42 (0.31–0.57)**	**<0.0001**	**0.34 (0.23–0.48)**	**<0.0001**	**0.47 (0.26–0.84)**	**0.02**
Q5 (3944.5–8765)	164 (23.94)	**0.65 (0.44–0.95)**	**0.03**	**0.45 (0.29–0.69)**	**<0.001**	0.75 (0.32–1.74)	0.44
	AFT						
Dietary sodium (mg/day)	**Case, n (%)**	**Model 1***	***P*-value**	**Model 2***	***P*-value**	**Model 3***	***P*-value**
Q1 (286–2076)	178 (30.54)	1.00 (Ref.)	–	1.00 (Ref.)	–	1.00 (Ref.)	–
Q2 (2076–2604)	147 (23.62)	0.70 (0.42,1.17)	0.17	0.69 (0.40,1.19)	0.17	0.93 (0.43,2.03)	0.83
Q3 (2604–3226)	129 (23.91)	0.71 (0.44,1.15)	0.16	0.71 (0.42,1.20)	0.19	1.05 (0.46,2.38)	0.89
Q4 (3226–3944.5)	109 (16.05)	**0.43 (0.26,0.73)**	**0.003**	**0.42 (0.24,0.74)**	**0.004**	0.69 (0.28,1.70)	0.35
Q5 (3944.5–8765)	106 (17.68)	**0.49 (0.31,0.78)**	**0.004**	**0.47 (0.28,0.79)**	**0.01**	0.92 (0.40,2.15)	0.82
	CERAD test						
Dietary sodium (mg/day)	**Case, *n* (%)**	**Model 1***	***P*-value**	**Model 2***	***P*-value**	**Model 3***	***P*-value**
Q1 (286–2076)	166 (30.06)	1.00 (Ref.)	–	1.00 (Ref.)	–	1.00 (Ref.)	–
Q2 (2076–2604)	136 (28.00)	0.90 (0.58–1.42)	0.65	0.82 (0.51–1.33)	0.4	1.06 (0.59–1.91)	0.83
Q3 (2604–3226)	131 (21.44)	**0.64 (0.42–0.96)**	**0.03**	**0.53 (0.31–0.89)**	**0.02**	0.69 (0.34–1.41)	0.25
	CERAD test						
Dietary sodium (mg/day)	**Case, *n* (%)**	**Model 1***	***P*-value**	**Model 2***	***P*-value**	**Model 3***	***P*-value**
Q4 (3226–3944.5)	127 (23.09)	0.70 (0.46–1.06)	0.09	**0.55 (0.36–0.83)**	**0.01**	0.80 (0.41–1.58)	0.45
Q5 (3944.5–8765)	132 (24.11)	0.74 (0.47–1.16)	0.18	**0.51 (0.28–0.91)**	**0.02**	0.82 (0.39–1.73)	0.53

Abbreviations: AFT, Animal fluency score total; CERAD, Consortium to Establish a Registry for Alzheimer's Disease; DSST, Digit Symbol Score Test.
*Model 1 calculated by binary logistic regression; Model 2 adjusted for age and sex Model 3 was adjusted for age, sex, race, education, BMI, SBP, DBP, total energy intake, dietary potassium intake, serum sodium, creatinine, smoking, alcohol use, diabetes, stroke, and antihypertensive drug use.

was observed in AFT (OR = 0.43, 0.26–0.73). Q3 showed the lowest odds ratios (OR = 0.64, 0.42–0.96) of cognitive impairment in CERAD compared with Q1. After adjusting for age, sex, race, education, BMI, SBP, DBP, total energy intake, dietary potassium intake, serum sodium, creatinine, smoking, alcohol use, diabetes, stroke, and use of antihypertensive drugs, the lowest weighted multivariable-adjusted OR of cognitive impairment in the DSST was also observed in Q4 (OR = 0.47, 0.26–0.84) compared with the lowest quartile of dietary sodium intake.

Additionally, we used a logistic regression model in participants with hypertension (Table 3). Compared with the lowest quartile of dietary sodium intake, the lowest weighted odds ratio of cognitive impairment in DSST was observed in Q4 (OR = 0.45, 0.29–0.70), and a similar trend was observed in AFT (OR = 0.34, 0.18–0.65). After adjusting the covariates, the lowest weighted multivariable-adjusted OR of cognitive impairment in DSST was also observed in Q4 (OR = 0.47, 0.26–0.84) compared with the lowest quartile of dietary sodium intake.

Furthermore, RCS was examined to explore the nonlinear relationship between dietary sodium intake and the risk of cognitive impairment (Figure 2a). We found that dietary sodium intake was U-shaped and associated with the risk of cognitive impairment (determined by the DSST) ($P_{non\text{-}linearity}$ = 0.0067). Figure 2b shows the relationship between dietary sodium intake and DSST score ($P_{non\text{-}linearity}$ = 0.0067).

3.3 | Dietary potassium intake and cognition

A logistic regression model was used to assess the association between dietary potassium intake and cognitive performance in all the participants (Table 4). Compared with the lowest quartile of dietary potassium intake, the crude OR of Q5 was (OR = 0.42, 0.26–0.67) for DSST, and Q3 was (OR = 0.40, 0.21–0.77) for AFT. After adjusting for age, sex, race, education, BMI, SBP, DBP, total energy intake, dietary potassium intake, serum sodium, creatinine, smoking, alcohol use, diabetes, stroke, and use of antihypertensive drugs, no significant association was observed between dietary potassium intake and the different dimensions of cognitive performance.

Furthermore, RCS was examined to explore the nonlinear relationship between dietary potassium intake and different dimensions of cognitive performance (Figure S1a–c). We found that there

TABLE 3 Weighted odds ratios (95% confidence interval) of cognitive impairment by quartiles of sodium intake in participants with hypertension.

	DSST						
Dietary sodium (mg/day)	Case, *n* (%)	Model 1*	*P-value*	Model 2*	*P-value*	Model 3*	*P-value*
Q1 (286–2076)	174 (36.20)	1.00 (Ref.)	–	1.00 (Ref.)	–	1.00 (Ref.)	–
Q2 (2076–2604)	132 (23.22)	**0.53 (0.35–0.81)**	**0.005**	**0.52 (0.34–0.78)**	**0.003**	0.67 (0.33– 1.40)	0.23
Q3 (2604–3226)	117 (23.65)	**0.55 (0.38–0.79)**	**0.002**	**0.47 (0.32–0.70)**	**<0.001**	0.62 (0.29– 1.30)	0.17
Q4 (3226–3944.5)	111 (20.38)	**0.45 (0.29–0.70)**	**<0.001**	**0.38 (0.25–0.58)**	**<0.0001**	**0.47 (0.25– 0.86)**	**0.02**
Q5 (3944.5–8765)	112 (23.59)	**0.54 (0.34–0.86)**	**0.01**	**0.41 (0.25–0.69)**	**0.001**	0.54 (0.18– 1.64)	0.23
	AFT						
Dietary sodium (mg/day)	Case, *n* (%)	Model 1*	*P-value*	Model 2*	*P-value*	Model 3*	*P-value*
Q1 (286–2076)	146 (36.14)	1.00 (Ref.)	–	1.00 (Ref.)	–	1.00 (Ref.)	–
Q2 (2076–2604)	113 (25.83)	0.62 (0.35,1.09)	0.09	0.64 (0.35,1.15)	0.13	0.87 (0.39,1.91)	0.67
Q3 (2604–3226)	103 (28.64)	0.71 (0.43,1.17)	0.17	0.73 (0.43,1.23)	0.23	0.97 (0.40,2.36)	0.94
Q4 (3226–3944.5)	80 (16.20)	**0.34 (0.18,0.65)**	**0.002**	**0.34 (0.18,0.67)**	**0.003**	0.47 (0.16,1.33)	0.13
Q5 (3944.5–8765)	72 (20.11)	**0.44 (0.26,0.77)**	**0.01**	**0.47 (0.28,0.81)**	**0.01**	0.77 (0.28,2.17)	0.56
	CERAD test						
Dietary sodium (mg/day)	Case, *n* (%)	Model 1*	*P-value*	Model 2*	*P-value*	Model 3*	*P-value*
Q1 (286–2076)	130 (34.84)	1.00 (Ref.)	–	1.00 (Ref.)	–	1.00 (Ref.)	–
Q2 (2076–2604)	98 (32.34)	0.89 (0.54–1.48)	0.65	0.91 (0.55–1.49)	0.69	1.13 (0.62–2.04)	0.63
Q3 (2604–3226)	106 (27.03)	0.69 (0.44–1.09)	0.11	0.59 (0.34–1.03)	0.06	0.72 (0.34–1.51)	0.31
	CERAD test						
Dietary sodium (mg/day)	Case, *n* (%)	Model 1*	*P-value*	Model 2*	*P-value*	Model 3*	*P-value*
Q4 (3226–3944.5)	88 (26.06)	0.66 (0.39–1.12)	0.12	**0.53 (0.32–0.87)**	**0.01**	0.69 (0.34–1.42)	0.26
Q5 (3944.5–8765)	88 (27.58)	0.71 (0.43–1.18)	0.18	**0.54 (0.31–0.95)**	**0.03**	0.78 (0.38–1.58)	0.42

Abbreviations: AFT, Animal fluency score total; CERAD, Consortium to Establish a Registry for Alzheimer's Disease; DSST, Digit Symbol Score Test.
*Model 1 calculated by binary logistic regression; Model 2 adjusted for age and sex Model 3 was adjusted for age, sex, race, education, BMI, SBP, DBP, total energy intake, dietary potassium intake, serum sodium, creatinine, smoking, alcohol use, diabetes, stroke, and antihypertensive drug use.

were no nonlinear relationships between dietary potassium intake and cognition (DSST score: *P* non–linearity = 0.1401; AFT: *P* non–linearity = 0.1055; CERAD test: *P* non–linearity = 0.0815).

4 | DISCUSSION

A previous study showed that high dietary sodium intake might influence cognitive performance.[30] Additionally, dietary sodium is a risk factor for hypertension.[31,32] In this study, we combined data from NHANES 2011 to 2012 and 2013–2014 and involved 2276 Americans (1,670 participants with hypertension) aged ≥60 years. After adjusting for all confounding factors, the associations between dietary sodium intake and cognitive performance were significant in participants with or without hypertension, and a parabolic-shaped dose–response relationship was also detected. In the United States, excessively high or low dietary sodium levels were associated with impaired processing speed, sustained attention, and working memory for hypertension in the older patients. Dietary sodium intake was unrelated to learning ability or executive function in this study. Here, we also analyzed the relationship between dietary potassium intake and cognitive performance. We found that potassium intake did not modify the risk of cognitive decline in the older patients with hypertension.

A dietary sodium intake of less than 2300 mg/day is recommended for the general population and 1500 mg/day for certain groups at risk, including individuals above 51 years of age.[33] In mice, excess dietary salt results in reduced cerebral blood flow, endothelial function, and cognitive impairment.[20] Another animal study showed that dietary salt could disrupt the tricarboxylic acid cycle[34] and lead to tau hyperphosphorylation, a significant biomarker of Alzheimer's pathology.[21] In mice, the researchers observed that excess dietary salt promotes plasma interleukin-17 production by proinflammatory cytokine interleukin-17, which promotes endothelial dysfunction and cognitive impairment.[19]

There is no doubt that a reduction in dietary sodium has beneficial cardiovascular effects.[35–37] However, the relationship between dietary sodium intake and cognitive impairment remains unclear. Anna et al.[38] examined the effects of hypertension and dietary salt

TABLE 4 Weighted odds ratios (95% confidence interval) of cognitive impairment by quartiles of potassium intake in participants with hypertension.

	DSST						
Dietary potassium (mg/day)	Case, *n* (%)	Model 1*	*P-value*	Model 2*	*P-value*	Model 3*	*P-value*
Q1 (108.5–1762)	177 (37.87)	1.00 (Ref.)	–	1.00 (Ref.)	–	1.00 (Ref.)	–
Q2 (1762–2235)	137 (24.35)	**0.53 (0.33–0.85)**	**0.01**	**0.52 (0.31–0.84)**	**0.01**	0.70 (0.38– 1.31)	0.22
Q3 (2235–2690)	126 (25.50)	**0.56 (0.38–0.84)**	**0.01**	**0.52 (0.35–0.78)**	**0.003**	0.87 (0.44– 1.72)	0.64
Q4 (2690–3280)	106 (21.05)	**0.44 (0.27–0.70)**	**0.001**	**0.35 (0.21–0.57)**	**<0.001**	0.70 (0.30– 1.62)	0.34
Q5 (3280–22666)	100 (20.20)	**0.42 (0.26–0.67)**	**<0.001**	**0.33 (0.20–0.56)**	**<0.001**	0.70 (0.24– 2.00)	0.43
	AFT						
Dietary potassium (mg/day)	Case, *n* (%)	Model 1*	*P-value*	Model 2*	*P-value*	Model 3*	*P-value*
Q1 (108.5–1762)	151 (35.59)	1.00 (Ref.)	–	1.00 (Ref.)	–	1.00 (Ref.)	–
Q2 (1762–2235)	124 (29.80)	0.77 (0.49,1.20)	0.24	0.83 (0.55,1.26)	0.38	1.23 (0.70,2.14)	0.4
Q3 (2235–2690)	90 (21.45)	**0.49 (0.32,0.76)**	**0.002**	**0.50 (0.33,0.76)**	**0.002**	0.79 (0.45,1.39)	0.35
Q4 (2690–3280)	79 (18.04)	**0.40 (0.21,0.77)**	**0.01**	**0.39 (0.21,0.71)**	**0.003**	0.77 (0.32,1.83)	0.48
Q5 (3280–22666)	70 (22.04)	**0.51 (0.26,1.00)**	**0.05**	**0.51 (0.27,0.98)**	**0.04**	1.19 (0.45,3.14)	0.67
	CERAD test						
Dietary potassium (mg/day)	Case, *n* (%)	Model 1*	*P-value*	Model 2*	*P-value*	Model 3*	*P-value*
Q1 (108.5–1762)	118 (32.66)	1.00 (Ref.)	–	1.00 (Ref.)	–	1.00 (Ref.)	–
Q2 (1762–2235)	115 (30.21)	0.89 (0.49–1.63)	0.7	0.97 (0.53–1.79)	0.92	1.24 (0.62–2.49)	0.47
Q3 (2235–2690)	100 (27.57)	0.78 (0.47–1.30)	0.33	0.74 (0.43–1.28)	0.27	1.00 (0.54–1.85)	0.99
	CERAD test						
Dietary potassium (mg/day)	Case, *n* (%)	Model 1*	*P-value*	Model 2*	*P-value*	Model 3*	*P-value*
Q4 (2690–3280)	86 (26.21)	0.73 (0.40–1.32)	0.29	0.56 (0.30–1.04)	0.07	0.87 (0.40–1.91)	0.69
Q5 (3280–22666)	91 (30.56)	0.91 (0.52–1.58)	0.72	0.72 (0.40–1.30)	0.27	1.31 (0.74–2.32)	0.29

Abbreviations: AFT, Animal fluency score total; CERAD, Consortium to Establish a Registry for Alzheimer's Disease; DSST, Digit Symbol Score Test.

*Model 1 calculated by binary logistic regression; Model 2 adjusted for age and sex Model 3 was adjusted for age, sex, race, education, BMI, SBP, DBP, total energy intake, dietary potassium intake, serum sodium, creatinine, smoking, alcohol use, diabetes, stroke, and antihypertensive drug use.

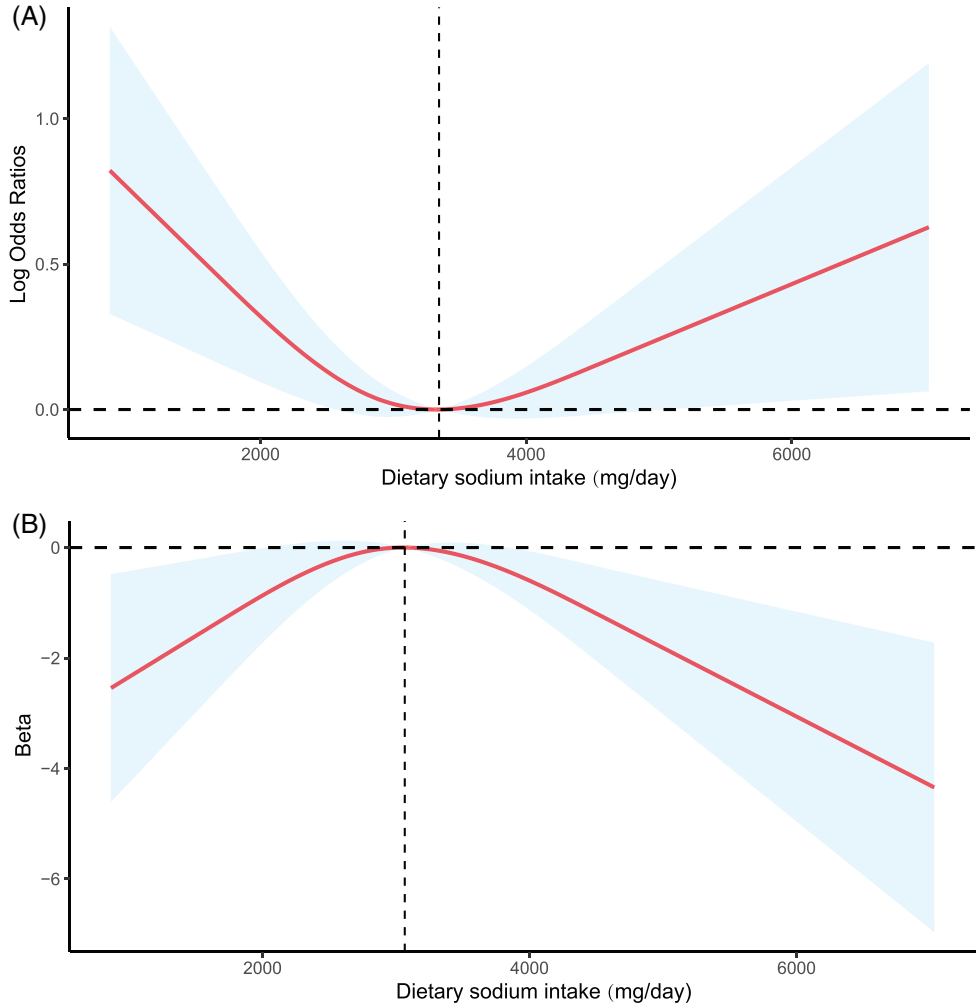

FIGURE 2 (A) Relationship between sodium intake and risk of cognitive impairment (determined by DSST). The model was based on logistic regression models and adjusted for age, sex, race, education, BMI, SBP, DBP, total energy intake, dietary potassium intake, serum sodium, creatinine, smoking, alcohol use, diabetes, stroke, and antihypertensive drug use. (B) Relationship between sodium intake and DSST score. The model was based on liner regression models and adjusted for age, sex, race, education, BMI, SBP, DBP, total energy intake, dietary potassium intake, serum sodium, creatinine, smoking, alcohol use, diabetes, stroke, and antihypertensive drug use.

intake on cerebrovascular disease and found that dietary salt intake positively correlated with white-matter hyperintensity (WMH) volume. Stephen et al.[39] also found that patients who did not reduce their salt intake in the long term were more likely to have lacunar strokes, lacunes, microbleeds, and severe WMH. In contrast, a recent dietary study based on community-dwelling older adults found that sodium/potassium intake was not associated with micro- or macro-structural brain magnetic resonance imaging (MRI) indices.[22] A cross-sectional study involving 925 community participants (74.5 ± 8.7 years) showed that lower sodium intake was associated with worse cognitive function.[23] The results of our study differ from those of the research mentioned above. This could be because of the different cognitive tests used in these studies. It is worth mentioning that a cross-sectional study observed that lower serum sodium (126–140 mmol/L) is associated with both prevalent cognitive impairment and cognitive decline in community-dwelling older men,[40] which implies that a lack of sodium has a harmful effect on cognition.

The relationship between dietary potassium intake and cognitive impairment has been controversial in previous studies. The Hisayama Study,[41] a prospective cohort study, showed that a higher self-reported dietary intake of potassium could reduce the risk of dementia in the general Japanese population. Another cohort study based on community-dwelling older adults showed that potassium intake was not associated with a decline in cognitive function.[22] We also observed that dietary potassium intake was not associated with cognitive impairments.

Our study has some limitations that should be acknowledged. First, because this was a cross-sectional study, it was difficult to determine a causal relationship between dietary sodium intake and cognitive performance. Furthermore, the assessment of cognitive performance is complicated and cognitive function is best assessed using multiple methods. However, only the DSST score was associated with dietary sodium intake in this study, which limited our ability to examine the association between sodium intake and cognition. Finally, 24-h dietary

recall is used as a simple and effective method in epidemiological studies, but it may not capture long-term dietary exposures and result in recall bias,[42] and it is an indirect method to evaluate dietary intake compared with 24-h urine sodium excretion. Therefore, the results of this study need to be interpreted with caution, and more large prospective studies are needed to validate these conclusions further.

In conclusion, both excessively high and low dietary sodium levels were associated with cognitive impairment, specifically in processing speed, sustained attention, and working memory, for hypertension in the older patients in the United States. Therefore, in addition to avoiding high-sodium dietary patterns, older patients with hypertension should also avoid extremely low sodium intake to prevent cognitive impairment. However, we did not observe an association between dietary potassium intake and cognition. Further large-scale prospective studies are still needed to clarify the effect of dietary sodium/potassium on cognition.

AUTHOR CONTRIBUTIONS

Jing Yu conceived and designed the study. Chengkun Kou and Xu Zhao contributed to data collection and analysis, and wrote the manuscript. Xin Fan, Xin Lin and Qiongying Wang contributed to study design, data analysis, and manuscript revision. Chengkun Kou and Xu Zhao contributed equally to this work.

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CONFLICTS OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

The original data in this study are openly available in National Center for Health Statistics (https://wwwn.cdc.gov/nchs/nhanes/Default.aspx). And there is no additional data available.

REFERENCES

1. He FJ, Tan M, Ma Y, MacGregor GA. Salt reduction to prevent hypertension and cardiovascular disease: JACC state-of-the-art review. *J Am Coll Cardiol*. 2020;75(6):632-647.
2. Babcock MC, Robinson AT, Migdal KU, et al. Reducing dietary sodium to 1000 mg per day reduces neurovascular transduction without stimulating sympathetic outflow. *Hypertension*. 2019;73(3):587-593.
3. Fuchs FD, Whelton PK. High blood pressure and cardiovascular disease. *Hypertension*. 2020;75(2):285-292.
4. GBD 2017 Diet. Health effects of dietary risks in 195 countries, 1990–2017: a systematic analysis for the global burden of disease study 2017. *Lancet*. 2019;393(10184):1958-1972.
5. Wang JG, Zhang W, Li Y, Liu L. Hypertension in China: epidemiology and treatment initiatives [published online ahead of print, 2023 Jan 11]. *Nat Rev Cardiol*. 2023. doi: 10.1038/s41569-022-00829-z
6. Titze J, Luft FC. Speculations on salt and the genesis of arterial hypertension. *Kidney Int*. 2017;91(6):1324-1335.
7. Huang L, Trieu K, Yoshimura S, et al. Effect of dose and duration of reduction in dietary sodium on blood pressure levels: systematic review and meta-analysis of randomised trials. *BMJ*. 2020;368:m315.
8. Brand A, Visser ME, Schoonees A, Naude CE. Replacing salt with low-sodium salt substitutes (LSSS) for cardiovascular health in adults, children and pregnant women. *Cochrane Database Syst Rev*. 2022;8(8):CD015207.
9. Yin X, Paige E, Tian M, et al. The proportion of dietary salt replaced with potassium-enriched salt in the SSaSS: implications for scale-up. *Hypertension*. 2023; 80(5):956-965.
10. Aburto NJ, Hanson S, Gutierrez H, et al. Effect of increased potassium intake on cardiovascular risk factors and disease: systematic review and meta-analyses. *BMJ*. 2013;346:f1378.
11. Xie Y, Qi H, Peng W, et al. Higher potassium intake and lower sodium intake may help in reducing CVD risk by lowering salt sensitivity of blood pressure in the Han Chinese population. *Nutrients*. 2022;14(20):4436.
12. Marklund M, Tullu F, Raj TS, et al. Estimated benefits and risks of using a reduced-sodium, potassium-enriched salt substitute in india: a modeling study. *Hypertension*. 2022;79(10):2188-2198.
13. Binia A, Jaeger J, Hu Y, Singh A, Zimmermann D. Daily potassium intake and sodium-to-potassium ratio in the reduction of blood pressure: a meta-analysis of randomized controlled trials. *J Hypertens*. 2015;33(8):1509-1520.
14. Marklund M, Singh G, Greer R, et al. Estimated population wide benefits and risks in China of lowering sodium through potassium enriched salt substitution: modelling study. *BMJ*. 2020;369:m824.
15. Yang Z, Wang H, Edwards D, et al. Association of blood lipids, atherosclerosis and statin use with dementia and cognitive impairment after stroke: a systematic review and meta-analysis. *Ageing Res Rev*. 2020;57:100962.
16. Milte CM, Ball K, Crawford D, McNaughton SA. Diet quality and cognitive function in mid-aged and older men and women. *Bmc Geriatr*. 2019;19(1):361.
17. Chen X, Liu Z, Sachdev PS, et al. Dietary patterns and cognitive health in older adults: findings from the Sydney memory and ageing study. *J Nutr Health Aging*. 2021;25(2):255-262.
18. Ozawa H, Miyazawa T, Miyazawa T. Effects of dietary food components on cognitive functions in older adults. *Nutrients*. 2021;13(8):2804.
19. O'Donnell MJ, Yusuf S, Mente A, et al. Urinary sodium and potassium excretion and risk of cardiovascular events. *JAMA*. 2011;306(20):2229-2238.
20. Faraco G, Brea D, Garcia-Bonilla L, et al. Dietary salt promotes neurovascular and cognitive dysfunction through a gut-initiated TH17 response. *Nat Neurosci*. 2018;21(2):240-249.
21. Faraco G, Hochrainer K, Segarra SG, et al. Dietary salt promotes cognitive impairment through tau phosphorylation. *Nature*. 2019;574(7780):686-690.
22. Nowak KL, Fried L, Jovanovich A, et al. Dietary sodium/potassium intake does not affect cognitive function or brain imaging indices. *Am J Nephrol*. 2018;47(1):57-65.
23. Rush TM, Kritz-Silverstein D, Laughlin GA, et al. Association between dietary sodium intake and cognitive function in older adults. *J Nutr Health Aging*. 2017;21(3):276-283.

24. Morris JC, Heyman A, Mohs RC, et al. The Consortium to Establish a Registry for Alzheimer's Disease (CERAD). Part I. Clinical and neuropsychological assessment of Alzheimer's disease. *Neurology*. 1989;39(9):1159-1165.
25. Strauss E, Sherman EMS, Spreen O. *A Compendium of Neuropsychological Tests: Administration, Norms, and Commentary*. Oxford University Press; 2006.
26. Wechsler D. *WAIS-III and WMS-III Technical Manual*. Psychological Corporation; 1997.
27. Lee H, Cho HJ, Bae E, et al. Not salt taste perception but self-reported salt eating habit predicts actual salt intake. *J Korean Med Sci*. 2014;29 Suppl 2(Suppl 2):S91-96.
28. Ikehara S, Iso H, Date C, et al. Salt preference and mortality from stroke and coronary heart disease for Japanese men and women: the JACC study. *Prev Med*. 2012;54(1):32-37.
29. Li Y, Huang Z, Jin C, et al. Longitudinal change of perceived salt intake and stroke risk in a Chinese population. *Stroke*. 2018;49(6):1332-1339.
30. Santisteban MM, Iadecola C. Hypertension, dietary salt and cognitive impairment. *J Cereb Blood Flow Metab*. 2018;38(12):2112-2128.
31. Little R, Murali SK, Poulsen SB, et al. Dissociation of sodium-chloride cotransporter expression and blood pressure during chronic high dietary potassium supplementation. *JCI Insight*. 2023:e156437.
32. Zhao D, Sun H, Li H, Li C, Zhou B. A prediction model for the impact of environmental and genetic factors on cardiovascular events: development in a salt substitutes population. *J Transl Med*. 2023;21(1):62.
33. Haring B, Wu C, Coker LH, et al. Hypertension, dietary sodium, and cognitive decline: results from the Women's Health Initiative Memory Study. *Am J Hypertens*. 2016;29(2):202-216.
34. Yuan M, Wang Y, Wen J, et al. Dietary salt disrupts tricarboxylic acid cycle and induces tau hyperphosphorylation and synapse dysfunction during aging. *Aging Dis*. 2022;13(5):1532-1545.
35. Hunter RW, Dhaun N, Bailey MA. The impact of excessive salt intake on human health. *Nat Rev Nephrol*. 2022;18(5):321-335.
36. Jaques DA, Wuerzner G, Ponte B. Sodium intake as a cardiovascular risk factor: a narrative review. *Nutrients*. 2021;13(9):3177.
37. Rossitto G, Delles C. Mechanisms of sodium-mediated injury in cardiovascular disease: old play, new scripts. *Febs J*. 2022;289(23):7260-7273.
38. Heye AK, Thrippleton MJ, Chappell FM, et al. Blood pressure and sodium: association with MRI markers in cerebral small vessel disease. *J Cereb Blood Flow Metab*. 2016;36(1):264-274.
39. Makin S, Mubki GF, Doubal FN, et al. Small vessel disease and dietary salt intake: cross-sectional study and systematic review. *J Stroke Cerebrovasc Dis*. 2017;26(12):3020-3028.
40. Nowak KL, Yaffe K, Orwoll ES, et al. Serum sodium and cognition in older community-dwelling men. *Clin J Am Soc Nephrol*. 2018;13(3):366-374.
41. Ozawa M, Ninomiya T, Ohara T, et al. Self-reported dietary intake of potassium, calcium, and magnesium and risk of dementia in the Japanese: the Hisayama Study. *J Am Geriatr Soc*. 2012;60(8):1515-1520.
42. Peeri NC, Egan KM, Chai W, Tao MH. Association of magnesium intake and vitamin D status with cognitive function in older adults: an analysis of US National Health and Nutrition Examination Survey (NHANES) 2011 to 2014. *Eur J Nutr*. 2021;60(1):465-474.

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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