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# Association between oral intake magnesium and sarcopenia: a cross-sectional study

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#### **Abstract**

**Background:** Sarcopenia is a common skeletal muscle disorder in the elderly population. The patients with sarcopenia increased the cost of care and the risk for hospitalization. Magnesium deficiency might increase reactive oxygen species and protein damage. The purpose of our study was to demonstrate the relation between oral intake magnesium and sarcopenia by European Working Group on Sarcopenia in Older People (EWGSOP) 2 definition.

**Methods:** Our study included 2532 participants with 1310 males and 1222 females. The multiple logistic regression model was designed to test the cross-sectional protective outcome of oral intake magnesium for sarcopenia.

**Results:** Oral intake magnesium had a protective outcome with sarcopenia (odd ratio (OR) = 0.997, 95% CI = 0.996, 0.998, P < 0.001). After fully adjusted, the significance persisted with OR = 0.998 (95% CI = 0.996, 0.999, P < 0.001).

**Conclusion:** Results of the present study showed the dose dependent relationship between oral intake magnesium and sarcopenia. Sufficient oral intake magnesium might prevent patient from sarcopenia.

Keywords: Magnesium, Sarcopenia

#### Introduction

Sarcopenia, one of the primary diseases of the elderly population, is a common skeletal muscle disorder that involves the progressive loss of skeletal muscle function and mass [1]. To date, the most widely cited diagnostic criteria is that proposed by the European Working Group on Sarcopenia in Older People (EWGSOP) and updated as EWGSOP2 in 2019 [2]. According to EWGSOP2, muscle strength, muscle quantity and physical performance were measured to evaluate sarcopenia [2]. Loss of skeletal muscle mass and function causes the risk of falls, fractures, physical disability, use of hospital services, poor quality of life, and death [3]. The patients with sarcopenia increase the risk for hospitalization and the cost of care

during hospitalization [4]. In 2000, there was approximately \$18.5 billion (\$10.8 billion in men, \$7.7 billion in women) health-care cost in the America due to sarcopenia [5]. Malnutrition plays a key role in the pathogenesis of sarcopenia [6] [7] [8]. There was a growing evidence base linking nutrition with greater muscle strength and better physical performance outcomes in patient with sarcopenia [9]. However, these articles paid more attention on protein [10], long-chain polyunsaturated fatty acids [11], vitamin D [12], and antioxidant nutrients [13]. Best to our knowledge, no previous data had discussed oral intake electrolyte and sarcopenia.

Magnesium is the second most intracellular cation after potassium in human. Several metabolic pathways and fundamental cellular activities including peptide and DNA synthesis, signal transduction, blood glucose control, stimulus-contraction coupling, stimulus-secretion coupling, and ion channel translocation related to magnesium [14]. Total body magnesium content is mainly located in bone and soft tissue [15]. The recommended daily intake of magnesium is 320 and 420 mg for adult

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women and men, respectively. Absorption mainly (80–90%) occurs in the jejunum and colon through passive and active transport [16]. Reabsorption of magnesium mainly occurred in the thin ascending limb of Henle (70%) and partially occurred in the proximal tubule and distal collecting tubule. Hypomagnesemia related to several clinical manifestations including neuromuscular and cardiovascular manifestations. Muscle spasms, muscle cramps, tremors, atrial fibrillation and ventricular arrhythmias were the specific symptoms of hypomagnesemia [17]. Further, electrolyte and hormone imbalanced including hypocalcemia, hypokalemia, and hypoparathyroidism were also associated with hypomagnesemia.

According to a previous study, magnesium deficiency might increase reactive oxygen species and lipid and protein damage and associate with damage of muscle [18]. The purpose of our study was to demonstrate the relation between oral intake magnesium and sarcopenia by EWG-SOP2 definition.

#### **Materials and methods**

#### Study design and participants

We conducted a cross-sectional study using the National Health and Nutrition Examination Survey (NHANES) database in 1999-2002 period. The data included overall home interviews and laboratory assessments conducted by the National Center for Health Statistics (NCHS). Institutional Review Board (IRB) approval and

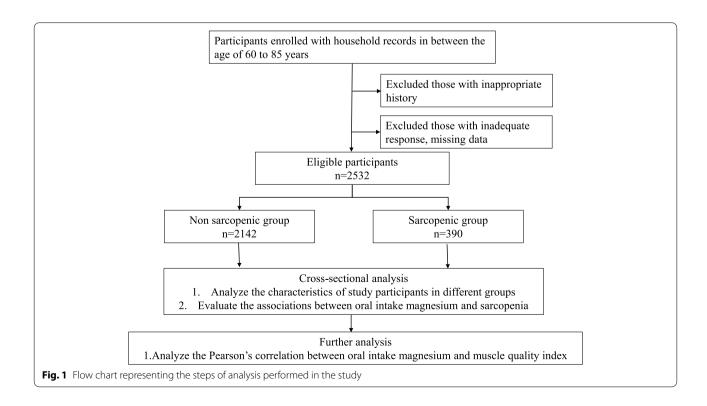
documented consent was obtained from participants. The exclusion criteria were dysphagia; history of gastrointestinal tract disease; inflammatory bowel disease; history of cancer; participation in resistance training; participation regularly using steroids, diuretics, hormones, and growth factors; and subjects without complete clinical data, laboratory results, or medical history. (Fig. 1).

#### **EWGSOP2** guideline

The EWGSOP published a sarcopenia definition in 2010 [19]. In early 2018, the Working Group met again (EWGSOP2) to update the definition of sarcopenia. In the EWGSOP2, low muscle strength as the primary parameter of sarcopenia. A sarcopenia diagnosis is confirmed by low muscle strength with the presence of low muscle quantity or quality. Severe sarcopenia was considered with three components, including low muscle strength, low muscle quantity/quality and low physical performance.

#### Measurement: muscle quality index

The NHANES documentation provided measurement of the muscle quality index. We listed these methods and cut points to estimate components of muscle quality index in Table-S1.



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#### Measurement of oral intake magnesium

Total magnesium intake in milligrams per day calculated from dietary information with 24 hours dietary recalls. Subjects were required to recall all intakes in one day period prior before the questionnaire. A measuring guides and a food model booklet was available for participants to help in reporting food amounts.

#### **Assessment of Covariables**

A computer-aided individual interviewing method was used for the collection of participants' information. Demographic information, including sex, age, race was assembled. Smoking and drinking status were assessed by a detailed survey. We also recorded self-reported comorbidities, including cancer and cardiovascular disease. Based on the guidelines of Centers for Disease Control and Prevention, biochemical analyses in the database used standard methods.

#### Statistical analysis

SPSS (Version 18.0 for Windows, SPSS, Inc., Chicago, IL) was used for all the statistical analyses. Descriptive information of continuous and categorical covariates is presented as the mean (standard deviation) and several observations with percentage (%). Continuous variables were used Student's t tests and categorical variables were used the  $\chi^2$  test for the comparison of characteristics and covariates across subgroups. The odds ratio (OR) with 95% confidence interval (CI) for sarcopenia was estimated by multivariate logistic regression analysis. We analyzed the association between oral intake magnesium and muscle quality index using a linear regression model. The beta coefficient is the degree of change in the muscle quality index for every 1-unit of change in the oral intake magnesium. The 2-sided P values less than 0.05 indicated significant differences. Four extended models were used for covariate adjustments. Model 1 examined unadjusted odd ratio (ORs). Model 2 presented ORs after adjusting sex, age, and race. Model 3 was adjusted for the variables included in Model 2+the body mass index (BMI), Albumin, total calcium, creatinine, fasting glucose, C-reactive protein (CRP), daily carbohydrate intake, daily total fat intake, and daily protein intake. The variables in model 3+the smoking history, comorbidities and education level were adjusted for model 4.

#### Result

#### Characteristics of the subjects

2532 subjects with 1310 males and 1222 females were included in our study. We separated subjects into two groups based on the EWGSOP definition of sarcopenia. (Table 1) The mean age were  $73.14\pm9.59\,\mathrm{years}$  in the sarcopenic group and  $63.90\pm9.32\,\mathrm{years}$  in the

non-sarcopenic group, respectively. It was also observed that the mean oral intake magnesium amount was higher for non-sarcopenic group at  $276.06 \pm 142.04$  compared to  $235.97 \pm 112.61$  in the sarcopenic group (P < 0.001).

## Association between oral intake magnesium and sarcopenia

In Table 2, we showed the association between different oral intake elements and sarcopenia. After adjusted all covariates, the association only showed in oral intake magnesium and potassium.

In Table 3, we performed logistic regression to exam the association between oral intake magnesium and sarcopenia. In Model 1, oral intake magnesium was significantly associated with sarcopenia (OR=0.997, 95% confidence interval (CI)=0.996, 0.998, P<0.001) The significance persisted after adjusted all covariates in Model 4 (OR=0.998, 95% CI=0.996, 0.999, P=0.007).

#### Sensitivity analysis

Logistic regression was used for sensitivity analysis to compare the sarcopenic and non-sarcopenic groups and quartiles of oral intake magnesium. Similar outcomes were found for the relation between oral intake magnesium and sarcopenia in Table 4. In the unadjusted model, the highest quartile of oral intake magnesium had an odds ratio of 0.400 to be associated with sarcopenia (95% CI = 0.282, 0.568, P < 0.001). The odds ratio of subjects with the highest quartile of oral intake magnesium would have sarcopenia was 0.468 (0.274, 0.799) times fewer than subjects with the lowest quartile of oral intake magnesium after fully adjusted.

## Correlation between oral intake magnesium and muscle quality index

In Table 5, the coefficients of oral intake magnesium and muscle strength, SMI, and gait speed were 0.207, 0.134, and -0.137. The linear regression analysis of the relation of oral intake magnesium and muscle quality index was showed in Table 6. The association of oral intake magnesium and muscle strength ( $\beta$ =0.145, 95% CI=0.118, 0.172), SMI ( $\beta$ =0.001, 95% CI=0.001, 0.002), and gait speed ( $\beta$ =-0.000365, 95% CI=-0.000471, -0.000258) had statistically significant result (P<0.001). After fully adjusted, the association of oral intake magnesium and muscle strength ( $\beta$ =0.048, 95% CI=0.012, 0.083), SMI ( $\beta$ =-0.000149, 95% CI=-000434, 0.000136), and gait speed ( $\beta$ =-0.000171, 95% CI=-0.000338, -0.000005).

#### Discussion

In our presented study, the relationship between oral intake magnesium and the components of sarcopenia was closely inspected in 2532 adult population. Our

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**Table 1** Characteristics of study participants

Characteristics of study participants	Sarcopenia	Non-sarcopenia	<i>P</i> value
	(n = 390)	(n=2142)	
Age at screening	73.14 (9.59)	63.90 (9.32)	0.227
Body mass index (kg/m²)	27.89 (4.70)	28.79 (5.58)	0.421
Creatinine (mg/dL)	0.92 (0.67)	0.88 (0.50)	0.001
Glucose (mg/dL)	107.81 (43.25)	102.39 (36.39)	0.001
C-reactive protein (mg/dL)	0.57 (0.98)	0.46 (0.75)	0.008
Albumin(g/dL)	4.22 (0.31)	4.31 (0.28)	0.211
Total Calcium (mg/dL)	9.47 (0.44)	9.43 (0.40)	0.178
Magnesium (mg/dL)	235.97 (112.61)	276.06 (142.04)	< 0.001
Carbohydrate (gm)	210.63 (94.13)	239.32 (114.20)	0.002
Total fat (gm)	61.79 (31.69)	72.23 (43.07)	< 0.001
Protein (gm)	64.26 (28.22)	74.22 (37.50)	< 0.001
Dietary fiber (gm)	14.16 (8.09)	16.78 (10.76)	< 0.001
Comorbidity	0.92 (0.99)	0.60 (0.89)	0883
Gender (male)	34.1%	54.9%	< 0.001
Race/ethnicity			
Mexican American	15.1%	19.2%	0.280
Other Hispanic	4.4%	4.2%	
Non-Hispanic White	63.1%	59.8%	
Non-Hispanic Black	14.1%	14.6%	
Smoking	52.1%	43.5%	0.008
Drinking	53.5%	67.6%	< 0.001
Education level past high school	57.8%	65.8%	0.003
Congestive heart failure	7.4%	3.2%	< 0.001
Coronary heart disease	9.2%	6.8%	0.109
Angina	6.9%	5.6%	0.291
Heart attack	9.0%	5.7%	0.016
Stroke	0.5%	0.7%	1.000
Emphysema	6.2%	2.4%	< 0.001

 Table 2
 The association between different oral intake elements and sarcopenia

Components of sarcopenia									
Oral intake elements	Model 1	P Value	Model 2	P Value	Model 3	P Value	Model 4	<i>P</i> Value	
magnesium (mg)	0.997 (0.996-0.998)	< 0.001	0.999 (0.998-1.000)	0.028	0.997 (0.996-0.999)	0.003	0.998 (0.996-0.999)	0.007	
Calcium (mg)	1.000 (0.999-1.000)	0.047	1.000 (1.000-1.000)	0.835	1.000 (1.000-1.000)	0.988	1.000 (1.000-1.000)	0.883	
Phosphorate (mg)	1.000 (0.999-1.000)	< 0.001	1.000 (1.000-1.000)	0.491	1.000 (0.999-1.000)	0.357	1.000 (0.999-1.000)	0.442	
Iron (mg)	0.981 (0.968-0.996)	0.010	0.993 (0.979-1.008)	0.360	0.992 (0.973-1.011)	0.415	0.994 (0.975-1.014)	0.567	
Zinc (mg)	0.966 (0.948-0.985)	< 0.001	0.992 (0.975-1.009)	0.335	0.986 (0.963-1.010)	0.262	0.988 (0.966-1.011)	0.305	
Copper (mg)	0.925 (0.804-1.064)	0.276	1.042 (0.966-1.125)	0.288	1.040 (0.962-1.124)	0.326	1.046 (0.968-1.131)	0.255	
Sodium (mg)	1.000 (1.000-1.000)	< 0.001	1.000 (1.000-1.000)	0.871	1.000 (1.000-1.000)	0.533	1.000 (1.000-1.000)	0.508	
Potassium (mg)	1.000 (1.000-1.000)	< 0.001	1.000 (1.000-1.000)	0.047	1.000 (1.000-1.000)	0.017	1.000 (1.000-1.000)	0.034	

BMI Body mass index, CRP C-reactive protein

Adjusted covariates:

 $Model\ 1 = Unadjusted$ 

Model 2 = Adjusted age, sex, race/ethnicity

 $Model \ 3 = Model \ 2 + BMI, Albumin, total \ calcium, creatinine, fasting \ glucose, CRP, daily \ carbohydrate intake, daily total fat intake, and daily protein intake$ 

Model~4 = Model~3 + drinking~history, smoking~history, comorbidities, and~education~level~

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**Table 3** Result of logistic regression predicting the presence of sarcopenia by oral intake magnesium

#### Components of sarcopenia

	Model 1	P Value	Model 2	P Value	Model 3	P Value	Model 4	P Value
Oral intake magnesium (mg)	0.997 (0.996-0.998)	< 0.001	0.999 (0.998-1.000)	0.028	0.997 (0.996-0.999)	0.003	0.998 (0.996-0.999)	0.007

BMI Body mass index, CRP C-reactive protein

Adjusted covariates:

Model 1 = Unadjusted

Model 2 = Adjusted age, sex, race/ethnicity

Model 3 = Model 2 + BMI, Albumin, total calcium, creatinine, fasting glucose, CRP, daily carbohydrate intake, daily total fat intake, and daily protein intake

Model 4 = Model 3 + drinking history, smoking history, comorbidities, and education level

**Table 4** Association between quartiles of oral intake magnesium and sarcopenia

9	'		
		Sarcopenia	
Models	Quartiles of oral intake magnesium	OR (95% CI)	P Value
Model 1	Q2 v.s. Q1	0.937 (0.701-1.251)	0.658
	Q3 v.s. Q1	0.767 (0.568-1.036)	0.084
	Q4 v.s. Q1	0.400 (0.282-0.568)	< 0.001
Model 2	Q2 v.s. Q1	0.870 (0.635-1.192)	0.387
	Q3 v.s. Q1	0.871 (0.628-1.208)	0.409
	Q4 v.s. Q1	0.615 (0.421-0.900)	0.012
Model 3	Q2 v.s. Q1	0.782 (0.555-1.102)	0.160
	Q3 v.s. Q1	0.767 (0.518-1.134)	0.183
	Q4 v.s. Q1	0.453 (0.267-0.767)	0.003
Model 4	Q2 v.s. Q1	0.775 (0.548-1.097)	0.150
	Q3 v.s. Q1	0.794 (0.534-1.181)	0.255
	Q4 v.s. Q1	0.468 (0.274-0.799)	0.005

BMI Body mass index, CRP C-reactive protein, CI Confidence interval, OR Odds ratio, Q Quartile

Adjusted covariates:

Model 1 = Unadjusted

Model 2 = Adjusted age, sex, race/ethnicity

Model 3 = Model 2 + BMI, Albumin, total calcium, creatinine, fasting glucose, CRP, daily carbohydrate intake, daily total fat intake, and daily protein intake

 $\label{eq:model-def} \mbox{Model 4} = \mbox{Model 3} + \mbox{drinking history, smoking history, comorbidities, and education level}$ 

results demonstrated strong relationships between oral intake magnesium and sarcopenia. Otherwise, based on the EWGSOP2, three components of sarcopenia all presented correlation with oral intake magnesium. To date, our study was the first article investigating the relation of oral intake magnesium and sarcopenia in a representative sample of the United States adult population.

Due to complicated pathogenesis, the development of sarcopenia remained controversial in the medical field. Previous study had mentioned different factors which associated with sarcopenia [20]. One of the main factors induced sarcopenia was the mitochondrial dysfunction [21]. Mitochondrial oxidative stress and dysfunction could be induced by intracellular magnesium deficiency [22]. Downregulating the electron transport chain increased the production of reactive oxygen species [23]. Low level of intracellular magnesium also suppressed the antioxidant defense system and reduced proteins such as superoxide dismutase, catalase, and glutathione [24]. Intracellular magnesium sufficient, in contrast, was found to suppress mitochondrial reactive oxygen species and improve mitochondrial function [25].

Intracellular and extracellular concentration of magnesium had thought to impact the synthesis of DNA and protein [26]. Magnesium regulated the onset of protein synthesis in activated frog oocytes and its rate in lymphocytes. Magnesium determines the onset of protein synthesis through the phosphatidylinositol-3-OH kinase

Table 5 Pearson correlation coefficient between oral intake magnesium and muscle quality index

Components of sarcopenia									
	Average peak force (Newtons)	Skeletal muscle index			Gait speed				
	Correlation	<i>P</i> Value	Correlation	P Value	Correlation	<i>P</i> Value			
Oral intake magnesium (mg)	0.207	< 0.001	0.134	< 0.001	-0.137	< 0.001			

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**Table 6** Association between oral intake magnesium and muscle quality index

#### Muscle quality index

	Average peak force	(Newtons)	Skeletal muscle index		Low gait speed		
Models	OR (95% CI)	P Value	OR (95% CI)	P Value	OR (95% CI)	P Value	
Model 1	0.145 (0.118-0.172)	< 0.001	0.001 (0.001-0.002)	< 0.001	- 0.000365 (- 0.000471 0.000258)	< 0.001	
Model 2	0.043 (0.020-0.066)	< 0.001	-0.000356 (-0.0010.000032)	0.031	-0.000253 (-0.000359 - 0.000147)	< 0.001	
Model 3	0.060 (0.025-0.096)	0.001	-0.000188 (-0.000472-0.000096)	0.195	-0.000264 (-0.0004330.000096)	0.002	
Model 4	0.048 (0.012-0.083)	0.009	-0.000149 (- 000434-0.000136)	0.306	-0.000171 (-0.0003380.000005)	0.044	

BMI Body mass index, CRP C-reactive protein, CI Confidence interval, OR Odds ratio

Adjusted covariates:

Model 1 = Unadjusted

Model 2 = Adjusted age, sex, race/ethnicity

Model 3 = Model 2 + BMI, Albumin, total calcium, creatinine, fasting glucose, CRP, daily carbohydrate intake, daily total fat intake, and daily protein intake

Model 4 = Model 3 + drinking history, smoking history, comorbidities, and education level

(PI-3-K) pathway at mechanistic target of rapamycin (mTOR) phosphorylation of two translation-regulating proteins [27, 28]. One study measured intracellular concentration of magnesium and the rate of protein and DNA synthesis [29]. At the same magnesium concentration, the rate of DNA synthesis peaked as protein synthesis. This article provided strong evidence of magnesium as a regulator of DNA and protein synthesis.

Low magnesium status is also reported to associate with increasing reactive oxygen species and inflammation. Several articles had discussed that higher magnesium consumption was related to lower serum CRP [30]. People who takes magnesium at least 50 mg/day were 22% less likely to have higher level of plasma CRP compared with those dietary magnesium taking less than 50% of the recommended dietary allowance [31]. Magnesium supplement is associated with a lower serum CRP in these studies. One linear regression analysis of 3173 females aged 50–79 years found that magnesium supplement was associated with lower serum inflammatory biomarkers, such as CRP, interlukin-6 (IL-6), and tumor necrosis factor- $\alpha$  receptor 2 (TNF- $\alpha$ -R2, [32]].

Our study has several strengths. We screened the sarcopenia clearly by using EWGSOP2 guideline. We also screened other oral intake elements initially in our study. A large adult sample was included to discuss the protective outcome of oral intake magnesium for sarcopenia. In the current study, sufficient oral intake magnesium could reduce the incidence of sarcopenia. This result is not only effective, but also economical for preventing from sarcopenia in the public health system.

However, some limitations in our study should be identified. First, we used a cross-sectional database in which the oral intake magnesium was determined at single measure instead of a long observational period. Then, some information such as participants' education levels

and medical histories were based on questionnaires. The recall bias and other unknown bias might distort our results. Third, total magnesium intake was calculated from 24 hours dietary recalls and measured by guides and a food model booklet. This method caused inaccuracy of daily oral intake magnesium. Lastly, the factors of drug consumption, participants' cognitive and nutritional status were not included our results. Thus, the confounding bias could not be estimated.

#### Conclusion

In our presented study, we identified the protective outcome of oral intake magnesium with sarcopenia. The significance especially showed in muscle strength and gait speed. Otherwise, dose dependent relationship was observed between oral intake magnesium and sarcopenia. Our findings suggested that sufficient oral intake magnesium might prevent patient from sarcopenia. Further prospective studies may be warranted to find the curative effect of oral intake magnesium in the patient with sarcopenia.

#### Abbreviations

EWGSOP: European Working Group on Sarcopenia in Older People; NHANES: National Health and Nutritional Examination Survey; NCHS: National Center for Health Statistics; DXA: Dual-energy X-ray absorptiometry; SMI: Skeletal muscle index; BMI: Body mass index; CRP: C-Reactive protein; OR: Odds ratio; CI: Confidence interval; ROC: Receiver operating characteristic curve; AUROC: Area under receiver operating characteristic curve; IL-6: Interlukin-6; TNF-α-R2: Tumor necrosis factor-α receptor 2.

#### **Supplementary Information**

The online version contains supplementary material available at https://doi.org/10.1186/s12877-022-03522-5.

**Additional file 1 Supplement Table 1.** Standard measurement protocol of muscle quality index in NHANES.

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Not applicable.

#### Authors' contributions

Shih-Wei Yang and Wei-Liang Chen designed the initial study. Shih-Wei Yang also managed and retrieved the data, contributed to primary data analysis and explanation, and drafted the initial script. Shih-Wei Yang, Yuan-Yuei Chen, and Wei-Liang Chen decided on the methods of data collection. Shih-Wei Yang and Wei-Liang Chen were both responsible for the decisions of data analysis. Wei-Liang Chen conceptualized the study, inspected all sides of the study, critically reviewed, and revised the initial script, and approved the final manuscript as submitted. All authors meet the ICMJE criteria for authorship.

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#### Availability of data and materials

The datasets used and/or analyzed during the current study are available from the NHANES database, https://www.cdc.gov/nchs/nhanes/index.htm.

#### Declarations

#### Ethics approval and consent to participate

The health data collection protocols were performed by the CDC's NCHS and approved by the NCHS IRB (Protocol Number: Protocol #98-12). All informed consents had been obtained from the eligible subjects before initiating data collection and NHANES health examinations. All methods were performed in accordance with the relevant guidelines and regulations.

#### Consent for publication

Not applicable.

#### Competing interests

There is no support, financial or otherwise received from any organization that may have an interest in the submitted work, and there are no other relationships or activities that could appear to have influenced the submitted work.

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