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The relationship between dietary selenium intake and telomere length among diabetes

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Abstract

Se is an indispensable trace element for the human body, and telomere length is considered a marker of biological ageing. Previous studies have shown that dietary Se intake is associated with telomere length. However, the relationship between Se intake and telomere length in patients with diabetes has not been well studied. Therefore, this study aimed to investigate the relationship between dietary Se intake and telomere length in patients with diabetes. We extracted 878 participants with diabetes from the National Health and Nutrition Examination Survey database for 1990–2002. Dietary Se intake was assessed using the 24 h dietary recall method, and telomere length was measured using quantitative PCR. Generalised linear models were constructed to assess the relationship between dietary Se intake and telomere length. After controlling for the confounders, 1 μg increase in dietary Se intake in female patients with diabetes, and telomere length increased by 1·84 base pairs ($\beta = 1{\cdot}84$ (95 % CI: 0·15, 3·53)), there was a line relationship between dietary Se intake and telomere length in female patients with diabetes and telomere length increased with increasing dietary Se intake within the range of 0–250 μg. The study demonstrates that dietary Se intake is significantly associated with telomere length only in the female population with diabetes in the USA. However, further prospective studies are required to confirm this finding.

Keywords: **Telomere length: Dietary Se: Diabetes: NHANES**

The human body contains few trace elements, but they have an important protective effect on human health and metabolism. As an indispensable trace element, Se mainly produces antioxidant defences and anti-inflammation and generates active thyroid hormones in the body in the form of selenoproteins[1]. The human body mainly absorbs Se in the form of a diet[2], and Se deficiency may harm human health[3]. Studies have shown that the intake of a certain amount of dietary Se has the potential to prevent the occurrence of bladder cancer, lung cancer and autoimmune thyroid disease and may also reduce the risk of diabetes[4–7].

Telomeres are composed of TTAGGG repeats at the ends of eukaryotic chromosomes, which shorten telomere length through mitosis in somatic cells[8]. Telomere length has an impact on human longevity and health status and is considered a biomarker of ageing[9]. A shorter telomere length is associated with an increased risk of age-related diseases, such as CVD, diabetes and dementia[10–12]. In addition, a healthy lifestyle (higher levels of physical activity or higher adherence to Mediterranean diet has been associated to longer TL[13,14]. Previous studies have reported an association between dietary Se intake and telomere length in middle-aged and elderly populations[2]. However, the relationship between dietary Se intake and telomere length in the population with diabetes remains unknown. Therefore, we used the National Health and Nutrition Examination Survey database to examine the relationship between dietary Se and telomere length in patients with diabetes.

Methods

Study participants

The National Health and Nutrition Examination Survey (NHANES) data from 1999 to 2002 comes from the Centers for Disease Control and Prevention's survey, approved by the

Abbreviations: NHANES, National Health and Nutrition Examination Survey.

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research ethics review board and downloaded from http://www.cdc.gov/nchs/nhanes/index.htm. Written informed consent was obtained from all the participants. A total of 1332 participants with diabetes constituted the study. We set the criteria for selecting participants. The participants were included if they had telomere length, after removing telomere length and Se mean standard exceeding three standard deviations (*n* 981). Then, the participants were included if they had telomere length and dietary Se, after removing telomere length and dietary Se mean standard exceeding three standard deviations (*n* 936) and no missing information regarding BMI, physical activity, education, cancer or malignancy and congestive heart failure (*n* 878). Therefore, a total of 878 participants with diabetes were included in the study, as shown in Fig. 1.

Dietary selenium intake assessment

All participants had their first face-to-face recall interview in a MEC private room in NHANES, asking for detailed information on the type of food intake 24 h prior to the interview (midnight to midnight) and estimating the composition of the ingested food. For each participant, dietary intake data were recorded using the NHANES computer-assisted dietary interview system, and all data were collected and transmitted through an automated system. Dietary data were processed using the University of Texas Food Intake Analysis System and the USA Department of Agriculture Survey Nutrition Database, coding individual foods and portion sizes to calculate the nutritional values of nutritional intake. It contains dietary Se and other dietary components[15,16].

Telomere length assessment

Telomere length determination was performed in the laboratory of Dr. Elizabeth Blackburn, UC San Francisco, USA, selecting the blood sample of all participants in the NHANES database, using quantitative PCR methods to measure telomere length (T/S ratio) relative to standard reference DNA[17], as previously described in more detail[18,19]. The mean and standard deviation of the T/S ratio were calculated, and the interassay coefficient of variation

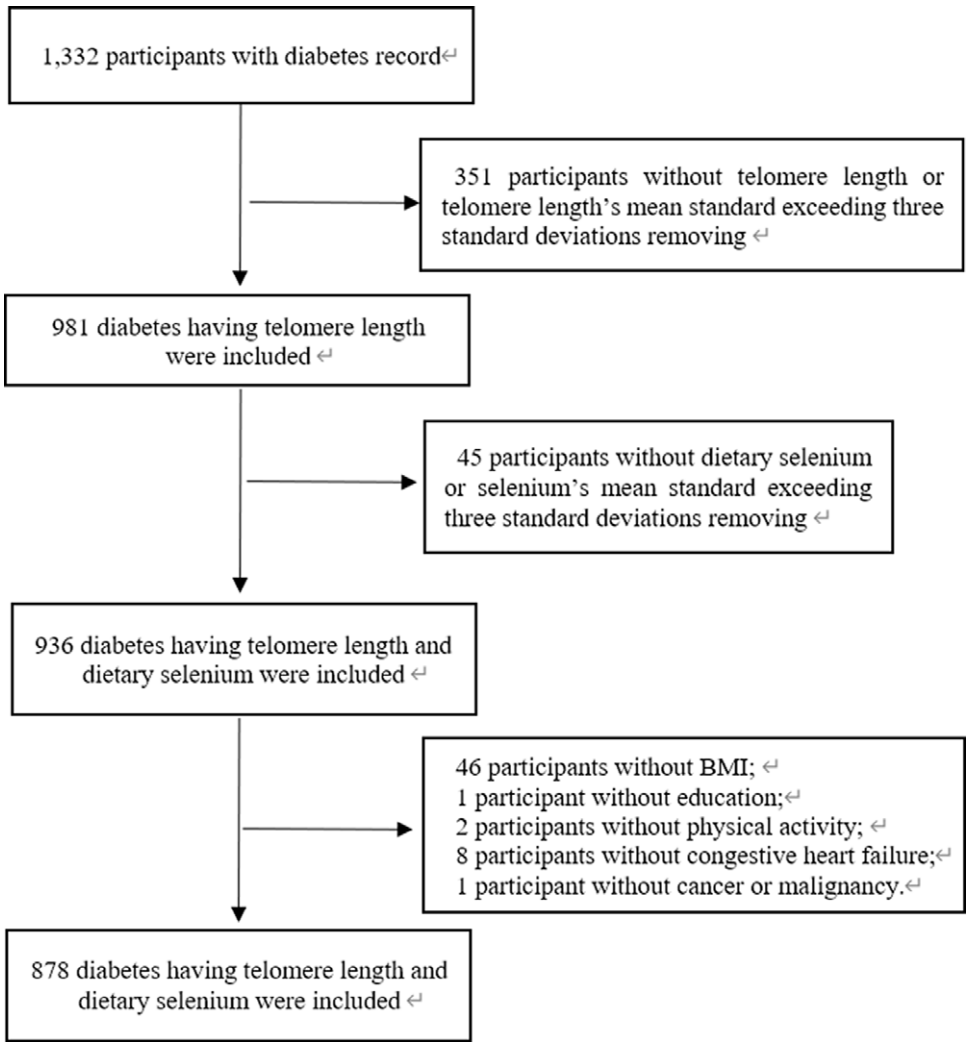

Fig. 1. The flow chart of participants selection.

Table 1. Characteristics of the participants with diabetes in different sex groups (Numbers and percentage, *n* 878)

	Total		Male (*n* 460)		Female (*n* 418)				
Characters	*n*	%	*n*	%	*n*	%	*β*	95 %CI	*P*
Age, year									
Mean	61·44		61·75		61·10		0·05	–0·09, 0·18	0·492
SD	13·82		12·50		15·15				
Age, CS							0·18	0·05, 0·31	0·008
≤ 45	134	15·26	56	12·17	78	18·66			
> 45	744	84·74	404	87·83	340	81·34			
Race							0·17	0·04, 0·30	0·176
Mexican American	256	29·16	131	28·48	125	29·90			
Other Hispanic	54	6·15	32	6·96	22	5·26			
Non-Hispanic white	340	38·72	191	41·52	149	35·65			
Non-Hispanic black	195	22·21	91	19·78	104	24·88			
Other Race	33	3·76	15	3·26	18	4·31			
BMI, kg/m^2									
Mean	31·34		30·55		32·22		0·24	0·11, 0·38	< 0·001
SD	6·89		6·26		7·43				
BMI, CS							0·22	0·09, 0·36	0·001
< 30	446	50·80	258	56·09	188	44·98			
≥ 30	432	49·20	202	43·91	230	55·02			
Physical Activity							0·35	0·21, 0·48	< 0·001
No aerobic activity	297	33·83	135	29·35	162	38·6			
Low activity	462	52·62	250	54·35	212	50·72			
Moderate activity	87	9·91	46	10·00	41	9·81			
High activity	32	3·64	29	6·30	3	0·72			
Education							0·11	–0·02, 0·24	0·254
Less than high school	433	49·32	222	48·26	211	50·48			
High school	183	20·84	90	19·57	93	22·25			
More than high school	262	29·84	148	32·17	114	27·27			
Energy intake, kcal									
Mean	1770·58		1992·92		1525·89		0·60	0·46, 0·73	< 0·001
SD	821·04		894·36		650·20				
Hypertension	615	70·05	305	66·30	310	74·16	0·17	0·04, 0·31	0·011
Congestive heart failure	70	7·97	38	8·26	32	7·66	0·02	–0·11, 0·15	0·741
Cancer or malignancy	104	11·85	56	12·17	48	11·48	0·02	–0·11, 0·15	0·752
Se intake, μg									
Mean	95·84		108·59		81·81		0·58	0·45, 0·72	< 0·001
SD	47·94		50·95		39·99				
Telomere Length, base pairs									
Mean	5558·90		5522·95		5598·45		0·14	0·00, 0·27	0·043
SD	552·80		530·17		574·71				

was 6·5 %. Finally, the T/S ratio was converted to bp (formula: base pairs = 3274 + 2413 × (T/S)), which was calculated by comparing the telomere restriction fragment length analysed using Southern blot and the T/S ratio of DNA samples using human diploid fibroblast IMR90[20]. The methods and data conversion for telomere length determination have been described in detail on the official website (–https://wwwn.cdc.gov/Nchs/Nhanes/2001–2002/TELO_B.htm).

Covariates assessment

The covariates included age, sex, education, race, BMI (BMI; weight divided by height squared (kg/m^2)), energy intake, hypertension, congestive heart failure, cancer or malignancy and physical activity. We categorised diabetes based on HbA1c level ≥ 6·5 %, fasting plasma glucose level ≥ 126 mg/dl or self-reported diabetes. We categorised ethnicity as non-Hispanic white, non-Hispanic black, Mexican American, other Hispanic or other races. Hypertension was categorised as systolic blood pressure ≥ 140 mmHg, diastolic blood pressure average ≥ 90 mmHg or self-reported hypertension. Physical activity was categorised as no aerobic activity, low activity, moderate activity or high activity. According to the NHANES, no aerobic exercise was defined as predominately sitting during the day and infrequent activity; standing or walking all the time during the day and not requiring frequent extraction of items is called low physical activity; carrying lightweight items or frequent mountain climbing is defined as moderate activity and having to work at high loads or carrying heavy objects is defined as high physical activity[21]. BMI was categorised as ≥ 30 kg/m^2 or < 30 kg/m^2. Age was categorised as > 45 years or ≤ 45 years.

Statistical analysis

First, the data were divided into two parts based on the male and female groups to find differences and general characteristics, using mean ± standard deviations or number and proportions

to expression. Second, the data were used in a stratified analysis to determine the relationship between Se intake and telomere length in different age groups, sex, race, BMI, physical activity, education, hypertension, congestive heart failure, cancer or malignancy. We then constructed generalised linear models to assess the association between dietary Se intake and telomere length in patients with diabetes. The first model was adjusted for age and sex to estimate this association, while the second model was further adjusted for general demographic characteristics, including age, sex, ethnicity and education. The third model additionally controlled for physical activity, BMI, energy intake, hypertension, congestive heart failure and cancer or malignancy. Lastly, after controlling for sex, we constructed generalised linear models to assess the association between dietary Se intake and telomere length in patients with diabetes. All analyses were based on a two-sided significance level ($P < 0{\cdot}05$). All analyses were performed using the statistical software packages R (http://www.R-project.org) and Empower stats (www.empowerstats.com, X&Y Solutions, Inc.).

Results

There were 878 participants with diabetes who were assessed for telomere length and dietary Se intake. The participants' average age was 61·44 ± 13·82 years. The characteristics of the participants with diabetes in different sex groups are shown in Table 1, and those in different age groups are shown in Table 2.

Subgroup analysis revealed a significant difference between Se intake and telomere length in women with diabetes (Table 3). The relationship between Se intake and telomere length in patients with diabetes in different models using a generalised linear model is shown in Table 4. In sex groups, we found 1 μg increase in dietary Se intake in female patients with diabetes, and then telomere length increased by 1·84 bp ($\beta = 1{\cdot}84$ (95 % CI: 0·15, 3·53)) after controlling for age, education, race, physical activity, BMI, energy intake, hypertension, congestive heart failure, and cancer or malignancy.

We also found that dietary Se intake has a line relation with telomere length in female patients with diabetes (Fig. 2); within the scope of 0–250 μg dietary Se intake, telomere length was longer following increased dietary Se intake.

Discussion

We used the NHANES database to recruit 878 patients with diabetes in the USA to investigate the relationship between dietary Se intake and telomere length. The findings showed a positive association between dietary Se intake and telomere length only in women with diabetes. In the dietary Se intake range of 0–250 μg, telomere length in the female population with diabetes increased with increasing dietary Se intake.

In the present study, we found an association between dietary Se intake and telomere length only in women with diabetes. These results were broadly consistent with those reported by Shu *et al.*[2]. Considering the population particularity, gender, age, race, education, physical activity, BMI, energy intake, hypertension, congestive heart failure, cancer or malignancy-related covariates were controlled in a generalised linear model; thus, the sample information was less than that of Shu *et al.*[2]. Changes in telomere length in women may be influenced by oestrogen levels in their bodies[22]. Oestrogen stimulates telomerase production and may prevent reactive oxygen species damage[23]. In addition, women have better antioxidant and physiological properties of selenoproteins, which may reduce telomere attrition[3,24]. Previous animal experiments have shown that female rats have a lower demand for Se than male rats[25,26], which is related to sex differences in telomere attrition[2]. However, the mechanism of action of sex on dietary Se intake and telomere length among diabetes remains unclear, and further studies are needed for confirmation.

Dietary Se intake is positively correlated with telomere length in patients with diabetes. This may be related to the following aspects. First, Se, an essential micronutrient for the human body, produces physiological characteristics in the form of selenoproteins and is absorbed by the body in the form of dietary Se. The anti-inflammatory properties of selenoproteins play a key role in telomere attrition, and the rapid replication of cells during inflammation accelerates cellular senescence and the extent of telomere attrition[27]. Second, the antioxidant defence properties of Se may prevent the development and progression of diabetes[28], and ingestion of a certain Se content is beneficial for the population with diabetes[29]. Third, long-term adherence to the Mediterranean diet can effectively reduce the risk of CVD and diabetes[30]. In addition, the Mediterranean diet has anti-inflammatory and antioxidant properties that may affect telomere length, with a higher intake being associated with longer telomere length[31]. We found a line relationship between dietary Se intake and telomere length among diabetes within the range of 0–250 μg from a smoothed curve fit. The intake of dietary Se is protective in humans. Insufficient Se intake can decrease immune and cognitive function and even cause death[1]. Laclaustra *et al.* found in the National Health and Nutrition Examination Survey that excessive Se levels in the body increased the prevalence of diabetes[32].

This study has three limitations. First, this study was a cross-sectional survey and did not directly infer a causal relationship between dietary Se intake and telomere length in patients with diabetes. Second, our study shows that dietary Se intake is significantly associated with telomere length only within the range of 0–250 μg, and relevant experimental studies are needed to demonstrate in the future beyond which range whether they are still relevant. Finally, the final sample size was limited to 878 participants.

Conclusions

Therefore, the present study demonstrates that dietary Se intake is significantly associated with telomere length only present in the female population with diabetes in the USA. However, further prospective studies are required to confirm this finding.

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Table 2. Characteristics of the participants with diabetes in different age groups
(Numbers and percentages, *n* 878)

	Total		Age ≤ 45 (*n* 134)		Age > 45 (*n* 744)				
Characters	*n*	%	*n*	%	*n*	%	*β*	95 %CI	*P*
Sex							0·25	0·07, 0·44	0·008
Male	460	52·39	56	41·79	404	54·30			
Female	418	47·61	78	58·21	340	45·70			
Race							0·33	0·15, 0·52	0·017
Mexican American	256	29·16	44	32·84	212	48·49			
Other Hispanic	54	6·15	9	6·72	45	6·05			
Non-Hispanic white	340	38·72	35	26·12	305	40·99			
Non-Hispanic black	195	22·21	38	28·36	157	21·10			
Other Race	33	3·76	8	5·97	25	3·36			
BMI, kg/m^2									
Mean	31·34		34·13		30·84		0·44	0·26, 0·63	< 0·001
SD	6·89		8·20		6·51				
BMI, CS							0·34	0·16, 0·53	< 0·001
<30	446	50·80	49	36·57	397	53·36			
≥30	432	49·20	85	63·43	347	46·64			
Physical Activity							0·31	0·12, 0·49	0·004
No aerobic activity	297	33·83	34	25·37	263	35·35			
Low activity	462	52·62	73	54·48	389	52·28			
Moderate activity	87	9·91	16	11·94	71	9·54			
High activity	32	3·64	11	8·21	21	2·82			
Education							0·24	0·06, 0·43	0·028
Less than high school	433	49·32	57	42·54	376	50·54			
High school	183	20·84	24	17·91	159	21·37			
More than high school	262	29·84	53	39·55	209	28·09			
Energy intake, kcal									
Mean	1770·58		2134·07		1705·11		0·49	0·30, 0·67	< 0·001
SD	821·04		978·27		772·18				
Hypertension	615	70·05	68	50·75	547	73·52	0·48	0·30, 0·67	< 0·001
Congestive heart failure	70	7·97	0	0	70	9·41	0·46	0·27, 0·64	< 0·001
Cancer or malignancy	104	11·85	5	3·73	99	13·31	0·35	0·16, 0·53	0·002
Se intake, μg									
Mean	95·84		105·57		94·09		0·23	0·04, 0·41	0·011
SD	47·94		54·46		46·49				
Telomere Length, base pairs									
Mean	5558·90		5897·26		5497·95		0·73	0·54, 0·91	< 0·001
SD	552·80		573·63		526·71				

Table 3. Relationship between selenium intake and telomere length in patients with diabetes
(Numbers and 95 % confidence intervals, *n* 878)

Characters	*n*	*β*	95 % CI	*P*	$P_{for\ interaction}$
Age, CS					0·680
≤ 45	134	0·96	−0·83, 2·75	0·296	
> 45	744	0·57	−0·25, 1·38	0·172	
Sex					0·008
Male	460	0·41	−0·54, 1·36	0·396	
Female	418	2·64	1·28, 4·00	< 0·001	
Race					0·820
Mexican American	256	1·02	−0·42, 2·45	0·165	
Other Hispanic	54	−0·48	−3·61, 2·65	0·765	
Non-Hispanic white	340	1·29	0·15, 2·44	0·027	
Non-Hispanic black	195	0·67	−1·10, 2·43	0·460	
Other Race	33	1·02	−2·33, 4·37	0·555	
BMI, CS					0·084
< 30	446	1·54	0·51, 2·56	0·004	
≥ 30	432	0·20	−0·91, 1·32	0·721	
Physical Activity					0·639
No aerobic activity	297	0·35	−0·99, 1·68	0·610	
Low activity	462	1·14	0·09, 2·18	0·034	
Moderate activity	87	−0·24	−3·02, 2·55	0·868	
High activity	32	1·89	−1·25, 5·03	0·247	
Education					0·472
Less than high school	433	0·41	−0·65, 1·48	0·450	
High school	183	1·58	−0·00, 3·17	0·052	
More than high school	262	0·90	−0·62, 2·42	0·247	
Hypertension					0·792
Yes	615	0·75	−0·21, 1·71	0·128	
No	263	0·96	−0·31, 2·23	0·142	
Congestive heart failure					0·064
Yes	70	−1·56	−4·08, 0·96	0·229	
No	808	1·06	0·26, 1·85	0·009	
Cancer or malignancy					0·523
Yes	104	0·11	−2·07, 2·30	0·920	
No	774	0·96	0·15, 1·77	0·021	

Table 4. Relationship between selenium and telomere length among diabetes in different models
(Numbers and 95 % confidence intervals)

Participants	Models	*β*	95 % CI	*P*	$P^*_{for\ trend}$
Sex					
Male	Model 1	−0·10	−1·00, 0·81	0·836	0·446
	Model 2	−0·07	−0·97, 0·84	0·881	0·583
	Model 3	−0·25	−1·46, 0·96	0·685	0·368
Female	Model 1	1·75	0·45, 3·05	0·009	0·026
	Model 2	1·79	0·48,3·11	0·008	0·028
	Model 3	1·84	0·15, 3·53	0·034	0·101
All participants					
	Model 1	0·56	−0·19, 1·31	0·143	0·274
	Model 2	0·55	−0·21, 1·30	0·156	0·235
	Model 3	0·42	−0·56, 1·41	0·399	0·466

Model 1 adjust for: sex; age.
Model 2 adjust for: sex; age; race; education.
Model 3 adjust for: sex; age; race; education; physical-activity; BMI; energy intake; hypertension; congestive heart failure; cancer or malignancy.

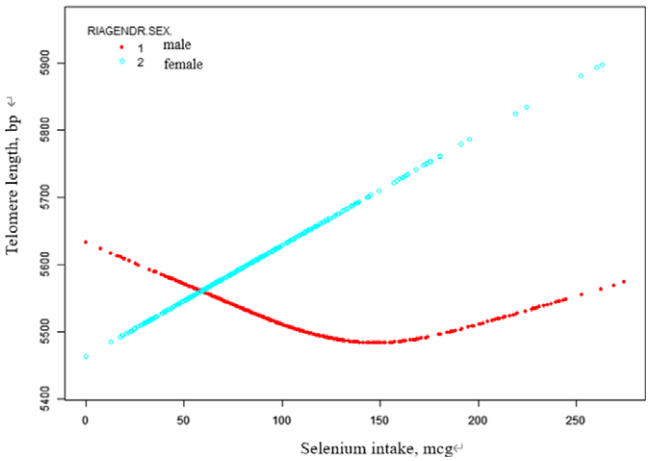

Fig. 2. Association between selenium and telomere length among diabetes in male and female.

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H-P. G., Q. Y., D-W. G. and W-X. H. contributed to the study design and writing; D-W. G. and Y-G. W. contributed to data analysis and discussion of the results; L-Z. D. and J-H. Z. contributed to article revision; J. W. and P. H. contributed to study design and manuscript revision. All authors have read and agreed to the published version of the manuscript.

References

1. Rayman MP (2012) Selenium and human health. *Lancet* **379**, 1256–1268.
2. Shu Y, Wu M, Yang S, *et al.* (2020) Association of dietary selenium intake with telomere length in middle-aged and older adults. *Clin Nutr* **39**, 3086–3091.
3. Ferguson LR, Karunasinghe N, Zhu S, *et al.* (2012) Selenium and its' role in the maintenance of genomic stability. *Mutat Res* **733**, 100–110.
4. Amaral AF, Cantor KP, Silverman DT, *et al.* (2010) Selenium and bladder cancer risk: a meta-analysis. *Cancer Epidemiol Biomarkers Prev* **19**, 2407–2415.
5. Zhuo H, Smith AH & Steinmaus C (2004) Selenium and lung cancer: a quantitative analysis of heterogeneity in the current epidemiological literature. *Cancer Epidemiol Biomarkers Prev* **13**, 771–778.
6. Nacamulli D, Mian C, Petricca D, *et al.* (2010) Influence of physiological dietary selenium supplementation on the natural course of autoimmune thyroiditis. *Clin Endocrinol* **73**, 535–539.
7. Kljai K & Runje R (2001) Selenium and glycogen levels in diabetic patients. *Biol Trace Elem Res* **83**, 223–229.
8. Cheng F, Carroll L, Joglekar MV, *et al.* (2021) Diabetes, metabolic disease, and telomere length. *Lancet Diabetes Endocrinol* **9**, 117–126.
9. Huang YQ, Liu L, Lo K, *et al.* (2020) The relationship between mean telomere length and blood pressure: results from the

National Health and Nutrition Examination Surveys. *Ann Transl Med* **8**, 535.
10. Fitzpatrick AL, Kronmal RA, Gardner JP, *et al.* (2007) Leukocyte telomere length and cardiovascular disease in the cardiovascular health study. *Am J Epidemiol* **165**, 14–21.
11. Zee RY, Castonguay AJ, Barton NS, *et al.* (2010) Mean leukocyte telomere length shortening and type 2 diabetes mellitus: a case-control study. *Transl Res* **155**, 166–169.
12. Yaffe K, Lindquist K, Kluse M, *et al.* (2011) Telomere length and cognitive function in community-dwelling elders: findings from the Health ABC Study. *Neurobiol Aging* **32**, 2055–2060.
13. Tucker LA (2017) Physical activity and telomere length in U.S. men and women: an NHANES investigation. *Prev Med* **100**, 145–151.
14. Nettleton JA, Diez-Roux A, Jenny NS, *et al.* (2008) Dietary patterns, food groups, and telomere length in the Multi-Ethnic Study of Atherosclerosis (MESA). *Am J Clin Nutr* **88**, 1405–1412.
15. NHANES (2002) Questionnaires, Datasets, and Related Documentation: Measuring Guides for the Dietary Recall Interview. https://www.cdc.gov/nchs/nhanes/measuring_guides_dri/measuringguides.htm (accessed May 2022).
16. NHANES (2004) Dietary Interview-Total Nutrient Intakes (DRXTOT_B): Data Documentation, Codebook, and Frequencies: Centers for Disease Control and Prevention. — https://wwwn.cdc.gov/Nchs/Nhanes/2001–2002/DRXTOT_B.htm (accessed May 2022).
17. NHANES (2004) Telomere Mean and Standard Deviation (Surplus) (TELO_B): Data Documentation, Codebook, and Frequencies: Centers for Disease Control and Prevention. — https://wwwn.cdc.gov/Nchs/Nhanes/2001–2002/TELO_B.htm (accessed May 2022).
18. Cawthon RM (2002) Telomere measurement by quantitative PCR. *Nucleic Acid Res* **30**, e47.
19. Needham BL, Adler N, Gregorich S, *et al.* (2013) Socioeconomic status, health behavior, and leukocyte telomere length in the National Health and Nutrition Examination Survey, 1999–2002. *Soc Sci Med* **85**, 1–8.
20. Needham BL, Rehkopf D, Adler N, *et al.* (2015) Leukocyte telomere length and mortality in the National Health and Nutrition Examination Survey, 1999–2002. *Epidemiol* **26**, 528–535.
21. NHANES (2004) Physical Activity (PAQ_B): Data Documentation, Codebook, and Frequencies: Centers for Disease Control and Prevention. —https://wwwn.cdc.gov/Nchs/Nhanes/1999–2000/PAQ_B.htm#PAQ180 (accessed May 2022).
22. Mayer S, Brüderlein S, Perner S, *et al.* (2006) Sex-specific telomere length profiles and age-dependent erosion dynamics of individual chromosome arms in humans. *Cytogenet Genome Res* **112**, 194–201.
23. Gardner M, Bann D, Wiley L, *et al.* (2014) Gender and telomere length: systematic review and meta-analysis. *Exp Gerontol* **51**, 15–27.
24. Carrero JJ, Stenvinkel P, Fellström B, *et al.* (2008) Telomere attrition is associated with inflammation, low fetuin-A levels and high mortality in prevalent haemodialysis patients. *J Intern Med* **263**, 302–312.
25. Brown DG & Burk RF (1973) Selenium retention in tissues and sperm of rats fed a Torula yeast diet. *J Nutr* **103**, 102–108.
26. Burk RF, Lawrence RA & Correia MA (1980) Sex differences in biochemical manifestations of selenium deficiency in rat liver with special reference to heme metabolism. *Biochem Pharmacol* **29**, 39–42.
27. Zhang J, Rane G, Dai X, *et al.* (2016) Ageing and the telomere connection: an intimate relationship with inflammation. *Ageing Res Rev* **25**, 55–69.
28. Siddiqi SM, Sun C, Wu X, *et al.* (2020) The correlation between dietary selenium intake and type 2 diabetes: a cross-sectional population-based study on North Chinese adults. *Biomed Res Int* **2020**, 8058463.
29. Rajpathak S, Rimm E, Morris JS3, *et al.* (2005) Toenail selenium and cardiovascular disease in men with diabetes. *J Am Coll Nutr* **24**, 250–256.
30. Martín-Peláez S, Fito M & Castaner O (2020) Mediterranean Diet Effects on Type 2 Diabetes Prevention, Disease Progression, and Related Mechanisms. *Nutrients* **122**, 236.
31. Canudas S, Becerra-Tomás N, Hernández-Alonso P, *et al.* (2020) Mediterranean Diet and telomere length: a systematic review and meta-analysis. *Adv Nutr* **111**, 544–1554.
32. Laclaustra M, Navas-Acien A, Stranges S, *et al.* (2009) Serum selenium concentrations and diabetes in USA adults: National Health and Nutrition Examination Survey (NHANES) 2003–2004. *Environ Health Perspect* **117**, 1409–1413.

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