



RESEARCH PAPER

Impact of a healthy lifestyle on all-cause and cardiovascular mortality after stroke in the USA

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ABSTRACT

Background Little is known about the effects of a healthy lifestyle on mortality after stroke. This study assessed whether five healthy lifestyle factors had independent and dose dependent associations with all-cause and cardiovascular mortality after stroke.

Methods In a nationally representative sample of the US population (n=15 299) with previous stroke (n=649) followed from survey participation (1988–1994) through to mortality assessment (2000), the relationship between five factors (eating ≥ 5 servings of fruits/vegetables per day, exercising >12 times/month, having a body mass index of 18.5–29.9 mg/kg², moderate alcohol use [1 drink/day for women and 2 drinks/day for men] and not smoking) and all-cause and cardiovascular mortality was assessed.

Results Mean age was 67.0 years (SE 1.1 years) and 53% were women. After adjusting for covariates, abstaining from smoking (HR 0.57, CI 0.34 to 0.98) and exercising regularly (HR 0.66, CI 0.44 to 0.99) were associated with lower all-cause mortality but no individual factors had independent associations with cardiovascular mortality. All-cause mortality decreased with higher numbers of healthy behaviours (1–3 factors vs none: HR 0.12, CI 0.03 to 0.47; 4–5 factors vs none: HR 0.04, CI 0.01 to 0.20; 4–5 factors vs 1–3 factors: HR 0.38, CI 0.22 to 0.66; trend p=0.04). Similar effects were observed for cardiovascular mortality (4–5 factors vs none: HR 0.08, CI 0.01 to 0.66; 1–3 factors vs none: HR 0.15, CI 0.02 to 1.15; 4–5 factors vs 1–3 factors: HR 0.53, CI 0.28 to 0.98; trend p=0.18).

Conclusions Regular exercise and abstinence from smoking were independently associated with lower all-cause mortality after stroke. Combinations of healthy lifestyle factors were associated with lower all-cause and cardiovascular mortality in a dose dependent fashion.

BACKGROUND

Stroke survivors have a higher mortality risk than the general population, even years after the index event.^{1–3} Studies have revealed that adherence to a combination of healthy lifestyle practices is associated with reduced stroke incidence^{4–5} and mortality risk in the general population^{6–11}; however, little is known about the effect of a healthy lifestyle on risk of death after stroke.

The objectives of this study were twofold: (1) to assess whether each of the individual five healthy lifestyle factors were independently associated with lower all-cause and cardiovascular mortality after stroke and (2) to investigate whether higher

numbers of healthy lifestyle behaviours were associated with a greater survival benefit.

METHODS

Population for study

The National Health and Nutrition Examination Survey (NHANES) are cross sectional samples of the US civilian, non-institutionalised population conducted by the National Center for Health Statistics, a branch of the Centers for Disease Control and Prevention. The protocols for conduct were approved by the National Center for Health Statistics institutional review board and informed consent was obtained from all participants.¹² The sampling plan followed a complex, stratified, multistage, probability cluster design, with oversampling of non-Hispanic blacks, Mexican Americans and the elderly, to enhance the precision of prevalence estimates in those groups. Details of the survey design and examination procedures have been previously published.¹²

In the third NHANES (NHANES III), conducted from 1988 to 1994, 33 199 adults were interviewed. The study outcomes—all-cause and cardiovascular mortality—were recorded from NHANES III mortality follow-up data, which relied on a probabilistic match between NHANES III and National Death Index death certificate records. Mortality records were available for 20 024 of 20 050 adults who completed both interviews and medical examinations. Mortality assessments, including cause specific mortality and mortality dates, were conducted from baseline interview to 31 December 2000. Cause specific mortality was coded using the ninth revision of the International Classification of Diseases, Injuries and Causes of Death (ICD-9) for deaths occurring between 1988 and 1998 and the 10th revision (ICD-10) for deaths occurring between 1999 and 2000. The Underlying Cause of Death 113 Groups All Years (UCOD-113) variable recoded all deaths prior to 1999 coded under ICD-9 guidelines into comparable ICD-10 codes.¹³

Of 649 persons with a self-reported history of stroke, 164 were assigned negative survey weights and were excluded by NHANES. We followed the NHANES survey design by excluding these individuals, leaving 485 persons for the analysis. All 485 persons had mortality follow-up data. Of these 485 persons, 97 (20%) had missing values for the covariates, leaving 388 persons for the complete case analysis. We compared the healthy lifestyle factors and covariates among the complete set (n=388) versus the incomplete set (n=97) to assess qualitative differences between groups.

Primary outcome variable

The primary outcome variable was all-cause mortality, analysed as a time to event outcome recorded in months (event was deceased from all causes versus alive).

Secondary outcome variable

The secondary outcome variable was cardiovascular mortality, analysed as a time to event outcome recorded in months (event was deceased due to cardiovascular causes versus alive while adjusting for competing non-cardiovascular causes). Cardiovascular deaths included deaths from any heart disease, cerebrovascular cause, atherosclerosis or hypertension (UCOD-113 codes 054-074). Stroke mortality (deaths from any cerebrovascular cause, UCOD-113 code 070) was not used as an outcome as it was too rare to formally control for covariates.

Primary predictor variables

Definitions of healthy lifestyle behaviours were consistent with a previous study of healthy lifestyle practices/factors: eating ≥ 5 servings of fruits/vegetables/day, exercising >12 times/month, body mass index (BMI) of 18.5–29.9 mg/kg², drinking alcohol in moderation (1 drink/day for women and 2 drinks/day for men) and not smoking.¹⁴ These variables have been evaluated in prior studies^{6 14} and are endorsed by national guidelines on stroke prevention.^{15 16} BMI was calculated from height and weight (kg/m²) measured using standardised protocols. The other variables were obtained by self-report.

Diet

Although studies have used different definitions of a healthy diet, several studies used fruit/vegetable intake,^{17–19} and the American Heart Association recommends five servings of fruits/vegetables/day as part of a healthy diet.²⁰

Exercise

Physical activity frequency was determined according to participation in leisure time physical activities within the previous month, including walking, jogging or running, riding a bicycle, swimming, aerobic exercise or other similar activities. Current guidelines recommend ≥ 30 min of moderate intensity activity ≥ 5 days/week²¹; however, a cardiovascular benefit is evident with as little as 1 h of running or 30 min of weight training per week.²² Physical activity was divided into two frequency groups (0–12 and >12 times/month), consistent with national recommendations at the time of NHANES 1988–1994.²³

Body mass index

Although a BMI of 18.5–24.9 kg/m² is considered optimal, there is no excess mortality risk for overweight individuals (BMI 25–29.9 kg/m²) compared with normal weight individuals (BMI 18.5–24.9 kg/m²);²⁴ therefore, a more liberal range of 18.5–29.9 kg/m² was used for this study.

Alcohol use

Moderate alcohol consumption was defined as 1 drink/day for women and 2 drinks/day for men, according to current USDA guidelines.²⁵

Covariates

Covariates assessed were: age, sex, race/ethnicity, history of myocardial infarction (MI), hypertension, diabetes mellitus (DM), hypercholesterolaemia, hypertriglyceridaemia and low level of high density lipoprotein (HDL) cholesterol.

Race/ethnicity was obtained by self-report. History of MI was defined by self-reported physician diagnosis. Hypertension was defined by self-reported physician diagnosis, self-reported current medical therapy or mean of the first three blood pressure readings >140 mm Hg systolic or 90 mm Hg diastolic. DM was defined by self-reported physician diagnosis, self-reported current medical therapy (insulin or oral agents) or glycosylated haemoglobin level $>7\%$. Hypercholesterolaemia was defined by self-reported physician diagnosis, self-reported current medical therapy or total cholesterol level >200 mg/dl. Hypertriglyceridaemia was defined as triglyceride level >150 mg/dl. Low HDL level was defined as HDL <50 mg/dl in women and <40 mg/dl in men.

Statistical analysis

Weighted estimates were applied to the descriptive prevalence analysis using NHANES mobile examination centre examined sample weight values. These weights adjusted for the differential probabilities of selection and non-response in the survey sample. To account for NHANES clustering, stratification and unequal weights on the Cox regression models below, the primary sampling unit variable, the stratification variable and the weight variable were adjusted for in the analysis. Statistical hypotheses were tested using $p < 0.05$ as the level of statistical significance.

Bivariate analysis

To assess the bivariate relationship between each covariate and all-cause mortality, the Cox regression model was used, adjusting for the survey design variables. For cardiovascular mortality, the Cox model was expanded to a competing risks Cox model as non-cardiovascular mortality is a simultaneous competing risk.

Multivariable analyses

The multivariable Cox regression and competing risks models were used to assess the simultaneous influence of all five healthy lifestyle factors on risk of all-cause and cardiovascular mortality, respectively, while adjusting for covariates. The final multivariable models excluded variables that were not significant at the $p < 0.25$ level using backwards selection. The relation between number and combination of health factors (versus none) and mortality outcomes was assessed using linear contrasts under the above additive models. As some excluded variables had missing data, the sample sizes for the final multivariable models increased slightly, with 428 individuals in the all-cause mortality model and 419 subjects in the cardiovascular mortality model.

To assess whether a higher number of health factors was associated with improved mortality outcomes, we divided the sample into groups based on number of health factors followed (0, 1–3, 4–5) and carried out a Cox regression analysis adjusting for demographic and clinical factors. Those who followed five health factors were rare and were thus grouped with those who followed four health factors. Since the analysis indicated that those who followed one, two or three health factors had similar mortality outcomes in the above Cox model, they were combined into a single category. Adjusted survival curves over time in the above groups were estimated under the above Cox regression model. For cardiovascular mortality, the corresponding cumulative incidence curves over time were constructed under the competing risks regression model after adjusting for the covariates.

Because of the potentially intersecting causal relationships among confounders and primary predictors, several nested

models were assessed. As exercise and diet likely influence BMI, we considered a model with only BMI as a health factor (without the other four healthy factors) after adjusting for demographic factors (age, sex, race) and clinical factors (the clinical factors in our final multivariable analysis) and a similar model without the clinical factors (hypothesised mediators). In addition, we considered a model with four healthy factors without BMI (hypothesised mediator) after adjusting for demographic and clinical factors and a similar model without the clinical factors (hypothesised mediators). Finally, we considered a model with all variables included. To strengthen the validity of our findings, analyses were performed both on the complete case sample (n=388) and after using single imputation for the missing values (n=485).

RESULTS

Among all adults with a history of stroke who participated in NHANES 1988–1994, mean age was 67.0 years (SE 1.1 years) and 50% were women. Table 1 depicts the demographic characteristics, medical comorbidities and lifestyle practices of individuals with a history of stroke. The majority of stroke survivors were white (79%), had hypertension (72.5%), hypercholesterolaemia (67%), hypertriglyceridaemia (59%) and low HDL cholesterol (52%). With respect to lifestyle factors, most stroke survivors were non-smokers (75%), ate 1–4 servings of fruits/vegetables per day (58%), had a BMI in the 18.5–29.9 kg/m² range (71%) and

did not drink (76%). The only differences between the complete and incomplete sets were that individuals in the complete set were more likely to be female and to exercise regularly.

Of the 388 individuals with a history of stroke, 208 persons died, of whom 126 died of cardiovascular causes. After bivariate analysis, healthy factors associated with lower all-cause mortality after stroke included moderate alcohol use (versus none) (HR 0.41, CI 0.22 to 0.76) and regular exercise (HR 0.59, CI 0.40 to 0.86) (table 2). Abstinence from smoking was associated with higher all-cause mortality; however, this effect only approached significance (HR 1.57, CI 0.98 to 2.52). After bivariate analysis, healthy practices associated with lower cardiovascular mortality after stroke included eating 1–4 servings of fruits/vegetables/day (versus none) (HR 0.30, CI 0.12 to 0.74), eating ≥5 servings of fruits/vegetables/day (versus none) (HR 0.44, CI 0.19 to 1.02) and moderate alcohol use (versus none) (HR 0.51, CI 0.25 to 1.05); however, the latter two variables only approached significance (table 2). Among covariates, increasing age, history of MI, hypertension and DM were associated with higher all-cause and cardiovascular mortality after stroke (table 2).

Regular exercise (HR 0.66, CI 0.44 to 0.99) and not smoking (HR 0.57, CI 0.34 to 0.98) were independently associated with lower all-cause mortality after adjusting for covariates (table 3). The nested multivariable models using the complete dataset and imputed missing variables showed similar results (see supplementary tables 1 and 2 available online only). None of the healthy lifestyle factors independently lowered the risk of cardiovascular mortality after stroke after adjusting for covariates; however, eating 1–4 servings of fruits/vegetables/day (versus none) (HR 0.30, CI 0.08 to 1.08) and eating ≥5 servings of fruits/vegetables/day (versus none) (HR 0.30, CI 0.09 to 1.04) had protective effects approaching significance (table 4). Again, the nested multivariable models using the complete dataset and imputed missing variables showed similar results (see supplementary tables 1 and 2 available online only). Covariates with independent adverse effects on all-cause mortality after stroke were increasing age, history of MI and DM (table 3); covariates with independent adverse effects on cardiovascular mortality after stroke were increasing age and hypercholesterolaemia, while female sex had a protective effect (table 4).

The nested models revealed that the effect of healthy lifestyle practices/factors was similar regardless of whether BMI and/or the six clinical factors were included (see supplementary tables 1 and 2 available online only). In addition, BMI as an individual factor was not important regardless of the inclusion or exclusion of other variables. In general, results from the imputed and complete case analyses were qualitatively similar.

Analysis of the relationship between number of healthy lifestyle factors and all-cause mortality revealed a cumulative effect (figure 1). The rate of all-cause mortality was reduced by 96% in those who followed at least four factors versus none (HR 0.04; CI 0.01 to 0.20) and by 88% in those who followed 1–3 factors versus none (HR 0.12; CI 0.03 to 0.47), after controlling for the other factors. Consistent with a cumulative effect, adherence to 4–5 factors was associated with significantly better mortality outcomes than adherence to only 1–3 factors, after controlling for the other factors (HR 0.38; CI 0.22 to 0.66). The results were similar even after controlling for the individual health factors, including exercise and smoking (HR 0.33; CI 0.15 to 0.71). Moreover, once the number of health factors was known, smoking and exercise were no longer significant.

For cardiovascular mortality, results were similar, although slightly less robust (figure 2). The rate of cardiovascular mortality was reduced by 85% for those who followed 1–3

Table 1 Descriptive summary of population—sample n=388 (weighted sample n=3 002 561)

Variable	Weighted frequency	Weighted per cent	SE (%)
Demographics			
Female	1 497 764	49.9	3.9
Hispanic	295 480	9.8	2.6
Black	328 902	11.0	1.6
White	2 378 179	79.2	2.9
Medical comorbidities			
Hypertension	2 175 824	72.5	3.5
Diabetes mellitus	761 041	25.3	2.6
Hypercholesterolaemia	2 003 838	66.7	3.4
Hypertriglyceridaemia	1 765 326	58.8	3.1
Low level of high density lipoprotein	1 574 440	52.4	4.7
History of myocardial infarction	689 934	23.0	3.3
Lifestyle factors			
Non-smoker	2 251 751	75.0	3.4
≥5 servings fruits/vegetables/day	1 188 398	39.6	4.0
1–4 servings fruits/vegetables/day	1 755 977	58.5	3.9
0 servings fruits/vegetables/day	58 186	1.9	0.6
Regular exercise	957 260	31.9	3.5
Body mass index ≥30 kg/m ²	847 054	28.2	3.9
Body mass index <18.5 kg/m ²	34 556	1.2	0.6
Body mass index 18.5–29.9 kg/m ²	2 120 951	70.6	3.9
Heavy alcohol intake	42 038	1.4	0.6
No alcohol intake	2 270 615	75.6	3.6
Moderate alcohol intake	689 908	23.0	3.6
No of lifestyle factors followed			
0	102 432	3.4	1.3
1	482 079	16.1	2.9
2	1 075 592	35.8	3.3
3	879 540	29.3	3.9
4	378 197	12.6	2.7
5	84 720	2.8	1.2

Table 2 Bivariate analyses of predictors of all-cause and cardiovascular mortality after stroke among individuals with a self-reported history of stroke

Predictor	All-cause mortality		Cardiovascular mortality	
	HR (95% CI)	p Value	HR (95% CI)	p Value
Body mass index ≥ 30 kg/m ² vs normal	0.76 (0.50 to 1.16)	0.20	0.78 (0.44 to 1.42)	0.42
Body mass index < 18.5 kg/m ² vs normal	0.78 (0.35 to 1.75)	0.54	1.03 (0.46 to 2.28)	0.95
Abstinence from smoking	1.57 (0.98 to 2.52)	0.06	1.70 (0.89 to 3.27)	0.11
≥ 5 servings fruits/vegetables vs none	0.73 (0.32 to 1.68)	0.46	0.44 (0.19 to 1.02)	0.06
1–4 servings fruits/vegetables vs none	0.56 (0.24 to 1.33)	0.19	0.30 (0.12 to 0.74)	0.009
Moderate vs heavy alcohol intake	0.53 (0.14 to 2.08)	0.36	5.10 (0.56 to 47.62)	0.15
Moderate vs no alcohol intake	0.41 (0.22 to 0.76)	0.005	0.51 (0.25 to 1.05)	0.07
Regular exercise	0.59 (0.40 to 0.86)	0.007	0.69 (0.41 to 1.17)	0.17
Age (per year)	1.07 (1.04 to 1.09)	< 0.001	1.05 (1.02 to 1.08)	< 0.001
Female vs male	1.05 (0.72 to 1.52)	0.79	0.80 (0.50 to 1.29)	0.36
Hispanic vs white non-Hispanic	0.52 (0.24 to 1.15)	0.10	0.49 (0.16 to 1.48)	0.20
Black vs white non-Hispanic	1.14 (0.79 to 1.64)	0.47	0.91 (0.60 to 1.39)	0.67
Hypertension	1.61 (1.05 to 2.47)	0.03	2.07 (1.10 to 3.87)	0.02
Diabetes mellitus	1.91 (1.28 to 2.83)	0.002	1.69 (1.08 to 2.67)	0.02
History of myocardial infarction	1.59 (1.13 to 2.22)	0.008	1.64 (1.12 to 2.40)	0.01
Hypercholesterolaemia	0.91 (0.66 to 1.25)	0.56	1.44 (0.89 to 2.31)	0.13
Hypertriglyceridaemia	0.99 (0.69 to 1.41)	0.95	0.98 (0.64 to 1.51)	0.92
Low level of high density lipoprotein	1.38 (0.92 to 2.06)	0.12	1.23 (0.79 to 1.92)	0.36

health factors versus none (HR 0.15; CI 0.02 to 1.15) and by 92% for those who followed 4–5 health factors versus none (HR 0.08; CI 0.01 to 0.66). Moreover, those who followed at least four health factors had a 47% reduction in the rate of cardiovascular mortality compared with those who followed only 1–3 health factors (HR 0.53; CI 0.28 to 0.98). Results were similar even after controlling for the individual health factors (HR 0.42; CI 0.18 to 0.98).

DISCUSSION

We found that a combination of healthy lifestyle factors is associated with lower all-cause and cardiovascular mortality after stroke. Among the individual healthy lifestyle factors, only regular exercise and not smoking were independently associated with lower all-cause mortality, while eating ≥ 1 serving of fruits/vegetables/day was associated with a trend towards lower cardiovascular mortality after stroke, after controlling for covariates. Higher numbers of healthy lifestyle factors amplified reductions in all-cause and cardiovascular mortality. The

dose–response association is in accord with other studies showing a graded cardiovascular benefit of healthy lifestyle practices.^{4 7 26 27}

This is the first study to our knowledge to assess the effect of a healthy lifestyle on mortality after stroke. Prior studies in the general population and in those with established coronary artery disease revealed that adopting a healthy lifestyle led to lower cardiovascular events, including stroke, and reduced cardiovascular and all-cause mortality.^{4–7 26–30} While previous studies revealed that each healthy behaviour independently lowered the risk for cardiovascular events,^{5 26 27} all-cause mortality⁷ and cardiovascular mortality,⁷ our study only showed an independent effect of regular exercise and not smoking on all-cause mortality after stroke. Most studies explored the influence of healthy factors in the general population; perhaps the role of these factors in persons with established symptomatic cerebrovascular disease is different. In addition, all except one prior study⁶ used different definitions of healthy lifestyle practices.

Table 3 Multivariable Cox hazard model* for predictors of all-cause mortality after stroke (n=428)

Predictor	HR (95% CI)	p Value
Normal BMI vs BMI ≥ 30 kg/m ²	0.96 (0.63 to 1.47)	0.86
Normal BMI vs BMI < 18.5 kg/m ²	1.67 (0.30 to 9.09)	0.56
Abstinence from smoking	0.57 (0.34 to 0.98)	0.04
≥ 5 servings fruits/vegetables vs none	0.46 (0.14 to 1.47)	0.19
1–4 servings fruits/vegetables vs none	0.41 (0.13 to 1.35)	0.14
Moderate vs heavy alcohol intake	0.61 (0.22 to 1.69)	0.34
Moderate vs no alcohol intake	0.65 (0.36 to 1.19)	0.16
Regular exercise	0.66 (0.44 to 0.99)	0.04
Age (per year)	1.09 (1.05 to 1.12)	< 0.001
Female vs male	0.74 (0.52 to 1.07)	0.10
Diabetes mellitus	2.09 (1.44 to 3.04)	< 0.001
History of myocardial infarction	1.54 (1.08 to 2.20)	0.02

*Model adjusted for: BMI, smoking, servings of fruits/vegetables, alcohol intake, exercise, age, sex, history of myocardial infarction and diabetes mellitus. BMI, body mass index.

Table 4 Multivariable competing risks regression model* for predictors of cardiovascular mortality after stroke (n=419)

Predictor	HR (95% CI)	p Value
Normal BMI vs BMI ≥ 30 kg/m ²	0.85 (0.46 to 1.56)	0.61
Normal BMI vs < 18.5 kg/m ²	0.77 (0.14 to 4.35)	0.76
Abstinence from smoking	0.85 (0.41 to 1.75)	0.66
≥ 5 servings fruits/vegetables vs none	0.30 (0.09 to 1.04)	0.06
1–4 servings fruits/vegetables vs none	0.30 (0.08 to 1.08)	0.07
Moderate vs heavy alcohol intake	7.14 (0.68 to 100.00)	0.10
Moderate vs no alcohol intake	0.62 (0.31 to 1.25)	0.19
Regular exercise	0.76 (0.47 to 1.23)	0.27
Age (per year)	1.07 (1.03 to 1.11)	< 0.005
Female vs male	0.48 (0.29 to 0.80)	0.004
History of myocardial infarction	1.45 (0.93 to 2.26)	0.10
Hypercholesterolaemia	1.79 (1.12 to 2.89)	0.02

*Model adjusted for: BMI, smoking, servings of fruits/vegetables, alcohol intake, exercise, age, sex, history of myocardial infarction and hypercholesterolaemia. BMI, body mass index.

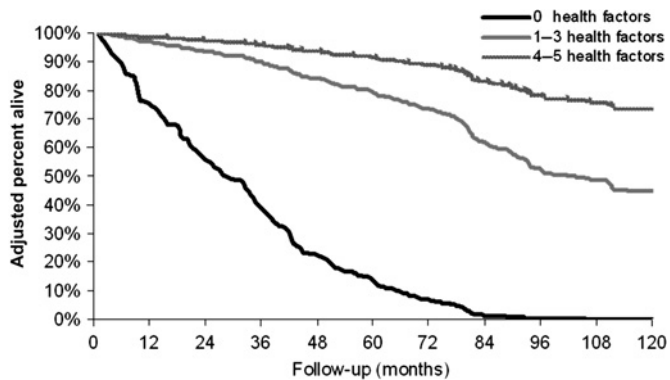


Figure 1 Protective effects of number of healthy lifestyle practices on all-cause mortality after adjustment for covariates.

Our study revealed an overall greater benefit of healthy behaviours compared with studies of primary stroke prevention^{4,5} and mortality reduction,^{6,7} but a similar effect size compared with studies of coronary heart disease prevention.^{26,27} However, different definitions of healthy lifestyle practices limit the extent to which comparisons can be made.

This study has several limitations. Firstly, since NHANES is cross sectional, participants' medical history, medication use and lifestyle practices prior to the stroke were unknown. In addition, the survey did not assess either stroke severity or post-stroke disability. These factors, which can potentially play a role in stroke mortality, were not controlled for. For example, healthy lifestyle factors may affect stroke severity which in turn affects stroke mortality. In addition, stroke severity affects the ability to adhere to lifestyle practices. Secondly, due to the cross sectional nature of the NHANES evaluation, we were only able to determine the presence or absence of healthy lifestyle factors at a time point after the stroke, without controlling for time since stroke or duration of adherence to healthy lifestyle practices. In addition, individuals' adherence to healthy lifestyle practices may have changed from the initial NHANES assessment (1988–1994) to the time of the outcomes assessment in 2000. Thirdly, NHANES relies on self-reported history of stroke, exercise frequency, alcohol use, smoking and fruit/vegetable intake. Although NHANES has not validated self-reporting of stroke, other studies found this method to have a sensitivity of 80–95% and a specificity of 96–99%.^{31,32} Fourthly, the effect of healthy lifestyle practices on mortality may differ in individuals

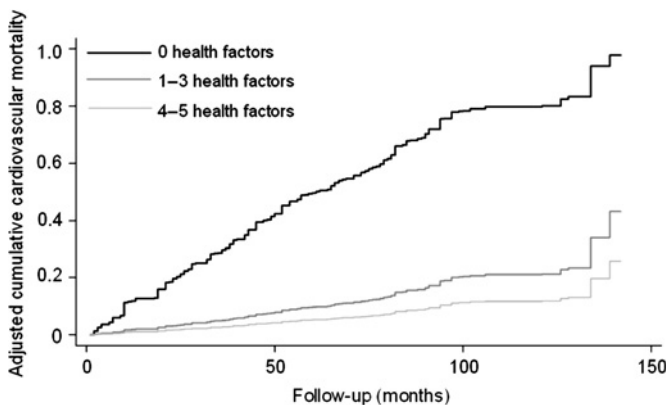


Figure 2 Protective effects of number of healthy lifestyle practices on cardiovascular mortality after adjustment for covariates.

with ischaemic versus haemorrhagic strokes, and the NHANES questionnaire does not differentiate between ischaemic and haemorrhagic stroke. Finally, only ~60% of stroke survivors who participated in NHANES were included in the final analysis; this relatively small number limited the power to detect effects from individual behaviours.

Nevertheless, this study implies that individuals with previous stroke have a lower risk of death from all-causes if they exhibit a higher number of healthy lifestyle factors, suggesting that interventions for improving adherence to healthy lifestyle behaviours among stroke patients may be warranted. Given the difficulties in accomplishing lifestyle change, interventions will likely require a multifaceted approach, incorporating education, social support and community involvement.

Competing interests None.

Ethics approval This was a cross sectional study performed by the Centers for Disease Control and all approvals were obtained.

Contributors AT: conception and design, analysis and interpretation of the data, drafting the article and final approval of the version to be published. DM: acquisition of the data, statistical analysis, analysis and interpretation of the data, critical revision of the manuscript for important intellectual content and final approval of the version to be published. BO: conception and design, analysis and interpretation of the data, critical revision of the manuscript for important intellectual content and final approval of the version to be published.

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