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Impact of socioeconomic, behavioral and clinical risk factors on mortality

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ABSTRACT

This study investigates the relative contributions of socioeconomic status (SES), behavioral and clinical risk factors on mortality. The Third National Health and Nutrition Survey Linked Mortality File was used to examine the association of SES (race, insurance, education, income), behavioral (smoking, obesity, physical activity), and clinical (elevated blood pressure, triglyceride level, lipid levels, C-reactive protein (CRP)) risk factors with 6–12-year all-cause mortality. Respondents were stratified by known chronic diseases into one of the following categories: no chronic disease, non-cardiovascular chronic disease, cardiovascular disease, and diabetes. The overall weighted mortality rate was 9.5% with the highest mortality rate among diabetics. Race, insurance coverage, income, smoking status, inadequate physical activity, elevated blood pressure and elevated CRP were independently associated with mortality in the overall population. When stratified by chronic disease, SES factors remained associated with mortality, most strongly in the healthy population. Current smoking and inadequate physical activity were also associated with mortality across disease groups while clinical risk factors were less consistent. SES factors, health behaviors and clinical risk factors were all associated with mortality even when baseline health status and chronic diseases are taken into account. Efforts to reduce mortality will require a multi-faceted approach incorporating healthy behaviors and accessible health care systems in addition to clinical advances

Keywords chronic disease, individual behavior, mortality

Background

A growing body of literature demonstrates that despite marked progress in medical technology, reductions in USA overall mortality rates have thus far been modest relative to other industrialized countries.[1,2] Advances in molecular biology, genetics and computational biology have increased our understanding of the biological precursors of disease that may underlie mortality. Clinical risk factors can identify individuals who should be targeted for personalized risk reduction strategies.[3–5] At the same time, ongoing research suggests that at least two other domains may be critical for predicting morbidity and mortality. First, mortality is strongly related to socioeconomic status (SES), as measured by income, education or occupation, with lower SES associated with higher mortality.[1,6–8] Second, healthy behaviors have also been shown to be modifiable risk factors for premature mortality. McGinnis and Foege[9] and Mokdad *et al.*[10] identified the major non-genetic modifiable risk factors that contributed to mortality in the USA and this work was later updated with 2000 data. They estimated that approximately half of all deaths could be attributed to risky health behaviors; the most prevalent being smoking, poor diet and physical inactivity.

Despite the wealth of research showing that SES, health behaviors and clinical risk factors individually contribute toward mortality, these three domains are almost always examined separately. Whereas studies of biochemical markers and other clinical factors have predominantly focused on high-risk populations who are receiving medical care, research on SES and health behaviors have primarily examined healthier, population-based samples. Few studies have attempted to measure the relative contributions of each of these risk factors on all-cause mortality in the same sample.[11] One study used county-level analyses in the USA to identify regions with excess mortality due to chronic diseases and injuries.[8] Another study used a population-based survey and registry to identify predictors of hospitalization.[12] Behavioral, biological and SES risk factors from a 1998 survey were used to simultaneously predict time to first hospitalization, demonstrating that risk factors from all three

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domains were significantly associated with hospitalization. The current study builds upon the previous work using a nationally representative survey that includes clinical, SES and health behavior data along with mortality follow-up to: (1) investigate the relative contribution of SES, behavioral and clinical factors on mortality at the individual level and (2) investigate how the relative contributions of each of these categories varies with the presence or absence of known chronic disease.

Methods

This study uses data from the Third National Health and Nutrition Survey (NHANES III) Linked Mortality File, a population-based survey and clinical assessment linked with mortality follow-up. The NHANES III was conducted by the National Center for Health Statistics (NCHS) in two phases between 1988 and 1994. The survey was a multistage cluster national probability sample of 33 994 civilian non-institutionalized Americans that over sampled Hispanic, African American and elderly populations, and consisted of interviews, physical examinations and laboratory tests. The sampling methods for NHANES III have been described previously.[13,14] The NHANES III Linked Mortality File matched NHANES III participants aged 17 and older with death records from the National Death Index (NDI) through 2000. Because of the staggered interviews, the mortality follow-up period ranges from 6 to 12 years post interview. The linking of NHANES III and NDI records was conducted by probabilistic matching and was completed in 2005.[15] The study population was limited to adults aged 18 years and older with valid follow-up mortality information. Sampling weights were incorporated in all analyses to provide estimates that are representative of the entire US adult population aged 18 years and older. Weights are also used to account for the unequal selection probabilities that result from the clustered design. Persons with a cancer diagnosis other than skin cancer were excluded from analysis.

Summary statistics were generated for continuous measures as means (standard error) and for categorical or dichotomized variables as frequencies (weighted percentage). Multivariable logistic regression was used to measure the primary outcome: all-cause mortality status as of 31 December 2000. Parameter estimates and odds ratios model the likelihood of mortality. Analyses were performed on the full population controlling for age (continuous), gender and self-reported health status. Self-reported global health status might be expected to be an important mediator or moderator for the association between each of the three categories of risk factors and mortality. The factors driving mortality might also be different in respondents with particular clusters of conditions. Respondents were thus categorized into one of the four disease populations: diabetes, chronic cardiovascular disease (CVD) (high blood pressure, congestive heart failure, previous heart attack, previous stroke), chronic non-CVD (e.g. skin cancer, emphysema, bronchitis or lupus) or healthy (reported no chronic diseases). Persons with more than one self-reported disease were placed in the most severe classification in the following order: diabetes, CVD and then chronic non-CVD. SES risk factors selected for this study included characteristics that have been shown in longitudinal studies to be associated with mortality: race, income relative to the federal poverty level and adjusted for household size, health insurance coverage within the previous 12 months, and level of education.[2] Health behavior covariates included characteristics shown to contribute to preventable mortality: current smoking status, physical activity level and waist circumference as a measure of obesity.[6,9] Waist circumference was used because it has been shown to be a marker for increased risk of diabetes, hypertension and CVD even in normal weight participants.[16] Separate models were run with BMI entered in place of waist circumference as a measure of obesity. There was no change in the main results, so only the waist circumference findings are reported here. Clinical risk factors included laboratory or physical findings available from the NHANES survey that have been associated with increased mortality in clinical trials and population-based studies: blood pressure, lipid levels (triglyceride, total cholesterol and HDL) and C-reactive protein (CRP), a measure of systemic inflammation that has been associated with both CVD and cardiovascular events.[17] Analyses were also done including calculated non-HDL cholesterol (total cholesterol – HDL cholesterol) and the total cholesterol/HDL cholesterol ratio without changes in the results reported here. Category cut-off values and additional information on all covariates included in our model are summarized in online Supplementary material Appendix S1.

Logistic regression was used to determine *P*-values for individual covariates, and odds ratios within the model (outcome: mortality) were obtained using SUDAAN PROC RLOGIST. The *C*-statistic was calculated for each model across the disease states. The *C*-statistic is used to measure the area under the receiver operating characteristic (ROC) curve, measuring the ability of each model to discriminate mortality outcomes (death/no death) and to evaluate the model's practical ability for correctly discriminating a subject's outcome. Values vary between 0.5 and 1.0; an area of 0.5 indicates that the model performs as well as a coin toss. All analyses were performed using SAS-Callable SUDAAN, Version 9.0.0. All analyses incorporated sampling weights

and primary sampling units (PSUs) to yield population-based estimates.

Results

Limiting our sample to NHANES III participants aged 18 years and more ($n = 19\,136$) with valid strata, PSU, weight and mortality responses yielded a sample size of 17 413, which represented 91% of the eligible population. Our disease subpopulation classifications showed 1241 (7.1%) subjects in the diabetic population, 2281 (12.0%) in the CVD population, 708 (4.7%) in the chronic non-CVD disease population and 13 183 (78.6 %) in the no chronic disease or healthy population. Subjects' demographic characteristics are summarized in Table 1. The population was 52.3% female with a mean age, at survey, of 45.3 (0.43) years. Responders under the age of 25 represented 10% of our population while another 10% of the population was 70 years or older. The study sample was predominantly white and privately insured. Over half of the sample had a waist circumference that met the criteria for obesity and just under one-third were active smokers at the initial interview. Most did not meet the CDC recommendations for physical activity. The overall weighted mortality rate was 9.5%. Self-reported global fair or poor health was associated with higher mortality (OR = 2.90; 95% CI 2.45–3.42) relative to those in good or excellent health. When stratified by self-reported diseases, the weighted mortality was 33.5% for diabetics (OR = 3.27; 95% CI 2.66–4.02), 15.3% for those with CVD (OR = 1.32; 95% CI 1.09–1.60), and 12.7% for those with non-CVD chronic diseases (OR = 1.44; 95% CI 1.15–1.80), when compared with 7.0% for those with no chronic diseases.

The results of individual analyses for each risk factor, adjusted for age, gender and self-reported health status are summarized in the first column of Table 2. Since the SES variables have multiple classification levels, the overall *P*-value for the variable, e.g. 'race', is also shown. All of the individual SES variables were significant predictors of mortality at the overall level, with black race and public insurance (either Medicare or Medicaid) associated with higher mortality. Higher education levels and higher family incomes were associated with lower mortality. In terms of health behaviors, being a smoker or physically inactive was associated with higher mortality but waist circumference was not. Among the clinical factors, elevated blood pressure, high triglyceride levels and elevated CRP levels were associated with higher mortality whereas total cholesterol and HDL levels were not.

When all of the covariates were entered simultaneously into a multivariable logistic regression for the full study population, race, insurance coverage and income remained statistically significant, but years of education did not (Table 2). The model was tested for collinearity and over-specification without evidence of either in the specifications presented. Black race was not statistically associated with mortality after controlling for the other covariates while other race remained associated with lower mortality. Income showed a graded response with higher incomes correlated with lower mortality. Health behaviors remained significant

Table 1 Demographics summary for 17 413 NHANES III participants

Gender (weighted %)	
Male	8136 (47.74)
Mean age in years (SE)	45.3 (0.43)
Race	
White	7179 (76.0)
Black	4866 (11.1)
Other	5368 (13.0)
Insurance coverage (weighted %)	
Medicare	984 (2.9)
Medicaid	1602 (5.8)
Uninsured	1969 (8.6)
Private	11 221 (82.7)
Income relative to poverty level (weighted %)	
< 100%	3743 (12.8)
100 to <200	4432 (21.4)
200 to <300	2917 (21.2)
≥ 300	4623 (44.7)
Abnormal waist circumference	
Females >88 cm, males >102 cm	10 209 (53.4)
Smoking (weighted %)	
Current smoker	4363 (28.1)
Inadequate physical activity	
< 5/week moderate or 3/week vigorous	11 356 (61.8)
CRP	
> 3 mg/l	2682 (12.2)
Triglycerides (mg/dl)	
≥ 250 mg/dl	6262 (34.6)
Blood pressure	
≥ 90/140 or on medication	8857 (49.3)
HDL	
Male <40 or female <50	6007 (34.8)
Serum cholesterol	
≥ 240 mg/dl	9212 (51.2)
Self-reported health status (weighted %)	
Fair or poor	4341 (15.5)
Self-reported chronic disease (weighted %)	
Diabetes	1241 (7.1%)
CVD	2281 (12.0%)
Non-cardiovascular chronic disease	708 (4.6%)
Healthy	13 183 (78.6%)

Table 2 Association of risk factors to all-cause mortality in the NHANES III population

	Individual associations with all-cause mortality[a]		*Simultaneous associations with all-cause mortality*[b]	
	OR[c]	*95% CI*[d] *on OR*	*OR*[c]	*95% CI*[d] *on OR*
SES risk factors				
Race	$P = 0.001$		$P = 0.01$	
Black versus white	1.27	1.05–1.55	0.95	0.77–1.18
Other versus white	0.58	0.41–0.82	0.52	0.36–0.75
Insurance	$P < 0.0001$		$P < 0.0001$	
Medicare versus private	1.87	1.42–2.46	1.43	1.01–2.02
Medicaid versus private	2.13	1.66–2.74	1.59	1.23–2.06
Uninsured versus private	0.68	0.49–0.94	0.56	0.43–0.74
Education	$P < 0.0001$		$P = 0.20$	
High school versus less than high school	0.81	0.70–0.94	0.89	0.73–1.08
More than high school versus less than high school	0.65	0.55–0.78	0.82	0.66–1.01
Income relative to poverty level	$P < 0.0001$		$P = 0.002$	
100–200 versus <100%	0.91	0.73–1.13	0.89	0.68–1.16
200–300 versus <100%	0.61	0.46–0.81	0.63	0.44–0.89
>300 versus <100%	0.49	0.38–0.65	0.57	0.39–0.81
Behavioral risk factors				
Smoking status				
Current versus never/former smoker	1.60	1.35–1.89	1.49	1.23–1.82
Waist circumference				
Obese versus normal/borderline	1.04	0.91–1.20	0.85	0.71–1.01
Physical activity				
Inadequate versus recommended level	1.36	1.16–1.60	1.30	1.05–1.60
Clinical risk factors				
Blood pressure				
High versus normal/borderline	1.69	1.37–2.07	1.68	1.30–2.17
Triglycerides				
High versus normal/borderline	1.21	1.05–1.41	1.09	0.89–1.34
Cholesterol				
High versus normal/borderline	1.01	0.86–1.17	0.85	0.69–1.03
HDL				
Low versus normal/borderline	1.08	0.92–1.26	1.00	0.82–1.23
C-reactive protein				
High versus normal/borderline	2.11	1.81–2.47	2.00	1.66–2.42

NHANES III population age 18+ with valid mortality follow-up, n = 17 413.

[a]Individual associations between risk factor and mortality, adjusted for age, gender and self-reported health status.

[b]Simultaneous associations between risk factor and mortality while adjusted for all other variables in table, and also including age, gender and self-reported health status.

[c]OR = odds ratio. A value greater than 1 indicates an increased risk of mortality.

[d]95% CI = 95% confidence interval on the odds ratio. An interval that does not include the null value 1.0 is considered to be statistically significant at the 5% level.

predictors of mortality with smoking and low levels of physical activity associated with higher mortality. Of the clinical factors studied, only high blood pressure and elevated CRP remained significantly associated with mortality.

In order to explore how these results might vary in populations with and without known chronic diseases, the same logistic regression models were run separately for respondents with diabetes, CVD, non-CVD chronic disease or no

Table 3 SES risk factor associations with mortality by self-reported disease state

Risk factor	*Diabetes*		*CVD*		*Chronic non-CVD*		*Healthy*	
	OR	*95% CI*	*OR*	*95% CI*	*OR*	*95% CI*	*OR*	*95% CI*
Race[a]	$P = 0.01$						$P = 0.04$	
Black	1.03	0.69–1.53	0.94	0.66–1.36	0.81	0.23–2.87	0.96	0.71–1.29
Other	0.42	0.24–0.74	0.47	0.25–0.89	0.25	0.01–4.46	0.59	0.38–0.91
Insurance[a]					$P = 0.05$		$P < 0.001$	
Medicare	1.38	0.67–2.85	2.75	1.11–6.79	0.22	0.06–0.85	1.26	0.73–2.18
Medicaid	1.56	0.84–2.87	1.57	0.94–2.64	1.29	0.35–4.67	1.78	1.16–2.71
Uninsured	0.85	0.28–2.61	1.46	0.64–3.35	0.46	0.05–4.00	0.43	0.30–0.62
Education								
High school	0.85	0.55–1.30	0.64	0.37–1.13	0.19	0.30–2.05	0.95	0.75–1.20
Beyond HS	0.80	0.48–1.32	1.48	0.80–2.74	0.52	0.20–1.37	0.84	0.65–1.09
Income[a]			$P = 0.01$		$P = 0.01$		$P = 0.03$	
Up to 200% poverty level	0.88	0.42–1.83	1.45	0.92–2.27	1.33	0.36–4.97	0.73	0.47–1.13
200–300%	0.61	0.21–1.76	0.64	0.37–1.13	0.46	0.11–1.86	0.64	0.38–1.07
Over 300%	0.45	0.17–1.14	1.48	0.80–2.74	0.32	0.08–1.21	0.48	0.28–0.81

Values reflect the association between the individual risk factor and mortality, simultaneously adjusted for age, gender, self-reported health status, and all other SES, behavioral, and clinical risk factors.

OR, odds ratio; 95% CI, 95% confidence interval.

[a]*P*-values reflect the overall significance of the multi-level covariates on mortality, adjusted for all other SES covariates, self-reported health status, clinical risk factors, age, and gender. *P*-values not included for associations not significant at the 5% level.

known chronic diseases (healthy). Stratifying the population by the four chronic disease groups led to somewhat different results. Table 3 shows the impact of SES risk factor for respondents in each of the four health categories. The overall *P*-values show the simultaneous impact of each individual risk factor in a model adjusting for all other covariates including behavioral and clinical risk factors. *P*-values that were not significant at the 5% level are not shown. Race is significant in two disease groups, driven by a lower mortality rate for other race in each population. The impact of insurance on mortality decreases across the populations, having its strongest association in the healthy population. The protective effect of income is seen in three of the population groups but not in diabetics. Years of education consistently do not predict subsequent mortality. For the SES variables in general, there is a somewhat graded response with the strongest effects seen in the healthy group, intermediate effects in the chronic disease groups and smallest effects in the diabetic group.

Table 4 shows the multivariable associations for behavioral and clinical risk factors. As in Table 3, the values associated with each risk factor are adjusted for all other covariates, including SES. Smoking was associated with higher mortality in all disease groups except diabetics. Waist circumference was not significant except in the healthy population where it was paradoxically associated with lower mortality. Low rates of physical activity were associated with higher mortality in diabetics and in chronic non-CVD subjects. Clinical risk factors had a variable pattern across disease states. Both elevated blood pressure and high CRP were associated with higher mortality in the healthy population. Only elevated triglycerides were associated with increased mortality in diabetics while elevated cholesterol was paradoxically associated with lower mortality. High CRP was the only significant clinical risk factor in the CVD population.

Logistic regression modeling was then used to evaluate the additive impact of the three different categories (SES, behaviors, clinical) on predicting mortality in each of the disease subpopulations. All models included age (continuous), gender and self-reported health status and covariates were added in a block by category, for example, SES variables. All covariates within each category were added irrespective of whether they had been shown to be statistically significant in the bivariate or multivariable analysis. The models consistently identified mortality more precisely in the healthy population (*C*-statistic = 0.862 for SES predictors) than in diabetics (*C*-statistic = 0.713 for SES predictors). However, each of the three categories of risk factors correctly indicated similar proportions of mortality within each disease state. For example, in the diabetic population, the respective

Table 4 Behavioral and clinical risk factors for mortality by self-reported disease state

Risk factor	*Diabetes*		*Cardiovascular disease*		*Chronic non-CVD*		*Healthy*	
	OR	*95% CI*	*OR*	*95% CI*	*OR*	*95% CI*	*OR*	*95% CI*
Behavior								
Current smoker	0.91	0.49–1.69	1.93	1.22–3.04	2.15	1.09–4.26	1.55	1.21–1.98
Waist circumference	0.74	0.39–1.42	0.98	0.69–1.40	1.03	0.52–2.05	0.76	0.60–0.96
Inadequate physical activity	1.75	1.10–2.79	1.63	0.99–2.68	2.13	1.11–4.09	1.10	0.85–1.43
Clinical								
High blood pressure	1.40	0.86–2.30	1.10	0.63–1.93	1.37	0.60–3.12	1.77	1.30–2.41
High triglycerides	2.24	1.22–4.12	0.92	0.63–1.35	1.16	0.49–2.76	0.96	0.71–1.31
High cholesterol	0.38	0.24–0.59	1.00	0.63–1.57	0.64	0.25–1.63	1.01	0.77–1.34
Low HDL	0.87	0.49–1.56	0.97	0.64–1.49	0.50	0.23–1.07	1.01	0.73–1.39
High C-reactive protein	1.72	0.97–3.05	2.40	1.45–3.98	2.36	0.67–8.36	1.82	1.45–2.28

Values reflect the association between the individual risk factor and mortality, simultaneously adjusted for age, gender, self-reported health status, and all other SES, behavioral and clinical risk factors.

OR, odds ratio; 95% CI, 95% confidence interval.

C-statistics for SES, behaviors and clinical risk factors were 0.713, 0.707 and 0.695. The highest *C*-statistic in each disease group was generated by the model containing all possible covariate groups. The *C*-statistics for every model are shown in online Supplementary material Appendix S2.

Finally, to address concerns that a portion of subjects who were at high risk for mortality across all factors could be driving the results, we investigated the degree of overlap between SES, behavioral and clinical high-risk groups. On the basis of the individual risk factors that were statistically significant in the bivariate analysis, we considered 'high-risk SES' to be subjects in both the lowest income and education level; 'high-risk behavior' were subjects who were active smokers with inadequate physical activity levels, and 'high-risk clinical' were subjects with abnormal triglycerides, CRP and blood pressure. Only 0.11% of the overall population fell simultaneously into all three high-risk groups. This proportion ranged from a high of 0.42% in diabetics to a low of 0.06% in the healthy population. Using a less conservative assumption, only 0.39% of the overall population fell into the high-risk SES and one other high-risk group. This proportion ranged from a low of 0.12% in the chronic disease population to a high of 2.16% in the diabetic population.

Discussion

Main findings of this study

SES, health behaviors and clinical risk factors were each significantly associated with mortality in the overall population even when simultaneously adjusting for all of the other risk factors. Race, insurance coverage and income were associated with mortality even when controlling for age, health status and behavioral and clinical risk factors. Smoking and low levels of physical activity were also significantly associated with mortality. Of the clinical risk factors, elevated blood pressure and CRP were independently associated with mortality. In multivariable models stratified by disease state, the effect of SES and behavioral risk factors remained strong across disease groups. SES risk factors (race, insurance and income) were most strongly associated with mortality in the healthy population but did affect mortality in populations with chronic disease. Smoking was the strongest behavioral risk factor associated with mortality across the disease groups while inadequate physical activity was associated with mortality in chronic disease populations. Clinical risk factors had a less consistent pattern across disease groups. Elevated blood pressure and CRP were associated with mortality in the healthy population while only elevated triglycerides were associated with higher mortality in the diabetic population. Although each group of individual risk factors (SES, behavioral, clinical) was statistically significantly associated with mortality, when added together, they improved little upon the ability of each separate category to predict overall mortality.

What is already known on this topic

A wealth of research has shown that SES, health behaviors and clinical risk factors individually contribute toward mortality. In the majority of the literature, however, these three domains have been examined separately. Whereas studies of biochemical markers and other clinical factors have predominantly focused

on high-risk populations receiving medical care, research on SES and health behaviors have primarily examined healthier, population-based samples. Few studies have attempted to measure the relative contributions of each of these risk factors on all-cause mortality in the same sample.

What this study adds

This study's findings corroborate previous longitudinal studies, showing that SES is a powerful predictor of future mortality. The relationship between SES factors and mortality is complex, with many studies showing a higher burden of disease, impaired functional status and risky health behaviors in many low SES populations.[6,18–20] However, in this study, the relationship between measures of SES and mortality remained the same after controlling for self-reported global health, known clinical risk factors, pre-existing disease and health behaviors. Of note, controlling for other factors eliminated the higher mortality associated with black race in the bivariate analysis while a lower mortality rate for those of other ethnicity remained the same. Previous studies have found that SES differences do explain some of the ethnic disparities in health outcomes. A study of stroke risk factors found that ethnic disparities in stroke prevalence persisted after accounting for clinical risk factors, but the disparity disappeared after controlling for income.[21] A lower mortality rate for Hispanics has been noted before and referred to by some investigators as the 'Hispanic paradox' or the 'healthy immigrant effect'.[20] The protective effect of higher incomes on mortality was consistent across all population groups except diabetics. This is consistent with previous studies showing that income has a protective effect on mortality that is independent of educational attainment.[22] Other studies have explored the education–health and income–health interactions in the USA, demonstrating that investments in education and poverty relief programs could have profound impacts on the health of the nation.[1,6] In diabetic respondents, the marginal effect of income may be dwarfed by the impact of the disease itself on mortality. In terms of predictive ability, it is interesting that SES characteristics were at least as strong as the behavioral and clinical risk factors included in this study even among respondents with known CVD or diabetes. This argues that SES factors remain powerful determinants of individual mortality and should not be ignored as scientific advances identify new and promising biologic predictors of clinical disease. The ability to realize the benefits of early detection and treatment are critically dependent upon knowledge about, access to and the affordability of health care.

Despite statistical significance, there was only modest improvement in discriminative ability as measured by the *C*-statistic when additional risk factors were added to the multivariable model. This is likely because a risk factor that performs well as a prognostic test for an individual patient must have a cutoff value that will discriminate between those who will and those who will not develop the disease with high sensitivity and specificity. One estimate is that a risk factor must be so strongly associated with a disease that the relative risk approaches 200 in order for it to be worthwhile for use as a screening test.[23] Few available biomarkers and none of the predictors included in this study perform at that level.[3,4] In cross-sectional population studies, the proportion of variance in mortality that can be explained is likely to be limited, and the ability to predict mortality will quickly reach an asymptote.

Waist circumference and cholesterol measures (HDL and total cholesterol) were generally not significantly associated with mortality. This may be in part due to what is referred to as the 'Obesity paradox',[24] which suggests that obese patients tend to receive better medical treatment and management because their health risks seem more obvious. Previous studies have shown occasional inconsistencies in the association of both obesity and cholesterol measures with cardiovascular events or mortality.[25–28]

Limitations of this study

These analyses have several limitations that must be noted. The main outcome of interest was all-cause mortality. The large number of missing causes of death precluded subgroup analyses by cause of death. Second, only a limited number of SES, behavioral and clinical variables were available for analysis in each of the domains. Simultaneous analyses of variables across domains are a unique contribution of this project, but further studies with more detailed variables and specifications are needed to explore causality and the mediating aspects of each of these domains on risk factors and outcomes. Third, ascertainment of SES, clinical factors and health behaviors was cross-sectional at the time of initial interview. These characteristics may change over time and thus our results may underestimate the impact of any of these factors on mortality. Future studies should address the impact of risk factor trajectories and their impact on mortality completely.[18,29] Finally, only 6–12 years of mortality follow-up was available for this study. Longer follow-up might offer additional insights.

Conclusions

The results suggest that SES factors, behaviors and known clinical risk factors all contribute significantly to subsequent

mortality even when baseline health status and known chronic diseases are taken into account. Despite accelerating scientific advances, our ability to identify clinical risk factors and translate that knowledge into improved health is likely to remain dependent in part on successful maintenance of healthy behaviors as well as the accessibility of appropriate health care.

Supplementary data

Supplementary data are available at the *Journal of Public Health* online.

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