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ORIGINAL ARTICLE

The Role of Health Behaviours Across the Life Course in the Socioeconomic Patterning of All-Cause Mortality: The West of Scotland Twenty-07 Prospective Cohort Study

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Abstract
Background Socioeconomic differentials in mortality are increasing in many industrialised countries.
Purpose This study aims to examine the role of behaviours (smoking, alcohol, exercise, and diet) in explaining socioeconomic differentials in mortality and whether this varies over the life course, between cohorts and by gender.
Methods Analysis of two representative population cohorts of men and women, born in the 1950s and 1930s, were performed. Health behaviours were assessed on five occasions over 20 years.
Results Health behaviours explained a substantial part of the socioeconomic differentials in mortality. Cumulative behaviours and those that were more strongly associated with socioeconomic status had the greatest impact. For example, in the 1950s cohort, the age-sex adjusted hazard ratio comparing respondents with manual versus non-manual occupational status was 1.80 (1.25, 2.58); adjustment for cumulative smoking over 20 years attenuated the association by 49 %, diet by 43 %, drinking by 13 % and inactivity by only 1%.
Conclusions Health behaviours have an important role in explaining socioeconomic differentials in mortality.

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Introduction

Socioeconomic differentials in mortality are well established, with the highest mortality rates observed in lower socioeconomic groups [1–3]. Differentials have been identified in many populations and are increasing in many industrialised countries despite falling mortality rates overall [4, 5]. Explanations for these inequalities include different patterns of material, cognitive, biomedical, psychosocial and behavioural risk factors in different socioeconomic groups, but the extent to which these factors explain socioeconomic differences in mortality remains unclear. Behavioural risk factors are most amenable to change and are of particular interest in terms of reducing inequalities. Previous studies have explored the role of health behaviours in explaining mortality associations with occupational socioeconomic status (SES) [6–11], education [7, 10–19] and income [7, 12, 17–21], and generally indicate that health behaviours have an impact, although the relative importance of individual behaviours varies by study.

Existing studies have a number of limitations. First, the majority have focussed on SES and behaviours measured at one time-point. However, exploration of social inequalities in health is increasingly based on experiences across the whole life course [22], and many risk factors have a cumulative impact on health [23]. Evidence suggests that lower SES individuals may adopt more adverse health behaviours [24, 25] and be less likely to make positive changes to their behaviours over time [26, 27]. Patterns of behaviours over time may therefore be important, e.g. unhealthy behaviours at particular life stages may have latent effects for later outcomes, and in a recent study of an occupational group [28],

cumulative behaviours explained more of SES–mortality associations than single measures. However, this finding is yet to be replicated in a population sample. Second, most studies focus on all health behaviours combined and do not consider individual effects. Third, evidence from industrialised countries [29–31] demonstrates that, over time, social inequalities in many health behaviours have narrowed, widened or, in some cases, reversed. The age of individuals at the time of these temporal changes may affect their contribution to explaining SES–mortality associations. We are unaware of any studies that have directly considered these effects. Finally, few studies have explored gender differences although it has been suggested that socioeconomic differentials in mortality may be greater in men [32].

In the present analyses, we explore the role of four health behaviours (smoking, alcohol consumption, physical activity and diet) in explaining SES–mortality associations (measured by occupation, education and income) in two population cohorts of men and women, born 20 years apart, in the West of Scotland. An unusual feature of our study is that data on health behaviours were collected on five occasions with up to 24 years of follow-up for mortality in a general population-based sample. We have examined the individual and combined impact of each health behaviour, measured distally, proximally and cumulatively, and we have explored differences by age cohort and gender.

Methods

The West of Scotland Twenty-07 study is a population-based multiple-cohort study and has previously been described in detail [33]. The study followed men and women living in the West of Scotland in three age cohorts born around 1932, 1952 and 1972. Baseline interviews were conducted in 1987/1988, when the three cohorts were approximately 55, 35 and 15 years of age, and respondents are representative of the general population of the sampled area [34]. There were four follow-up waves in 1990/1992, 1995/1997, 2000/2004 and 2007/2008. Ethics approval was gained for each wave from the National Health Service (NHS) and/or Glasgow University Ethics Committees. The current analyses are based on the two oldest cohorts as mortality in the youngest cohort is very low to date. Cohort members are flagged with the NHS Central Register for mortality follow-up. The current analysis is based on deaths up to 7th November 2011. International Classification of Diseases (ICD) 9th and 10th revisions were used to define cardiovascular disease (CVD) (ICD-9: 390-459.9; ICD-10: I00-I99), cancer (ICD-9: 140-209.9; ICD-10: C00-C97) and respiratory (ICD-9: 460–519.9; ICD-10: J00-J99) mortality.

We employed three alternative measures of SES in our analyses. The paper primarily focusses on respondent's occupational SES at each wave, which was coded according to the Registrar General's 1980 classification [35]. For couple households, the highest status occupation of the two partners was used; if the respondent (or partner) was not working, their most recent job was used. Preliminary analyses considered SES in six categories and results demonstrated that the strongest mortality difference was between respondents with manual versus non-manual occupational status. For clarity, we present results for occupational SES coded as a dichotomous variable comparing manual (V/IV/IIIM) versus non-manual (IIINM/II/I) classes based on highest household occupational status. Analyses were repeated using income and education in place of occupational SES. Income was based on total household earnings after tax, including any benefits; respondents were asked to specify an actual amount in pounds sterling per week, month or year, or, if they were unwilling to do so, to identify an appropriate income band on a pre-printed card. Analyses compared respondents whose income was below versus above the median. Education was based on age at which respondents left school and was subdivided into ≤14 versus 15+ years. Over two thirds of respondents had left school by age 14.

Data on smoking, alcohol consumption, physical activity, and diet were based on self-report, although questions were not identical at every wave. Behaviour variables created for these analyses were based, where possible, on contemporary guidelines, as well as making variables homogeneous between cohorts and waves, and comparable with previous studies [7, 28]. Smoking status at each wave was defined as current, ex- or non-smoker. Weekly alcohol consumption was used to define respondents as abstainers (no alcohol), or drinkers within or above gender-specific recommended weekly limits (≤21 vs. 22+ units for men; ≤14 vs. 15+ units for women) [36]. Alcohol strength changed for some drinks during follow-up [37], and we recalculated this variable in waves 4 and 5 (2000s); this change had no impact on our results. Physical activity was based on the number of occasions per week that respondents took part in an activity "lasting more than 20 min" that made them "sweat or out of breath", reflecting guidelines at the time to undertake exercise like this three times per week. Respondents were then subdivided into high (3+ per week), moderate (1–2 per week) or low (0 per week) physical activity. Diet, from food-frequency questionnaires, was based on the number of days per week on which participants reported eating fruit and vegetables. Fruit and vegetable consumption in the study fell well short of recommendations [38], but, in line with the previous work [7, 28], respondents were classified as high (eat fruit and vegetables almost daily), moderate or low (never eat fruit or vegetables or both). It is worth noting that, while the highest group will include respondents who ate just one portion of fruit and vegetables per day, it will also include those who ate many more. We performed a number of sensitivity analyses in which these thresholds were varied, and results were very similar to those presented here.

Data on SES and health behaviours were not complete for all respondents at all waves. Missing data were imputed using multivariate imputation by chained equations in Stata v11.0 [39–41]. This imputation is valid under the assumption that data are "missing at random" in the sense that variables predictive of missing data are included in the imputation model [42]. Among those who were interviewed at each wave, missing values for health behaviour variables were generally low and missingness was dominated by attrition, particularly in later waves. Predictors of missingness in the Twenty-07 cohorts have previously been explored in detail [43] and were found to include religion, marital status, long-standing illness and return of a self-complete form. These variables were therefore included in imputation models, along with variables from analysis models (SES and health behaviours at all waves, mortality and time to death). Data were imputed 20 times for respondents known to be alive at each wave. Parameter estimates were averaged across the 20 imputed datasets and standard errors computed according to the "Rubin rules" [44]. Results from analyses based on complete data (not shown) were very similar to those based on imputed data.

Statistical Analysis

SES–mortality associations were explored using Cox proportional hazards regression, and the contribution of health behaviours to explaining these associations was determined by the percentage attenuation of the hazard ratio (HR) due to adjustment:

$$\left(\frac{HR_{unadjusted}-HR_{adjusted}}{HR_{unadjusted}-1}\right)\times 100\%$$

The impact of individual and combined health behaviours on SES–mortality associations were explored in three ways: (1) distally, using baseline behaviours; (2) proximally, using most recent behaviours, assessed at final wave or wave immediately preceding death,; and (iii) cumulatively, using "mean" behaviours over all live waves, where the most healthy behaviours were coded as 0 and the least healthy coded as 2. Preliminary analyses confirmed that attenuation of SES–mortality associations after adjustment for these single summary measures of cumulative behaviour was very similar to attenuation after adjustment for all five wave-specific measures. Results are presented separately for the two age cohorts and, in the 1932 cohort, for men versus women; this restriction to the oldest cohort is necessary as there are currently too few deaths in the 1952 cohort to support gender-specific analyses. The main analyses focussed on mortality from all causes combined, although we also examined the role of behaviours in SES associations with CVD, cancer and respiratory mortality in the 1932 cohort; there are currently too few deaths in the 1952 cohort to support cause-specific analyses. In sensitivity analyses, we excluded individuals with cancer, cardiovascular or respiratory disease at baseline to allow for changes in behaviours due to ill health. Finally, although intuitive, the method described here, comparing adjusted and unadjusted models, has limitations and may be prone to bias [45, 46]. We therefore repeated our case-complete analyses using additive hazard models [46].

Results

Analyses are based on cohorts born around 1952 and 1932. The wave 1 (baseline) response rate among those invited for interview was 87 %, and respondents were found to be representative of the general population of the sampled area [34]. Inevitably, there was some attrition in later waves, and 70–85 % of 1952 and 66–85 % of 1932 respondents who were known to be alive were interviewed in waves 2–5. Respondents who did not take part in later waves were more likely to have manual occupational status in wave 1. Gender and health behaviours in wave 1 were similar in those who did and did not take part in subsequent waves. At baseline, data were missing for between 0.2 % (smoking) and 8.2 % (diet) of subjects. In subsequent follow-up waves, the extent of missing data among those who were interviewed was as follows: wave 2—between 0.0 % (drinking) and 1.6 % (diet); wave 3—between 0.4 % (drinking) and 2.8 % (physical activity); wave 4—between 0.3 % (physical activity) and 5.3 % (diet); and wave 5—between 0.0 % (drinking/physical activity) and 1.7 % (diet). Health behaviours at baseline among those with non-missing baseline SES are shown in Table 1. In total, 35 % of the younger (1952) cohort had manual occupational status compared with 46 % of the older (1932) cohort. Poorer health behaviours were generally more common in respondents with manual occupational status, most markedly for smoking and diet. Generally, behaviours improved in subsequent waves, with the exception of inactivity, which increased in the older cohort, and heavy drinking, which increased in the younger group (not shown).

After a median follow-up of 24 years (range, 0–24), 120 (8.3 %) and 719 (46.4 %), respectively, of the younger and older cohorts had died. In the 1932 cohort, 263 (36.6 %), 258 (35.9 %) and 82 (11.4 %) deaths were due to CVD, cancer and respiratory disease, respectively; in the 1952 cohort, the corresponding figures were 33 (27.5 %), 40 (33.3 %), and 10 (8.3 %). Among the older cohort, mortality was greater in men (54.7 %) than in women (39.5 %). Table 2 presents all-cause mortality associations with manual versus non-manual occupational status, and the impact on these associations of

simultaneous adjustment for all four health behaviours at different time-points. Respondents with manual occupational status had around 60–80 % greater mortality compared with respondents with non-manual occupational status, and these associations were consistently attenuated by adjustment for behaviours. Adjustment for distal behaviours had the least impact (24–44 % attenuation), followed by proximal behaviours (24–55 %). Adjustment for cumulative behaviours had the greatest attenuating effect (38–77 %). The impact of adjustment was greater in the younger cohort and attenuation in the older cohort was greater for men than women.

Results for individual health behaviours are presented in Table 3. Again, adjustment for cumulative behaviours had the greatest impact and we present these results. However, comparable, weaker results were observed when adjusting for baseline and most recent behaviours. Adjustment for inactivity had no notable impact on SES–mortality associations, and adjustment for drinking had only a modest effect in the younger cohort. Adjustments for smoking and diet had a more marked impact, both attenuating SES-mortality associations by around a third in the older cohort and up to half in the younger cohort. Again, the impact of smoking and diet in the older cohort was greater in men. The attenuation due to adjustment for all four behaviours was less than the sum of the attenuation due to each individual behaviour, indicating that attenuating effects of each behaviour were not independent.

Results for cause-specific mortality are presented in Table 4. For all causes, there was a clear excess mortality in respondents with manual occupational status. Overall adjustment for all four health behaviours had the greatest impact on cancer mortality, which showed the least marked association with SES. Adjustment for smoking and diet generally had the greatest individual impact, attenuating all associations by between a quarter and a half. The impact of diet and smoking was broadly similar for CVD and cancer mortality, but, unsurprisingly, smoking had a greater impact on SES–respiratory mortality associations.

As would be expected in two adult cohorts, the (tetrachoric) correlations of SES between waves were strong, ranging from 0.71 (wave 1 vs. wave 5) to 0.93 in the 1952s and 0.93 (wave 1 vs. wave 5) to 1.00 in the 1932s cohort. Mortality associations with time-varying SES, income and education were fairly similar to those presented in Tables 2 and 3 for baseline occupational SES, although hazard ratios for mortality were larger for more proximal SES in the younger cohort and lower for education in the older cohort (not shown). However, the impact of adjustments for behaviours was consistently similar to those presented for baseline SES in Table 3. Very few respondents reported having cancer, cardiovascular or respiratory disease at baseline, and excluding these individuals had no impact on our results (not shown). Finally, results from additive hazards models confirmed that the proportions of the SES effect on mortality due to each behaviour were almost identical to those presented in Tables 2 and 3 (not shown).

Table 1 *N* (%) baseline health behaviours by socioeconomic status defined by manual versus non-manual occupational class in those with non-missing baseline occupational status[a]

	1952 men Manual/non-manual	1952 women Manual/non-manual	1932 men Manual/non-manual	1932 women Manual/non-manual
Smoking status				
Non-smoker	56 (24.5)/153 (37.1)	80 (30.2)/233 (45.0)	55 (15.9)/109 (30.4)	125 (34.3)/221 (46.1)
Ex-smoker	35 (15.3)/92 (22.3)	28 (10.6)/87 (16.8)	67 (19.4)/113 (31.5)	60 (16.4)/104 (21.7)
Current smoker	138 (60.3)/168 (40.7)	157 (59.3)/198 (38.2)	223 (64.6)/137 (38.2)	180 (49.3)/154 (32.2)
Alcohol consumption				
Abstainer	40 (17.5)/54 (13.1)	96 (36.2)/149 (28.7)	87 (25.2)/87 (24.4)	183 (50.1)/187 (39.0)
Within guidelines	115 (50.2)/234 (56.7)	152 (57.4)/326 (62.8)	159 (46.1)/197 (55.2)	164 (44.9)/281 (58.7)
Above guidelines	74 (32.3)/125 (30.3)	17 (6.4)/44 (8.5)	99 (28.7)/73 (20.5)	18 (4.9)/11 (2.3)
Physical activity				
High (3+ times per week)	45 (21.6)/53 (13.5)	34 (13.9)/49 (9.8)	67 (19.5)/59 (16.4)	47 (12.9)/64 (13.4)
Moderate (1–2 times per week)	44 (21.2)/138 (35.2)	54 (22.1)/154 (30.7)	39 (11.3)/68 (18.9)	74 (20.3)/103 (21.5)
Low (none)	119 (57.2)/201 (51.3)	156 (63.9)/299 (59.6)	238 (69.2)/232 (64.6)	244 (66.9)/312 (65.1)
Fruit and vegetable intake				
High (daily)	17 (8.6)/51 (14.1)	35 (15.7)/122 (25.0)	32 (10.0)/66 (19.2)	85 (25.2)/159 (34.5)
Moderate	94 (47.7)/214 (59.1)	134 (60.1)/266 (54.4)	179 (55.8)/206 (60.1)	169 (50.0)/232 (50.3)
Low (never)	86 (43.7)/97 (26.8)	54 (24.2)/101 (20.7)	110 (34.3)/71 (20.7)	84 (24.9)/70 (15.2)

[a] Total numbers vary due to missing values among specific health behaviour variables

Table 2 Impact of adjustment of occupational status-all cause mortality associations for distal, proximal and cumulative health behaviours by cohort and sex

	1952 cohort (*N*=1,444)	1932 cohort (*N*=1,550)
N (%) died	120 (8.3)	719 (46.4)
Hazard ratio (95 % confidence interval) for all-cause mortality in respondents with manual versus non-manual occupational status		
Age- and sex-adjusted[a]	1.80 (1.25, 2.58)	1.66 (1.43, 1.93)
Adjusted for 4 baseline behaviours[b]	1.39 (0.95, 2.02)	1.43 (1.23, 1.66)
% attenuation[c]	44 %	30 %
Adjusted for 4 most recent behaviours[d]	1.30 (0.88, 1.92)	1.40 (1.19, 1.63)
% attenuation[c]	55 %	34 %
Adjusted for 4 cumulativebehaviours[e]	1.14 (0.77, 1.69)	1.28 (1.10, 1.50)
% attenuation[c]	77 %	51 %
	1932 men (*N*=704)	1932 women (*N*=846)
N (%) died	385 (54.7)	334 (39.5)
Hazard ratio (95 % confidence interval) for all-cause mortality in respondents with manual versus non-manual occupational status		
Age-adjusted[a]	1.59 (1.30, 1.95)	1.74 (1.41, 2.16)
Adjusted for 4 baseline behaviours[b]	1.31 (1.06, 1.62)	1.53 (1.23, 1.91)
% attenuation[c]	42 %	24 %
Adjusted for 4 most recent behaviours[d]	1.30 (1.05, 1.62)	1.53 (1.21, 1.92)
% attenuation[c]	43 %	24 %
Adjusted for 4 cumulativebehaviours[e]	1.18 (0.95, 1.46)	1.41 (1.12, 1.77)
% attenuation[c]	65 %	38 %

[a] Hazard ratio comparing all cause mortality in respondents with manual versus non-manual occupational status

[b] Hazard ratio additionally adjusted for each of smoking, drinking, physical activity and fruit and vegetable intake at baseline

[c] % attenuation $=100\times(HR_{unadjusted}-HR_{adjusted})/(HR_{unadjusted}-1)$

[d] Hazard ratio additionally adjusted for each of smoking, drinking, physical activity, and fruit and vegetable intake at last wave or wave immediately preceding death

[e] Hazard ratio additionally adjusted for mean smoking, drinking, physical activity and fruit and vegetable intake over all waves at which respondent was known to be alive

Discussion

We explored the role of health behaviours in explaining mortality associations with SES, measured by occupation, income and education, in two representative population cohorts of men and women in the West of Scotland. In common with existing literature [6–11], we confirmed that health behaviours, particularly smoking and low fruit and vegetable intake, impact on SES–mortality associations, and we have expanded on these findings in a number of ways.

Most previous studies focus on baseline health behaviours and do not allow for changing behaviours over time. Results from an occupational (British Whitehall II) cohort [28] demonstrate the superior predictive ability of repeated longitudinal measures of health behaviours over a single baseline measure. We are the first, to our knowledge, to confirm this finding in (two) population cohorts, using repeated measures of health behaviours over 20 years of follow-up and multiple measures of SES. Our results demonstrate that, while baseline behaviours have a role in explaining SES–mortality associations, they have less explanatory potential than more proximal and, particularly, cumulative behaviours. This suggests that, rather than acting during some critical exposure period, health behaviours have a continuing impact on mortality risk throughout the life course. There is growing interest in the beneficial effects of improving behaviours [23, 47–50], and our results suggest that adopting healthier behaviours, even in middle- and early-older age is still worthwhile in terms of reducing mortality risk.

In addition to changes in an individual's behaviours, temporal changes in the association between SES and behaviours at a population level may also influence the relative impact of behaviours on SES–mortality associations over time. We explored associations separately in two cohorts who were born 20 years apart and potentially grew up in different social climates, not least in the prevalence and meanings of smoking [51] and heavy drinking [52] in men and women. The negative impact of lower SES on mortality was stronger in the

Table 3 Impact of adjustment of occupational status–all cause mortality associations for cumulative smoking, drinking, physical activity and fruit and vegetable intake by cohort and sex

	1952 cohort (N=1,444)	1932 cohort (N=1,550)
N (%) died	120 (8.3)	719 (46.4)
Hazard ratio (95 % confidence interval) for all-cause mortality in respondents with manual versus non-manual occupational status		
Age- and sex-adjusted[a]	1.80 (1.25, 2.58)	1.66 (1.43, 1.93)
Adjusted for smoking[b]	1.34 (0.92, 1.96)	1.40 (1.20, 1.63)
% attenuation[c]	49 %	34 %
Adjusted for drinking[d]	1.66 (1.15, 2.40)	1.66 (1.43, 1.93)
% attenuation[c]	13 %	-
Adjusted for physical activity[e]	1.78 (1.24, 2.56)	1.64 (1.41, 1.90)
% attenuation[c]	1 %	3 %
Adjusted for fruit and vegetable intake[f]	1.40 (0.96, 2.03)	1.39 (1.20, 1.62)
% attenuation[c]	43 %	35 %
Adjusted for all 4 behaviours[g]	1.14 (0.77, 1.69)	1.28 (1.10, 1.50)
% attenuation[c]	77 %	51 %
	1932 men (N=704)	1932 women (N=846)
N (%) died	385 (54.7)	334 (39.5)
Hazard ratio (95 % confidence interval) for all-cause mortality in respondents with manual versus non-manual occupational status		
Age-adjusted[a]	1.59 (1.30, 1.95)	1.74 (1.41, 2.16)
Adjusted for smoking[b]	1.30 (1.06, 1.61)	1.51 (1.21, 1.88)
% attenuation[c]	43 %	26 %
Adjusted for drinking[d]	1.58 (1.29, 1.94)	1.78 (1.43, 2,22)
% attenuation[c]	2 %	-
Adjusted for physical activity[e]	1.57 (1.28, 1.92)	1.73 (1.39, 2.14)
% attenuation[c]	2 %	2 %
Adjusted for fruit and vegetable intake[f]	1.31 (1.06, 1.61)	1.50 (1.20, 1.87)
% attenuation[c]	42 %	28 %
Adjusted for all 4 behaviours[g]	1.18 (0.95, 1.46)	1.41 (1.12, 1.77)
% attenuation[c]	65 %	38 %

[a] Hazard ratio comparing all cause mortality in respondents with manual versus non-manual occupational status

[b] Hazard ratio additionally adjusted for cumulative smoking

[c] % attenuation=$100\times(HR_{unadjusted}-HR_{adjusted})/(HR_{unadjusted}-1)$

[d] Hazard ratio additionally adjusted for cumulative drinking

[e] Hazard ratio additionally adjusted for cumulative physical activity

[f] Hazard ratio additionally adjusted for cumulative fruit and vegetable intake

[g] Hazard ratio additionally adjusted for cumulative smoking, drinking, physical activity and fruit and vegetable intake

younger cohort and attenuation due to smoking, drinking, and diet was also greater. This may reflect a change in the association between SES and behaviours over time. For example in the UK, in the early 1950s, when the older cohort were young adults, cigarette smoking was more common in higher SES groups, while by 1971, when the younger cohort were young adults, cigarette smoking was more common in lower SES groups [30, 31]. We therefore observed a greater impact of smoking in the cohort who are likely to have adopted the behaviour when it was more strongly associated with lower SES. Recent discussion of the apparent widening of social inequalities in health [53] has described, among others, the theory of "diffusion of innovation", which suggests that higher SES groups are quicker to adopt new, healthier behaviours. This theory might explain the greater impact of behaviours in the younger cohort who were at an earlier stage of making healthy changes as compared to the older cohort in whom all likely healthy changes, e.g. quitting smoking and improving diet, had already been made.

These cohort differences are novel and require duplication in other populations. In addition, the results are open to alternative explanations. The difference in attenuation between the two

Table 4 Impact of adjustment of occupational status–cause specific mortality associations for cumulative smoking, drinking, physical activity and fruit and vegetable intake in 1932 cohort (both genders combined; $N=1,550$)

Cause of death	CVD	All cancers	Respiratory disease
N (%) died	263 (17.0)	258 (16.7)	82 (5.3)
Hazard ratio (95 % confidence interval) for cause-specific mortality in respondents with manual versus non-manual occupational status			
Unadjusted[a]	2.05 (1.60, 2.63)	1.48 (1.16, 1.89)	1.92 (1.24, 2.99)
Adjusted for smoking[b]	1.71 (1.32, 2.20)	1.27 (0.99, 1.63)	1.47 (0.94, 2.31)
% attenuation[c]	25 %	39 %	41 %
Adjusted for drinking[d]	2.02 (1.57, 2.60)	1.52 (1.18, 1.94)	1.97 (1.26, 3.07)
% attenuation[c]	2 %	–	–
Adjusted for physical activity[e]	2.02 (1.57, 2.60)	1.47 (1.15, 1.88)	1.85 (1.19, 2.89)
% attenuation[c]	2 %	1 %	6 %
Adjusted for fruit and vegetable intake[f]	1.70 (1.32, 2.20)	1.23 (0.95, 1.58)	1.62 (1.03, 2.55)
% attenuation[c]	26 %	47 %	26 %
Adjusted for all 4 behaviours[g]	1.55 (1.19, 2.01)	1.17 (0.90, 1.51)	1.40 (0.89, 2.21)
% attenuation[c]	39 %	61 %	48 %

[a] Hazard ratio comparing cause specific mortality in respondents with manual versus non-manual occupational status

[b] Hazard ratio additionally adjusted for cumulative smoking

[c] % attenuation $=100\times(HR_{unadjusted}-HR_{adjusted})/(HR_{unadjusted}-1)$

[d] Hazard ratio additionally adjusted for cumulative drinking

[e] Hazard ratio additionally adjusted for cumulative physical activity

[f] Hazard ratio additionally adjusted for cumulative fruit and vegetable intake

[g] Hazard ratio additionally adjusted for cumulative smoking, drinking, physical activity and fruit and vegetable intake

cohorts may be due to the relative impact of behaviours on diseases at different ages, although our results demonstrating that cumulative behaviours are more important than single measures make this less likely. In addition, we cannot rule out artefact or bias arising from the selection of the two cohorts. For example, survival bias may have impacted on results in the older cohort as some of the most disadvantaged, heaviest smoking individuals born in 1932 might have already died before recruitment to the study. Alternatively, mortality in the younger cohort was inevitably dominated by premature deaths and the different cause profile might account for differences in the impact of behaviours, although it is worth noting that the main causes of death in the older cohort (CVD, cancer and respiratory disease) also accounted for the majority of deaths in the younger cohort.

Societal and cultural variation may also lead to differences in the impact of SES on individual health behaviours, and this may affect the potential for these factors to explain SES–mortality associations. For example, results from two occupational cohorts from the UK (Whitehall II) and France (GAZEL) [7] suggest that, while behavioural factors (smoking, alcohol, physical activity, and diet) were strong predictors of mortality in both cohorts, their associations with SES were markedly different, and this was reflected in the differing impact of behaviours in explaining associations between occupational position and all-cause mortality. The same four behaviours have also been explored in population cohorts in France and Northern Ireland [12], and in Finland [13]. Results from our analyses are broadly consistent with those from other population studies and from Whitehall II. Specifically, the behaviours most consistently associated with SES, smoking and diet had the greatest impact on SES associations with all-cause mortality.

Few studies have considered gender differences in the explanatory potential of health behaviours, in spite of gender differences in the impact of SES on mortality [32] and behaviours [54, 55], and evidence that behaviours explain a substantial part of gender differentials in mortality [56]. In our older cohort, there was little attenuation due to adjustment for alcohol or physical activity in either gender. However, smoking and diet had a greater impact on SES–mortality associations in men than in women. This is consistent with previous studies that reported greater attenuation of SES/education-mortality associations in men after adjustments for health behaviours [13] and risk factors including behaviours [9].

Strengths and Limitations

Our data are from two large representative population-based cohorts, living in the same geographical area, and followed up on five occasions, allowing us to compare distal, proximal and cumulative behaviours and to investigate their impact on three different measures of SES. We have explored two distinct age cohorts, born 20 years apart but followed-up over the same

calendar period, thus accounting, in part, for population trends in behaviours over time. We are not aware of any other studies that have explored temporal trends in this way. In addition, few existing studies have looked at gender differences in the impact of health behaviours on socioeconomic inequalities in mortality.

However, there are also some weaknesses to consider. We did not have complete SES and behaviours data at every wave. However, we explored a wide range of factors that predicted missing data, and these were included in our imputation models [42]. In addition, as with most studies, our health behaviour measures were simple and not always based on identical questions at each wave. If, as a result, there was measurement error in these variables, we may have underestimated the true impact of health behaviours in explaining SES–mortality associations. The cut-offs used to define healthier behaviours were based, as far as possible, on guidelines at the time of data collection and may not reflect current recommendations. For example, our drinking variable is based on previous weekly guidelines [36] and, therefore, may not identify binge drinkers who are highlighted in more recent guidance based on daily limits [57]. Similarly, guidelines for physical activity have now been revised [58] with higher targets for moderate and vigorous exercise than those used here. In addition, it was not always possible to match guidelines exactly. For example, our fruit and vegetable variable was based on days per week on which fruit and vegetables were consumed rather than portions per day and will therefore include some respondents who were eating only two or three portions as well as those meeting the recommended five-a-day. Again, this may have led to an underestimate of effect. The healthiest groups identified here may not be directly comparable to the healthiest group in another population, and the cut-offs used in these data should not be interpreted as targets that, if met, will counteract the negative health effects of lower SES. Our aim in these analyses was to compare more versus less healthy behaviours within our population and it is of note that the behaviours that had the greatest impact, i.e. smoking and diet, are the same as those identified previously.(12, 13) It is also important to recognise that the healthy behaviours identified in these data may not reduce SES–mortality risk directly and that they may be a marker for other, unmeasured, healthy lifestyle aspects.

Our primary SES measure was based on baseline occupation and may not fully capture the experience of being in a low or high socioeconomic group across the life-course, particularly in the context of changing labour markets or, in the older cohort, retirement. However, our aim was to explore the impact of health behaviours on SES–mortality associations in two distinct groups, rather than tracking SES–mortality associations over time. It is reassuring that analyses based on SES at each follow-up wave, and measured all three ways, demonstrated that, although SES–mortality associations were not fixed over time, the role of cumulative behaviours in explaining these associations was consistent. Our cause-specific analyses provide some insight into mechanisms but we did not have sufficient numbers to explore individual causes of specifically premature deaths in the younger cohort. It would also be of potential interest to look separately at causes of death not as strongly associated with behaviours, e.g. external causes, but again we did not have sufficient numbers. Our aim was to explore the impact of health behaviours on SES–mortality associations. However, it is important to recognise that other factors may also impact on these associations and on health behaviours. These include access to and use of health services, psychosocial factors and their biological consequences, physical environments in the home, work and neighbourhood, personal social networks and support and wider neighbourhood influences. Finally, although our cohorts were representative of the populations from which they were drawn, our results are not necessarily generalizable to the wider population, for example, theTwenty-07 study does not cover rural areas. In addition, lower SES respondents were more likely to have missing data in later waves. However, again, it is of note that our results are in line with those observed in other populations.

Conclusion

Health behaviours explain some, but not all, of the socioeconomic differentials in mortality in middle and early-older age. Behaviours that are more strongly associated with SES, and cumulate over time, tend to have the greatest impact. Interventions to improve health behaviours, and reduce inequalities in them, need to consider not only the differences between social groups but also how these vary by population group, by gender and over time.

Conflict of Interest The authors have no conflict of interest to disclose.

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