The effect of socioeconomic deprivation on the association between an extended measurement of unhealthy lifestyle factors and health outcomes: a prospective analysis of the UK Biobank cohort

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Summary

Background Combinations of lifestyle factors interact to increase mortality. Combinations of traditional factors such as smoking and alcohol are well described, but the additional effects of emerging factors such as television viewing time are not. The effect of socioeconomic deprivation on these extended lifestyle risks also remains unclear. We aimed to examine whether deprivation modifies the association between an extended score of lifestyle-related risk factors and health outcomes.

Methods Data for this prospective analysis were sourced from the UK Biobank, a prospective population-based cohort study. We assigned all participants an extended lifestyle score, with 1 point for each unhealthy lifestyle factor (incorporating sleep duration and high television viewing time, in addition to smoking, excessive alcohol, poor diet [low intake of oily fish or fruits and vegetables, and high intake of red meat or processed meats], and low physical activity), categorised as most healthy (score 0–2), moderately healthy (score 3–5), or least healthy (score 6–9). Cox proportional hazards models were used to examine the association between lifestyle score and health outcomes (all-cause mortality and cardiovascular disease mortality and incidence), and whether this association was modified by deprivation. All analyses were landmark analyses, in which participants were excluded if they had an event (death or cardiovascular disease event) within 2 years of recruitment. Participants with non-communicable diseases (except hypertension) and missing covariate data were excluded from analyses. Participants were also excluded if they reported implausible values for physical activity, sleep duration, and total screen time. All analyses were adjusted for age, sex, ethnicity, month of assessment, history of hypertension, systolic blood pressure, medication for hypercholesterolaemia or hypertension, and body-mass index categories.

Findings 328 594 participants aged 40–69 years were included in the study, with a mean follow-up period of 4·9 years (SD 0·83) after the landmark period for all-cause and cardiovascular disease mortality, and 4·1 years (0·81) for cardiovascular disease incidence. In the least deprived quintile, the adjusted hazard ratio (HR) in the least healthy lifestyle category, compared with the most healthy category, was 1·65 (95% CI 1·25–2·19) for all-cause mortality, 1·93 (1·16–3·20) for cardiovascular disease mortality, and 1·29 (1·10–1·52) for cardiovascular disease incidence. Equivalent HRs in the most deprived quintile were 2·47 (95% CI 2·04–3·00), 3·36 (2·36–4·76), and 1·41 (1·25–1·60), respectively. The HR for trend for one increment change towards least healthy in the least deprived quintile compared with that in the most deprived quintile was 1·25 (95% CI 1·12–1·39) versus 1·55 (1·40–1·70) for all-cause mortality, 1·30 (1·05–1·61) versus 1·83 (1·54–2·18) for cardiovascular disease mortality, and 1·10 (1·04–1·17) versus 1·16 (1·09–1·23) for cardiovascular disease incidence. A significant interaction was found between lifestyle and deprivation for all-cause and cardiovascular disease mortality (both $p_{interaction}<0·0001$), but not for cardiovascular disease incidence ($p_{interaction}=0·11$).

Interpretation Wide combinations of lifestyle factors are associated with disproportionate harm in deprived populations. Social and fiscal policies that reduce poverty are needed alongside public health and individual-level interventions that address a wider range of lifestyle factors in areas of deprivation.

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Introduction

Non-communicable diseases are the commonest cause of death worldwide, and these diseases are mainly caused by unhealthy lifestyles.[1] The contribution of socioeconomic deprivation to mortality risk is similar to that observed for some lifestyle factors (eg, physical inactivity).[2] However, the relationships between lifestyle factors, deprivation, and mortality remain unclear.

Research in context

Evidence before this study

We searched PubMed in October, 2017, for articles published in English between Sept 8, 2007, and Sept 8, 2017, using MeSH terms for "healthy lifestyle", "life style", "health behaviour", "risk-taking", "mortality", and "socioeconomic factors". Previous studies have shown disproportionate lifestyle-related harm in deprived groups associated with traditional lifestyle factors (eg, smoking, alcohol consumption, and physical inactivity); however, none has examined more extensive combinations of lifestyle factors that include emerging lifestyle factors, such as sleep duration, sedentary time, or television viewing time. Combinations of lifestyle factors are known to interact synergistically to raise associated mortality and morbidity. The addition of emerging lifestyle factors to traditional lifestyle factor combinations has been shown to strengthen the association with mortality; however, the contribution of socioeconomic deprivation to lifestyle-related mortality is uncertain. Specifically, no study has examined how deprivation modifies the association between an extended combination of lifestyle factors and adverse health outcomes.

Added value of this study

To our knowledge, this prospective study is the first to investigate how socioeconomic deprivation affects the association between a broad combination of lifestyle factors, including newer emerging lifestyle factors, and adverse health outcomes. This study uses high-quality data from a large UK population cohort study with linked hospital admission and mortality data. We carried out landmark survival analyses and excluded participants with non-communicable diseases (but not those with hypertension) at baseline to reduce the chance of our results being attributable to reverse causality. We used the most conservative models to minimise the influence of important potential confounders. We found a significant interaction between an extended lifestyle score and socioeconomic deprivation that synergistically raised all-cause mortality and cardiovascular disease mortality (both $p_{interaction}<0·0001$), but not cardiovascular disease incidence ($p_{interaction}=0·11$).

Implications of all the available evidence

Lifestyle-related harm is a substantial and growing burden on society, but there is disproportionate lifestyle-related harm in more deprived groups. This study shows disproportionate harm is associated with both emerging lifestyle factors such as television viewing time and sleep duration as well as more traditional lifestyle factors such as smoking. Thus, policies should target a wider range of lifestyle factors than they do currently. However, to avoid increasing health inequalities, lifestyle interventions need to incorporate an understanding of the upstream socioeconomic determinants of lifestyle. Our findings support the societal and political imperative to reduce poverty, because—despite similar levels of multiple unhealthy lifestyle factors—deprived populations suffer disproportionately worse outcomes.

For example, although deprived populations have the highest prevalence of multiple co-occurring adverse lifestyle factors and the highest premature mortality,[2,3] the prevalence of lifestyle factors only partially explains the increased mortality associated with deprivation.[4]

Study of the synergistic interaction between lifestyle factors and deprivation might improve our understanding of the excess mortality associated with deprivation. For example, individuals from deprived groups have an increased risk of harm from the same amount of alcohol consumed even after controlling for drinking patterns, body-mass index, and smoking.[5] Similarly, deprived populations are disproportionately affected by the harmful effects of smoking and physical inactivity.[6,7] Mechanisms underlying this disproportionate lifestyle harm remain unclear but might include extremes of unhealthy lifestyle factors,[8] interactions with other factors associated with deprivation (such as psychosocial stress),[9] and reduced access to health services.[10] However, previous studies of interactions between lifestyle factors and deprivation have examined only smoking, alcohol intake, and physical inactivity.[5–7]

Numerous individual lifestyle factors are associated with mortality; however, combinations of lifestyle factors interact synergistically, resulting in stronger associations with mortality.[11] Evidence from the past 3 years has emphasised a role for emerging factors, such as sleep duration and television viewing time.[12,13] Combining emerging and traditional factors (smoking, alcohol intake, physical activity, and diet) in extended lifestyle scores shows that a high proportion of deaths are due to modifiable factors and are therefore avoidable, highlighting new targets for public health intervention.[14]

To our knowledge, interactions between deprivation and extended combinations of lifestyle factors that include emerging factors have not been investigated. An understanding of how health outcomes associated with a broad combination of lifestyle factors vary with deprivation could inform future public health policy, highlighting new targets for individual-level and population-level interventions.

Our aim was to examine how deprivation affects the association between a previously devised extended lifestyle score, which incorporates emerging and traditional lifestyle factors, and health outcomes (all-cause mortality, and cardiovascular disease mortality and incidence). We had two hypotheses: the health risk for individuals with an unhealthy lifestyle score increases disproportionately with increasing level of deprivation; and this disproportionate increase in risk is greater when lifestyle is measured by an

extended score as opposed to a traditional score that omits emerging lifestyle factors.

Methods

Study design and participants

This was a prospective, population-based cohort study of participants enrolled in the UK Biobank. 502 655 participants were recruited by postal invitation between March 13, 2006, and Oct 1, 2010. Participants attended one of 22 assessment centres across England, Scotland, and Wales, where they completed touchscreen and nurse-led questionnaires, had physical measurements taken, and provided biological samples.[15] All individuals registered with the National Health Service (NHS) aged 40–69 years who were living within 25 miles from one of the 22 study assessment centres were invited to participate; those who responded and had capacity to consent were included. Participants were excluded from analyses if they reported implausible values for physical activity, television viewing time, or sleep duration, defined as the sum of their total physical activity, sleep duration, and total screen time exceeding 24 h. Further details of these measurements can be found in the UK Biobank online protocol.[16] Participants with missing lifestyle or sociodemographic data, an event (death or cardiovascular disease event) within 2 years of recruitment, and any self-reported non-communicable disease (excluding hypertension) at baseline were also excluded from analyses. The UK Biobank study was approved by the North West Multi-Centre Research Ethics Committee; participants provided written informed consent for data collection, data analysis, and record linkage. This study is part of UK Biobank project 7155 (NHS National Research Ethics Service 16/NW/0274).

Procedures

Dates of death were obtained from death certificates held by the NHS Information Centre (England and Wales) and the NHS Central Register (Scotland). Dates and causes of hospital admissions were identified via record linkage to Health Episode Statistics (England and Wales) and to the Scottish Morbidity Records (SMR01). Analyses for each outcome were censored at different dates because different data sources were used for each outcome. All-cause mortality analyses were censored at Jan 31, 2016, or date of death if death occurred earlier. Cardiovascular disease analyses were censored at March 31, 2015 (latest date available), or date of event if the event occurred earlier. Incident cardiovascular disease was defined as a hospital admission or death with International Classification of Diseases, 10th revision, code I05–I89.9. Therefore, incident cardiovascular disease included both fatal and non-fatal events.

Townsend deprivation index scores were derived from national census data about car ownership, household overcrowding, owner occupation, and unemployment aggregated for postcodes of residence.[17] Higher Townsend scores equate to higher levels of socioeconomic deprivation. Data about household income were self-reported (appendix). Educational attainment was derived from the highest self-reported qualification based on the International Standard Classification of Education (appendix).[18] Age was calculated from dates of birth and baseline assessment. Ethnicity was self-reported as white, mixed, Asian or Asian British, black or black British, Chinese, or other ethnic group; data for ethnic subgroups were available, but with few participants in each, we did not analyse participants by ethnic subgroup. Smoking status was categorised into never, former, and current smoking. Non-communicable diseases (depression, bipolar disorder, schizophrenia, alcohol problems, substance abuse, eating disorders, cognitive impartment, dementia, Parkinson's disease, chronic pain syndrome, chronic obstructive pulmonary disease, chronic asthma, chronic liver diseases, hypertension, heart diseases, stroke, diabetes, inflammatory diseases, arthritis, and cancer) were self-reported on the baseline questionnaire. Trained nurses measured blood pressure, height, and bodyweight during the initial assessment. WHO criteria were applied to classify body-mass index into underweight (<18·5 kg/m²), normal weight (18·5–24·9 kg/m²), overweight (25·0–29·9 kg/m²), and obese (≥30·0 kg/m²).[19]

Available data for lifestyle factors in UK Biobank were used to generate a lifestyle score using smoking status, alcohol intake, physical activity, television viewing time, sleep duration, fruit and vegetable intake, oily fish intake, and red and processed meat intake. This score was based on a previously published score from an Australian cohort of 267 079 adults, which represented the sum of each dichotomised lifestyle variable (0 points if not at risk, 1 point if at risk), resulting in a risk score range of 0–6, in which a higher score indicated higher risk (appendix).[14] UK Biobank data collection precluded using exactly the same lifestyle variables so the score was adapted. Lifestyle variables comprising the score were derived from questionnaire responses (appendix). If available, we used national guidelines to generate healthy and unhealthy categories for each lifestyle factor. We assigned 1 point to participants for each unhealthy category (current smoker; alcohol consumed daily or almost daily; <150 min per week of moderate intensity physical activity or <75 min per week of vigorous intensity physical activity; ≥4 h per day of television viewing time; <7 h or >9 h of sleep per day; <400 g of fruits and vegetables per day; less than one portion of oily fish per week; more than three portions of red meat per week; more than one portion of processed meat per week). Participants' points were summed to create an unweighted score. The minimum score was 0, indicating a healthier lifestyle, and the maximum score was 9, indicating an unhealthier lifestyle. To examine the associations of lifestyle with health outcomes, participants were classified into three categories according to their lifestyle score. Participants who scored 0, 1, or 2 were classed as most healthy; those who scored 3, 4,

See Online for appendix

For the **UK Biobank** see http://www.ukbiobank.ac.uk

	Most healthy lifestyle (n=141 419)	Moderately healthy lifestyle (n=173 455)	Least healthy lifestyle (n=13 720)
Sociodemographics			
Age, years	55·9 (8·13)	55·6 (8·12)	55·2 (8·08)
Sex			
Women	88 219 (62·4%)	86 563 (49·9%)	4593 (33·5%)
Men	53 200 (37·6%)	86 892 (50·1%)	9127 (66·5%)
Educational attainment			
No relevant qualifications	15 947 (11·3%)	26 833 (15·5%)	3085 (22·5%)
CSEs or equivalent	6962 (4·9%)	10 875 (6·3%)	1098 (8·0%)
O levels, GCSEs, or equivalent	29 172 (20·6%)	38 933 (22·4%)	3231 (23·5%)
A levels, AS levels, or equivalent	16 690 (11·8%)	19 762 (11·4%)	1427 (10·4%)
College or university degree	55 768 (39·4%)	54 865 (31·6%)	2974 (21·7%)
Data missing	16 880 (11·9%)	22 187 (12·8%)	1905 (13·9%)
Income			
Less than £18 000	20 361 (14·4%)	29 223 (16·8%)	3083 (22·5%)
£18 000–29 999	29 954 (21·2%)	37 466 (21·6%)	3027 (22·1%)
£30 000–51 999	34 084 (24·1%)	41 550 (24·0%)	3047 (22·2%)
£52 000–100 000	29 757 (21·0%)	33 476 (19·3%)	2107 (15·4%)
More than £100 000	8782 (6·2%)	8561 (4·9%)	505 (3·7%)
Data missing	18 481 (13·1%)	23 179 (13·4%)	1951 (14·2%)
Socioeconomic deprivation quintile			
1 (least deprived)	31 817 (22·5%)	35 501 (20·5%)	2175 (15·9%)
2	30 739 (21·7%)	35 596 (20·5%)	2302 (16·8%)
3	29 376 (20·8%)	35 465 (20·4%)	2479 (18·1%)
4	27 561 (19·5%)	34 758 (20·0%)	2972 (21·7%)
5 (most deprived)	21 926 (15·5%)	32 135 (18·5%)	3792 (27·6%)
Ethnicity			
White	133 609 (94·5%)	164 803 (95·0%)	13 055 (95·2%)
Mixed background	838 (0·6%)	1083 (0·6%)	104 (0·8%)
South Asian	2821 (2·0%)	2818 (1·6%)	162 (1·2%)
Black	2197 (1·6%)	2799 (1·6%)	272 (2·0%)
Chinese	514 (0·4%)	618 (0·4%)	33 (0·2%)
Other	1440 (1·0%)	1334 (0·8%)	94 (0·7%)
Obesity-related markers			
Weight, kg	74·7 (14·36)	78·7 (15·57)	82·2 (16·66)
Height, cm	168·0 (9·09)	169·3 (9·37)	170·8 (9·23)
Body-mass index	26·4 (4·21)	27·4 (4·58)	28·1 (4·90)
Body-mass index categories			
Under weight (<18·5 kg/m^2)	796 (0·6%)	736 (0·4%)	86 (0·6%)
Normal weight (18·5–24·9 kg/m^2)	57 090 (40·4%)	54 556 (31·5%)	3624 (26·4%)
Overweight (25·0–29·9 kg/m^2)	59 256 (41·9%)	76 790 (44·3%)	5919 (43·1%)
Obese (≥30·0 kg/m^2)	24 277 (17·2%)	41 373 (23·9%)	4091 (29·8%)
Waist circumference, cm	86·5 (12·20)	90·7 (12·90)	94·8 (13·15)
Body fat percentage	30·9 (8·35)	30·9 (8·50)	30·0 (8·37)
Smoking and alcohol			
Smoking status			
Never	90 312 (63·9%)	94 541 (54·5%)	3915 (28·5%)
Previous	47 464 (33·6%)	57 336 (33·1%)	3511 (25·6%)
Current	3643 (2·6%)	21 578 (12·4%)	6294 (45·9%)
Alcohol intake, proportion of total energy intake	3·0 (1·38)	2·7 (1·51)	2·1 (1·52)

(Table 1 continues on next page)

or 5 were classed as moderately healthy; and those who scored 6, 7, 8, or 9 were classed as least healthy. Distributions of the lifestyle score and lifestyle categories are shown in the appendix.

Outcomes

The outcomes assessed in this analysis were all-cause mortality, and cardiovascular disease mortality and incidence; exposures of interest were lifestyle category and quintiles of area-based deprivation.

Statistical analysis

We explored the associations between lifestyle category, deprivation quintile, and health outcomes using Cox proportional hazards models, with years of follow-up as the time-varying covariate. Sociodemographic factors (age, sex, and ethnicity), month of assessment, history of hypertension, systolic blood pressure, medication for hypercholesterolaemia or hypertension, and body-mass index categories were treated as confounders being associated with both exposures and outcomes. To reduce the chance of reverse causality, all analyses were landmark analyses, with follow-up commencing 2 years after recruitment, thereby excluding participants with an event (death or cardiovascular disease event) within 2 years of recruitment. In addition, participants with any self-reported non-communicable disease (excluding hypertension) at baseline were excluded from analyses. Participants who reported a history of hypertension at baseline (n=70 854) were not excluded because power would be reduced considerably.

First, we investigated the separate associations of lifestyle score (0–9; ordinal variable), lifestyle category (most healthy, moderately healthy, and least healthy), and deprivation quintile with health outcomes. Second, we investigated whether deprivation modified the associations observed between lifestyle category and health outcomes. Significant interactions between the lifestyle category and deprivation on health outcomes were tested by fitting an interaction term between the two variables (ie, lifestyle category by deprivation quintile). To determine the interaction effect, we used ordinal coding, with participants in the least deprived quintile who were in the most healthy lifestyle category as the reference group. All analyses were adjusted for age, sex, ethnicity, month of assessment, history of physician-diagnosed hypertension, systolic blood pressure, medication for hypercholesterolaemia or hypertension, and body-mass index categories. When the associations between lifestyle category and health outcomes were investigated separately, models were adjusted for deprivation quintile, and vice versa.

The Townsend index was our primary socioeconomic exposure because it was available for nearly all participants. However, we also ran models with individual-level measures of socioeconomic status (household income and educational attainment). To highlight the effect of

emerging lifestyle factors, we ran analyses using a traditional score, which excluded sleep and television viewing time. Additionally, we tested a score that only included lifestyle factors that were significantly associated with all outcomes. We also ran analyses excluding participants with hypertension. To examine interaction effects, we ran models using continuous variables for lifestyle and deprivation, and calculated a synergy index.

The proportional hazards assumption was checked with tests based on Schoenfeld residuals. We used Stata, version 14, for all analyses.

Role of the funding source

There was no funding source for this study. This study used UK Biobank data but was designed, conducted, analysed, and interpreted by the authors. All authors had full access to all the data in the study. The corresponding author had final responsibility for the decision to submit for publication.

Results

Of the 502 655 participants recruited to UK Biobank, 134 614 (26·8%) reported non-communicable diseases at baseline, 39 447 (7·8%) participants had an event within 2 years of recruitment, 39 415 (7·8%) had missing covariate data, and 705 (0·1%) reported implausible values for physical activity, sleep duration, and total screen time. Some participants met more than one of these exclusion criteria. Excluded participants were broadly similar to those included (appendix). Therefore, 328 594 (65·4%) participants had full data available for the landmark analyses. The mean follow-up period was 4·9 years (SD 0·83; range 3·3–7·9) after the landmark period for all-cause and cardiovascular disease mortality, and 4·1 years (0·81; range 2·4–7·0) for cardiovascular disease incidence. Over the follow-up period, 17 380 (5·3%) participants had incident cardiovascular disease and 5553 participants (1·7%) died (1528 [0·5%] from cardiovascular disease).

Overall, 141 419 (43·0%) participants were in the most healthy category, 173 455 (52·8%) in the moderately healthy category, and 13 720 (4·2%) in the least healthy category (table 1). Characteristics of participants by deprivation quintile are summarised in the appendix. The most deprived quintile had a lower prevalence of healthy lifestyle behaviours as well as a greater proportion of participants who were obese or underweight than did the other quintiles (appendix). Participants in the most deprived quintile had a larger waist circumference and slightly higher body fat percentage than those in the other quintiles (appendix).

Participants in the least healthy category had a higher hazard ratio (HR) for all outcomes than did those in the reference, most healthy, category (table 2). One increment change towards the least healthy category was associated with a HR for trend of 1·34 (95% CI 1·28–1·41) for all-cause mortality, 1·44 (1·32–1·57) for cardiovascular disease mortality, and 1·10 (1·07–1·13) for cardiovascular disease incidence (table 2). There was a clear and significant trend of increased risk for all outcomes for participants with an increasingly unhealthier lifestyle score compared with those with a minimum score of 0 (appendix). One increment change in lifestyle score was associated with a HR for trend of 1·13 (95% CI 1·12–1·15) for all-cause mortality, 1·16 (1·12–1·20) for cardiovascular disease mortality, and 1·04 (1·03–1·05) for cardiovascular disease incidence (appendix). Deprivation was associated with a higher hazard for all-cause mortality, and cardiovascular disease mortality and incidence (appendix). One quintile increment in deprivation was associated with a HR for trend of 1·11 (95% CI 1·08–1·13) for all-cause mortality, 1·16 (1·12–1·21) for cardiovascular disease mortality, and 1·05 (1·04–1·06) for cardiovascular disease incidence (appendix).

Associations between lifestyle categories and health outcomes were stratified by quintiles of deprivation (table 3). Relative to the most healthy category, there were marked increases in HR for all-cause and cardiovascular disease mortality across the lifestyle categories for most quintiles of deprivation; however, the pattern was less clear for cardiovascular disease incidence. Significant interactions between lifestyle category and deprivation were found for all-cause and cardiovascular disease mortality (both $p_{interaction}<0·0001$) but not for cardiovascular

	Most healthy lifestyle (n=141 419)	Moderately healthy lifestyle (n=173 455)	Least healthy lifestyle (n=13 720)
(Continued from previous page)			
Physical activity and television viewing time			
Total physical activity, MET-h per week	8·1 (9·19)	5·6 (8·86)	4·2 (8·98)
Television viewing, h per day	2·2 (1·24)	2·9 (1·60)	4·0 (1·86)
Sleep-related characteristics			
Sleep duration, h per day	7·3 (0·85)	7·0 (1·07)	6·6 (1·31)
Sleep duration categories			
Short sleepers (<7 h per day)	17 420 (12·3%)	52 783 (30·4%)	7942 (57·9%)
Normal sleepers (7–9 h per day)	123 379 (87·2%)	118 245 (68·2%)	5199 (37·9%)
Long sleepers (>9 h per day)	620 (0·4%)	2427 (1·4%)	579 (4·2%)
Dietary factors			
Fruit and vegetables intake, g per day	412·5 (205·20)	273·8 (154·17)	191·2 (118·89)
Oily fish, portions per week	1·4 (1·12)	0·9 (0·89)	0·5 (0·62)
Red meat, portions per week	1·6 (1·00)	2·1 (1·50)	3·2 (2·04)
Processed meat intake, portions per week	1·4 (0·90)	2·1 (1·05)	2·9 (0·88)
Health status			
History of physician-diagnosed hypertension	28 167 (19·9%)	39 155 (22·6%)	3532 (25·7%)
Systolic blood pressure, mm Hg	138·6 (19·74)	140·1 (19·55)	141·8 (19·31)
Cholesterol-lowering medication	6625 (4·7%)	7095 (4·1%)	456 (3·3%)
Blood pressure medication	8038 (5·7%)	8712 (5·0%)	519 (3·8%)
None of the above medications	126 756 (89·6%)	157 648 (90·9%)	12 745 (92·9%)

Data are mean (SD) or n (%) for continuous and categorical variables, as appropriate. MET=metabolic equivalent.

Table 1: **Cohort characteristics by lifestyle category**

	Total (n=328 594)	Most healthy lifestyle (n=141 419)		Moderately healthy lifestyle (n=173 455)		Least healthy lifestyle (n=13 720)		HR for trend (95% CI)
		Events	Adjusted HR	Events	Adjusted HR (95% CI)	Events	Adjusted HR (95% CI)	
All-cause mortality	5553 (1·7%)	1896 (1·3%)	1 (ref)	3176 (1·8%)	1·23 (1·16–1·31)	481 (3·5%)	2·06 (1·86–2·29)	1·34 (1·28–1·41)
Cardiovascular disease mortality	1528 (0·5%)	461 (0·3%)	1 (ref)	919 (0·5%)	1·35 (1·21–1·51)	148 (1·1%)	2·21 (1·83–2·67)	1·44 (1·32–1·57)
Cardiovascular disease incidence	17 380 (5·3%)	6560 (4·6%)	1 (ref)	9718 (5·6%)	1·06 (1·03–1·10)	1102 (8·0%)	1·31 (1·23–1·40)	1·10 (1·07–1·13)

Data are n (%) unless otherwise stated. The most healthy category was used as reference category. HR for trend indicates the change in HR by one lifestyle category change towards least healthy. All analyses were 2-year landmark analyses and with exclusion of all participants with non-communicable diseases at baseline (n=134 614). All analyses were adjusted for age, sex, ethnicity, month of assessment, history of physician-diagnosed hypertension, systolic blood pressure, medication for cardiovascular disease, body-mass index category, and socioeconomic deprivation quintile. HR=hazard ratio.

Table 2: **Cox proportional hazards models of the overall associations between lifestyle category and health outcomes**

	Total (n=328 594)	Most healthy lifestyle (n=141 419)		Moderately healthy lifestyle (n=173 455)		Least healthy lifestyle (n=13 720)		HR for trend (95% CI)
		Events	Adjusted HR	Events	Adjusted HR (95% CI)	Events	Adjusted HR (95% CI)	
All-cause mortality								
1 (least deprived)	1091/69 493 (1·6%)	415 (0·3%)	1 (ref)	620 (0·4%)	1·22 (1·07–1·38)	56 (0·4%)	1·65 (1·25–2·19)	1·25 (1·12–1·39)
2	1057/68 637 (1·5%)	420 (0·3%)	1 (ref)	569 (0·3%)	1·07 (0·94–1·22)	68 (0·5%)	1·88 (1·45–2·44)	1·19 (1·07–1·33)
3	1102/67 320 (1·6%)	407 (0·3%)	1 (ref)	611 (0·4%)	1·16 (1·03–1·32)	84 (0·6%)	2·02 (1·59–2·56)	1·29 (1·16–1·43)
4	1142/65 291 (1·7%)	368 (0·3%)	1 (ref)	674 (0·4%)	1·34 (1·18–1·52)	100 (0·7%)	2·08 (1·66–2·61)	1·40 (1·27–1·55)
5 (most deprived)	1161/57 853 (2·0%)	286 (0·2%)	1 (ref)	702 (0·4%)	1·42 (1·24–1·63)	173 (1·3%)	2·47 (2·04–3·00)	1·55 (1·40–1·70)
Cardiovascular disease mortality								
1 (least deprived)	274/69 493 (0·4%)	97 (0·1%)	1 (ref)	159 (0·1%)	1·23 (0·95–1·58)	18 (0·1%)	1·93 (1·16–3·20)	1·30 (1·05–1·61)
2	260/68 637 (0·4%)	107 (0·1%)	1 (ref)	132 (0·1%)	0·91 (0·70–1·18)	21 (0·2%)	1·96 (1·21–3·16)	1·12 (0·90–1·39)
3	312/67 320 (0·5%)	98 (0·1%)	1 (ref)	199 (0·1%)	1·48 (1·16–1·89)	15 (0·1%)	1·30 (0·75–2·26)	1·31 (1·08–1·59)
4	321/65 291 (0·5%)	92 (0·1%)	1 (ref)	198 (0·1%)	1·47 (1·15–1·89)	31 (0·2%)	2·25 (1·49–3·40)	1·49 (1·23–1·80)
5 (most deprived)	361/57 853 (0·6%)	67 (<0·1%)	1 (ref)	231 (0·1%)	1·84 (1·40–2·43)	63 (0·5%)	3·36 (2·36–4·76)	1·83 (1·54–2·18)
Cardiovascular disease incidence								
1 (least deprived)	3550/69 493 (5·1%)	1437 (1·0%)	1 (ref)	1951 (1·1%)	1·08 (1·01–1·15)	162 (1·2%)	1·29 (1·10–1·52)	1·10 (1·04–1·17)
2	3640/68 637 (5·3%)	1498 (1·1%)	1 (ref)	1970 (1·1%)	1·01 (0·94–1·08)	172 (1·3%)	1·23 (1·05–1·45)	1·04 (0·99–1·11)
3	3568/67 320 (5·3%)	1373 (1·0%)	1 (ref)	1994 (1·1%)	1·07 (1·00–1·15)	201 (1·5%)	1·32 (1·14–1·53)	1·10 (1·04–1·17)
4	3329/65 291 (5·1%)	1238 (0·9%)	1 (ref)	1869 (1·1%)	1·06 (0·99–1·14)	222 (1·6%)	1·27 (1·10–1·47)	1·10 (1·03–1·16)
5 (most deprived)	3293/57 853 (5·7%)	1014 (0·7%)	1 (ref)	1934 (1·1%)	1·10 (1·01–1·18)	345 (2·5%)	1·41 (1·25–1·60)	1·16 (1·09–1·23)

Data are n/N (%) or n (%) unless otherwise stated. The most healthy lifestyle category for each quintile of socioeconomic deprivation was used as the reference category. The HR for trend indicates the change in HR by one lifestyle category change towards least healthy. All analyses were 2-year landmark analyses and with exclusion of all participants with non-communicable diseases at baseline (n=134 614). All analyses were adjusted for age, sex, ethnicity, month of assessment, history of physician-diagnosed hypertension, systolic blood pressure, medication for cardiovascular disease, and body-mass index category. HR=hazard ratio.

Table 3: **Cox proportional hazards models of the association between lifestyle category and health outcomes, stratified by socioeconomic deprivation quintile**

disease incidence ($p_{interaction}$=0·11; figure; appendix). In the least deprived quintile, the adjusted HR for individuals in the least healthy category compared with those in the most healthy category was 1·65 (95% CI 1·25–2·19) for all-cause mortality, 1·93 (1·16–3·20) for cardiovascular disease mortality, and 1·29 (1·10–1·52) for cardiovascular disease incidence (table 3). By contrast, individuals in the least healthy category but also in the most deprived quintile had an adjusted HR of 2·47 (95% CI 2·04–3·00) for all-cause mortality, 3·36 (2·36–4·76) for cardiovascular disease mortality, and 1·41 (1·25–1·60) for cardiovascular disease incidence compared with those in the same quintile (most deprived) but in the most healthy category (table 3). When all groups were compared with a single combined most healthy and least deprived reference group, there was a dose–response increment for all-cause and cardiovascular disease mortality HR across most lifestyle categories and deprivation quintiles (figure; appendix). A similar, albeit non-significant, association was also observed for cardiovascular disease incidence (figure; appendix). The HR for trend for one increment change towards least healthy in the least deprived quintile compared with that in the most deprived quintile was 1·25 (95% CI 1·12–1·39) versus 1·55 (1·40–1·70) for all-cause mortality, 1·30 (1·05–1·61) versus 1·83 (1·54–2·18) for cardiovascular disease mortality, and 1·10 (1·04–1·17) versus 1·16 (1·09–1·23) for cardiovascular disease incidence (table 3; figure; appendix).

Sensitivity analyses using the traditional score showed the same pattern; however, for individuals in the least healthy category, HRs for all outcomes across all levels of deprivation were lower with the traditional score than with the extended score (appendix). Analyses of individual lifestyle factors showed considerable variation in HRs for each outcome (appendix). However, analyses using a lifestyle score comprising only those lifestyle factors associated with all outcomes of interest produced similar results to our main results (appendix). Excluding individuals with hypertension also produced similar results, albeit with wider CIs than analyses that included these individuals (appendix). Broadly similar estimates were found for individual-level measures of socioeconomic status; individuals with the lowest income or educational attainment in the least healthy category had the highest HR for all outcomes (appendix). The patterns of interactions between lifestyle category and individual-level measures of socioeconomic status, and those of the additional tests for interaction and synergy, were similar to the pattern between lifestyle category and the Townsend index (appendix).

Discussion

Our findings show that an extended score of unhealthy lifestyle factors—incorporating the emerging risk factors of sleep duration and television viewing time together with more traditional lifestyle factors of smoking, alcohol, physical activity, and diet—has a strong association with all-cause mortality, and cardiovascular disease mortality and incidence. We found that the association between an unhealthy lifestyle and all-cause and cardiovascular disease mortality becomes stronger with increasing levels of deprivation. This disproportionate lifestyle-associated risk in more deprived groups was seen with both area-level and individual-level measures of socioeconomic status. Furthermore, this risk was even greater in more deprived groups when lifestyle was measured by an extended score that included emerging factors than by a traditional score. These inequalities in lifestyle-related risk were not accounted for by differences in sex, age, ethnicity, blood pressure, concurrent cholesterol or blood pressure medication, or body-mass index. The steeper all-cause and cardiovascular disease mortality trends (figure) across lifestyle categories in more deprived groups support a vulnerability hypothesis whereby deprived groups are more vulnerable to the effects of unhealthy lifestyles than are groups that are not deprived.[6,20] According to this hypothesis, deprived groups are likely to experience disproportionate harm from unhealthy lifestyles, which is not simply explained by greater prevalence of unhealthy lifestyles.

These findings are consistent with previous studies that have shown disproportionate harm associated with some traditional lifestyle factors. For example, US survey data from approximately 41 000 adults showed that those of lower socioeconomic status had disproportionately poorer

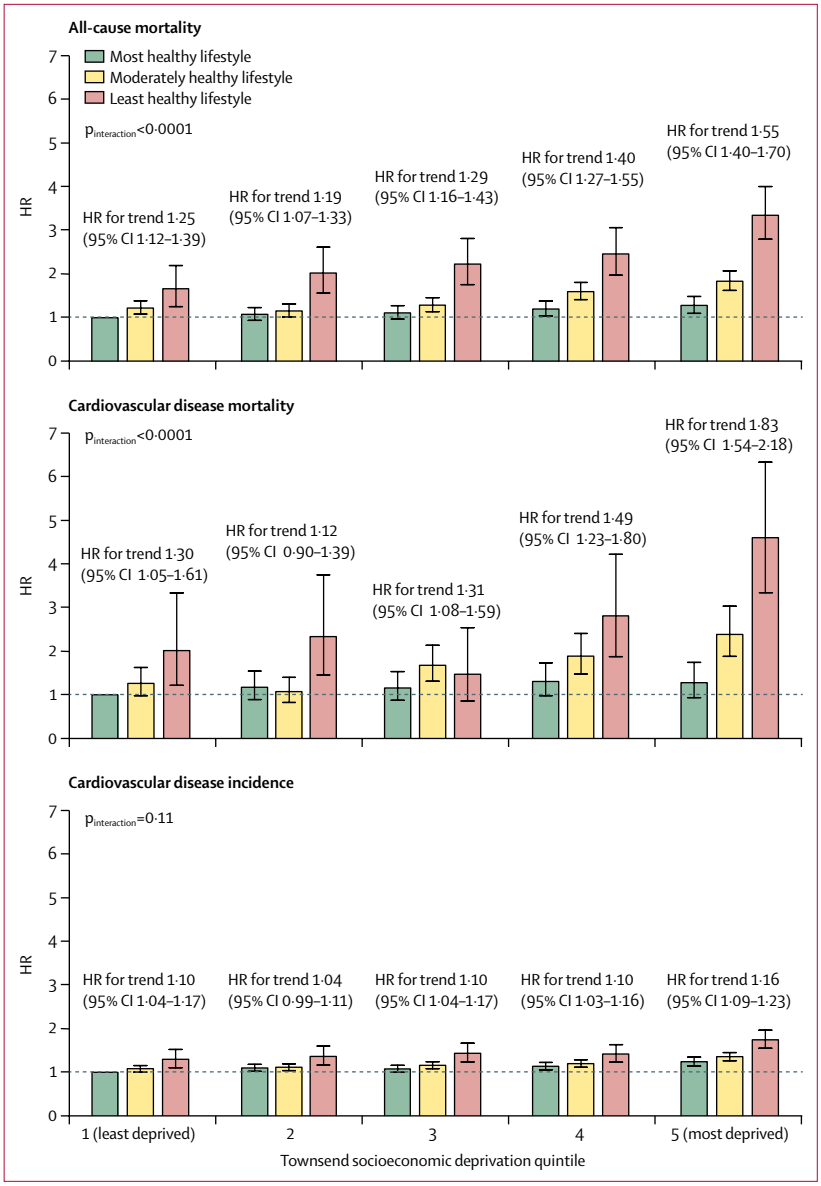

Figure: **Cox proportional hazards models of the association between lifestyle category, socioeconomic deprivation quintile, and health outcomes**
Most healthy, least deprived group is the reference group. Error bars indicate 95% CIs. HR=hazard ratio.

measures of morbidity and self-rated health associated with traditional lifestyle factors.[6] However, no significant interaction between low socioeconomic status and lifestyle factors in terms of mortality was reported. Additionally, a study[7] of survey data from 23 564 adults from one area of Canada found disproportionate levels of poor health reported by smokers compared with non-smokers in those with low incomes and without employment. Interactions between alcohol intake and socioeconomic status have been investigated more frequently than other lifestyle factors as researchers attempt to explain the well described alcohol paradox, in which similar or lower levels of alcohol consumption in

more deprived groups are associated with disproportionate alcohol-related harm.[21] Annual surveys from 50 236 participants in Scotland linked to mortality registers, hospital admissions, and prescription data have shown that the disproportionate alcohol-related harm associated with more deprived areas was attenuated but remained significant after controlling for body-mass index, smoking status, and drinking patterns.[5] Less evidence exists for an interaction between physical inactivity and low socioeconomic status. In our study, the significant interaction between extended lifestyle score and deprivation suggests that deprived populations are more vulnerable to a wider range of lifestyle factors than previously understood, including emerging lifestyle factors such as television viewing time and sleep duration.

Mechanisms underpinning disproportionate lifestyle-related harm in deprived groups remain unclear. One possible explanation might be a socioeconomic gradient of intensiveness for each lifestyle factor.[8] For example, more deprived participants who smoke might smoke more cigarettes than their affluent counterparts. Another possible explanation includes an interaction between unhealthy lifestyle factors and increased psychosocial stress, leading both directly (via inflammatory mediators) and indirectly (via low mood) to disproportionate cardiovascular pathology.[9] Other purported mechanisms include interactions between unhealthy lifestyle factors and structural factors associated with deprivation, such as poorer access to health care and lack of social support.[10]

The implications of these disproportionately worse outcomes in more deprived individuals, despite a similar, broadly defined lifestyle, are threefold. There should be a renewed call to government-level action on poverty to reduce health inequalities and reverse adverse impacts from austerity measures;[22,23] public health interventions could increase their impact by broadening the range of lifestyle factors targeted while acknowledging social determinants of lifestyle;[24] and individual-level lifestyle interventions could be both broadened to include emerging lifestyle factors and strengthened in areas of deprivation to be proportionate to need.[25]

Although there was a clear trend of increasing risk of cardiovascular disease incidence with least healthy lifestyle category and increasing deprivation, we found no significant interaction between lifestyle and deprivation for cardiovascular disease incidence. There were many more cardiovascular disease incidence events (17 380) than all-cause (5553) and cardiovascular disease (1528) mortality events, and therefore the estimates for cardiovascular disease incidence might be more accurate. However, cardiovascular disease mortality is a composite measure of both cardiovascular disease incidence and survival; because deprivation is associated with both of these outcomes, the modifying effect of deprivation might, as a result, be stronger for mortality than for incidence, which includes both fatal and non-fatal events. An alternative explanation for this non-significant interaction might lie in the socioeconomic status differential in health-care-seeking behaviour, which results in under-reporting of non-fatal cardiovascular disease events in more deprived groups, whereas fatal events are less likely to be under-reported because all deaths are recorded in the UK. Previous qualitative research has shown that individuals from more deprived areas tend not to present to health services in response to chest pain, sometimes because of a perception of health service overuse.[26] Here, the number and proportion of events generally increased with deprivation for all-cause and cardiovascular disease mortality despite fewer participants in more deprived groups (table 3). Although a similar pattern was seen for cardiovascular disease incidence, with the proportion of events increasing with deprivation, the absolute number of events did not increase with deprivation. This could represent under-reporting of non-fatal cardiovascular disease events in more deprived groups. Therefore, the incidence of cardiovascular disease in more deprived groups recorded in our study might be lower than the true incidence, and the resulting association with lifestyle and deprivation might be attenuated.

UK Biobank is a large prospective cohort with linked data for registered deaths and hospital admissions, offering a unique opportunity to explore risks associated with lifestyles and deprivation. The large size of the cohort allowed us to exclude individuals with self-reported non-communicable diseases at baseline as well as those who died within 2 years of recruitment while maintaining sufficient participant numbers and statistical power. These strategies reduce the chance that our results are due to reverse causality (ie, poor health leading to unhealthy lifestyles and adverse outcomes), although the possibility remains in this observational study. We undertook analyses using both area-level and individual-level measures of socioeconomic status, which increases the robustness of our results. Participants with missing covariate data (about 8%) were excluded, which might have influenced results, but excluded participants were broadly similar to those included (appendix). UK Biobank is acknowledged to have a low response rate (5%), with participants being more affluent and more likely to be from a white ethnic background than the UK population as a whole. Consequently, summary statistics such as prevalence cannot be generalised to the wider UK population; however, estimates of the strength of associations can be generalised.[27] Presence of a non-communicable disease at baseline and therefore exclusion from the final survival models was based on self-report; therefore, misreporting might have biased results. However, misreporting might be less likely because diagnosis was based on a physician's diagnosis and was discussed at interview with a trained nurse. Self-reported lifestyle data are prone to healthy reporting bias,[28] and there were much larger numbers of participants in the moderately healthy and most healthy lifestyle categories (table 1; appendix), consistent with a misreporting phenomenon. Misreporting might have affected the

associations described here if there were systematic differences in misreporting between affluent and deprived groups. Although evidence for a socioeconomic gradient of healthy recall bias is scarce, there might be more misreporting of lifestyle behaviours generally among more deprived groups than among affluent groups.[29] Increased misreporting of lifestyle behaviours in deprived groups would flatten the associations between lifestyle and health outcomes among the deprived group, suggesting that the associations identified here would be even stronger with more accurate reporting. The lifestyle score used assumes the same level of risk associated with each component lifestyle factor.[14] However, HRs varied by individual factor (appendix). Applying weighting for the risk associated with each component could produce different results. Moreover, identification of specific high-risk permutations or combinations of lifestyle factors, although not practical here (768 possible permutations for the nine lifestyle factors examined in this study), could help to identify high-risk groups in future research. The manner in which data were collected resulted in crude categories for some factors. For example, alcohol consumption daily or almost daily defined the unhealthy behaviour category, which might erroneously include low-risk drinkers and thereby reduce the association of alcohol consumption with adverse outcomes.

In conclusion, this study is, to our knowledge, the first to highlight the disproportionate risk associated with a broad range of unhealthy lifestyle factors among more deprived socioeconomic groups. If this association is causal, policies to improve a broad range of lifestyle factors among these groups could lead to substantial improvements in health outcomes. This improvement in health outcomes would not only come about because of the higher prevalence of unhealthy lifestyles in deprived areas than in affluent areas but also because those in deprived areas appear to be more vulnerable to the deleterious effects of unhealthy lifestyle factors, including emerging factors such as sleep duration and television viewing time, than those in affluent areas. However, improvement of specific lifestyle factors alone in areas of deprivation is unlikely to reduce health inequalities. On the basis of the increased vulnerability seen in this study, deprived populations would continue to have worse outcomes despite similar levels of unhealthy lifestyle factors seen in affluent populations. This finding clearly strengthens the arguments for government policies that tackle upstream determinants of ill-health and aim to reduce poverty, and for health policies that offer increased support in areas of deprivation.[22,23,25] Public health and individual-level interventions should aim to improve wider combinations of lifestyle factors but need to recognise and address the social and environmental determinants of lifestyle to avoid increasing health inequalities.[30] Further research is required to understand the mechanisms behind the interaction between lifestyle and deprivation, as well as to identify those combinations of unhealthy lifestyle factors that incur the greatest risk across socioeconomic groups.

Contributors

HMEF, CAC-M, JMRG, BIN, CAO'D, and FSM were involved in study concept and design. HMEF, CAC-M, JMRG, BIN, CAO'D, JPP, and FSM were involved in acquisition, analysis, or interpretation of data. Drafting of the manuscript was led by HMEF and CAC-M, with support from JMRG, BIN, CAO'D, and FSM. All authors were involved in critical revision of the manuscript for important intellectual content. HMEF and CAC-M did the statistical analysis, with support from JMRG, BIN, CAO'D, and FSM; FP-R tabulated the data. Administrative, technical, or material support was provided by HMEF, CAC-M, FP-R, JMRG, BIN, CAO'D, and FSM. CAC-M, JMRG, CAO'D, and FSM supervised the study. FSM is the guarantor.

Declaration of interests

We declare no competing interests.

Data sharing

UK Biobank data are available via www.ukbiobank.ac.uk. Syntax for the generation of derived variables and for the analysis used for this study will be submitted to UK Biobank for record.

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