

Best-practice interventions to reduce socioeconomic inequalities of coronary heart disease mortality in UK: a prospective occupational cohort study

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Summary

Background How much the successful implementation of the most effective (ie, best-practice) interventions could reduce socioeconomic inequalities of coronary heart disease mortality is not known. We assessed this issue in an occupational cohort study comparing low with high socioeconomic groups.

Methods We undertook a prospective cohort study on 17 186 male civil servants aged 40–69 years between 1967 and 1970 in the UK (the Whitehall study). Socioeconomic position was based on employment grade. We compared the potential reduction in excess coronary heart disease mortality in men of low with those of high socioeconomic position with either best-practice interventions (reduction of systolic blood pressure by 10 mm Hg, of total cholesterol by 2 mmol/L, and of blood glucose by 1 mmol/L in pre-diabetic people; halving the prevalence of non-insulin-dependent diabetes; and complete cessation of cigarette smoking) or primordial prevention.

Findings 15-year absolute risk of death due to coronary heart disease per 100 men, standardised to age 55 years, was 11·0 for men in the low employment grade group and 7·5 for those in the high grade group. Population-wide best-practice interventions would reduce coronary heart disease mortality by 57%, and the difference in mortality between socioeconomic groups by 69%. For primordial prevention, the corresponding reductions would be 73% and 86%, respectively.

Interpretation Our results suggest that current best-practice interventions to reduce classic coronary risk factors, if successfully implemented in both high and low socioeconomic groups, could eliminate most of the socioeconomic differences in coronary heart disease mortality. Modest further benefits would result if the classic coronary risk factors could be reduced to primordial levels for the whole population.

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Introduction

The most effective ways to reduce socioeconomic inequalities in coronary heart disease—a leading cause of death—are not known.^{1–3} Studies have assessed the contribution of various risk factors to socioeconomic inequalities by comparing the relative risk of coronary heart disease between high and low socioeconomic groups before and after adjustment for these risk factors.^{4–7} The drawback of this method is that it does not take into account the extent to which reduction in each risk factor is feasible because it simply approximates what would happen after standardisation to the risk-factor distribution in the total study population.

Classic, well-established risk factors of coronary heart disease are high blood pressure, high cholesterol, diabetes mellitus, and cigarette smoking.^{8–10} The most successful (ie, best-practice) pharmacological and lifestyle interventions to date have reduced systolic blood pressure by about 10 mm Hg,¹¹ total cholesterol by 2 mmol/L,¹² blood glucose in pre-diabetic people by 1 mmol/L, and have halved the prevalence of non-insulin-dependent diabetes mellitus (non-IDDM) in adults.¹³ With existing

databases, the extent to which successful implementation of these best-practice interventions could reduce socioeconomic inequalities in coronary heart disease at the population level can be estimated.^{9,14}

Here, we assessed—both in absolute and relative terms—what would happen to socioeconomic inequalities in coronary heart disease if these best-practice risk reductions applied to both high and low socioeconomic groups. We also measured potential risk reduction if all smokers quit. New aggressive anti-smoking policies and reduction in smoking from 82% to 26% among men in the UK over the past 60 years gave at least an approximation to this scenario that is more plausible than envisaged.¹⁵

We also assessed the effects on socioeconomic inequalities of a primordial prevention strategy based on risk-factor levels below those currently observed in developed societies. The increase in blood pressure with age is not physiologically inevitable,¹⁶ and diabetes prevalence is low in the absence of diets and obesity rates that are typical of developed countries.^{17,18} Apart from a small proportion of people with a strong genetic predisposition to hypercholesterolaemia, cholesterol

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	High employment grade (N=12 644)	Low employment grade (N=4542)	Difference (low grade–high grade)	p value
Age (years)	50.9 (0.1)	55.5 (0.1)	4.6	<0.001
Systolic blood pressure (mm Hg)	136.3 (0.2)	137.3 (0.3)	1.0	0.01
Total cholesterol (mmol/L)	5.12 (0.01)	5.02 (0.02)	–0.10	<0.001
IDDM	0.3% (32)	0.2% (9)	–0.1%	0.55
Non-IDDM	0.7% (82)	1.6% (91)	0.9%	<0.001
Post-load blood glucose for non-diabetics (mmol/L)	4.15 (0.01)	4.20 (0.01)	0.05	<0.001
Current smoking	36.7% (4625)	56.0% (2550)	19.3%	<0.001
Low-risk group*	1.8% (244)	1.1% (34)	–0.7%	0.003
15-year mortality rate†	4.91 (748)	7.06 (514)	2.15	<0.001

Data are mean (SE) or percentage (number). IDDM=insulin-dependent diabetes mellitus. Non-IDDM=non-insulin-dependent diabetes mellitus. *Blood pressure of 120 mmHg or less; cholesterol concentration of 5.0 mmol/L or less; post-load blood glucose of 4.6 mmol/L or less; and never smoked. †Age-standardised coronary heart disease mortality rate per 1000 person-years (number of deaths).

Table 1: Age-adjusted risk factors and coronary heart disease mortality by employment grade

concentrations could be kept low throughout life,¹⁹ and tobacco use could be eliminated. We therefore quantified risk reduction associated with systolic blood pressure of 120 mmHg or less, cholesterol concentration of 5.0 mmol/L or less, post-load blood glucose of 4.6 mmol/L or less,²⁰ and no smoking on socioeconomic inequalities in coronary heart disease.

Methods

Study population

We examined 19 019 non-industrial London-based male government employees aged 40–69 years between Sept 14, 1967, and Jan 15, 1970 (response rate 74%).²¹ Socioeconomic position was based on civil-service employment grade. To obtain sufficient numbers for each socioeconomic group and to simplify data interpretation, we dichotomised employment grade into high (administrative, professional, or executive) and low (clerical, messenger, and other unskilled manual grades). 886 men from the diplomatic service and the British Council could not be given a comparable employment grade; therefore, these individuals were excluded from all analyses.

Procedures

A trained observer took a single blood-pressure reading from the participant's left arm with a London School of Hygiene sphygmomanometer.²² We recorded systolic blood pressure at the first appearance of the Korotkoff sounds. We took a capillary blood sample from the earlobe after an overnight fast. Participants then drank a 50-g anhydrous dextrose preparation. We measured plasma cholesterol concentration with the standard Technicon method N24a,²¹ and we took a second blood sample 2 h later. We measured post-load blood glucose concentration by the ferricyanide reduction method on an autoanalyser (Technicon method N-9a, NY, USA). Those with self-reported diabetes did not undertake the glucose-tolerance test and those with a post-load blood

glucose of 11.1 mmol/L or more were categorised as non-IDDM patients. Diabetic participants on insulin treatment were classified as insulin-dependent diabetes mellitus (IDDM) patients. Smoking status was self-reported.

For 99% of men, 15-year mortality data were obtained from the National Health Service (NHS) Central Register with the NHS identification number assigned to each citizen in the UK. Because these records do not cover deaths of people who emigrated from the UK and died abroad, participants flagged as embarkees were censored at the date of embarkation. Death due to coronary heart disease was indicated by codes 410–414 (International Classification of Diseases [ICD]-8 and ICD-9) and codes I20–I25 (ICD-10).

We excluded from the analysis men who had any missing values for the risk factors of interest—systolic blood pressure (6), cholesterol (709), smoking status (8), blood glucose or diabetes (135). Difficulties in retrieving a sufficient sample of blood with the capillary method from an incision in the earlobe may have contributed to the high rate of missing values for cholesterol (3.7%), but this issue existed for both socioeconomic groups ($p=0.38$). We also excluded 16 men who had died but for whom the cause of death was unknown. Overall, 17 186 men were included in the analysis.

Statistical analysis

We chose a follow-up of 15 years to keep the possible dilution of effects associated with extended mortality surveillance to a minimum and to cover a period when statins had yet to become a commonly prescribed treatment for high cholesterol.^{21,23} In analyses of baseline characteristics and mortality, prevalence of risk factors and 15-year coronary heart disease mortality rates between men in the high and those in the low employment grade were adjusted for age (with 5-year age groups) by the direct standardisation method, and compared with the Mantel–Haenszel test. For continuous measures, we used

linear regression to calculate age-adjusted least square means, and tested differences between grades with the dichotomous grade term.

We estimated the relation between risk factors and coronary heart disease mortality with the parametric Weibull distribution to model the survival function. With this model, hazard ratios (HRs) associated with each risk factor and their 95% CIs were calculated for 10 mm Hg increase for systolic blood pressure, for 2 mmol/L increase for total cholesterol, and for current smokers versus ex-smokers or non-smokers. Due to the threshold effect of glucose on coronary heart disease mortality observed previously in this study,²⁰ we calculated the HR for 1 mmol/L increase for post-load blood glucose concentrations above 4.6 mmol/L, and the HR for individuals with IDDM and non-IDDM compared with those with blood glucose concentrations below 4.6 mmol/L. We adjusted for age by both a linear and quadratic term, and included a dichotomous term for employment grade. All the HRs and CIs were almost identical to those computed with a Cox proportional hazards model. With the Weibull model, the risk of death from coronary heart disease over any period can be calculated as follows:

$$p(t)=1-\exp\left(-\exp\frac{\log(t)-\chi\beta}{\sigma}\right)$$

where χ is the vector of risk factors, β is the vector of coefficients, and σ is a scale parameter, which is also estimated. This model has previously been used in the Framingham study to describe risk profiles and the effects of risk factors on coronary outcomes through the Framingham risk score.²⁴

For each person in the analysis, we used the above equation to calculate the risk of coronary heart disease on the basis of his observed risk factors, with age between 40 and 55 years. These risks were summed up for all individuals and tabulated by employment grade to give the baseline expected number of deaths before any intervention. To estimate the effect on risk of each

risk-factor intervention, we applied the intervention to each individual and recomputed the risk of coronary heart disease with the risk equation. To estimate the effect of a 50% reduction in non-IDDM prevalence, we randomly gave 50% of non-IDDM men the non-diabetic status when calculating the risk of coronary heart disease. We repeated this random allocation five times and averaged the expected risk. Sensitivity analyses constraining the age to either 50 or 60 years changed the risk of coronary heart disease, but had little effect on the percentage changes in excess risk or the risk ratio between low and high employment grade. Separate equations to measure the effects of best-practice interventions for each specific risk factor on coronary heart disease mortality yielded results similar to those based on a model with all risk factors included simultaneously. Thus, we only reported the latter analysis.

Primordial prevention aims to change the conditions that determine risk-factor development, thus successful prevention will eliminate the risk factors themselves. We modelled the effect of such prevention by applying the best-practice intervention to all individuals, irrespective of their risk levels, and additionally setting a low risk profile (ie, systolic blood pressure ≤ 120 mm Hg, total cholesterol ≤ 5.0 mmol/L, post-load blood glucose ≤ 4.6 mmol/L, no non-IDDM, and never smoked) for those who had residual non-optimal risk levels or were ex-smokers after the best-practice interventions (IDDM remained unchanged). In all other respects, the modelling corresponds to that described for the best-practice interventions.

Role of the funding source

The study sponsors did not contribute to the study design and had no role in data collection, data analysis, data interpretation, or writing of the report. The corresponding author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

	HR* (95% CI) for 15-year CHD mortality	Best-practice intervention		Primordial prevention	
		Effect	Reduction in risk of CHD mortality	Post-prevention	Reduction in risk of CHD mortality
Systolic blood pressure	1.18 (1.15–1.20)	↓ 10 mm Hg	14%	≤ 120 mm Hg	32%
Total cholesterol	1.51 (1.39–1.65)	↓ 2 mmol/L	33%	≤ 5 mmol/L	33%
IDDM vs glucose reference†	1.79 (0.74–4.32)	No change in IDDM	0%	No change in IDDM	0%
Non-IDDM vs glucose reference†	2.55 (1.84–3.54)	↓ 50% prevalence		No non-IDDM	
Post-load blood glucose, per unit increase above 4.6 mmol/L	1.14 (1.03–1.25)	↓ 1 mmol/L post-load glucose for non-diabetics	2%‡	≤ 4.6 mmol/L	3%‡
Current smoking	1.78 (1.59–2.00)	Smoking cessation	23%	Never smoked	37%
All risk factors	..	All above	57%	All above	73%

CHD=coronary heart disease. HR=hazard ratio. IDDM=insulin-dependent diabetes mellitus. Non-IDDM=non-insulin-dependent diabetes mellitus. ↓=reduction. *Hazard ratios are mutually adjusted, and also adjusted for age and employment grade. †Reference group includes those with blood glucose concentration of 4.6 mmol/L or less. ‡Combined effect of interventions targeting non-IDDM and high post-load blood glucose.

Table 2: Best-practice interventions and primordial prevention effects on coronary heart disease mortality

	15-year risk* of CHD mortality		Excess risk	Reduction in excess risk	Relative risk ratio	Change in relative risk ratio
	High employment grade	Low employment grade				
Best-practice intervention						
None	7.5	11.0	3.5	Reference	1.458	Reference
Systolic blood pressure (↓ 10 mm Hg)	6.4	9.4	3.0	13%	1.465	2%
Total cholesterol (↓ 2 mmol/L)	5.1	7.5	2.4	31%	1.473	3%
IDDM prevalence (no change)	7.5	11.0	3.5	0%	1.458	0%
Non IDDM prevalence (↓ 50%)	7.4	10.7	3.3	6%	1.441	-4%
Non-diabetics (↓ 1 mmol/L glucose†)						
Smoking cessation	6.0	7.9	2.0	43%	1.331	-28%
All above	3.3	4.4	1.1	69%	1.323	-30%
Primordial prevention						
None	7.8	11.5	3.8	Reference	1.486	Reference
Systolic blood pressure to ≤120 mm Hg	5.4	7.5	2.2	43%	1.402	-17%
Total cholesterol to ≤5.0 mmol/L	5.2	7.8	2.6	31%	1.504	4%
Non IDDM (no change)	7.8	11.5	3.8	0%	1.486	0%
No non-IDDM and post-load glucose to ≤4.6 mmol/L glucose†	7.6	11.0	3.4	9%	1.453	-7%
Never smoked	5.1	6.9	1.8	52%	1.356	-27%
All above	2.2	2.7	0.5	86%	1.231	-53%

CHD=coronary heart disease. IDDM=insulin-dependent diabetes mellitus. Non-IDDM=non-insulin-dependent diabetes mellitus. ↓=reduction. *15-year risk of CHD mortality per 100 men, adjusted to age 55 years. †Combined effect of interventions targeting non-IDDM and high post-load blood glucose.

Table 3: Best-practice interventions and primordial prevention effects on excess risk and relative risk of coronary heart disease mortality for low versus high employment grades

Results

Men with low employment grades generally had a less favourable risk profile than those with high grades (table 1). Systolic blood pressure, glucose, and proportions of current smokers and non-IDDM patients were higher in the low employment grade group than in the high grade group. However, total cholesterol concentrations were slightly greater in the high grade group than in the low grade group.

During the 15-year follow-up, 1262 men died from coronary heart disease. As expected, low employment grade and all risk factors were associated with high mortality (table 2). The inclusion of best-practice interventions in the model resulted in an overall decrease of more than 50% in the risk of coronary heart disease mortality at age 55 years (table 2).

Furthermore, the primordial prevention model suggests that removing all risk factors from the study population reduces mortality by nearly three-quarters (table 2).

Table 3 shows 15-year risk of death at age 55 years for 100 men in the low employment grades and for 100 men in the high grades, and the risk difference between groups. All best-practice interventions lowered the 15-year risk of mortality in both groups (table 3). In relative terms, the ratio of 15-year coronary heart disease mortality risk between the high and low employment grade groups is 1.46, and the corresponding post-

intervention risk ratio is 1.32 (table 3). The overall reduction in the risk ratio was almost entirely attributable to the effect of smoking cessation (table 3). Because employment grade differences in the prevalence of the other coronary risk factors were small, best-practice interventions that reduced these risk factors had little effect on the relative risk associated with low socioeconomic position.

Interventions that reduced all the outstanding high levels of risk and transformed them into low levels, and completely eliminated non-IDDM and smoking (ie, all men regarded as non-smokers) further reduced the population systolic blood pressure to 115.0 mm Hg, cholesterol to 3.1 mmol/L, post-load glucose in non-diabetics to 4.0 mmol/L, and did not expose to cigarette smoking. The 15-year coronary heart disease mortality risk under these circumstances is 2.2 for 100 men of the high grade employment group and 2.7 for 100 men of the low grade group (table 3). For the relative risk, smoking and blood pressure were the most important contributing factors.

Discussion

Our aim was to assess the extent to which the successful implementation of best-practice interventions to modify coronary heart disease risk factors could reduce socioeconomic inequalities in mortality at a population

level. According to estimates from the population of the Whitehall study,²¹ best-practice interventions to reduce classic coronary risk factors, if successfully implemented in both high and low socioeconomic groups, could eliminate 69% of the difference in risk between these groups. Reduction of cholesterol concentration and smoking cessation account for the largest part of the change. Primordial prevention would confer only modest additional benefits. In relative terms, best-practice interventions and primordial prevention were not very successful in reducing relative inequalities in coronary heart disease between socioeconomic groups.

Numerous population-based cohort studies have assessed the contribution of the classic risk factors to relative socioeconomic inequalities, ignoring the contribution to absolute differences in coronary heart disease between socioeconomic groups. Our quantification suggests that interventions that caused the greatest reduction in coronary heart disease in randomised controlled trials (best-practice interventions) decrease the relative difference in coronary heart disease between high and low socioeconomic groups only by 30%. Previous studies with adjustment-based approaches suggested a similar reduction of 15% to 40% in relative risk.^{5,7} The adjustment-based method differs from ours in that it approximates what would happen after standardisation to the risk-factor distribution in the total study population, irrespective of whether this is possible.

Smoking cessation made the greatest contribution to both relative and absolute risk difference in coronary heart disease between high and low socioeconomic groups. Reductions in the other risk factors had only a modest effect on relative inequalities. Although smoking cessation for all smokers is the ultimate aim of all health policies, this may seem a very optimistic goal at a population level. Re-calculation of the overall best-practice intervention effect with smoking prevalence only halved suggests a 55% (rather than 69%) drop in mortality differences between high and low employment grades and a 12% (rather than 30%) reduction in the relative risk. In this scenario, total cholesterol reduction becomes the most important factor to lower absolute socioeconomic disparities in coronary heart disease mortality.

The relative approach to socioeconomic inequalities is only sensitive to reductions in risk factors that are socially patterned. Because cholesterol concentrations were not higher in low than in high socioeconomic groups, cholesterol reduction did not decrease relative socioeconomic inequalities in coronary heart disease mortality, despite substantially lowering mortality difference between socioeconomic groups. This result shows that the determinants of relative socioeconomic inequalities may differ from those of absolute differences.⁵

The proportion of people who had low risk-factor levels was small (<2% in our cohort). In other studies, with less stringent criteria, this proportion was 15% in a Finnish sample⁵ and 5% in a multiracial study of men in the US

Atherosclerosis Risk in Communities study.⁹ In a study in Norway—one of the healthiest nations in the world—about 86% of men at age 40 years had some high risk-factor levels and none had low levels in all risk factors, according to the 2003 European guidelines criteria.²⁵

Although no evidence-based strategies or ways to eliminate completely the classic coronary risk factors from the population currently exist, this may not be the case in the future. In addition to best-practice prevention, interventions that target upstream risk factors—such as psychosocial and material circumstances in which people live and work—could be effective in reducing relative inequalities.^{26–28} Lifetime risk-factor prevention is regarded as key to lowering coronary heart disease mortality and reducing absolute differences between socioeconomic groups.²⁹ These two prevention strategies are not mutually exclusive. For example, attempts to reduce smoking through fiscal policy will both prevent initiation of smoking (and ultimately might eliminate smoking) and reduce absolute differences in smoking between different social groups (even if relative differences increase).

We suggest that any prevention programme that completely eliminates the four classic coronary risk factors from the population would reduce coronary heart disease mortality by 73% and absolute socioeconomic disparities by 86%. True primordial prevention strategies may have greater effect than we have suggested here, because for several factors we only categorised risk according to measures taken in adult life. We do not have estimates of risk associated with lifetime low concentrations of circulating cholesterol, which will be lower than those associated with low concentrations in only one period of life. Studies^{30,31} that have assessed genetic variants associated with different lifetime concentrations of cholesterol have shown that reductions in coronary heart disease risk in randomised controlled trials of short (<10 years) duration and in observational studies initiated when study participants were middle-aged underestimated the absolute differences in coronary-heart disease rates associated with long-term differences in cholesterol concentrations. Similar results apply to other classic risk factors.

In isolated, non-developed populations with a non-sedentary lifestyle and a low-fat diet free of added salt and refined carbohydrate—such as the Yanomamo Indians—average systolic blood pressure is about 100 mm Hg, total cholesterol 3.1 mmol/L, and cardiovascular death is rare.^{19,32} Thus, we may be underestimating the potential of true primordial prevention in our analyses. A caveat that would affect estimates in the opposite direction, but probably to a lesser extent, is that a small proportion of individuals with high genetically-related increases in cholesterol, glucose, or blood pressure^{33–36} would have levels above those used as our cut-off points.

The validity of our estimates depends on some assumptions and the data used. Instead of risk-factor

categories, we used continuous measures of blood pressure, cholesterol, and blood glucose, which may have improved precision. Our analyses of possible risk-factor reductions were based on the effects of best-practice interventions derived from randomised controlled trials or, where possible, meta-analyses of such trials—ie, the strongest possible evidence.^{11–13} This approach may provide a more realistic estimation for policy than that based on risk-factor standardisation or comparisons of exposed and unexposed groups. Differential treatment between socioeconomic groups is an unlikely source of bias in this study because treatment for blood pressure was rare (1.7% in both high and low grade groups) and effective cholesterol-lowering interventions had yet to become available.²¹ Prevalence of risk factors and associated excess risks of coronary heart disease from the same population (rather than from different populations) is also a strength of the study.

However, our study is not without limitations. First, the findings are based on data from an occupational cohort of men and results may not be valid for women or non-working people. Nonetheless, differences between the participants and British men aged 45–64 in 2003 were small for total cholesterol (5.1 mmol/L vs 5.5 mmol/L) and systolic blood pressure (137 mm Hg vs 135 mm Hg),¹⁵ and there is little evidence of differences in the effects on cardiovascular disease of the studied risk factors between men and women, except for greater diabetes-related risk of fatal coronary heart disease in women than in men.^{37,38} Prevalence of smoking was high in this study population compared with that of present populations, and is likely to overemphasise the role of smoking in the creation of absolute socioeconomic inequalities.

Second, we estimated the benefits from interventions for each risk factor separately while controlling for others. However, we did not take into account the potential interaction effects between different risk reductions because comparable clinical evidence relates to single-factor rather than multiple-factor interventions. Our predictive models could not capture lag effects or time trends in risk factors and coronary heart disease mortality.³⁹ Furthermore, we estimated the benefits of best-practice risk factor reductions in terms of lower coronary heart disease mortality from observational data that are open to confounding.

Despite these potential limitations, our estimates of intervention effects did not substantially deviate from those reported for coronary heart disease morbidity and mortality in clinical trials—eg, 33% reduction in coronary heart disease from lowering cholesterol in this study versus 20% to 36% reductions observed in lipid lowering trials,⁴⁰ and 15% reduction for lowering systolic blood pressure in our analysis versus 10% to 20% reductions in relative risk of cardiovascular events in blood-pressure lowering trials.⁴¹ Although observational data suggest that these reductions depend on age, trials provided little

evidence of any age-specific effects, supporting our analysis in which we combined all age groups.^{40,41} With lifetime reductions in these risk factors, greater effects than those predicted in our observational data or seen in short-term randomised controlled trials would be expected.

Finally, we assumed that best-practice interventions are implemented equitably and have similar effectiveness in both high and low socioeconomic groups. This goal is a major challenge for policy because there are many issues to realise the full potential of interventions in all groups, which include unhealthy environments that are not conducive to healthy living, together with restricted financial and health-care resources. These constraints disproportionately affect low socioeconomic groups.³

This study provides an example of a novel way of quantifying socioeconomic inequalities in health. Although the aim of health practitioners and governments is to reduce the classic coronary risk factors and eliminate cigarette smoking, population-wide comprehensive screening and best-practice interventions still need to be implemented. We estimated from a working population that such interventions, if implemented equally successfully in high and low socioeconomic groups, would eliminate most of the socioeconomic inequalities of coronary heart disease.

Contributors

MK, GDS, ASM, and MJS designed the original hypothesis, which other authors further elaborated. MJS, MK, and GDS ran all the analyses. MK wrote the first draft of the report, apart from the statistical section, which was written by MJS. All authors interpreted the results, revised the report, and approved the final draft of the report.

Conflict of interest statement

We declare that we have no conflict of interest.

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