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# Do the Poor Gain More? The Impact of Secondary-Care Expenditure on Health Inequality

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## Abstract

**Background** Quasi-experimental studies of mortality variation and trends among large administrative areas of England in the 2000s and early 2010s have suggested that more deprived populations gain larger mortality benefits from marginal increases in public expenditure on secondary care.

**Objective** To identify causal effects of marginal changes in expenditure on mortality variation in 2018 among 32,784 more and less deprived small areas of England, with a mean population of 1700, allowing more fine-grained measurements of deprivation and mortality.

**Methods** We used cross-sectional data on secondary-care funding allocated to 195 National Health Service administrative areas in England in 2018/19 and employed a well-established instrumental variable approach based on the “distance from target” component of the funding formula, which generates quasi-exogenous variation in funding based on historical factors unrelated to current need for secondary care.

**Results** We found an inverted U-shape pattern of mortality gains by deprivation group, whereby the middle group gained significantly more than others. However, we could not reject the null hypothesis that the two more deprived groups received the same mortality gain as the two less deprived groups. These findings were robust to extensive sensitivity analysis using different levels of analysis, control variables, mortality outcomes, functional forms, first-stage regression specifications, and exclusions, and our preferred specifications all satisfied standard instrumental variable diagnostic tests.

**Conclusions** We found that the poor do not always gain more from marginal increases in public expenditure on secondary care and, conversely, might not always bear the largest share of the health opportunity costs of cost-increasing programmes.

## Key Points for Decision Makers

Large-area level studies of England in the 2000s found a “pro-poor” pattern whereby more deprived groups gained larger mortality benefits from increased hospital expenditure than less deprived ones.

Our small-area level study of England in 2018 found a “pro-middle” pattern of mortality gains, with no sign that more deprived groups gained more than less deprived groups.

Increasing hospital expenditure may not reduce health inequality, and the social distribution of the health opportunity cost of healthcare interventions may not be “pro-poor”.

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## 1 Introduction

Policy concern about health inequality is increasingly prominent following the COVID-19 crisis [1]. However, although numerous studies have examined the overall impact of marginal changes in health spending on mortality [2, 3], less is known about the health inequality impact, i.e., how the overall impact varies between more and less socially advantaged groups [4].

This paper uses applied health econometric methods to address this issue, which is relevant to policy questions about how health inequality considerations should inform (1) priority-setting decisions about the funding and delivery of specific health interventions [5], (2) geographical resource allocation between sub-national areas [6], and (3) total national healthcare expenditure decisions [7]. The study is timely, since in 2025 the National Institute for Health and Care Excellence (NICE) announced that it will begin using Distributional Cost-Effectiveness Analysis (DCEA) in technology evaluation, allowing manufacturers to submit evidence about the magnitude of health inequality impact and potentially altering its recommendations if the impact is considered to be substantial [8]. An important building block of DCEA is the assumed social distribution of health opportunity cost. Our findings provide new empirical evidence that challenges the previous assumption that poorer groups bear a disproportionately large share of forgone health, supporting instead a flat distributional assumption. This study therefore has important implications for the estimated health inequality impacts of new technologies, as well as for decision making more generally.

There is a growing international literature linking variation in health expenditure and mortality across large sub-national administrative areas, including cross-sectional instrumental variable studies in Australia, Sweden and England and panel data studies in Spain, the Netherlands and South Africa [9]. England has been examined in particular detail in this literature, exploiting variation in health expenditure among many sub-national administrative areas—usually about 150 or more—with two landmark early studies published in 2008 [10] and 2015 [11] and several further studies published subsequently [12–17]. Early studies of England used census-based socio-economic variables to instrument sub-national variation in expenditure, but the well-established current approach following a methodological study published in 2017 is to use “funding rule” instruments which exploit quasi-exogenous variations in expenditure due to quirks in the way that funding is allocated to sub-national areas [18]. Findings have been broadly comparable between all instrumental variable studies of England, including the early studies:

estimated all-age all-cause mortality elasticities typically range from about  $-0.5$  to  $-1.5$ , with elasticities consistently above this in some disease areas (e.g., circulatory disease) and below in others (e.g., cancer) [12, 14]. Estimates have been found to be robust to changes in geography, identification strategy, sensitivity analysis of the validity of instruments used for identification, and comparison of disease area mortality with all-cause mortality [19]. Effects are also comparable between total health-care expenditure and secondary-care expenditure, though the effect of public health expenditure (i.e., preventive services beyond healthcare) is found to be considerably greater per pound spent [20]. Estimated mortality elasticities for England using instrumental variable methods are generally higher than those found in the large number of studies that do not control for endogeneity bias, which includes most cross-country studies and most studies in low- and middle-income countries [2, 3].

However, only a handful of studies have examined how mortality effects vary between more and less socially advantaged groups. One study used an indirect approach that combined data on overall inpatient hospital utilisation by neighbourhood deprivation with previous estimates of mortality effects by broad disease categories [21]. Two studies used a large-area instrumental variable approach and navigated the challenges of sample size using quantile regression [15, 22]. Finally, two studies used a time series approach that examined changing patterns of expenditure and mortality across large administrative areas with different levels of deprivation [23, 24]. All of these studies, conducted in England, concluded that the mortality effect per unit of marginal healthcare expenditure is larger in more deprived (or higher mortality) large-area populations—implying that marginal increases in healthcare expenditure will tend to reduce health inequality. However, these studies were all vulnerable to potential biases that we address in this study, as explained below and in the Discussion, Sect. 5, and were all conducted in the 2000s and 2010s, prior to the sustained period of deterioration in the UK economy and public services during the 2010s. Furthermore, the quasi-experimental studies only looked at variations between large areas with heterogeneous populations, which risks masking important effects on health inequality within those areas [25, 26]. Even if the large-area findings were correct, it does not necessarily follow that deprived small areas benefited more from increases in healthcare expenditure than affluent small areas. Most health inequality occurs within rather than between large administrative areas, and so studies that do not look at small-area inequalities potentially miss the most important part of the picture [25].

This is the first study to estimate the causal effect of healthcare expenditure on social inequality in health using quasi-experimental methods based on small-area level data

on deprivation and mortality. We use the same well-established instrumental variable approach used in numerous studies of the overall mortality effects of health expenditure in England but innovate by estimating sub-group differences by deprivation and mortality at the small-area level as well as the large-area level. We also use more robust methods than previous large-area quasi-experimental studies of the social gradient in mortality effects by: (1) focusing directly on healthcare expenditure, unlike previous time-series studies which risk confounding by non-healthcare expenditure trends; (2) focusing directly on deprivation quintiles rather than mortality quintiles, unlike previous large-area quantile regression studies; (3) using actual NHS expenditure geography rather than indirect mapping to local authority geographies; and (4) following Brindley et al. [27] in using only a single funding rule instrument, known as the “Distance from Target Index”, which generates quasi-exogenous variation in funding based on historical factors unrelated to the need for secondary care expenditure. We believe that this is a more robust instrumental variable approach than those used in the two previous studies of this kind, one of which used census-based socioeconomic variables as instruments [21] and one of which inappropriately used funding rule components related to the need for secondary care as instruments rather than control variables [15]. As explained in detail in the Methods, Section 4.1, we believe that need factors should be treated as controls rather than instruments, since they are causally linked to the outcome (mortality) as well as to the exposure (expenditure). We also report extensive diagnostic tests and sensitivity analysis.

We use a small-area level of analysis for mortality outcomes and a large-area level of analysis for health expenditure inputs. The large-area level is appropriate for analysing expenditure, because this is the level at which expenditure is actually allocated within the English NHS. The small-area level is more appropriate for analysing mortality effects by deprivation group, however, because this is the outcome of interest from a health inequality perspective. We focus on secondary-care expenditure (which we sometimes refer to as “hospital” expenditure, for short) because this is by far the largest component of health expenditure in England [27] and may potentially have more immediate effects on mortality than primary-care and public health expenditure focused on long-term prevention.

## 2 Conceptual Framework

This section describes the main causal mechanisms that might plausibly generate differential mortality effects of marginal secondary-care expenditure among different social advantage groups. In line with previous empirical findings, our tentative prior hypothesis was a “pro-poor”

gradient—that is, a monotonic positive relationship between mortality effect and deprivation, with larger mortality effects in more deprived groups. A causal mechanism that would generate this pattern is diminishing marginal returns to additional expenditure on secondary care relative to need. In high-income countries with universal health systems, a “disproportionate care law” operates whereby socially disadvantaged people tend to receive a smaller proportion of the secondary care they need than socially advantaged people, especially in terms of elective care [4]. Socially advantaged people thus receive more “need-adjusted” expenditure than socially disadvantaged people, where “need-adjusted” expenditure is the proportion of needed expenditure received multiplied by mean per capita expenditure. If there are diminishing marginal returns to “need-adjusted” expenditure, then the same increase in mean per capita expenditure will produce a smaller mortality gain for socially advantaged people than socially disadvantaged people.

However, three other mechanisms could generate a more complex pattern of effects. First, there may be a “sharp elbows” mechanism that influences how increases in the mean per capita secondary-care funding available to a large area are shared out among individual patients living within that large area. Most of the additional expenditure is likely to go on elective outpatient and inpatient activity, and emergency activity might conceivably even fall if emergencies are prevented. Yet socially disadvantaged people may be less adept than socially advantaged people at seeking and demanding secondary-care activity. Second, there may be a “crowding out” mechanism whereby public expenditure crowds out private expenditure on secondary care for the most socially advantaged, having little impact on their overall (public plus private) expenditure and outcomes. For instance, elective procedures such as hip replacements are more likely to be financed privately by wealthier individuals when NHS waiting times are long; conversely, higher public expenditure that reduces waiting times may lessen their need to seek treatment privately. Third, there may be a “co-morbidity and co-investment” mechanism influencing the long-term health benefits of healthcare expenditure, whereby disadvantaged populations have worse health outcomes per additional unit of publicly funded secondary-care expenditure than affluent populations. This is because socially disadvantaged populations tend to have greater co-morbidity and lesser ability to co-invest their own resources in health improvement alongside the publicly funded healthcare inputs, for example by adhering to medication and rehabilitation regimes, providing healthy living and working conditions conducive to recovery, helping to share information and coordinate healthcare inputs provided by different healthcare staff, and topping up publicly funded care where necessary through privately funded care [4].

There is currently no well-developed theory about what patterns to expect in what contexts, so our aim was to examine the effects empirically in the context of England in 2018.

### 3 Data

#### 3.1 Secondary-Care Funding Allocations Data

We used data on healthcare funding allocations in the financial year 2018/19 [28] to 195 NHS administrative areas in England, known as “Clinical Commissioning Groups” (CCGs), with responsibility for purchasing and planning secondary care for their local populations; data were accessed from <https://www.england.nhs.uk/allocations/> (accessed 20 April 2023). We focused on the main funding stream, known as “Core Allocations”, of which approximately two-thirds is assigned to general and acute hospital care [29]. We excluded two separate funding streams for “Primary Care” and “Specialised Services”; the latter involves unusual conditions with high costs for individual patients and few providers. In 2018/19 “Core Allocations” represented 75% of CCG funding, while “Primary Care” and “Specialised Services” accounted for 8% and 17%, respectively [27].

#### 3.2 Population and Mortality Data

We used Office for National Statistics (ONS) mid-year population estimates for 2018 and death counts by sex, age (in 5-year bands from 0–4 to 90+ years) and 2011 Lower Super Output Area (LSOA) for calendar year 2018 in England, both available on public websites [30, 31] (accessed 30 March 2022). 2011 LSOAs are small areas with a mean population of about 1700 and a minimum threshold population of 1000. Our full sample of 32,844 LSOAs covered a population of 55,977,178 with 513,422 deaths recorded.

We linked these data with the 2019 English Indices of Deprivation, which measure relative deprivation across English LSOAs [32], from the Ministry of Housing Communities and Local Government (accessed 20 March 2022) (see Appendix Note A1 (Electronic Supplementary Material, ESM) on why small-area deprivation is commonly used to measure health inequality in England). We use the overall index at LSOA level to construct deprivation quintile groups. This distinguishes our work from the existing literature [15, 21–24], which uses a large-area (CCG-level) measure of deprivation. We linked LSOAs with their corresponding CCGs using official NHS technical guide codes for CCG boundaries for the 2018/19 financial year [33].

This small-area stratification allowed us to measure the effect of large-area funding allocation on mortality across different small-area deprivation groups, regardless of whether they reside in an affluent or deprived large area

CCG. Figure 1 illustrates the difference between previous studies using large-area stratification (195 CCGs) and our approach using small-area data (32,784 LSOAs). Whereas large-area stratification assigns each CCG to a single deprivation quintile, the small-area approach captures the considerable heterogeneity that exists within CCGs. This enables us to assess, for a given change in CCG-level expenditure, whether health benefits are greater in more or less deprived areas. We also conducted sensitivity analysis using large-area deprivation.

#### 3.3 Outcome Measures

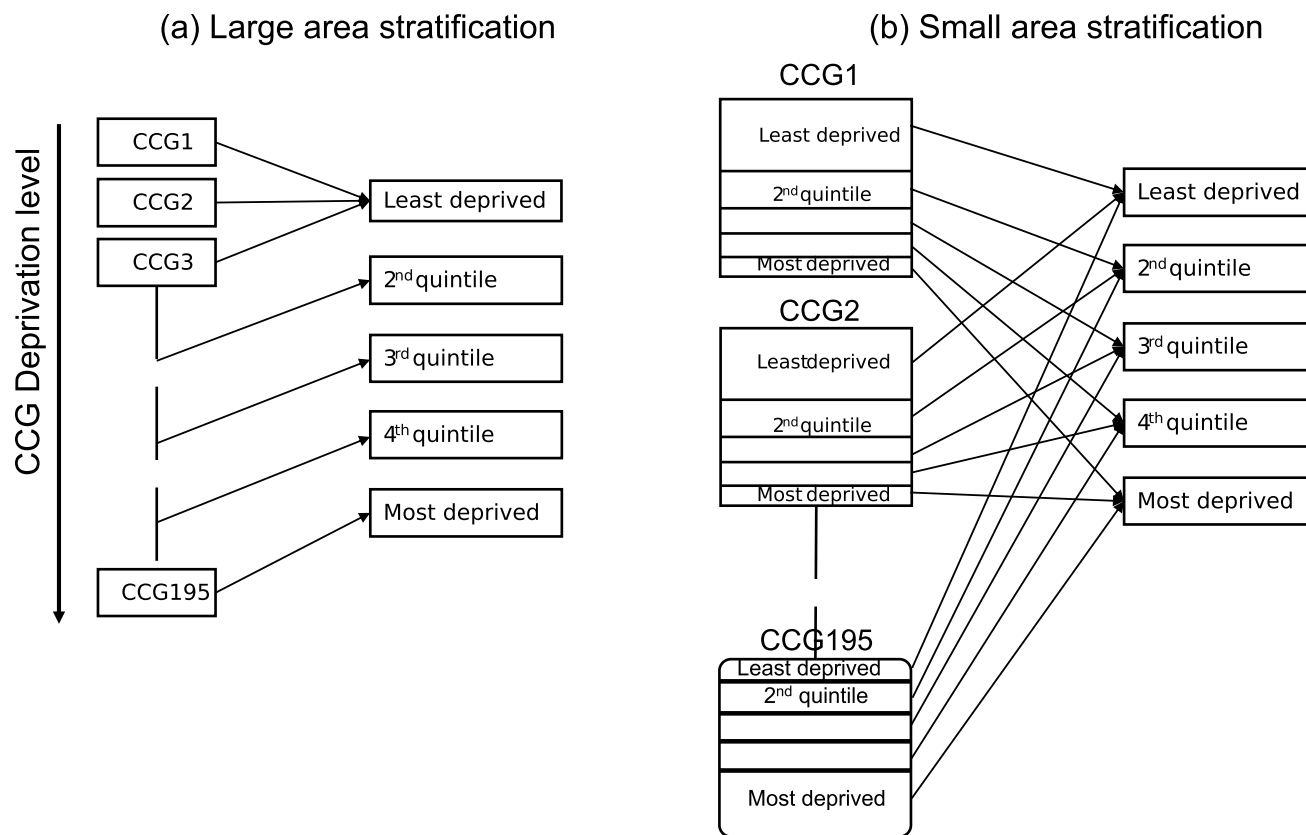
The main outcome of interest was the age-sex standardised all-age mortality rate per 100,000 general population for each LSOA. We dropped 60 LSOAs with zero death counts in 2018 (0.18 of 1% of all 32,844 LSOAs) before taking the logarithm of mortality, and so our analytical sample contained 32,784 LSOAs. We used direct standardisation, referenced to the 2018 English population. In sensitivity analysis, we also examined life years lost under age 75 years per 100,000 general population, to facilitate comparison with other studies.

Table 1 shows descriptive statistics (mean and standard deviation) for hospital expenditure and the two mortality measures at the England level, by five area deprivation quintile groups and both small-area (LSOA) and large-area (CCG) units of analysis, which are discussed in Appendix Note A2 (ESM).

Figure A1 in the Appendix (ESM) shows the relationship between age-standardised mortality, hospital expenditure and deprivation quintile group by large and small areas of analysis, including 95% confidence intervals based on fractional-polynomial prediction. There are substantial differences between expenditure and mortality patterns at large- and small-area levels, and the confidence intervals are smaller at small-area level due to increased statistical precision. This suggests that taking a fine-grained look at small-area deprivation has the potential to reveal mortality effects that might otherwise remain hidden by large-area aggregation.

#### 3.4 Control Variables

We included control variables that we consider to be confounding factors that are causally linked with both our exposure (large-area expenditure) and our outcome (small-area mortality). Our preferred specification has two kinds of control variables: First, large-area level controls for two components of the funding formula related to the need for hospital expenditure and thereby causally linked to mortality as well as expenditure—the Age-Cost Index and the Market Forces Factor. Second, small-area level controls



**Fig. 1** Small-area versus large-area stratification. Large-area stratification creates groups of large-areas Clinical Commissioning Groups (CCGs) based on large-area deprivation. Small-area stratification cre-

ates groups of small-area Lower Super Output Areas (LSOAs) based on small-area deprivation

**Table 1** Main variables of interest by large- and small-area deprivation group

	Large area unit of analysis			Small area unit of analysis		
	Hospital expenditure per capita	Adjusted mortality rate per 100,000	Life years lost per 100,000	Hospital expenditure per capita	Adjusted mortality rate per 100,000	Life years lost per 100,000
Least deprived	1,170	806	3,259	1,218	777	2,721
2nd quintile	1,223	884	3,738	1,246	877	3,193
3rd quintile	1,296	928	4,131	1,255	962	3,761
4th quintile	1,298	949	4,287	1,270	1125	4,527
Most deprived	1,373	1069	5,029	1,310	1379	6,211
Overall	1,272	927	4,089	1,260	1,024	4,084

The deprivation quintile groups are stratified by 195 large NHS administrative areas Clinical Commissioning Groups (CCGs) and 32,784 small-area Lower Super Output Area (LSOAs), respectively. The adjusted mortality rate is per 100,000 population, adjusted for age and sex, and the years of life lost are calculated for under age 75 years per 100,000 population

for all six domains of the Index of Multiple Deprivation (IMD) 2019 other than health, i.e., education, barriers to housing, environment, employment, income, and crime domain scores. We did not control for the health domain of the IMD based on morbidity data or the Additional Needs component of the funding formula based on standardised mortality data, since they are mediators or proxy

outcomes rather than confounders: controlling for these health variables would risk under-estimating the mortality effect due to over-adjustment (insofar as morbidity lies on the causal pathway from expenditure to mortality) and circularity (insofar as we would be adjusting mortality by mortality). However, we included the health domain in sensitivity analysis.

We also conducted sensitivity analysis around using the IMD domains as large-area level controls. There is room for debate about which level is more appropriate, since the small-area level measures the small-area mortality aspect of confounding more accurately, whereas the large-area level measures the large-area expenditure aspect more accurately.

## 4 Methods

We are interested in the causal relationship between secondary-care funding allocation and mortality. We estimated this using a well-established cross-sectional instrumental variable (IV) approach that has been developed and refined in numerous previous studies [12, 14, 18, 27].

A naïve regression equation (without IV) to estimate the overall marginal effect of allocation on mortality would take the following form:

$$Y_{ij} = \beta_0 + \beta_1 E_j + x_{ij}'\beta_2 + \epsilon_{ij} \quad (1)$$

with the dependent variable  $Y_{ij}$  denoting the log of the outcome (e.g., age-sex standardised mortality rate per 100,000) for LSOA  $i$  ( $i = 1, \dots, 32,784$ ) in large area  $j$  and  $E_j$  the log of the allocation of hospital expenditure per capita in  $j$ .  $x_{ij}$  is a vector of control variables at LSOA level and  $\epsilon_{ij}$  is the error term.

However, the variable  $E_j$  is potentially endogenous as funding allocations are influenced by need factors such as morbidity, which are also correlated with mortality. This introduces endogeneity into the relationship between funding and mortality, meaning that cross-sectional associations capture not only the effect of healthcare funding on health outcomes but also the reverse effect of health outcomes on healthcare funding. To address this endogeneity, we instrumented hospital funding using a component of the funding formula, known as the *Distance from Target Index (DfT)*, as explained later. The revised regression equation to estimate the overall effect on mortality takes the following form:

$$Y_{ij} = \beta_0 + \beta_1 \hat{E}_j + x_{ij}'\beta_2 + \epsilon_{ij} \quad (2)$$

where  $\hat{E}_j$  is the log of the instrumented allocation of hospital expenditure from a first stage regression,  $E_{ij} = \lambda_0 + \lambda_1 Z_j + x_{ij}'\lambda_2 + \omega_{ij}$ , where  $Z_j$  is the distance from target instrumental variable, and  $x_{ij}$  the same set of control variables.

We estimated both equations using a standard two-stage least-squares method, with robust standard errors clustered by CCG because health expenditure is allocated at CCG level [34]. Our main parameter of interest,  $\beta_1$ , is the elasticity of mortality with respect to expenditure allocation (i.e., the % change in mortality for a 1% change in expenditure). We prefer a log-log functional form because both mortality

and expenditure are closer to log normal than normal in distribution, but we also report results using linear-linear, log-linear and linear-log forms. With the linear-linear form,  $\beta_1$  estimates the absolute change in mortality for a £100 change in per capita expenditure after rescaling expenditure into units of £100 for convenience.

### 4.1 Strength of the Instrumental Variable

We use the so-called “funding rule” instrumental variable (IV) approach, based on quirks of the NHS funding formula that induce quasi-exogenous large-area variations in expenditure. This is a well-established approach that has been used in a large body of empirical work in England for the past decade [13–15, 18, 27]. This IV approach was introduced by Andrews and colleagues [18], who used theoretical simulations as well as empirical tests to argue that funding rule instruments should not be supplemented with less informative secondary IVs based on census socioeconomic characteristics, as this tends to bias estimates downwards. The target budget is based on various factors indicating relative need for healthcare. The target budget is then adjusted using the “Distance from Target Index” (DfT) to reduce the pace of change in expenditure and avoid large and unexpected fluctuations in budgets, so that planning areas move towards their target budget in small steps rather than giant leaps. This index is based on the difference between the formula target and the historical allocation, and means that planning areas can be persistently “above” or “below” target for many years [15, 18].

A valid instrument needs to satisfy two main requirements: (1) the relevance condition, that it influences secondary-care funding (i.e., the instrumented variable), and (2) the exclusion restriction, that it influences mortality (the outcome) only via secondary-care funding and is uncorrelated with unobserved variables that may also influence mortality. We follow Bridley and colleagues in arguing that this is the only component of the funding formula that meets both requirements [27], and discuss why the other components do not meet these requirements in Appendix Note A3 (ESM). We use DfT as a sole instrument and the Age-Cost Index and MFF as control variables in our preferred specification, but report sensitivity analysis including Age-Cost Index and MFF as instruments in Appendix Table A6 (ESM).

The relevance condition for DfT can be formally tested. Table 2 reports the results of the first-stage regression for our preferred model (small-area stratification, small-area controls, log-log functional form). This shows that DfT exhibits a statistically significant effect on hospital spending. The Kleibergen–Paap test for under-identification confirms that DfT is a strong predictor of hospital spending ( $p$ -value < 0.001), with a first-stage F-statistic exceeding 106—comfortably above the conventional threshold of 10—thereby

**Table 2** First-stage regression model (outcome: natural log of hospital expenditure per capita)

	(1)	(2)
Variables	Coef	SE
<i>Instrumental variable</i>		
Natural log of Distance from Target Index	0.719***	0.070
<i>Deprivation</i>		
Least deprived	- 0.024***	0.005
2nd quintile	- 0.021***	0.004
3rd quintile	- 0.020***	0.003
4th quintile	- 0.012***	0.002
Most deprived	Ref.	Ref.
<i>Control variables</i>		
Lacking Education (score 0 to 100)	- 0.089***	0.014
Lacking Employment (proportion 0 to 1)	9.832***	3.712
Poor Living Environment (0 to 100)	- 0.006	0.013
Barriers to Housing and Services (0 to 100)	- 0.086***	0.015
Risk of Crime (z-score in sd units, -4 to +4)	0.956***	0.256
Low Income (proportion 0 to 1)	6.586*	3.446
Market forces factor index	0.533***	0.084
Age-cost index	0.756***	0.040
Constant	5.878***	0.120
Observations	32,784	32,784
First-stage F stat	106.3	
Kleibergen-Paap test for relevance	$p$ -value<0.001 $H_0$ : IV is weak	
Hausman test for endogeneity	$p$ -value<0.001 $H_0$ : Spending is exogenous	

Robust standard errors clustered by Clinical Commissioning Groups (CCGs). \* $p < 0.10$ , \*\* $p < 0.05$ , \*\*\* $p < 0.01$

This is linear regression with a log-linear functional form, based on the natural logarithm of the outcome, which shows the percentage point change in hospital expenditure per capita for a one-unit change in the covariate. The Distance from Target Index (DfT) has mean 0 and range -0.05 to 0.30 where the units represent proportional distances from target (e.g., 0.30 represents 30% above target). The original DfT variable is right-skewed with a long right-hand tail of CCGs with substantial distance from target. For linear estimation purposes, we re-scale this explanatory variable to yield a more symmetric distribution by adding 1 so that units represent shares of target (e.g., 1.30 represents 30% of the target share) and taking the logarithm. Deprivation groups are indicator variables taking the values of 0 or 1, Employment and Income are proportions from 0 to 1, Education, Living Environment and Barriers to Housing and Services are scores from 0 to 100, Crime is a z-score with mean value 1 and range about - 4 to +4

avoiding the “weak instrument problem” [35]. These findings confirm that DfT is a strong predictor of health expenditure allocation. Moreover, the Hausman test indicates that hospital spending is endogenous ( $p$ -value < 0.000), which suggests that the ordinary least squares (OLS) regression is biased and justifies the use of an instrumental variable approach.

There is no formal test to verify the exclusion restriction, so we must rely on causal inference reasoning. DfT is used to implement a “pace of change” policy designed to avoid overly rapid changes in expenditure. It estimates how far the current funding differs from the target funding allocation based on assessed need only (i.e., all the other components of the formula). Planning areas that are substantially below target—i.e, historically “under-funded”—receive less than the full needed increase, and vice versa. Over time, this smooths out the funding shifts so that planning areas gradually move towards their target allocation in small steps rather than giant leaps. DfT thus captures historical influences on expenditure, which are distinct from the current need factors incorporated in the main part of the formula. These historical influences may not be quasi-random, in the sense that they may be systematically determined by dynamic processes involving interacting historical factors, including historical need factors as well as historical political and sociological factors. However, they are at least quasi-exogenous, in the sense that they involve historical factors that are not closely correlated with current need and that are unlikely to exert any direct causal influence on current mortality.

Following Basu [36], we test whether DfT is correlated with observable confounding variables hypothesised to influence mortality, which might suggest an indirect impact on mortality through other channels. This diagnostic information is reported in Appendix Table A1 (ESM), in the form of linear regressions of IVs against control variables. Our results indicate that DfT is less strongly correlated with confounding variables than Age-Cost Index and MFF. We acknowledge that some correlation of DfT with confounding variables exists. However, we argue that DfT is conditionally exogenous once we control for these confounding factors in our regression.

To assess potential indirect pathways through which DfT might affect mortality other than health expenditure, we examine the funding formula in Appendix Note A3 (ESM), which shows that the target budget per capita depends explicitly on the Age-Cost Index and the MFF Index. The Age-Cost Index reflects healthcare need based on demographic and morbidity profiles based on detailed person-level hospital diagnosis codes—factors which are, unsurprisingly, correlated with mortality, while the MFF captures geographic variation in wages and input prices that may influence mortality indirectly through migration rather than spending. By controlling for both indices and for all six non-health domains of the Index of Multiple Deprivation (IMD) 2019, as shown in Table 2, we account for the main observable determinants of DfT. Even after doing so, DfT remains a strong predictor of healthcare expenditure, reflecting central policy-makers’ discretionary decisions in setting permissible deviations from target allocations that are unrelated to the controlled variables. These considerations

strengthen the case that DfT is conditionally exogenous and therefore a valid instrumental variable.

## 4.2 Sub-Group Analysis by Deprivation Group

To analyse the mortality elasticity for each deprivation group (also known as “conditional average treatment effects”), we estimate the following specification:

$$Y_{ij} = \beta_0 + \beta_1 \widehat{E}_j + \beta_2 (\widehat{E}_j \circ q_{ij}^2) + \dots + \beta_5 (\widehat{E}_j \circ q_{ij}^5) + q_{ij}' \beta_6 + x_{ij}' \beta_7 + \epsilon_{ij} \quad (3)$$

where the instrumented allocation,  $\widehat{E}_j$ , for large area  $j$  is interacted with four binary indicators of small-area deprivation quintile group  $q_{ij}^2, \dots, q_{ij}^5$ , with the reference category  $q_{ij}^1$  (least deprived) dropped and deprivation group main effects included (using vector notation  $q_{ij}$  for all four deprivation indicators). This model augments our previous equation by assuming that small-area mortality is a function of the interaction between expenditure and small-area deprivation group, and of small-area deprivation group, as well as large-area expenditure and small-area confounding factors.

In sensitivity analysis (Appendix Table A5, ESM), we also explore a more flexible approach that allows the instrumental variable relationship between DfT and funding allocation to interact with small-area deprivation quintile group, as discussed in Appendix Note A4 (ESM).

The elasticity of mortality with respect to expenditure allocation to the first quintile group (the dropped category) is the baseline expenditure coefficient  $\xi_{Y,E|Q1} = \widehat{\beta}_1$ , where  $\widehat{\beta}_1$  is the two-stage least-squares regression in Eq. 3. Similarly,  $\xi_{Y,E|Q2} = \widehat{\beta}_1 + \widehat{\beta}_2$ ,  $\xi_{Y,E|Q3} = \widehat{\beta}_1 + \widehat{\beta}_3$ ,  $\dots$ ,  $\xi_{Y,E|Q5} = \widehat{\beta}_1 + \widehat{\beta}_5$ . In other words, the elasticity of mortality with respect to allocation for the second quintile is the sum of the first quintile elasticity plus the coefficient on expenditure interacted with the second quintile, and similarly for the remaining quintiles.

We examined whether there is a significant pro-deprived or anti-deprived linear slope in mortality effects across deprivation groups by testing the null hypothesis (*least deprived + 2nd quintile*) - (*4th quintile + most deprived*) = 0 which is equivalent to test  $\widehat{\beta}_2 - \widehat{\beta}_4 - \widehat{\beta}_5 = 0$ , and we examined whether the middle group had a significantly larger effect than other groups by testing the null hypothesis *3rd quintile - (least deprived + 2nd quintile + 4th quintile + most deprived)/4 = 0* which is equivalent to test  $\widehat{\beta}_3 - \frac{\widehat{\beta}_2 + \widehat{\beta}_4 + \widehat{\beta}_5}{4} = 0$ .

## 5 Results

### 5.1 Main Results

Table 3 presents results using twelve variants of our preferred instrumental variable approach using Distance from

Target Index (DfT) as the sole instrumental variable and a single first-stage regression. We report variants using three different models—M1 large-area stratification, M2 small-area stratification with large-area controls, and M3 small-area stratification with small-area controls—two different mortality variables—standardised all-age mortality and years of life lost under age 75 years—and two different functional forms—log-log showing elasticities, and the linear-linear form showing absolute effects of £100 expenditure. Model M1 is our least preferred model, since this does not account for inequalities within large-area stratification, and, also, the statistical power is low with only 195 observations split into 39 observations per CCG quintile group. If forced to choose, we prefer M3 over M2, as using control variables at the small-area level arguably allows for a more precise adjustment of mortality outcomes, which are also measured at that level in the second-stage regression, while still serving—albeit more indirectly—to account for their effect on hospital expenditure in the first stage. Full two-stage least-squares results for this model in log-log form with all-age mortality and years of life lost can be found in Appendix Tables A2 and A3 (ESM), respectively.

The overall effect is significant, and our preferred model shows that a 1% increase in expenditure reduces mortality by 0.92% (95% confidence interval (CI) 0.37–1.47). The pattern of effects by deprivation quintile group exhibits an inverted U-shape pattern in all 12 variants, and the effect is significantly larger in the middle group in 10 out of 12 variants. However, there is no sign of a significant pro-poor gradient in any of the 12 model variants. We cannot reject the null hypothesis of no significant linear slope in 7 out of the 12 variants—i.e., the two most deprived groups gain the same as the two least deprived groups. Furthermore, in the other five variants, we find a significantly “pro-rich” slope—all four variants of model 2 and one variant of model 3 (mortality outcome, linear-linear form).

Figure 2 visualises the results for our preferred model 3, showing the inverted U-shape, where the third quintile group benefits more from marginal health expenditure. Figure 3 visualises the results for all three models, showing the same basic pattern in each case.

(four variants of preferred model 3). To improve readability, we inverted signs. Our preferred specification is model 3, which uses data at the Lower Super Output Area (LSOA) level, this corresponds to employing small-area stratification and small-area controls. The elasticity shows the proportional effect on the mortality outcome of a 1% increase in hospital expenditure using a log-log specification, while the absolute effect shows the effect of £100 per capita increase in expenditure using a linear-linear specification in terms of age-sex adjusted mortality per 100,000 and years of life lost under 75 per 100,000

**Table 3** Estimated coefficients for 12 main specifications

	Model 1		Model 2		Model 3	
	Large-area unit of analysis (CCG)		Small-area unit of analysis (LSOA) with GGC controls		Small-area unit or analysis (LSOA) with LSOA controls	
	Estimate	SE	Estimate	SE	Estimate	SE
<i>Outcome: Adjusted mortality elasticities (log-log)</i>						
Least deprived	-1.068***	0.410	-1.138***	0.384	-0.879***	0.277
2nd quintile	-1.445***	0.422	-1.264***	0.393	-1.041***	0.293
3rd quintile	-1.299***	0.375	-1.265***	0.384	-1.010***	0.283
4th quintile	-1.149***	0.413	-1.151***	0.379	-0.935***	0.284
Most deprived	-1.116***	0.384	-0.764**	0.383	-0.758***	0.290
Overall	-1.215***	0.383	-1.116***	0.380	-0.925***	0.279
Flat slope test /1	-0.248	0.291	-0.487***	0.165	-0.228	0.169
Middle slope/2	-0.105	0.133	-0.186***	0.067	-0.107	0.066
<i>Outcome: Adjusted mortality as absolute effect of £100 per capita (linear-linear)</i>						
Least deprived	-60.958***	23.560	-77.851***	24.536	-53.088***	16.672
2nd quintile	-83.861***	24.441	-78.484***	24.253	-54.606***	17.076
3rd quintile	-74.172***	20.834	-78.588***	24.353	-52.250***	17.330
4th quintile	-66.377***	23.419	-70.555***	24.190	-45.412**	17.639
Most deprived	-62.896***	20.988	-32.213	24.661	-20.463	18.694
Overall	-69.653***	21.189	-67.524***	23.985	-45.154***	16.913
Flat slope test /1	-15.546	19.830	-53.568***	12.137	-41.819***	12.556
Middle slope/2	-5.649	9.230	-13.812***	4.822	-8.858*	4.750
<i>Outcome: Years of life lost elasticities (log-log)</i>						
Least deprived	-1.029**	0.414	-1.460***	0.456	-1.282***	0.319
2nd quintile	-1.082***	0.403	-1.392***	0.437	-1.270***	0.310
3rd quintile	-0.960***	0.350	-1.491***	0.449	-1.347***	0.314
4th quintile	-1.020***	0.368	-1.630***	0.444	-1.602***	0.311
Most deprived	-0.691*	0.368	-0.759*	0.428	-1.240***	0.308
Overall	-0.956***	0.354	-1.346***	0.430	-1.348***	0.293
Flat slope test /1	-0.400	0.370	-0.463**	0.230	0.291	0.236
Middle slope/2	-0.005	0.167	-0.181	0.140	0.002	0.135
<i>Outcome: Years of life lost as absolute effect of £100 per capita (linear-linear)</i>						
Least deprived	-328.68**	134.72	-432.65***	123.26	-333.83***	75.46
2nd quintile	-334.02**	129.85	-412.54***	118.25	-323.15***	73.31
3rd quintile	-274.13**	115.25	-439.09***	122.64	-354.67***	76.64
4th quintile	-286.99**	118.40	-436.62***	123.34	-398.31***	77.95
Most deprived	-159.55	108.37	-67.79	112.89	-220.82***	79.50
Overall	-276.67**	113.231	-357.62***	116.452	-326.12***	70.918
Flat slope test /1	-216.16*	116.674	-340.78***	69.721	-37.859	69.704
Middle slope/2	3.178	52.237	-101.69***	34.818	-35.640	34.524
Observations	195		32,784		32,784	

Robust standard errors clustered by Clinical Commissioning Groups (CCGs). \* $p < 0.10$ , \*\* $p < 0.05$ , \*\*\* $p < 0.01$

Estimated coefficients are marginal effects by deprivation group estimated by two-stage least-squares with distance from target index (DFT) as the sole instrumental variable and expenditure interacted with deprivation quintile (full results for elasticities are reported in Appendix Tables A1 and A2 (ESM), for adjusted mortality and life years lost, respectively); 1/ Flat slope test (least deprived + 2nd quintile)—(4th quintile + most deprived) = 0; 2/ Middle slope test: 3rd quintile—(least deprived + 2nd quintile + 4th quintile + most deprived)/4 = 0

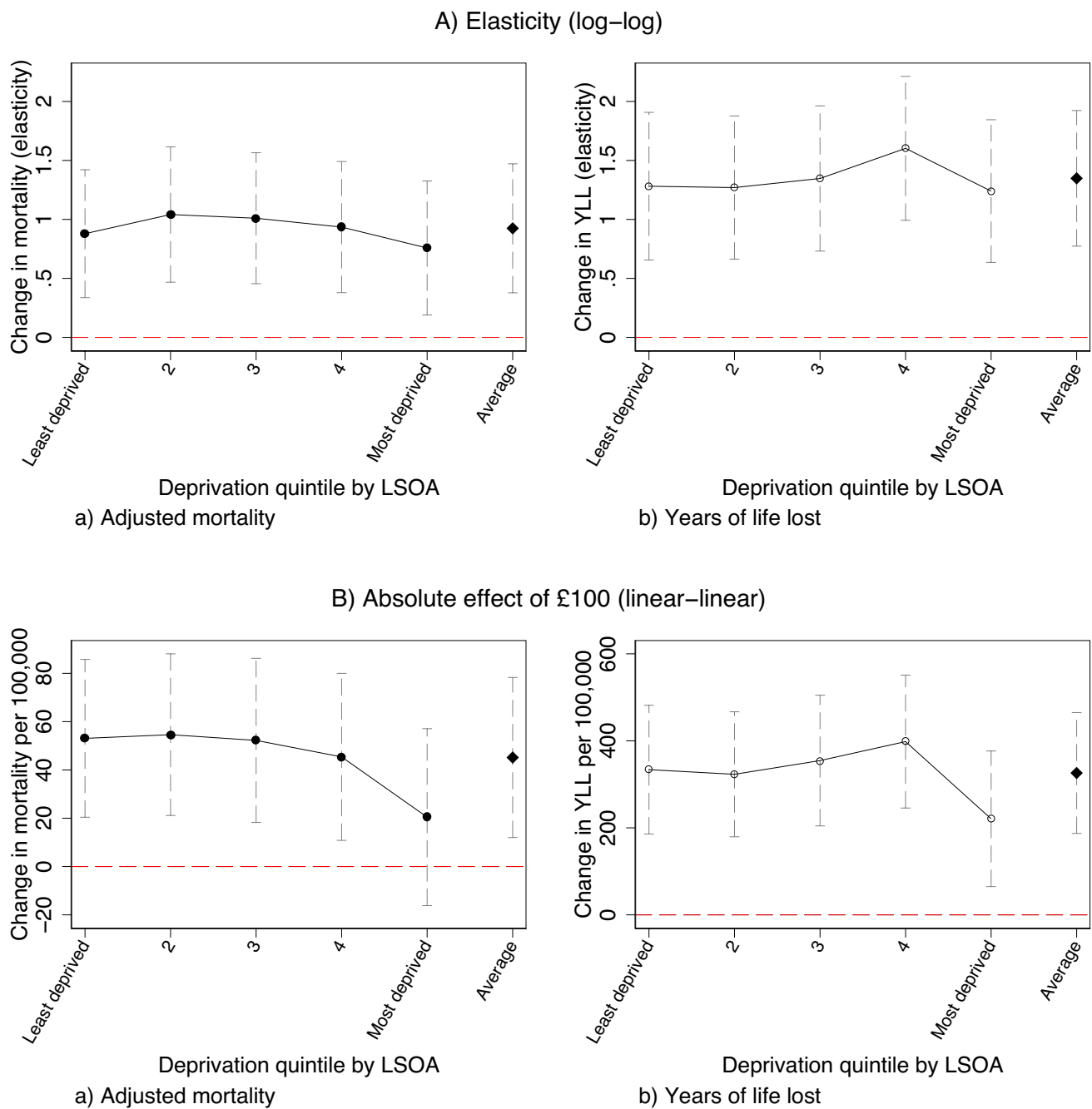


Fig. 2 Mortality effect of hospital expenditure by small-area deprivation group

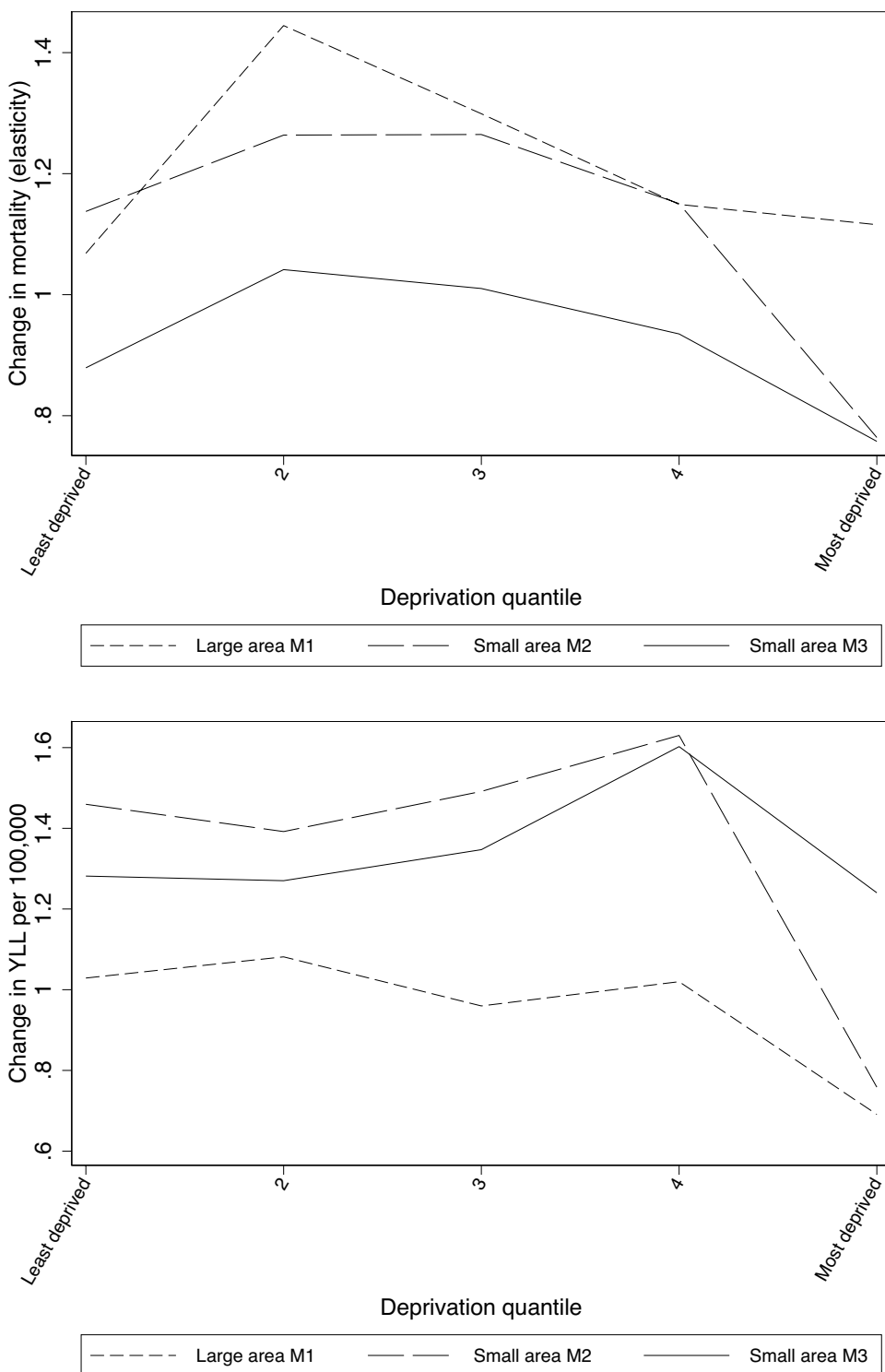
### 5.2 Sensitivity Analyses

Sensitivity analyses regarding the choice of controls are presented in Appendix Table A4 (ESM), showing minimal impact on our results. Figure A2 in the Appendix (ESM) compares the preferred result with two additional specifications that incorporate five first stages, as shown in

Appendix Table A5 (ESM), further reinforcing our general findings.

Appendix Table A6 (ESM) shows sensitivity analyses around the IV approach, including a naïve regression with no IV, an expanded IV set with DfT, Age-Cost and MFF, and an extended control set including IMD domains, Age-Cost and MFF. The basic inverted-U pattern of effects is similar

**Fig. 3** Comparison of social gradient patterns from all three main models (elasticity point estimates for mortality and years of life lost). This figure visualises the effect gradients reported in Table 3. “Large area M1” corresponds to Column 1, “Small Area M2” corresponds to Column 2, and “Small Area M3” corresponds to Column 3 of Table 3. The confidence intervals from Table 3 are not displayed here since this would clutter the graph and make it hard to visualise the basic patterns



in all cases, though in the naïve and expanded IV set models none of the effects is significant, including the overall effect.

Appendix Table A7 (ESM) shows that removing all controls slightly reduces effect sizes but retains the inverted-U pattern; the same holds when adding the health domain as a control. Appendix Figure A3 (ESM) demonstrates that

using robust standard errors instead of clustering by CCG results in narrower confidence intervals.

Our key findings remain robust to several additional specifications: allowing for a distinct treatment effect for London (Appendix Table A8, ESM), applying alternative functional forms (log-linear and linear-log; Appendix

Table A9), and using a “MFF-adjusted” NHS budget allocation per capita to capture real purchasing power (Appendix Table A10, ESM).

## 6 Discussion

### 6.1 Main Findings

We found that the mortality effects of a marginal change in public expenditure on secondary care in England in 2018/19 did not have a “pro-poor” pattern favouring more deprived groups. Instead, they had a “pro-middle” pattern with an inverted U shape, whereby the effect was largest in the middle deprivation group. This pattern persisted whether effects were measured in proportional or absolute terms, and whether mortality was measured as an age-sex-adjusted all-age mortality rate or as years of life lost under age 75 years. It was also robust to numerous sensitivity analyses. If anything, there was more sign of a “pro-rich” pattern than a “pro-poor” pattern, in that five of our 12 main specifications showed a significant “pro-rich” pattern.

This finding contradicted our prior expectation that more deprived people would gain more health from additional public expenditure on secondary care than less deprived people, due to diminishing marginal returns to additional expenditure on secondary care relative to need. It also contradicted the findings of previous quasi-experimental studies of the marginal effect of secondary-care expenditure on mortality in England in the 2000s and early 2010s [13, 15, 21, 23]. We speculate that a combination of three main mechanisms may be driving our results. First, a “sharp elbows” mechanism whereby more deprived groups are less likely than the middle group to benefit from increased public expenditure on non-emergency hospital services. Second, a “crowding out” mechanism whereby increases in public expenditure have smaller impacts on non-emergency utilisation and outcomes among the least deprived groups than the middle group due to countervailing decreases in private expenditure. Third, a “co-morbidity and co-investment” mechanism whereby the most deprived groups have worse healthcare outcomes than other groups, due to greater co-morbidity and lesser ability to co-invest their own private resources in care co-ordination, compliance, recovery, rehabilitation and relapse prevention and compliance.

### 6.2 Strengths and Limitations

The main strength of our study is the use of small-area data to estimate how the mortality effects of public expenditure vary between more and less deprived populations. This

allows us to measure deprivation more precisely than previous studies using large-area data, accounting for impacts on health inequality within large areas. A second strength is that we use a well-established instrumental variable approach to allow for endogeneity bias, based on a quirk of the NHS funding formula that generates quasi-exogenous variation in funding, with careful theoretical discussion, diagnostic checks, and sensitivity analysis. A third strength is that we report findings for two different mortality outcomes—all-age age-sex standardised mortality and years of life lost under age 75 years—in terms of both elasticities and absolute effects.

The main limitation is that our approach rests on causal inference assumptions that cannot be directly tested. We assume that our “Distance from Target” instrument reflects variation in expenditure that is both (i) quasi-exogenous and (ii) sustained. These assumptions are justified in Sect. 4.1. The latter assumption means that, like the authors of many previous studies in this literature, we interpret our findings as the annual mortality effect of sustained variation in annual expenditure that started several years before the mortality data period, rather than temporary variation that started in the current year. That is why the 3-month discrepancy in time periods between our funding and mortality data (financial year and calendar year 2018, respectively) is not material to our conclusions. A second limitation is that we do not have data on public or private hospital expenditure at small-area level. This means we cannot tell how far our results are due to each of the three potential mechanisms described above, i.e. “sharp elbows”, “crowding out” and “co-morbidity and co-investment”. However, this limitation does not bias or invalidate our main conclusion. We can safely conclude that, around the year 2018/19, people in the two most deprived quintile groups of small areas of England did not gain more health than those in other groups from increases in secondary-care funding and did not lose more health from decreases.

### 6.3 Comparison with Existing Literature

Five studies of English data from the 2000s to early 2010s all concluded that changes in secondary-care expenditure tend to have larger mortality effects in more deprived or higher-mortality populations. This discrepancy with our findings may be due to a structural change in the social patterning of hospital expenditure effects in England from pro-deprived to pro-middle during the 2010s, following a sustained deterioration in per capita national income and public services that may have strengthened all three mechanisms described above (i.e., “sharp elbows”, “crowding out”, and “co-morbidity and co-investment”). Supporting evidence for this speculation is that more deprived groups

have tended to use disproportionately more emergency care and disproportionately less elective and outpatient care since the early 2010s [37, 38]. If this hypothesis is correct, there is little prospect of rapid reversion to a pro-deprived pattern of mortality effects, since UK economic performance has remained lacklustre since the year of our study (2017-18) and independent forecasters predict only modest growth in the coming decade.

However, it is also possible that all five previous studies were biased in favour of finding a pro-deprived gradient, especially the study by Love-Koh and colleagues [21], which found that 26% of the total health effect from an expenditure shift would benefit the most deprived small-area quintile group and only 14% to the least deprived. This gradient was mostly driven by emergency utilisation and if the study were repeated using more recent data would be entirely driven by emergency utilisation [37–39]. However, additional expenditure is more likely to be used to increase the supply of elective and outpatient care than emergency care, and the impact on emergency admissions could conceivably even be negative if acute emergencies are prevented by improved elective care. Furthermore, deprived populations might have worse health outcomes per marginal unit of expenditure than affluent populations due to the co-morbidity and co-investment mechanism previously described [4].

Two large-area studies using quantile regression [15, 22] found that secondary-care allocations had a greater proportionate effect in large areas with higher mortality rates. However, when we use quantile regression to analyse our data we find the opposite, at both large- and small-area levels of analysis—see Fig. A4 in the Appendix (ESM). We believe the discrepancy is due to the use of less robust instrumental variable approaches in these past studies, one of which used census-based socioeconomic variables as instruments rather than funding components, and one of which used the Age-Cost Index and Market Forces Factor as instrumental variables, which is inappropriate for the reasons explained in Methods, Sect. 4.1.

Finally two previous studies used a time series approach, exploiting differential changes in expenditure in more and less deprived large areas [23, 24]. They found that more deprived local government areas in England tended to experience larger changes in amenable mortality age under 75 years than less deprived administrative areas per unit change in health expenditure, both during the 2000s (a period of high healthcare expenditure growth) and the 2010s (a period of low healthcare expenditure growth). However, these studies looked at all health expenditure, not just hospital expenditure, and may have been confounded by wider public expenditure changes beyond the health system.

## 6.4 Conclusions and Policy Implications

Our main conclusion is that there is not a “pro-poor” social gradient in the mortality effects of changes in secondary-care expenditure in England, whereby more deprived individuals gain substantially larger health benefits than less deprived individuals. This conclusion is specific to secondary-care expenditure, and we cannot draw conclusions about the health inequality impacts of changes in primary-care, public health, or social-care expenditure. It is also specific to England in the late 2010s since we cannot rule out the possibility of a structural shift during the 2010s due to deteriorating economic conditions and public services. Furthermore, we do not know the relative contribution of the three plausible mechanisms that may be generating this finding—(i) the “sharp elbows” mechanism relating to how far increases in hospital funding for large administrative areas of England are unequally shared between more and less deprived people living within those areas, (ii) the “crowding out” mechanism relating to how far private expenditure on hospital care by the least deprived groups is crowded out by increases in public expenditure, and (iii) the “co-morbidity and co-investment” mechanism relating to how far more deprived people gain less health per additional pound of hospital expenditure than less deprived people.

Our findings challenge the conventional wisdom that the poor always gain more from marginal increases in public expenditure on secondary care. Importantly, however, we did not examine the effects of a marginal increase in public expenditure on primary care and public health, which may tend to have a more “pro-poor” pattern of impacts than secondary care. However, primary care and public health only makes up a small proportion of total government health expenditure in England [40]. Our findings also challenge the conventional wisdom that there is a “pro-poor” gradient in the health opportunity costs of introducing cost-increasing technologies and programmes into universal publicly funded health services, whereby more socially disadvantaged groups bear a larger share of the health opportunity costs. However, further research is needed to examine whether reductions in health expenditure could have different impacts from equivalent increases, thereby testing the potential existence of asymmetric effects in the relationship between expenditure and health outcomes.

Our main conclusion is more robust than the contradictory findings of previous studies, including a study by Love-Koh and colleagues which estimated an implausibly steep “pro-poor” gradient based on emergency utilisation patterns [21], two quantile regression studies based on potentially biased instrumental variables [15, 22] and two time series studies [23, 24] that may have picked up effects of changes in expenditure on wider public services beyond healthcare.

Our findings have implications for distributional cost-effectiveness analysis (DCEA) of healthcare interventions, which requires an assumption about the social distribution of health opportunity costs of healthcare expenditure. Our findings suggest that it is not appropriate to assume a steep “pro-poor” gradient of this kind in England. Instead, a more reasonable base-case assumption for England may be a flat distribution with a 20% share in each group. This may be more reasonable than assuming a symmetric “pro-middle” pattern since (1) it is more parsimonious, (2) it will generally yield the same health inequality impact, and (3) the magnitude of the pro-middle differential is highly uncertain. However, it would be appropriate to conduct sensitivity analysis using alternative pessimistic and optimistic scenarios. For example, a suitable “pessimistic” scenario yielding a smaller health inequality reduction might be a slight pro-deprived gradient (e.g., 22%, 21%, 20%, 19%, 18%, respectively, for the most to least deprived group) and a suitable “optimistic” scenario yielding a larger health inequality reduction might be the corresponding slight anti-deprived gradient (e.g., 18%, 19%, 20%, 21% and 22%, respectively).

Our findings also have the policy implication that reducing health inequality requires more than simply increasing total secondary-care expenditure or re-directing secondary-care expenditure towards large administrative areas with high average levels of deprivation. Additional efforts are needed to ensure that non-emergency secondary care is targeted and used more effectively to improve the health of socially disadvantaged individuals living within large administrative areas [41]. And wider action is needed to prevent the need for secondary care by re-directing resources away from hospitals and towards primary and community care, public health, and wider public services that foster healthy living conditions and behaviours.

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## Declarations

**Conflict of Interest Statement** Dr. Grasic is currently an employee of Amgen, Dr. Cookson reports grants from NIHR during the conduct of the study and personal fees from pharmaceutical companies for advisory work on equity methods outside the submitted work. The remaining of authors do not have conflicts of interest to disclose.

**Ethics Approval** Not applicable.

**Consent to Participate** Not applicable.

**Consent for Publication** Not applicable.

**Data Availability** All data used in this report are publicly available: **CCG funding allocations (2018/19)** for 195 NHS areas in England: <https://www.england.nhs.uk/allocations/>. **2018 mid-year population estimates**, from the Office for National Statistics (ONS): <https://www.ons.gov.uk/peoplepopulationandcommunity/populationandmigration/populationestimates/datasets/lowersuperoutputareamidyearpopulationestimatesnationalstatistics>. **2018 death counts** by sex, age (5-year bands), and 2011 LSOA, from the ONS: <https://www.ons.gov.uk/peoplepopulationandcommunity/birthsdeathsandmarriages/deaths/adhocs/10829deathsbylowerlevelsuperoutputarealsoenglandandwales2018registrations>. **2019 English Indices of Deprivation**, by LSOA, from the Ministry of Housing, Communities and Local Government: <https://www.gov.uk/government/statistics/english-indices-of-deprivation-2019>. **CCG boundary codes (2018/19)** for matching LSOAs to CCGs: <https://www.england.nhs.uk/publication/technical-guide-to-ccg-allocations-2019-20-to-2023-24-spreadsheet-files-for-ccg-allocations-2019-20-to-2023-24/>.

**Code Availability** The code to reproduce the analysis is available from the corresponding author upon reasonable request.

**Authors’ Contributions** All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by Misael Anaya-Montes and Katja Grašič. The first draft of the manuscript was written by Richard Cookson and Katja Grašič, with contributions from Misael Anaya-Montes and James Lomas. The interpretation of the results were critically appraised and subsequent drafts improved by Laura Anselmi, Miqdad Asaria, Christodoulos Kyridemos and Chris Bentley. The robustness of the empirical methodology and interpretation of results were assessed by Benjamin Barr, Matthew Sutton and Richard Cookson. All authors read and approved the final manuscript.

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