The impact of health spending on health outcomes and health inequality

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Abstract

Recently there has been considerable interest in the relationship between health spending and health outcomes and in the determinants of health inequality. Building on earlier work by Martin et al[4], this paper aims to quantify the impact of health spending on health outcomes, and the contribution of health spending to health inequality.

The units of analysis were English Primary Care Trusts (PCTs). Health outcomes were measured using 2008 Standardised Mortality Ratios (SMRs) for cancers and circulatory diseases taken from the National Centre for Health Outcomes Development (NCHOD) website. Health spending figures for 2007/08 were based on Department of Health (DH) Programme Budgeting data. Additional covariates were taken from the DH Exposition Book.

We regress the SMRs against the spending figures plus covariates, using instrumental variables to account for the endogeneity of spending. We use a concentration index approach measure inequality in mortality and decomposition methods to calculate the contribution of spending to mortality.

Spending has a negative impact on mortality. Cancer mortality and spending are concentrated in more deprived PCTs. Circulatory disease mortality is concentrated in more deprived PCTs but circulatory disease spending is equally distributed across PCTs.

While spending has a negative impact on the level of mortality the impact of spending on inequality in mortality is mixed. Cancer

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spending makes a negative contribution to inequality in cancer mortality, whereas spending on circulatory diseases makes a positive, though insignificant contribution to circulatory disease mortality inequality.

1 Introduction

Of crucial importance in decisions on health care funding is the efficiency of spending in terms of how it affects health outcomes. Furthermore in discussions regarding health inequality this efficiency and the existing inequality in both expenditure and health outcome need to be accurately estimated. Using more recent data, this paper seeks to update previous analysis for 2008 mortality rates. In addition, the effect of funding on inequality in outcome will be studied.

A critical issue facing macro-level analysis of health care spending is the degree to which health funding and health outcomes are endogenous. With suitable instrumental variables endogeneity can be purged from the expenditure variable, so the identification and use of such instruments is important for analysis.

Another issue is the contribution of preventative medicine to lowering adverse health outcomes. Unfortunately, this would require a richer, long-term panel dataset which is not currently available. The cross-sectional dataset available does allow analysis of the impact of spending on those already suffering with health conditions. Due to the need to have well-defined health outcomes we focus on the cancer and circulatory disease programmes of care, with SMR health outcomes. Ideally palliative care and other expenditure could be distinguished, unfortunately that is not possible.

Finally, it is paramount to accurately control for the need for health care in a population. Areas with high levels of illness attract high levels of funding. This does not imply the funding causes the illness. Analysis of health care expenditure must take into account what the underlying level of illness there is – what the mortality would have been if there were no health care.

The paper is structured as follows. After a brief review of previous studies the data will be described, the model estimation strategy introduced, the results presented, and conclusions discussed.

2 Literature Review

Previous studies investigating the links between health care and health outcomes have generally focused on international comparisons between countries [1] [8] [3]. These analyses tend to show socio-economic and dietary factors as being larger determinants of health than health care. In contrast, intrana-

tional area-level analyses such as Cremieux et al[2] and Martin et al[4]l find stronger effects of health spending on health outcomes. This is likely to be due to the heterogeneity in health care and other health determinants between nations. Though still likely to exist, within a country such differences are likely to be much less. This allows better and more significant comparisons to be made between areas with more and less health care funding.

In the Martin, Rice and Smith 2007 paper an attempt is made to measure the effects of health care spending across English PCTs in 2004/05. In their study, they develop a two equation static theoretical model to explain firstly the health care provider's budget decision (Equation 1), and secondly the effect of health expenditure on health outcomes (Equation 2). Each equation is then estimated by two stage least squares using instruments derived from the 2001 Census and the Index of Multiple Deprivation 2000 (IMD2000). For the cancer model, these are the percentage of unpaid carers and the percentage of lone pension households. For the circulatory model, the instrument set is augmented with the the IMD2000. The model estimated is

$$\begin{aligned} ProgrammeExpenditure_i = \alpha + \beta_1 Need_i + \beta_2 TotalExpenditure_i \\ + \beta_3 NonProgrammeSMR_i + \nu_i \end{aligned} \tag{1}$$

$$ProgrammeSMR_i = a + b_1 Need_i + b_2 ProgrammeExpenditure_i + \epsilon_i \tag{2}$$

where the Need variable is measured by the needs component of the PCT funding allocation formula, which changes the funding for PCTs based on the expected health care need based on area-level characteristics such as age and deprivation. The model is estimated for the cancer and circulatory disease programmes of care, and the results show a substantial negative effect of health care spending on mortality. Furthermore, the Need variable shows a positive coefficient, which in turn indicates a correlation between mortality and deprivation.

This paper is followed and updated in a number of CHE discussion papers[5] [6], which largely support the previous results using newer data. Additionally, these paper attempt to predict the cost of saving a year of life from either disease, using the health outcome variable Years of Life Lost (YLL). This variable is constructed by summing the difference between age

of death and 75 for every under-75 death due to the condition. The model is extended to different programmes of care.

While previous studies have investigate the relationship between health funding and health outcomes, none have estimated the contribution of health funding to inequality in outcomes.

3 Data and Estimation Strategy

The relationship under consideration is between mortality, deprivation and expenditure, shown in equation (3).

$$ProgrammeSMR_i = \alpha + \beta_1 Deprivation_i + \beta_2 ProgrammeExpenditure_i + \epsilon_i \quad (3)$$

Condition-specific mortality is measured by the 2008 all-age Standardised Mortality Rate available on the NCHOD website. This is a simple ratio of observed deaths over expected deaths, both of which are also extracted to allow standardisation of the concentration index.

Deprivation is measured by the Index of Multiple Deprivation 2007 average score, which averages a range of deprivation domains. Importantly, as it is an index it cannot be considered a cardinal measure, if PCT 'A' has double the score as PCT 'B', 'A' is more deprived but not *twice* as deprived. Therefore when elasticities of the deprivation variable are considered, they do not have the same objective meaning as, say, the elasticity of spending with respect to mortality. In this sense the effect deprivation is being controlled for rather than estimated, though the index also provides a ranking variable crucial to concentration index analysis.

Condition-specific expenditure is based on the programme budgeting data released by the Department of Health, namely the programme expenditure per person for the financial year 2007/08. The PCT population figure used is the Unified Weighting. However, this funding measure by itself only gives the condition-specific expenditure of a PCT as a proportion of the total number of people in the PCT. Ideally, the expenditure variable would restrict the denominator of this measure to those people with the condition, as the numerator (programme spending) is not being spent on people without the condition or on prevention. Consider the case of two PCTs 'A' and 'B', with the same population, the same total spending on cancer and the same

standardised mortality, but 'A' has 10% cancer prevalence and 'B' has 2.5%. The spending variable should be four times higher for 'B' than 'A' as only a quarter of people are being treated, at the same price, for the same condition, with the same outcome. Though there is no 'expenditure per affected person' data, there is prevalence data for 2007 on the NCHOD website, from the Quality and Outcomes Framework (QOF). Assuming prevalence did not vary substantially between the periods January to March 2007 and January to March 2008, dividing expenditure per person by prevalence provides a reasonable measure of expenditure per affected person. Clearly, however, some level of time invariance is being assumed to allow for mortality data (which is reported in calendar years) to be regressed on expenditure data based (which is reported in financial years).

Unfortunately, the inclusion of prevalence limits the dataset. PCT boundary changes between 2006 and 2007 reduced the number of PCTs from 303 to 152. Prevalence data was recorded under the old structure. Where multiple PCTs were amalgamated or left alone, the prevalence data for the new PCT structure was possible to identify. Where a PCT was split up and divided into a number of new PCTs it was not possible to calculate the prevalence. This led to a reduction in the number of observations from 152 to 147. This is the reason the summary statistics for cancer and circulatory SMRs in Table 1 do not show a mean of 100, which would have been true by definition with the full number of PCTs. It is possible to infer from the fact that both SMR values are slightly greater than 100 that the areas excluded were, on average, areas with lower standardised mortality.

In the case of endogeneity being discovered in the expenditure variable, instruments are required to employ Instrumental Variable techniques. These instruments ought to be correlated with expenditure, and only correlated with the standardised mortality insofar as they affect expenditure. Two variables which fulfil these requirements are the percentage of unpaid carers in a PCT, and the expenditure on other programmes by the PCT, both of which were also used in Martin et al's paper [4]. The first of these is collected by the census, conveniently the NCHOD website contains the 2001 Census data for this by PCT. An assumption underlying its use as an instrument is that it is fairly invariant over time, so the 2001 situation is similar to the 2007. The reasoning behind its inclusion as an instrument is that a greater number of unpaid carers will reduce the amount spent by the PCT on palliative care, ceterus paribus, while not itself causing a change in mortality. The second of these is a simple subtraction of the condition-specific funding from

total funding in the DH programme budgeting dataset. This instrument measures the other calls on a PCT's resources, such calls ought to reduce the amount available to spend on a specific programme while not itself affecting the mortality rate for the condition. Note that in contrast to the condition-specific expenditure which we are instrumenting for, the other-programmes expenditure is not standardised for prevalence of any disease, so remains pounds per capita. Though the instruments used by Martin et al were tested, they were not sufficiently correlated with 2007/8 expenditure to be included.

The following table displays summary statistics on the data to be used.

Variable	Mean	S. D.	Min	Max
Cancer SMR	100.7	11.6	63.8	135.5
Cancer observed deaths	1143	674.1	241	3131
Cancer expected deaths	1167	750.6	224	3533
Circulatory SMR	100.8	11.2	60.5	135.1
Circulatory observed deaths	1399	851.1	269	3895
Circulatory expected deaths	1427	935.9	257	4345
Deprivation	21.8	9.2	8.1	48.3
Cancer spending per affected person	18072.29	3640.9	9894.67	30567.07
Cancer spending per person	90.95	16.6	45.79	152.12
Cancer prevalence	0.005	0.001	0.002	0.008
Circulatory spending per affected person	695.05	105.5	485.58	1260.23
Circulatory spending per person	125.57	19.1	58.36	221.76
Circulatory prevalence	0.18	0.03	0.11	0.25
Non-cancer spending per person	1360.85	68.6	1203.05	1899.94
Non-circulatory spending per person	1326.24	69.2	1164.54	1845.29
Proportion of Unpaid Carers	0.10	0.011	0.07	0.12
Population size	427767	236724.4	88599	1229304

Table 1: Summary Statistics (n=147)

4 Results

Table 2 shows the results for the cancer programme of care. All variables are log-transformed and so coefficients can be interpreted as elasticities.The column under"OLS" displays the results for Ordinary Least Squares estimation, which assumes exogeneity of the regressors. The model under "IV" uses the

first stage instruments $OtherExpenditure$ and $CarerPercentage$ to derive predicted values for $CancerExpenditure$ free from endogeneity.

	OLS		IV	
Variable	Coef	S.E	Coef	S.E
First Stage				
$CancerExpenditure$:				
$Constant$			15.858	(1.994)***
$Deprivation$			0.151	(0.039)***
$OtherExpenditure$			-1.044	(0.262)***
$CarerPercentage$			-0.473	(0.123)***
Second Stage				
$CancerSMR$:				
$Constant$	4.499	(0.396)***	8.392	(1.537)***
$Deprivation$	0.189	(0.018)***	0.244	(0.0275)***
$CancerExpenditure$	-0.047	(0.043)	-0.462	(0.162)***
Tests	Value	P value	Value	P value
First Stage F(2,143)			20.5	0.00
Second Stage F(2,144)			43.18	0.00
Hansen J overID			0.988	0.3203
Kleibergen-Paap underID			17.94	0.0001
Shea Partial R^2			0.1646	
Endogeneity test			6.969	0.008
RESET test	1.26	0.291	0.31	0.576

Table 2: Cancer Programme. *** is significant at the 1% level; ** at the 5% level; * at the 10% level (n=147)

The endogeneity test rejects the null hypothesis that expenditure is exogenous. Also the OLS estimates deviate substantially from the instrumented model, particularly the coefficient on expenditure which is not significantly different from zero. This implies the Instrumental Variables are required to get accurate results and the OLS estimates are biased. Within the IV model, all the coefficients attain significance at the 1% level. In the first stage, deprivation is positively related to spending on cancer treatment, whereas expenditure on other illnesses reduces the amount spent on cancer. Similarly, the percentage of unpaid carers reduces the amount spent on cancer.

In the second stage, cancer mortality is positively correlated with de-

privation and negatively correlated with spending on cancer, as one would expect. The elasticity of -0.462 on expenditure means that an increase in spending per cancer patient of 10% would result in a fall in SMR of 4.6%, other things being equal. As deprivation is measured by an index, a similar meaning cannot be infered from the elasticity of 0.244, though the sign and and significance of the coefficient leaves no doubt that deprivation is highly correlated with standardised mortality.

In terms of tests for specification and legitimacy, the model performs well. The Hansen statistic doesn't reject the null hypothesis that the instruments are valid, the Kleibergen-Paap rejects the null hypothesis that the equation is under-identified, and the RESET test does not reject the null hypothesis that the equation is linear in its regressors.

Unfortunately, there are problems with the circulatory model, as shown in Table 3. Firstly, the endogeneity test fails to reject the null hypothesis that the expenditure variable is exogenous. Secondly, the Hansen J statistic rejects the null hypothesis that the instruments are valid. The failure to reject exogeneity in the former test may be caused by the failure to find good enough instruments revealed in the latter test. The Kleibergen-Paap test for under-identification and the RESET test for specification are both passed. Overall, this raises a question as to how well specified the model is, as the Hansen J test indicates the instruments may be correlated with the error term.

In the first stage of the IV regression the instruments have similar coefficients to those in the cancer model. Both are negative and spending on other programmes has a substantially bigger coefficient than the percentage of unpaid carers in the PCT. However the relationship between deprivation and circulatory spending is markedly different to that between deprivation and cancer spending, as it is negative and fails to attain any statistical significance. In the IV estimation of the circulatory model this is no problem; the Needs variable is not an instrument. Though when decomposing the concentration index this negative relationship will be important.

The results suggest the effect of both deprivation and health care expenditure are less for the circulatory programme than that of cancer. The estimated elasticity of Circulatory Expenditure with respect to circulatory standardised mortality is -0.32, indicating a 10% increase in expenditure per affected person will reduce standardised mortality by 3.2%.

	OLS		IV	
Variable	Coef	S.E	Coef	S.E
First Stage				
CirculatoryExpenditure				
Constant			12.52	(1.91)***
Deprivation			-0.003	(0.02)
OtherExpenditure			-0.99	(0.26)***
CarerPercentage			-0 .48	(0.11)***
Second Stage				
CirculatorySMR				
Constant	4.47	(0.482)***	6.16	(0.90)***
Deprivation	0.177	(0.014)***	0.17	(0.016)***
CirculatoryExpenditure	-0.061	(0.072)	-0.32	(0.14)**
Tests	Value	P value	Value	P value
First Stage F(2,143)			17.87	0.00
Second Stage F(2,144)			78.18	0.00
Hansen J overID			5.51	0.02
Kleibergen-Paap underID			17.89	0.0
Shea Partial R^2			0.29	
Endogeneity test			1.021	0.312
RESET test	0.96	0.413	0.01	0.93

Table 3: Circulatory Programme. *** is significant at the 1% level; ** at the 5% level; * at the 10% level (n=147)

Figure 1 shows the concentration curves for observed cancer deaths and the expected cancer deaths based on the area's age distribution are both pro-rich, by plotting the cumulative share of deaths across PCTs in descending order of deprivation. It is clear to see that, while richer areas suffer higher rates of mortality, the age of those living in the area would suggest more deaths. This means that richer areas have relatively lower rates of mortality compared with deprived areas when age is controlled for. The 'amount' by which the poor are worse off is the area between the red and blue lines, between the mortality expected and the mortality observed.

The underlying model on which the decomposition is based is that of the IV model displayed in Table 2. The concentration index was calculated using the 'convenient regression' approach [7]. However, in order to use a

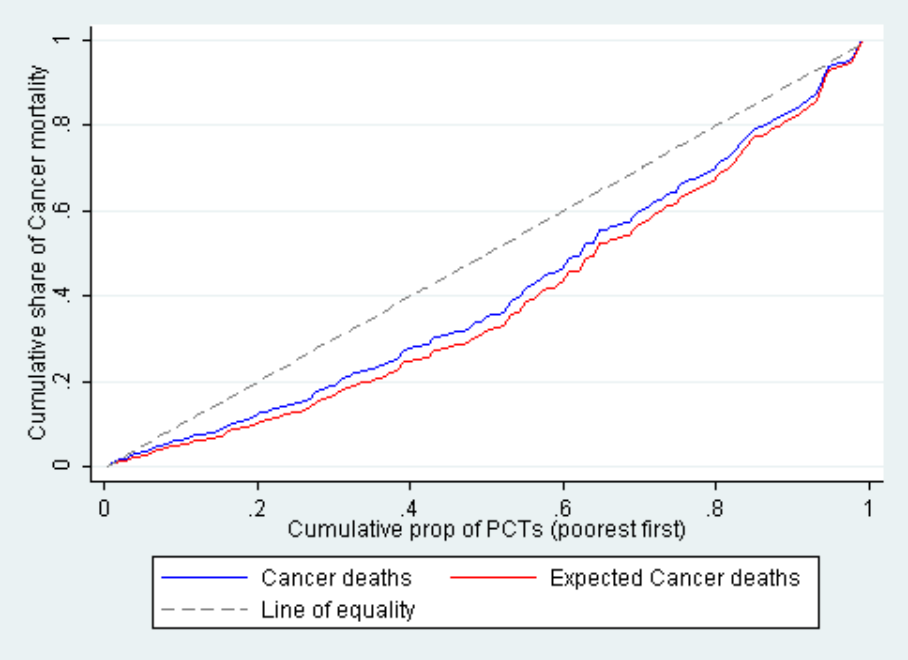

Figure 1: The observed and expected cancer mortality concentration curve

meaningfully summable measure of mortality, the condition-specific SMR cannot be used by itself. In its place, expected deaths is included as an additional covariate. This is extracted from the SMR by dividing observed deaths by the SMR. The variable produced is the number of deaths indirectly age-standardised.

$$\begin{aligned} ObservedCancerdeaths = \alpha + \beta_1 ExpectedCancerdeaths + \beta_2 Deprivation \\ + \beta_3 CancerExpenditure + \nu \end{aligned} \tag{4}$$

The results for regression in Equation (4) are presented in Table 4. This regression is used to find the elasticities in the decomposition of the cancer concentration index. Note none of the variables are in logarithms. Overall, the results are similar to the results in Table 2 in sign and significance. Note that expected deaths are positively correlated with observed deaths.

Variable	Coef	S.E
First Stage		
$CancerExpenditure$		
$Constant$	41477.8	(4226.1)***
$ExpectedCancerdeaths$	-0.822	(0.058)
$Deprivation$	85.4	(43.3)*
$OtherExpenditure$	-12.3	(3.1)***
$CarerPercentage$	-74801.4	(23827.4)***
Second Stage		
$Cancerdeaths$		
$Constant$	333.3	(123.2)***
$ExpectedCancerdeaths$	0.92	(0.024)***
$Deprivation$	8.4	(1.3)***
$CirculatoryExpenditure$	-0.02	(0.006)***
Tests	Value	P value
First Stage F(4,142)	14.48	0.00
Second Stage F(3,143)	975.2	0.00

Table 4: Cancer Concentration Decomposition regression. *** is significant at the 1% level; ** at the 5% level; * at the 10% level (n=147)

$$\begin{aligned} CI_{Obsdeaths} = {} & e_{Expdeaths} CI_{Expdeaths} + e_{Deprivation} CI_{Deprivation} \\ & + e_{Spending} CI_{Spending} + GCI_{\epsilon} \end{aligned} \tag{5}$$

$$\begin{aligned} CI_{Standardiseddeaths} &= CI_{Obsdeaths} - e_{Expdeaths} CI_{Expdeaths} \\ &= e_{Deprivation} CI_{Deprivation} + e_{Spending} CI_{Spending} + GCI_{\epsilon} \end{aligned} \tag{6}$$

Table 5 shows the results of the concentration index and decomposition of cancer mortality from Equation (5), where CI_x is the concentration index of x with respect to the deprivation rank, e_x is the elasticity of x with respect to observed cancer deaths and GCI_{ϵ} is the generalised concentration index of the error term [7]. Once the concentration index of cancer deaths has been decomposed into its covariates, the observed mortality concentration index can be standardised by subtracting the contribution of expected deaths as in

Equation (6). This is done in the upper and lower horizontal level of Table 5, where the double vertical line distinguishes the left hand side of the equation from the right hand side.

Cancer	Observed Deaths	Expected Deaths	Expenditure	Deprivation
C.I.	0.184	0.229	-0.032	-0.237
Bootstrap S.E	(0.036)***	(0.037)***	(0.005)***	(0.023)***
Elasticity	-	0.93	-0.401	0.171
B.S.E		(0.019)***	(0.102)***	(0.031)***
Contribution	-	0.213	0.013	-0.041
B.S.E		(0.034)***	(0.004)***	(0.008)***
	Standardised Deaths		Expenditure	Deprivation
C.I.	-0.029		-0.032	-0.237
B.S.E	(0.006)***		(0.005)***	(0.023)***
Elasticity	-		-0.401	0.171
B.S.E			(0.102)***	(0.031)***
Contribution	-		0.013	-0.041
B.S.E			(0.004)***	(0.008)***
% Contribution	-		-44.1	140

Table 5: Cancer Concentration Decomposition. *** is significant at the 1% level; ** at the 5% level; * at the 10% level. Bootstrapped standard errors are based on 1000 replications

The concentration index analysis reveals a pro-poor distribution of mortality from cancer and expenditure on cancer, as would be expected. From the previous regression model it is already apparent that funding cancer treatment reduces cancer mortality, so the negative elasticity of cancer expenditure coupled with cancer spending's pro-poor distribution results in Expenditure's positive contribution. In simpler terms, spending money on cancer treatment reduces cancer deaths, cancer deaths and cancer spending are concentrated in the poorer PCTs so overall the effect of cancer spending alleviates the pro-poor distribution of cancer deaths. A similar description of the results applies to deprivation - deprivation is higher in deprived areas, deprivation increases cancer mortality so deprivation exacerbates the pro-poor distribution of cancer mortality. However, deprivation is the ranking measure for social class and does not exist in a natural measure of units so the precise meaning of its estimates in the decomposition is not clear; it is

mainly of use to control for deprivation.

Overall, the decomposition works well, with contribution of expenditure and deprivation explaining 95.9% of the standardised concentration index, leaving only 4.1% unexplained as the generalised concentration index of the error term.

Similar to the cancer concentration curve, Figure 2 shows the concentration curves for observed circulatory deaths and the expected circulatory deaths based on the area's age distribution are both pro-rich also. Again, while richer areas suffer higher rates of mortality, the age of those living in the area would suggest more death. This means that richer areas have relatively lower rates of mortality compared with deprived areas when age is controlled for. The area between the red and blue lines is the degree of pro-poor standardised mortality.

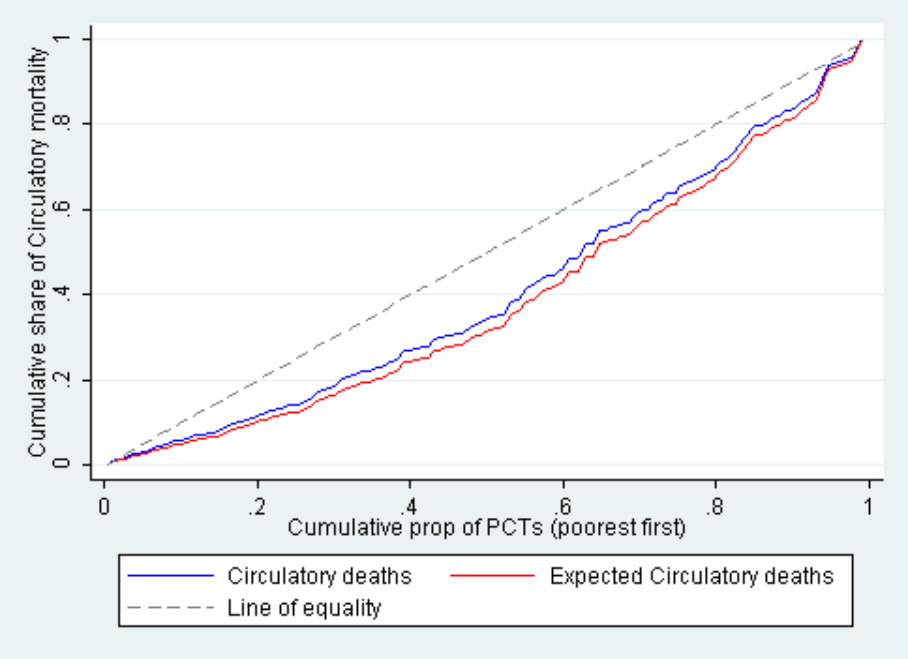

Figure 2: The observed and expected circulatory mortality concentration curve

As with cancer, the standardisation requires a regression of observed mortality on expected mortality, deprivation and programme expenditure, as in Equation (7). The results are reported in Table 6.

$$ObservedCirculatorydeaths = \alpha + \beta_1 ExpectedCirculatorydeaths + \beta_2 Deprivation + \beta_3 CirculatoryExpenditure + \nu \quad (7)$$

Variable	Coef	S.E
First Stage		
CirculatoryExpenditure		
Constant	1752.8	(235.6)***
ExpectedCirculatorydeaths	-0.02	(0.007)***
Deprivation	-1.1	(0.8)
OtherExpenditure	-0.5	(0.16)***
CarerPercentage	-3414.3	(825.6)***
Second Stage		
Circulatorydeaths		
Constant	368.6	(160.5)**
ExpectedCirculatorydeaths	0.93	(0.03)***
Deprivation	6.2	(1.1)***
CirculatoryExpenditure	-0.62	(0.19)***
Tests	Value	P value
First Stage F(4,142)	7.38	0.00
Second Stage F(3,143)	580.2	0.00

Table 6: Circulatory Concentration Decomposition regression. *** is significant at the 1% level; ** at the 5% level; * at the 10% level (n=147)

The results for regression in Equation (7) are presented in Table 6. As with cancer, this regression is used to find the elasticities in the decomposition of the circulatory concentration index and none of the variables are in logarithms. The results are similar to the results in Table 3 in sign and significance. Using the same standardisation as Equation (6), Table 7 shows the results of the concentration index and decomposition of circulatory disease mortality. As before, expected deaths are positively correlated with observed deaths.

Circulatory mortality displays a similar pro-poor distribution. As before, the elasticities of circulatory spending and depriviation coincide with the re-

Circulatory	Deaths	Expected Deaths	Expenditure	Deprivation
Concentration Index	0.192	0.234	0.004	-0.237
Bootstrap S.E	(0.036)***	(0.037)***	(0.003)	(0.023)***
Elasticity	-	0.941	-0.317	0.103
B.S.E		(0.019)***	(0.092)***	(0.022)***
Contribution	-	0.22	-0.001	-0.025
B.S.E		(0.034)***	(0.001)	(0.005)***
	Standardised Deaths		Expenditure	Deprivation
Concentration Index	-0.028		0.004	-0.237
B.S.E	(0.005)***		(0.003)	(0.023)***
Elasticity	-		-0.317	0.103
B.S.E			(0.092)***	(0.022)***
Contribution	-		-0.001	-0.025
B.S.E			(0.001)	(0.005)***
% Contribution	-		4.3	87.5

Table 7: Circulatory Concentration Decomposition. *** is significant at the 1% level; ** at the 5% level; * at the 10% level. Bootstrapped standard errors are based on 1000 replications

gression model's estimates. As before, the concentration index of deprivation indicates a pro-poor distribution of deprivation. However, in contrast to cancer spending's pro-poor distribution, circulatory disease's expenditure is not significantly pro-poor or pro-rich. This means that although spending on circulatory disease is effective, the overall effect of circulatory disease spending does not change the distribution of circulatory death between rich and poor. It should be noted, however, that the specific elasticity estimate is based on the model for circulatory disease which fails the Hansen J test of instrument validity, so should be treated with caution. Ideally we would like more, and better, instruments.

The circulatory decomposition does not do quite as well as the cancer decomposition in explaining the standardised mortality concentration index. Together, expenditure and deprivation only account for 91.8% of the concentration index, leaving 8.2% to be explained by the generalised concentration index of the error term.

5 Conclusion

This analysis shows there is a real benefit to health from health care spending across the cancer and circulatory disease programmes of care. Furthermore, it describes how the distribution of health care funding across PCTs in areas of affluence and poverty affects the inequality in health outcomes in England.

The model for cancer largely supports the results of Rice et al's paper. The coefficient on expenditure of -0.462 is very close to the original paper's value of -0.491. It is likely that the small difference between these estimates is due to either the transformation of expenditure from spending per person to spending per affected person, or a change over time in the elasticity of spending. The model now more specifically measures the effect of condition-specific clinical spending on health outcome.

The circulatory disease regression results suggest the effect of both deprivation and health care expenditure are less for the circulatory programme than that of cancer. Previously the opposite has been true – the effect of deprivation and health care expenditure have been substantially greater for the circulatory programme of care. In particular, the elasticity of spending with respect to mortality is -0.32, which sharply contrasts with the previous estimate of -1.387. This might be caused by the transformation of the expenditure variable from per person to per affected person, or be due to the poor quality of the instruments as evidenced by the Hansen J statistic. In previous papers the percentage of the population describing themselves as in the white ethnic group in the 2001 census has also been included as an instrument, but was not significant when included as an instrument in this model. This may be due to the change in distribution of ethnic minorities due to immigration over the previous eight years.

The concentration index decompositions, though standardised for gender and age in a different way, further support the elasticity estimates found in the regressions. The surprising result that circulatory disease spending is largely even between richer and poorer areas indicates the increased funding overall of poorer PCTs does not translate to high spending on circulatory diseases. This is possibly due to the relatively older age of circulatory disease deaths – 75% of circulatory deaths are over the age of 75, whereas 52% of cancer deaths are – which would mean ages with a higher life expectancy would find circulatory disease a relatively more pressing concern.

It is interesting that the results for cancer and circulatory disease appear to show cancer spending as being the more efficacious. This seems to contra-

dict previous results that show circulatory disease being the cheaper to treat to the same outcome. However, the estimate must be treated with caution as the unit in this study is spending per affected person instead of simply per person. Further data on prevalence in years other than 2007 would permit analysis to be undertaken as to whether this is due to the change in measure or a real change over time.

Furthermore, it would be interesting to have more outcome data for other programmes. This would allow analysis of other programmes which include more palliative care. Currently, models such as this one are constrained by the availability of simple health outcome data.

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