



Socioeconomic inequalities in the rate of stillbirths by cause: a population-based study

Sarah E Seaton,¹ David J Field,¹ Elizabeth S Draper,¹ Bradley N Manktelow,¹ Gordon C S Smith,² Anna Springett,³ Lucy K Smith¹

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¹Department of Health Sciences, University of Leicester, Leicester, UK ²Department of Obstetrics and Gynaecology, NIHR Biomedical Research Centre, University of Cambridge, Cambridge, UK ³Centre of Environmental and Preventive Medicine, Queen Mary, University of London, London, UK

Correspondence to Sarah E Seaton; sarah.seaton@le.ac.uk

ABSTRACT

Objective: To assess time trends in socioeconomic inequalities in overall and cause-specific stillbirth rates in England.

Design: Population-based retrospective study. **Setting:** England.

Participants: Stillbirths occurring among singleton infants born between 1 January 2000 and 31 December 2007.

Main outcome measure: Cause-specific stillbirth rate per 10 000 births by deprivation tenth and year of birth. Deprivation measured using the UK index of multiple deprivation at Super Output Area level.

Methods: Poisson regression models were used to estimate the relative deprivation gap (comparing the most and least deprived tenths) in rates of stillbirths (overall and cause-specific). Excess mortality was calculated by applying the rates seen in the least deprived tenth to the entire population at risk. Discussions with our local NHS multicentre ethics committee deemed that this analysis of national nonidentifiable data did not require separate ethics approval.

Results: There were 44 stillbirths per 10 000 births, with no evidence of a change in rates over time. Rates were twice as high in the most deprived tenth compared with the least (rate ratio (RR) 2.1, 95% Cl 2.0 to 2.2) with no evidence of a change over time. There was a significant deprivation gap for all specific causes except mechanical events (RR 1.2, 95% Cl 0.9 to 1.5). The widest gap was seen for stillbirths due to antepartum haemorrhages (RR 3.1, 95% Cl 2.8 to 3.5). No evidence of a change in the rate of stillbirth or deprivation gap over time was seen for any specific cause.

Conclusion: A wide deprivation gap exists in stillbirth rates for most causes and is not diminishing. Unexplained antepartum stillbirths accounted for 50% of the deprivation gap, and a better understanding of these stillbirths is necessary to reduce socioeconomic inequalities.

ARTICLE SUMMARY

Article focus

- To explore time trends in socioeconomic inequalities in cause-specific stillbirths in England over an 8-year period.
- To aid understanding of the deprivation gap in overall and cause-specific stillbirth rates.

Key messages

- A wide deprivation gap exists in the rates of stillbirth, and this has remained constant over time.
- Significant deprivation differences were seen between the most and least deprived groups in all causes except mechanical events that occurred during labour.
- Future collection of more detailed information is necessary in order to better identify modifiable risk factors and thus permit the introduction of appropriate targets and interventions.

Strengths and limitations of this study

- Individual-level information was not available, and therefore, we could not explore issues such as smoking status.
- However, these analyses based on routine data are straightforward to undertake and allow health service planners to monitor trends in stillbirths.

INTRODUCTION

Despite improvements in healthcare in developed countries, stillbirth remains a common adverse pregnancy outcome¹ with particularly high rates in the UK.² In a study of stillbirth rates in 13 developed countries around the world including the USA, Canada, Australia and European nations, the UK was shown to have the highest rate of stillbirth in recent years.² This problem remains apparently intractable with little or no improvement in rates over time unlike the reductions seen for neonatal mortality.³ Consequently, stillbirths have become the largest contributor to perinatal mortality (in 2009 stillbirths accounted for 68% of perinatal deaths)³ and are a major public health

burden that is frequently overlooked since still births are generally not included in international comparisons of maternal and infant health.^{1 4}

This burden does not affect all groups alike with socioeconomic inequalities in stillbirth rates existing in the UK and internationally^{2 5-10} with women at higher risk of stillbirth in deprived areas. These socioeconomic inequalities do not appear to be accounted for even after adjusting for factors such as attendance at antenatal appointments or previous reproductive history¹¹ although some of the deprivation gap is explained by smoking.¹² Little is known on differences in the deprivation gap by specific causes of stillbirth in the UK. Stillbirths are not a homogeneous group, with a variety of possible causes potentially resulting in a stillbirth. Classifying stillbirths into specific causes is extremely difficult, but they are known to be linked to certain factors such as placental abruption, congenital anomalies and intrapartum events. Variation in the deprivation gap for different causes has been noted in neonatal mortality,¹³ and these causespecific socioeconomic inequalities are likely to also exist in stillbirths. Research has noted that increased deprivation was associated with increased perinatal mortality due to non-chromosomal anomalies¹⁴ and also associated with stillbirths occurring for unknown reasons prior to labour.⁵ However, these and other studies have been based on relatively small populations and addressed a limited number of causes.⁵¹⁴¹⁵

In the UK, the Stillbirth and Neonatal Death Society (SANDS) have campaigned for stillbirths to be researched further, highlighting deprivation as a risk factor for stillbirth.¹⁶ There has been no recent evidence

related to the effect of deprivation on the overall stillbirth rate or indeed whether the deprivation gap has changed over time. Here, we explore time trends in socioeconomic inequalities in cause-specific stillbirths in England over an 8-year period to aid understanding of each cause's impact on the deprivation gap and the overall stillbirth rate.

METHODS

Data on all singleton stillbirths (losses from the 24th weeks of gestation) born to mothers resident in England between 1 January 2000 and 31 December 2007 were obtained from the Centre for Maternal and Child Enquiries (CMACE) that collected stillbirth data as part of its national perinatal mortality surveillance work funded by the National Patient Safety Agency. Data included cause of death, gestational age and Super Output Area (SOA) (geographical populations of approximately 1500 residents) of mother's residence. A local CMACE coordinator in each maternity hospital initially classified deaths by using the Obstetric (Aberdeen) classification system.⁷ A CMACE regional manager then checked them with reference to postmortem and coroner's reports where available. Finally, CMACE carried out central cross validation checks to ensure consistency. We amalgamated several of the rarer classification groups and divided unexplained antepartum deaths on the basis of birth weight (≤ 10 th centile or >10th centile) resulting in nine categories: congenital anomalies, pre-eclampsia, antepartum haemorrhage, mechanical, maternal disorder, miscellaneous, unexplained and small for gestational age, unexplained and

Category	Comprised deaths due to:				
Congenital anomalies	Neural tube defects				
	Other anomalies				
Pre-eclampsia	Pre-eclampsia without antepartum haemorrhaging				
	Pre-eclampsia complicated by antepartum haemorrhaging				
Antepartum haemorrhage	Antepartum haemorrhage with placental praevia				
	Antepartum haemorrhage with placental abruption				
	Antepartum haemorrhage of uncertain origin				
Mechanical	Cord prolapsed or compression with vertex or face presentation				
	Other vertex or face presentation				
	Breech presentation				
	Oblique or compound presentation, uterine rupture, etc.				
Maternal disorder	Maternal hypertensive disease				
	Other maternal disease				
	Maternal infection				
Miscellaneous	Isoimmunisation due to rhesus or other antigens				
	Neonatal infection				
	Other neonatal infection				
	Specific fetal condition				
Unexplained antepartum SGA	Unexplained antepartum (birth weight \leq 10th centile)				
Unexplained antepartum not SGA	Unexplained antepartum (birth weight >10th centile)				
Unclassifiable	Unclassified				
	Missing				

not small for gestational age and unclassifiable (table 1).¹⁷ As this study is based on routinely collected data that were anonymised, there was no requirement for ethical approval.

Denominator data on the total number of live singleton births by SOA and year of birth were obtained from the UK Office of National Statistics (www.statistics.gov.uk). The number of live births in each SOA was added to the number of stillbirths to produce denominator data of the total number of births. We only included singleton births since differential access to fertility treatment might have led to higher incidence of multiple births in less deprived areas, and the stillbirth rate of multiple births is known to be higher than that of singletons.

Socioeconomic differences were measured using an area-level measure, assigning the Index of Multiple Deprivation score (IMD) for 2004¹⁸ to the SOA provided by CMACE (geographical populations of approximately 1500 residents) of the mother's residence at the time of delivery. The IMD 2004 score is made up of seven factors relating to income; employment; health and disability; education, skills and training; barriers to housing; living environment and crime. Although some degree of heterogeneity will exist between areas, the small size of SOAs limits this. Only stillbirths with a valid SOA were included; otherwise, no deprivation score could be assigned. All SOAs in England were ranked by IMD 2004 score and divided into 10 groups with approximately equal numbers of live births (tenths) from 1: least deprived to 10: most deprived. Ten groups were used as we had a large number of births, and this allowed better investigation of the differences between the most and least deprived. If the stillbirth rate was the same irrespective of deprivation, we would therefore expect similar numbers of stillbirths across all tenths.

We calculated the rate of stillbirths, both overall and for each specific cause occurring in each deprivation tenth. Rates were calculated per 10000 births due to small numbers of stillbirths occurring in certain causes. Year of birth was categorised into two time periods: 2000-2003 and 2004-2007. Poisson regression models were fitted to assess changes over time in overall stillbirth rates and by specific cause. In order to measure the *relative* deprivation gap, rather than just comparing the most and least deprived tenths that would only partially use the data, we treated deprivation tenth as a linear term and then the mortality rate ratio (RR) between the fitted values for the most deprived and least deprived tenths was calculated. This is similar in approach to the relative index of inequality.¹⁹ A separate deprivation effect for each time period was tested to assess whether there was a significant change in the relative deprivation gap over time. The absolute change in stillbirth rates over time by deprivation tenth was also calculated. Excess mortality was calculated by considering how many stillbirths would be expected if the rate observed in the least deprived tenth was applied to the whole population and dividing that by the total number of deaths observed.

The proportion of the overall deprivation gap explained by each cause of stillbirth was calculated. For each cause, the rate in the least and most deprived tenths was estimated from the Poisson regression models. The absolute difference in these rates was then calculated and expressed as a proportion of the absolute difference in the rates overall. This was calculated for 2000–2003 and 2004–2007 and displayed graphically with a line drawn to join these two time periods.

RESULTS

All-cause stillbirth mortality

From 2000 to 2007, there were 21 472 singleton stillbirths reported to CMACE of which 120 (0.6%) had a missing SOA and 919 (4.3%) had missing or unclassifiable cause of death leaving 20433 for analyses. The overall stillbirth rate was 44/10000 births. There was no evidence of a change in stillbirth rate over time (2000-2003 rate: 44/10000, 2004-2007: 44/10000, p=0.80). The total number of stillbirths in each deprivation tenth increased as deprivation increased (table 2) with approximately double the number in the most deprived tenth compared with the least deprived. Women from the most deprived tenth were twice as likely to experience a stillbirth due to any cause as those from the least deprived (table 3: RR 2.1, 95% CI 2.0 to 2.2) (p<0.0001). There was no evidence that this changed over time (table 4: p=0.26). The percentage of all-cause excess deaths related to deprivation was 33% suggesting in total a third more stillbirths were observed than we would have expected if the stillbirth rate were the same for all deprivation groups as the least deprived tenth.

Cause-specific stillbirth mortality

Table 2 shows stillbirths by cause of death with antepartum deaths of unknown cause being the most common (59.2% (21.3% small for gestational age; 37.9% not small for gestational age)) followed by antepartum haemorrhage (13.0%), maternal disorders (9.1%), congenital anomalies (7.8%), pre-eclampsia (4.2%) and mechanical issues during labour (2.4%). The remaining 4.3% were due to miscellaneous or unclassified reasons and were excluded from the Poisson regression analyses.

There was no evidence of trends of increasing or decreasing rates of stillbirth over time for any specific cause (table 4); however, the deprivation gap varied by cause. Stillbirths relating to mechanical issues during labour were the only specific cause where there was no evidence of a deprivation gap (RR 1.2, 95% CI 0.9 to 1.5). All other causes showed a significant deprivation gap in stillbirth rates varying from a 1.7- to 3.1-fold difference (table 3). The widest deprivation gap was seen for deaths due to antepartum haemorrhage, and pregnancies from the most deprived tenth were 3.1 (95% CI 2.8 to 3.5) times more likely to result in stillbirth than those from the least deprived tenth. Wide deprivation gaps were also seen for deaths due to congenital anomalies (RR 2.8, 95% CI 2.4 to 3.3) and maternal disorders

Table 2 Number (%) of live births and stillbirths (by specific cause) and deprivation tenth											
	Depriva	eprivation tenth 1=least deprived, 10=most deprived									
	1	2	3	4	5	6	7	8	9	10	Total
Live births	463 148	464 092	464 487	465 295	465 814	466 377	467 227	467 767	468 144	469 044	4 661 395
Stillbirths											
All cause	1489	1526	1642	1783	1991	2099	2372	2647	2760	3043	21 352
Cause-specific											
stillbirths											
Congenital	106	111	116	114	159	132	184	229	258	258	1667 (7.8)
anomalies											
Pre-eclampsia	60	66	70	71	80	96	121	112	111		894 (4.2)
Antepartum	141	152	185	229	261	287	302	366	372	478	2773 (13.0)
haemorrhage											
Mechanical	52	40	46	46	51	67	52	55	47		512 (2.4)
Maternal disorder	130	126	157	156	190	181	222	258	275		1946 (9.1)
Miscellaneous	26	32	37	31	25	32	38	47	42	47	357 (1.7)
(including											
isoimmunisation)											
Unknown	293	290	351	385	374	438	527	574	613	709	4554 (21.3)
antepartum (SGA)	050		005	704	700		074	0.40		1051	
Unknown	650	682	635	701	790	802	874	942	960	1051	8087 (37.9)
antepartum											
(not SGA)	01	07	45	50	61	C 4	50	C 4	00	00	FCO (0 C)
Unclassifiable	31	27	45	50	61	64	52	64	82	86	562 (2.6)
SGA, small for gestation	nal age.										

Table 2 Number (%) of live births and stillbirths (by specific equips) and deprivation

such as hypertension (RR 2.2, 95% CI 1.9 to 2.5). The deprivation gap was wider for stillbirths that were small for gestational age (RR 2.5, 95% CI 2.3 to 2.7) than those that were not small for gestational age (RR 1.7, 95% CI 1.5 to 1.8).

Figure 1 demonstrates the percentage of the deprivation gap in all-cause stillbirth mortality explained by each specific cause estimated from the Poisson regression models. Deaths due to unexplained antepartum events explain 50% of the deprivation gap. Despite the small for gestational age births forming a smaller group (21.3% of stillbirths) than those that were appropriately grown (37.9% of stillbirths), they explain more of the deprivation gap since the associated deprivation gap is wider for the small for gestational age stillbirths. There was no evidence of a change in the proportion of the deprivation gap explained by any of the different causes

over time that can be seen by the lack of change in the gradient of the lines explaining each specific cause. Mechanical causes are seen to represent a very small, insignificant proportion of the deprivation gap.

DISCUSSION

This study estimated time trends in the deprivation gap in stillbirth by cause of death for which there has been limited recent published data. Here, we have shown wide socioeconomic inequalities in the rate of stillbirth with rates twice as high in the most deprived areas compared with the least deprived. If the stillbirth rates seen in the least deprived areas were seen throughout the population, there would be a third less stillbirths in England, nearly 900 fewer each year. Significant deprivation differences between the most and least deprived were seen in all causes except mechanical issues that occurred

	Excess mortality (%)	Rate ratio (95% CI)	Test for effect of deprivation tenth, p-value		
All causes	33	2.1 (2.0 to 2.2)	<0.0001		
Cause-specific stillbirths		· · ·			
Congenital anomalies	44	2.8 (2.4 to 3.3)	<0.0001		
Pre-eclampsia	30	2.0 (1.6 to 2.4)	<0.0001		
Antepartum haemorrhage	53	3.1 (2.8 to 3.5)	<0.0001		
Mechanical	8	1.2 (0.9 to 1.5)	0.241		
Maternal disorder	35	2.2 (1.9 to 2.5)	<0.0001		
Unknown antepartum (SGA)	39	2.5 (2.3 to 2.7)	<0.0001		
Unknown antepartum (not SGA)	23	1.7 (1.5 to 1.8)	<0.0001		

Table 4Observed rates of stillbirth per 10 000 births by deprivation tenth and year of delivery and estimated change in
mortality over time (based on Poisson regression model) with 95% CIs

			Change in mortality from 2000–2003 to 2004–2007		
	2000–2003	2004–2007	Absolute change per 10 000 births	Relative change (%)	
All stillbirths, N=20433					
Least deprived	29.3 (28.1 to 30.5)	29.3 (26.0 to 32.9)	-0.3 (-2.0 to 1.4)	1.0 (0.9 to 1.0)	
Most deprived	61.9 (59.9 to 64.0)	61.2 (59.3 to 63.2)	-0.7 (-3.5 to 2.2)	1.0 (0.9 to 1.0)	
Cause-specific stillbirths	, , , , , , , , , , , , , , , , , , ,	· · · · · · · · · · · · · · · · · · ·	, , , , , , , , , , , , , , , , , , ,	, , , , , , , , , , , , , , , , , , ,	
Congenital anomalies,					
N=1667 (8.1%)					
Least deprived	2.0 (1.8 to 2.4)	1.7 (1.1 to 2.6)	-0.1 (-0.5 to 0.4)	1.0 (0.8 to 1.2)	
Most deprived	6.3 (5.6 to 7.0)	5.2 (4.6 to 5.8)	-1.1 (-2.0 to -0.2)	0.8 (0.7 to 1.0)	
Pre-eclampsia,					
N=894 (4.0%)					
Least deprived	1.4 (1.2 to 1.7)	1.4 (0.8 to 2.5)	-0.2 (-0.6 to 0.2)	0.9 (0.7 to 1.1)	
Most deprived	2.8 (2.4 to 3.3)	2.4 (2.1 to 2.9)	-0.4 (-1.0 to 0.2)	0.9 (0.7 to 1.1)	
Antepartum haemorrhage,					
N=2773 (14.0%)					
Least deprived	3.3 (2.9 to 3.7)	3.1 (2.2 to 4.3)	-0.2 (-0.7 to 0.3)	0.9 (0.8 to 1.1)	
Most deprived	10.5 (9.7 to 11.4)	9.2 (8.5 to 10.1)	-1.2 (-2.4 to -0.1)	0.9 (0.8 to 1.0)	
Mechanical, N=512 (2.5%)					
Least deprived	0.9 (0.7 to 1.2)	0.7 (0.3 to 1.4)	0.2 (-0.2 to 0.5)	1.2 (0.8 to 1.6)	
Most deprived	1.3 (1.0 to 1.6)	1.1 (0.9 to 1.4)	-0.2 (-0.5 to 0.2)	0.9 (0.6 to 1.2)	
Maternal disorder,					
N=1946 (9.5%)					
Least deprived	2.5 (2.2 to 2.8)	2.2 (1.5 to 3.2)	0.5 (-0.03 to 1.0)	1.2 (1.0 to 1.4)	
Most deprived	5.9 (5.3 to 6.6)	6.1 (5.5 to 6.7)	0.2 (-0.7 to 1.1)	1.0 (0.9 to 1.2)	
Unknown antepartum SGA,					
N=4554 (22.3%)					
Least deprived	6.0 (5.5 to 6.6)	6.1 (4.7 to 7.8)	-0.2 (-0.9 to 0.6)	1.0 (0.9 to 1.1)	
Most deprived	14.9 (13.9 to 16.0)	14.7 (13.7 to 15.7)	-0.2 (-1.6 to 1.2)	1.0 (0.9 to 1.1)	
Unknown antepartum not SGA,					
N=8087 (39.6%)					
Least deprived	13.5 (12.6 to 14.3)	15.2 (12.7 to 18.3)	-0.3 (-1.5 to 0.8)	1.0 (0.9 to 1.1)	
Most deprived SGA, small for gestational age.	20.9 (19.7 to 22.1)	23 (21.9 to 24.2)	2.1 (0.5 to 3.8)	1.1 (1.0 to 1.2)	

SGA, small for gestational age.

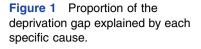
during labour. Half of the excess stillbirths attributed to deprivation were of unknown cause.

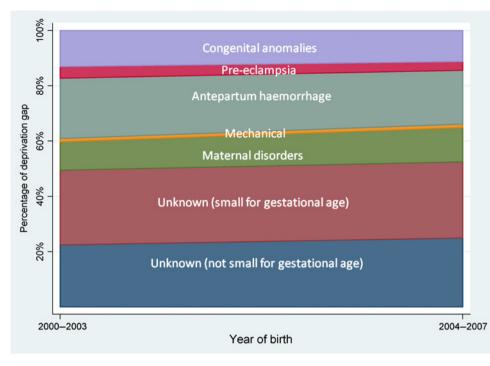
Our findings of wide inequalities in stillbirth rates confirm the continuation of patterns seen in previous research.^{3 5} However, the static nature of the overall stillbirth rate in England in recent years is in contrast to the views of the CMACE report of 2009.³ We have previously shown widening inequalities in neonatal mortality,¹³ with larger reductions over time in neonatal mortality for populations from the least deprived areas. These trends have not been mirrored among stillbirths where rates appear to have remained static for all sections of the population.

The widest relative deprivation gap was seen in stillbirths due to an antepartum haemorrhage. Recognised risk factors for this condition include women who have had previous pregnancies or several close pregnancies, who smoke or who are at the extremes of maternal age.²⁰ Work has also linked low socioeconomic status and placental praevia (which is strongly associated with antepartum haemorrhage).²¹ Other potential factors may include vasoconstrictive drugs such as cocaine that have been linked with abruption.²² A number of these factors are either known to be linked to socioeconomic deprivation or can be plausibly linked in terms of lifestyle and or behaviours. However, studies focusing on the potential link between deprivation and the mechanisms involved with the risk of stillbirth have found that such factors are only partially explanatory.¹¹

Similarly, stillbirths due to congenital anomalies were nearly three times more likely in the most deprived tenth and accounted for 10% of the deprivation gap as seen in studies of neonatal¹³ and perinatal mortality.¹⁴ This could be due to lower rates of termination among women from deprived areas who have been identified to have a fetus with a severe anomaly.²³ Our analysis excluded any late legal abortions, and therefore, all stillbirths seen in this work occurred naturally and spontaneously.

Stillbirths due to pre-eclampsia were twice as likely in the most deprived group. This may be due to





socioeconomic inequalities in the underlying rates of pre-eclampsia due to obesity and diabetes.²⁴ Inequalities in access to care for pre-eclampsia may also impact as shown in Belgium where severe pre-eclampsia was concentrated in the more socially deprived women with poorer access to care.²⁵

The only cause not to show a significant deprivation gap was deaths due to mechanical issues during labour. This suggests that this aspect of midwifery and obstetric care is not influenced by deprivation. Since such events are acute and generally not predictable prior to labour, this finding is reassuring that care in labour is not related to deprivation. We were unable to study intrapartum stillbirth due to non-mechanical causes as these are not separately defined in the data source. However, a study from Scotland demonstrated no association between deprivation and deaths due to intrapartum anoxia²⁶ and similar findings have been seen in neonatal deaths due to intrapartum events.¹³ It was noted in Sweden that even after adjusting for antenatal care attendance, women of a lower socioeconomic status were more likely to experience a stillbirth.¹¹ It would therefore appear that intrapartum stillbirths do not occur due to differences in the care these women receive in hospital and are not affected by social factors during pregnancy.

Our research has shown that there was a wide deprivation gap for those deaths that occurred in the antepartum period due to unknown causes as seen by Huang *et al*²⁷ This deprivation gap was wider for stillbirths where the fetus was small for gestational age. A study in the USA²⁸ found that even after adjusting for factors such as maternal smoking and hypertension, babies were more likely to be small for gestational age if their mothers lived in deprived areas. They concluded that some additional factor such as psychological stress was possibly causing the infants to be born small for gestational age. Sutan *et al*²⁹ suggested that risk factors for unexplained stillbirths in Scotland included maternal age, deprivation, smoking and height.

LIMITATIONS

A limitation of much stillbirth research including ours is that current stillbirth classifications, such as the Obstetric (Aberdeen) classification,⁷ classify the majority of stillbirths as occurring for unknown reasons, and it is difficult to focus on these deaths without improved classification systems. Alternative classifications of these deaths were not available for this work since for the time period studied national routinely collected data in England only used this classification for stillbirth. There are currently 35 published classification systems for stillbirth, many relying on advanced diagnostics that are not globally available.³⁰ These systems are not comparable, and there has been a strong case made to have one universal system for all countries.³¹ Consequently, Flenady *et al*² have called for a consensus on definitions and classifications in order to better understand the causes of stillbirth. Alternative systems such as the ReCoDe, Tulip or CODAC classifications³² provide a possible cause of death for approximately 85% of stillborn infants providing greater insight for those developing interventions to reduce future mortality.

Data on individual risk behaviour, lifestyle, health and ethnicity were not available for the mothers included in this work as it has been in other research.³³ Inevitably, this has limited the extent of our conclusions and has the potential to have produced a degree of confounding. For example, epidemiological work using individual-

level data has shown wide differences in stillbirth rates associated with maternal smoking during pregnancy,³⁴ hypertension³⁵ and maternal obesity.³⁶ Stillbirths are also known to be more common in sole registrations.³⁷ In women from deprived areas of Scotland,¹² maternal smoking status accounted for 38% of the inequalities seen in stillbirths. The lack of individual-level data also mean it was not possible to identify women who had more than one stillbirth over the time period; however, while women who have had a stillbirth are more likely to have a recurrence, the proportion of stillbirths that are likely to show this pattern is low and therefore negligible in terms of our findings.

Despite these shortcomings, we believe that our methods are relatively straightforward to undertake and provide an important approach for health service planners to monitor up-to-date trends in stillbirths.

IMPLICATIONS

This research confirms the continuation of previous trends in stillbirth rates and deprivation and suggests little change in the deprivation gap over time. However, recent reductions in other high-income countries² suggest that there exist modifiable risk factors and that by introducing targeted interventions, an improvement in stillbirth rates could be seen. Flenady *et al*² highlight the need to have an increased focus on appropriate interventions to reduce these disparities in stillbirths. Maternal smoking may be targeted successfully to impact on the rate of stillbirths, but we are currently lacking the effective tools needed to impact on maternal obesity and maternal age.³⁸

Our work highlighting the deprivation gap for different causes in stillbirths should assist the targeting of resources to specific geographical areas. These methods and findings are useful for monitoring inequalities in stillbirth in the future, but the collection of more detailed individual-level information for stillbirths and denominator data is required. Additionally, an improved classification system is necessary in order to better identify other modifiable risk factors and facilitate the introduction of appropriate targets and interventions.

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Contributors SES undertook the statistical analysis and wrote the first draft of the paper. LKS, BNM, ESD and DJF made substantial contributions to conception and design. LKS, ESD and AS were responsible for acquisition of data. LKS made a substantial contribution to the statistical analysis. All authors contributed to interpretation of the data, writing of the paper and revising it critically for important intellectual content. All authors approved the final version of the paper. SES is the guarantor.

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Competing interests None.

Ethics approval This study is based on routinely available national data that are anonymised, and hence, there is no requirement for ethical approval. We have clarified this in the manuscript.

Provenance and peer review Not commissioned; externally peer reviewed.

Data sharing statement There are no additional data available.

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	Item No	Recommendation	Page of document
Title and abstract	1	(a) Indicate the study's design with a commonly used term in the title	P3
		or the abstract	
		(b) Provide in the abstract an informative and balanced summary of	P3
		what was done and what was found	
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	P4
Objectives	3	State specific objectives, including any prespecified hypotheses	P4/5
Methods			
Study design	4	Present key elements of study design early in the paper	P6&7
Setting	5	Describe the setting, locations, and relevant dates, including periods	P6
6		of recruitment, exposure, follow-up, and data collection	
Participants	6	(<i>a</i>) <i>Cohort study</i> —Give the eligibility criteria, and the sources and	P6
r	~	methods of selection of participants. Describe methods of follow-up	-
		<i>Case-control study</i> —Give the eligibility criteria, and the sources and	
		methods of case ascertainment and control selection. Give the	
		rationale for the choice of cases and controls	
		Cross-sectional study—Give the eligibility criteria, and the sources	
		and methods of selection of participants	
		(b) Cohort study—For matched studies, give matching criteria and	
		number of exposed and unexposed	
		<i>Case-control study</i> —For matched studies, give matching criteria and	
		the number of controls per case	
Variables	7	Clearly define all outcomes, exposures, predictors, potential	P6/7
		confounders, and effect modifiers. Give diagnostic criteria, if	
		applicable	
Data sources/	8*	For each variable of interest, give sources of data and details of	P6
measurement		methods of assessment (measurement). Describe comparability of	
		assessment methods if there is more than one group	
Bias	9	Describe any efforts to address potential sources of bias	P12
Study size	10	Explain how the study size was arrived at	
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If	Table 1
		applicable, describe which groupings were chosen and why	
Statistical methods	12	(a) Describe all statistical methods, including those used to control	P6/7
		for confounding	
		(b) Describe any methods used to examine subgroups and	P6
		interactions	
		(c) Explain how missing data were addressed	P8
		(<i>d</i>) Cohort study—If applicable, explain how loss to follow-up was	
		addressed	
		Case-control study-If applicable, explain how matching of cases	
		and controls was addressed	
		Cross-sectional study—If applicable, describe analytical methods	

STROBE Statement-checklist of items that should be included in reports of observational studies

taking account of sampling strategy

 (\underline{e}) Describe any sensitivity analyses

Continued on next page

Results			
Participants	13*	(a) Report numbers of individuals at each stage of study-eg numbers potentially	P8
		eligible, examined for eligibility, confirmed eligible, included in the study,	
		completing follow-up, and analysed	
		(b) Give reasons for non-participation at each stage	
		(c) Consider use of a flow diagram	
Descriptive	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and	P8
data		information on exposures and potential confounders	
		(b) Indicate number of participants with missing data for each variable of interest	P8
		(c) Cohort study—Summarise follow-up time (eg, average and total amount)	
Outcome data	15*	Cohort study—Report numbers of outcome events or summary measures over time	
		Case-control study—Report numbers in each exposure category, or summary	
		measures of exposure	
		Cross-sectional study—Report numbers of outcome events or summary measures	P8
Main results	16	(a) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and	P8/9
		their precision (eg, 95% confidence interval). Make clear which confounders were	
		adjusted for and why they were included	
		(b) Report category boundaries when continuous variables were categorized	
		(c) If relevant, consider translating estimates of relative risk into absolute risk for a	Table
		meaningful time period	4
Other analyses	17	Report other analyses done-eg analyses of subgroups and interactions, and	
		sensitivity analyses	
Discussion			
Key results	18	Summarise key results with reference to study objectives	P10
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or	P12
		imprecision. Discuss both direction and magnitude of any potential bias	
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations,	P11-
		multiplicity of analyses, results from similar studies, and other relevant evidence	12
Generalisability	21	Discuss the generalisability (external validity) of the study results	
Other information	on		
Funding	22	Give the source of funding and the role of the funders for the present study and, if	P20

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.