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The value of customised centiles in assessing perinatal mortality risk associated with parity and maternal size

J Gardosi,[a] B Clausson,[b] A Francis[a]

[a] Perinatal Institute, Birmingham, UK [b] Mayday University Hospital, Croydon, UK
Correspondence: Prof. J Gardosi, Perinatal Institute, Crystal Court, Aston Cross, Birmingham B6 5RQ, UK. Email jason.gardosi@pi.nhs.uk

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Objective We wanted to compare customised and population standards for defining smallness for gestational age (SGA) in the assessment of perinatal mortality risk associated with parity and maternal size.

Design Population-based cohort study.

Setting Sweden.

Population Swedish Birth Registry database 1992–1995 with 354 205 complete records.

Method Coefficients were derived and applied to determine SGA by the fully customised method, or by adjustment for fetal sex only, and using the same fetal weight standard.

Main outcome measure Perinatal deaths and rates of small for gestational age (SGA) babies within subgroups stratified by parity, body mass index (BMI) and maternal size within the BMI range of 20.0–24.9.

Results Perinatal mortality rates (PMR) had a U-shaped distribution in parity groups, increased proportionately with maternal BMI, and had no association with maternal size within the normal BMI range. For each of these subgroups, SGA rates determined by the customised method showed strong association with the PMR. In contrast, SGA based on uncustomised, population-based centiles had poor correlation with perinatal mortality. The increased perinatal mortality risk in pregnancies of obese mothers was associated with an increased risk of SGA using customised centiles, and a decreased risk of SGA using population-based centiles.

Conclusion The use of customised centiles to determine SGA improves the identification of pregnancies which are at increased risk of perinatal death.

Keywords Body mass index, customised centiles, fetal growth restriction, intrauterine growth restriction, maternal size, parity, perinatal mortality, small for gestational age.

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Introduction

Fetal growth restriction is a principal concern in maternity care, and is associated with stillbirth, neonatal morbidity and mortality, cerebral palsy and delayed effects in childhood and adult life.[1–3] The diagnosis of small for gestational age (SGA) based on fetal or neonatal weight is an indicator for further prospective assessment, and is used in the retrospective analysis of the factors which affect perinatal outcome.

Conventionally, SGA was defined as below the lowest tenth centile of the fetal weight or birthweight range of the population, adjusted for gestational age. However, population standards include physiological as well as pathological factors that affect fetal growth.

Customised centiles are able to adjust for individual physiological variation and exclude pathological factors which may affect fetal growth and birthweight.[4,5] Coefficients have now been derived for different populations, and comparisons of expected birthweight for a 'standard mother' with the same parity, size and ethnic origin have shown remarkable similarities in different countries, suggesting that the principle can be applied as an international standard for birthweight and fetal growth.[6,7] Furthermore, compared with population-based birthweight standards to define SGA, the customised model improves the associations with pregnancy complications such as abnormal umbilical artery Doppler, caesarean section for fetal distress, low Apgar score, perinatal morbidity, admission and prolonged stay in the neonatal unit, as well as stillbirths and neonatal deaths.[8–12]

Because of the link between prematurity and IUGR,[13,14] the customised model to define growth potential does not

rely on preterm birthweights which, by definition, are derived from pregnancies with a pathological outcome. Instead, it uses Hadlock's ultrasound-based fetal weight curve[15] which is adjusted for gestational age using a proportionality formula.[5] As a result, investigators have been able to apply this tool to study the associations between prematurity and fetal growth.[16–18] About 26% of babies recognised by the customised standard but not by the population-based birthweight standard are born at preterm gestations.[12]

It has recently been claimed by Hutcheon and colleagues[19] that the main benefit of the customised model comes from its use of a fetal weight instead of a birthweight curve, and that after adjusting for fetal sex, further adjustment for maternal characteristics such as size or parity has little added benefit. However, the authors used a different, only partial form of customisation, and also did not control for the number of cases in each group when comparing perinatal mortality risk. Furthermore, their sample was not stratified to allow an examination of the effect that adjustment for maternal characteristics can have within the various groups of the population for which the customisation is particularly intended.

Therefore, we undertook a retrospective study of the variation of perinatal mortality risk within parity and maternal size groups, and the associated incidence of SGA birthweight, defined either by the fully customised model, or by one adjusted for fetal sex only, using the same ultrasound-based fetal weight curve.

Methods

Population

The study population was derived from a database of births in the Swedish Birth Registry 1992–1995 which we have previously described[8] and which is part of the longer database used by Hutcheon *et al.*[19] It contains prospectively collected information of births from 22 weeks and stillbirths from 28 weeks, according to the Swedish definition of stillbirth. Gestational age was determined according to the dating ultrasound scan, which was offered routinely and completed by 19 weeks gestation in over 90% of cases. In the absence of ultrasound, dating was carried out according to the given last menstrual period. Investigations by ultrasound and Doppler were undertaken if the fetus was suspected to be small for gestational age, but such information was not recorded in our database.

From a total of 439 358 singleton pregnancies, we excluded 916 cases with missing birthweight and/or gestational age data, and 84 237 cases with one or more missing variables needed to calculate customised centiles (fetal gender, maternal height, maternal weight as recorded at first visit, parity and ethnic origin). This resulted in a study population of 354 205 complete records, representing 81% of the original population. The sample included 1013 stillbirths and 786 neonatal deaths, which were combined and designated as 1799 perinatal deaths for the subgroup analysis.

Determination of 'small for gestational age' (SGA)

SGA was defined as below the tenth percentile birthweight, derived by one of two methods:

Model A (SGAcust): this represented the fully customised standard[5] to determine fetal growth potential at term, by

1 adjustment for significant maternal and pregnancy characteristics (height, weight, parity, ethnic origin (Nordic versus non-Nordic), and fetal sex);
2 exclusion of known pathological variables affecting birthweight such as diabetes and smoking; and
3 use of a fetal (ultrasound-based) weight standard to delineate the growth curve up to the predicted term weight.

Model B (SGApop): for comparison, a population-based standard was used, as described by Hutcheon *et al.*,[19] which made adjustment for fetal sex but not for maternal characteristics, and used the same fetal weight standard.

Both models were controlled for gestational age and used Hadlock's fetal weight curve,[15] which was converted into a proportionality curve to cover the whole gestational age range, as previously described.[5]

Coefficients for both models were derived by stepwise multiple regression, as previously described,[4,5] after excluding from the study population pregnancies which were complicated by stillbirth (n = 1013), congenital abnormality (13 325) or preterm birth (<259 days; n = 15 961). This left 323 955 cases for multiple regression analysis.

The variables entered into the model are listed in Table 1. They included maternal characteristics, smoking, diabetes, hypertensive diseases and antepartum haemorrhage. We also added a low (<20) and a high category (≥30) of maternal body mass index (BMI) as pathological variables, to study their effect on birthweight in addition to the influence of maternal height and weight.

Subgroups

To assess the association between perinatal mortality and SGA, defined with and without adjustment for maternal characteristics, the variables (i) parity, (ii) BMI and (iii) maternal size within normal BMI limits were examined. We were not able to look at ethnicity as 90% of the population was 'Nordic', and because the 'non-Nordic' group consisted of many ethnic minority groups which were individually too small for analyses by perinatal mortality rate (PMR).

Table 1. Characteristics of study population

Total *n* = 354 205	*n*	%	Mean	SD
Mother				
Age (years)			28.4	5.0
Height (cm)			166.2	6.4
Weight (kg)			65.2	11.3
Body mass index			23.6	3.8
<20	47 484	13.4		
≥20 and <30	282 960	79.9		
≥30	23 761	6.7		
Parity				
0	144 580	40.8		
1	130 137	36.7		
2	55 317	15.6		
3	16 741	4.7		
4+	7430	2.1		
Ethnic origin				
Nordic	317 705	89.7		
Non-Nordic	36 500	10.3		
Smoking (number/day)				
0	277 351	78.3		
1–9	43 402	12.3		
10+	25 046	7.1		
Hypertensive diseases	13 983	3.9		
Antepartum haemorrhage	5809	1.6		
Diabetes	16 206	4.6		
Baby				
Birthweight (g)			3551.7	559.3
Gestational age (weeks)			39.8	1.8
<37 weeks	15 961	4.5		
Gender				
Male	181 611	51.3		
Female	172 594	48.7		
Congenital anomaly	13 325	3.8		
Stillbirth	1013	2.86		
Neonatal death	786	2.22		
Perinatal death	1799	5.08		

For each maternal characteristic studied, the population (n = 354 205) was divided into four predetermined categories, which were clinically relevant and large enough to test for association with perinatal mortality.

1 Parity was defined as the number of previous births at the beginning of the index pregnancy, and categorised as 0, 1, 2 and 3 or more.
2 Maternal BMI [BMI: kg weight/(m height)^2] was based on measurements obtained routinely at the first visit in pregnancy. As in a previous analysis of BMI in Sweden,[20] four groups were defined: <20.0, 20.0–24.9, 25.0–29.9, and 30.0+.
3 Maternal size within normal BMI limits (20.0–24.9, n = 209 626), was divided according to maternal weight at first visit (recorded in integer kilograms) to provide four groups which were as equal as possible in number of cases: <58, 58–61, 62–65 and 66+ kg. As the BMI was restricted to the 20.0–24.9 range, the maternal height followed the maternal weight groups, rising incrementally to average heights of 160.1, 164.9, 167.7 and 171.8 cm in the four maternal weight groups (Table 3).

Statistical analysis

Multivariate backward linear regression was used to obtain coefficients for significant variables in Models A and B. Regression analyses were conducted using SPSS v17 (SPSS Inc, Chicago, IL, USA).

Within each of the subgroups described above, PMR and two SGA rates were calculated using the customised (Model A) and population methods (Model B). As both methods used the same fetal weight curve, all perinatal deaths were considered together without stratification for gestational age. Comparisons were made using relative risk and 95% confidence intervals.

To compare the change in perinatal mortality and how it was reflected by SGA rate, we made the assumption of linearity in increments between the four categories in each subgroup, and compared the resultant slope coefficients using t tests with 4 df (8 values minus 4 restrictions).

Risk analyses and tests were conducted using Excel (Microsoft Corporation, Redmond, WA, USA) with add-in functions.

Results

Table 1 describes the characteristics of the 354 205 cases. Table 2 lists the coefficients for the significant variables resulting from the multiple regression analyses for Models A and B, i.e. the fully customised model and the population-based model adjusted for gestational age and sex only. For Model B, the constant (intercept) of 3613.5 g represents the average birthweight expected at 280 days, adding or subtracting 64.4 g if the baby is male or female respectively.

In Model A, the constant of 3575.2 g similarly expresses the expected, fetal sex-neutral 280 day weight, but in addition it represents a mother in her first pregnancy, of average height (166 cm) and weight (65 kg), of Nordic ethnic origin. The coefficients to add or subtract from these standard characteristics are listed for each of these variables. In addition, smoking, hypertension and diabetes have returned significant coefficients in the regression model, which means that the constant represents an 'optimal' expected term birthweight, in a pregnancy free from such complications.

The predicted birthweight at 280 days resulting from each model can be compared after adjustment for parity, as Model A is centered on Para 0 while Model B represents the 'average' parity in the whole population. The total weighted average, using the frequencies (Table 1) and

Table 2. Multiple regression analysis using all variables (Model A) or fetal sex only (Model B)

n = 323 955		Model A*			Model B**		
		Coefficient	SE	95% CI	Coefficient	SE	95% CI
Constant		3575.2	1.7	3571.9 to 3578.5	3613.5	1.1	3611.3 to 3615.7
SE of constant		410.6			442.8		
Adjusted R^2		0.291			0.180		
Sex	Male	64.1	0.8	62.7 to 65.6	64.4	0.7	63.0 to 65.8
	Female	−64.1	0.8	−65.6 to −62.7	−64.4	0.7	−65.8 to −63.0
Height (cm)	Height	8.316	0.188	7.947 to 8.685			
	Height^3	−0.006	0.001	−0.008 to −0.004			
Weight (kg)	Weight	9.066	0.128	8.816 to 9.316			
	Weight^2	−0.067	0.004	−0.075 to −0.059			
Non-Nordic		−57.5	2.5	−62.4 to −52.6			
Parity	1	136.0	1.7	132.7 to 139.3			
	2	174.4	2.2	170.1 to 178.7			
	3	183.4	3.6	176.3 to 190.5			
	4+	189.0	5.3	178.7 to 199.3			
Smoking	1–9	−142.1	2.2	−146.5 to −137.7			
	10+	−207.9	2.9	−213.6 to −202.2			
Hypertensive disease		−131.0	4.1	−139.0 to −123.0			
Diabetes		110.7	3.6	103.7 to 117.7			
BMI	<20	−17.0	2.9	−22.6 to −11.4			
	≥30	−36.5	4.5	−45.4 to −27.6			

*Model A is centred on gestational age 280 days, sex 'neutral', parity 0, Nordic ethnicity, maternal height 166 cm, maternal weight 65 kg.
**Model B is centred on gestational age 280 days and sex 'neutral'.

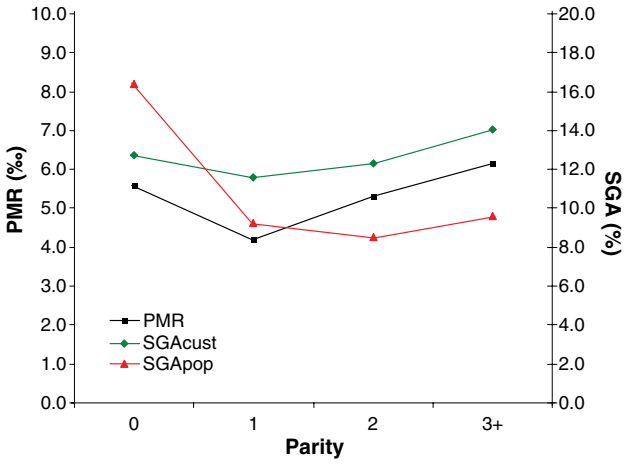

Figure 1. Perinatal mortality rate (PMR) and smallness for gestational age (SGA) by customised (SGAcust) and population-based centiles (SGApop), according to maternal parity at the beginning of pregnancy. *t* Test for difference of slopes: PMR versus SGAcust: $P = 0.778$; PMR versus SGApop: $P = 0.160$.

coefficients (Table 2) of the parity categories, is 91.3 g; adding this to the 'nulliparous' constant of Model A (3575.2 g) results in 3666.5 g which is 53 g higher than the constant for Model B. This increase is likely to be because of the exclusion of pathological factors in Model A, in particular smoking.

In Table 3, perinatal death rates are compared within four subgroups each of (i) parity, (ii) BMI and (iii) maternal size within normal BMI limits. For parity, the perinatal mortality risk has a U-shaped distribution, with babies in first pregnancies (para 0) and in higher order pregnancies (para 2, 3+) having an increased risk compared with second (para 1) pregnancies. For maternal BMI, the relationship with perinatal mortality is directly correlated, with low BMI pregnancies having a lower perinatal mortality risk and increasing incrementally with higher BMI categories. In contrast, small and large mothers with normal BMI showed similar PMR.

Table 3 also lists the SGA rates according to the fully customised method (SGAcust, Model A) and the method adjusted for sex only (SGApop, Model B). For each of the three variables, SGAcust is seen to reflect the perinatal mortality risk more closely than SGApop. This effect is particularly marked in the high parity and high BMI groups. Of 3395 SGAcust babies of para 3+ mothers (Table 3), 1200 (35.3%) were not small by SGApop, i.e. they were newly detected by the customised method; these babies had an increased risk of perinatal deaths (OR 3.1, CI 1.7–5.4). Similarly, the 3987 SGAcust babies of mothers with BMI 30+ (Table 3), included 1942 (48.7%) which were only SGA by

Table 3. Subgroup analysis of perinatal mortality and smallness for gestational age (SGA) rates (n = 354 205)

			Total, *n*	Perinatal deaths				Small for gestational age			
				n	Rate per 1000	RR**	95% CI	SGAcust		SGApop	
								n	%	*n*	%
Parity											
0			144 580	807	5.6	**1.32**	1.19–1.48	18 408	12.7	23 708	16.4
1*			130 137	549	4.2	1.00		15 054	11.6	12 052	9.3
2			55 317	294	5.3	**1.26**	1.09–1.45	6811	12.3	4706	8.5
3+			24 171	149	6.2	**1.46**	1.22–1.76	3395	14.0	2317	9.6
All/average			354 205	1799	5.1			43 668	12.3	42 783	12.1
Body mass index											
<20			47 484	185	3.9	**0.83**	0.71–0.98	5874	12.4	8548	18.0
20–24.9*			209 626	978	4.7	1.00		24 100	11.5	25 103	12.0
25–29.9			73 280	457	6.2	**1.34**	1.20–1.50	9654	13.2	7069	9.6
30+			23 761	179	7.5	**1.62**	1.38–1.90	3987	16.8	2060	8.7
All/average			354 151	1799	5.1			43 615	12.3	42 780	12.1
Maternal size (within BMI 20–24.9)											
Weight group (kg)	Mean weight (kg)	Mean height (cm)									
<58	54.5	160.1	46 981	227	4.8	1.09	0.91–1.31	5347	11.4	8347	17.8
58–61*	59.6	164.9	53 417	237	4.4	1.00		6195	11.6	6900	12.9
62–65	63.4	167.7	52 738	243	4.6	1.04	0.87–1.24	5991	11.4	5420	10.3
66+	69.3	171.8	56 490	271	4.8	1.08	0.91–1.29	6567	11.6	4436	7.9
All/average			209 626	978	4.7			24 100	11.5	25 103	12.0

CI, confidence interval; RR, relative risk.
*Reference group. **Values in bold are statistically significant.

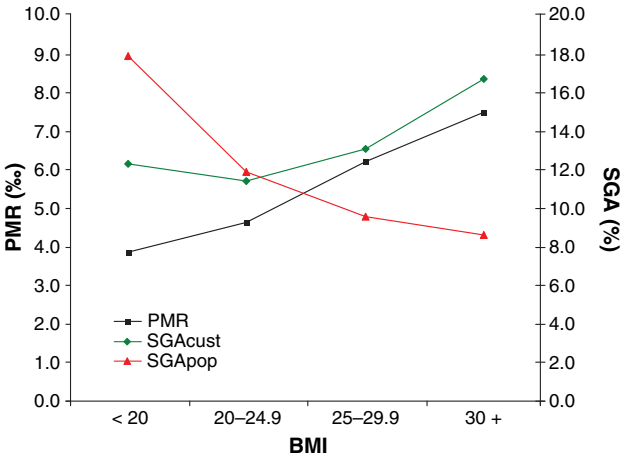

Figure 2. Perinatal mortality rate (PMR) and smallness for gestational age (SGA) by customised (SGAcust) and population-based centiles (SGApop), according to maternal body mass index (BMI). *t* Test for difference of slopes: PMR versus SGAcust: $P = 0.753$; PMR versus SGApop: $P = 0.007$.

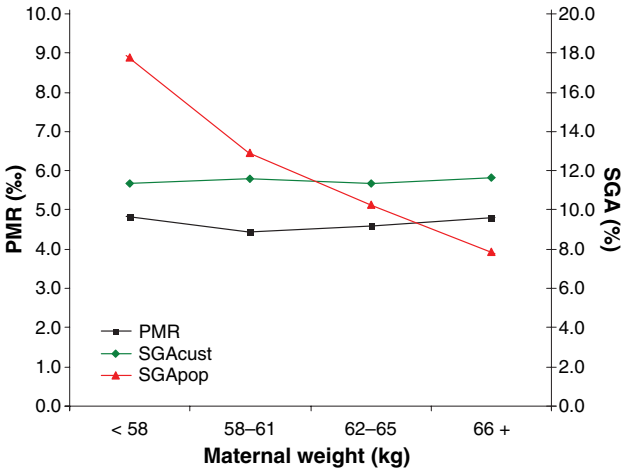

Figure 3. Perinatal mortality rate (PMR) and smallness for gestational age (SGA) by customised (SGAcust) and population-based centiles (SGApop), according to maternal weights within normal body mass index (BMI 20–24.9). *t* Test for difference of slopes: PMR versus SGAcust: $P = 0.743$; PMR versus SGApop: $P < 0.001$.

the customised method, and this group also had a significantly elevated risk of perinatal death: OR 1.8, CI 1.1–2.9).

The relationship between deaths and SGA rates is illustrated in Figures 1–3, showing in each instance a closer similarity between perinatal mortality and customised SGA than uncustomised SGA. To quantify this association, the graphs were analysed by linear regression and their slopes compared. For each of the variables, the SGAcust curve followed the perinatal mortality curve, with high P values ranging

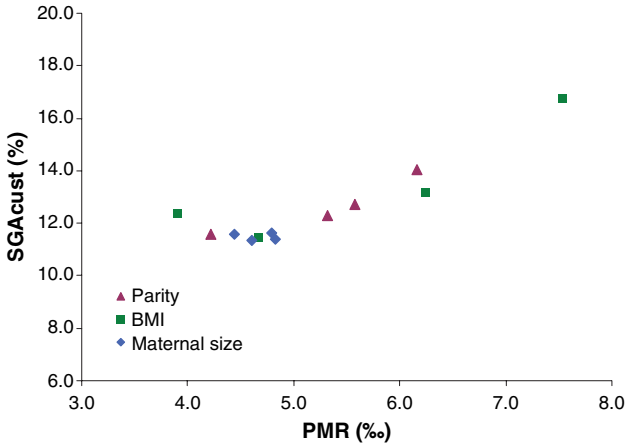

Figure 4. Scatter graph of 12 data pairs of perinatal mortality rate (PMR) versus small for gestational age rate by customised centiles (SGAcust), from the subgroups of the variables studied: parity, maternal BMI, maternal size with normal BMI. $R = 0.89$, $P < 0.01$.

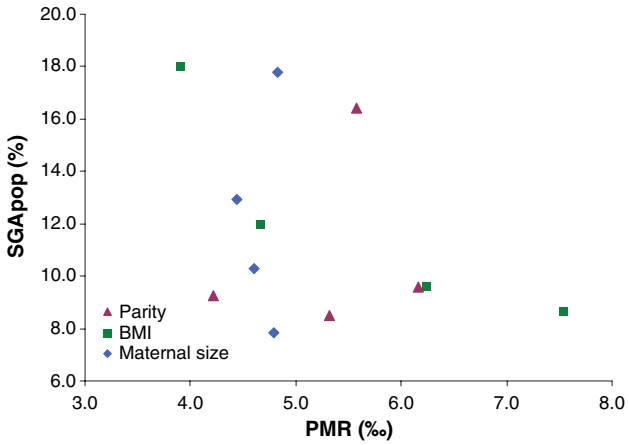

Figure 5. Scatter graph of 12 data pairs of perinatal mortality rate (PMR) versus small for gestational age rate by population centiles (SGApop), from the subgroups of the variables studied: parity, maternal BMI, maternal size with normal BMI. $R = -0.39$, $P = 0.21$.

from 0.74 to 0.78 (Figures 1–3). In contrast, there were marked differences between the perinatal mortality and each of the SGApop lines, which became significant for BMI ($P = 0.007$) and maternal size ($P < 0.001$) (Figures 2 and 3).

The 12 perinatal mortality—SGA data pairs (four in each of the three subgroups) were plotted for each of the models in Figures 4 and 5. Customised SGA showed a close correlation with perinatal mortality: $r = 0.89$, $P < 0.01$, while uncustomised, population-based SGA rates were more scattered, resulting in a correlation which was not significant and in fact tended towards the negative: $R = -0.39$, $P = 0.21$.

Discussion

This study has examined the perinatal mortality risk of several factors known at the beginning of pregnancy— parity, BMI and maternal size with normal BMI—and their association with SGA birthweight. For each of the variables studied, rates of SGA defined by customised centiles resulted in a strong association with perinatal mortality, while no such link was observed using SGA by centiles which did not adjust for maternal characteristics. The associations were different for each of the factors studied.

Parity

The relationship between parity and perinatal mortality is U-shaped, with first as well as third, fourth and higher order pregnancies being at higher risk (Table 3). This is in agreement with previous findings.[21] As Figure 1 shows, this trend is well reflected by the SGAcust curve but less so by SGApop.

Ego *et al.*[22] recently argued that parity should not be adjusted for when customising centiles, as nulliparity represents a higher risk pregnancy: the higher rate of SGA resulting from non-adjustment of centiles would have a beneficial effect in highlighting the higher risk. However, unadjusted centiles appear to increase the proportion of firstborn considered SGA to an excessive degree, which is not reflected by their PMR (Figure 1). While first pregnancies can have more complications such as pre-eclampsia and prolonged labour, increased clinical awareness and appropriate management should not have to be contingent on defining more babies as SGA, which can lead to unnecessary investigations and interventions. Furthermore, the authors[22] looked at the lower end of the parity spectrum only, but mothers with higher parity also have an increased risk. When all parity groups are considered, it is apparent that 'customised' SGA better reflects perinatal mortality risk across the whole parity spectrum. This has since been demonstrated when comparing customised centiles with and without adjustment for parity,[23] and here it is further confirmed by comparison between customised versus uncustomised, fetal weight-based centiles. The customised method is able to identify an additional 35% of mothers with significantly increased perinatal mortality risk.

Body mass index

Perinatal mortality is directly proportional to BMI (Table 3). In the obese BMI category, the higher PMR is associated with higher rates of SGA with customised but not with population centiles. An additional 49% of mothers with elevated perinatal mortality risk are identified using the customised method.

Reporting on data from the same Swedish register of births, Cnattingius and colleagues[20] found that obesity was protective of SGA. However, this is likely to have been an artefact associated with the use of uncustomised, population-based centiles which obscured the *relative* smallness of the baby. For example, a 3.5 kg baby will be within normal

limits for the general population and for an average size mother, but may be below the tenth centile for a mother with a high BMI. On its own, such a re-classification might be meaningless, but as Figure 2 shows, these 'SGAcust' babies have in fact a higher risk of perinatal death. In contrast, SGApop not only fails to follow this trend, but in fact drops as PMR increases with obesity.

It is interesting that mothers with BMI <20 had a *lower* rate of deaths. The reason for this is not clear, but preliminary, unpublished findings from a population-based study in the West Midlands suggest increased antenatal detection of fetal growth restriction in thin mothers. It is as yet uncertain whether this can be considered causal to reduced PMR.

To date, we truncated within the GROW software[24] the adjustments for maternal height and weight to within a BMI of BMI 20–30, to avoid adjusting for pathological factors when predicting the growth potential. However, the findings in this study suggest that adjustment *without* BMI limits is more useful, as it is able to identify these relatively small babies of big mothers who have a significantly increased risk of perinatal mortality, and new versions of GROW will reflect this.

Maternal size

There was no significant difference in perinatal mortality between different weight and height groups of mothers with normal BMI, and this was also reflected in the customised centile SGA rate. In contrast, population centile-based SGA showed a markedly different trend, with an excessive SGA rate in mothers who were small, and an incremental drop in rates as mothers got bigger, which was not reflected by the perinatal mortality risk. This is a good demonstration of how small-normal babies can be falsely considered SGA when maternal size is not adjusted for, potentially resulting in unnecessary intervention and maternal anxiety.

There may be several reasons why our findings contradict those of Hutcheon *et al.*,[19] who claimed that adjusting for maternal characteristics added little advantage. First, although the authors referred to our original method for customising centiles,[4,5] the model they applied was different in several respects. They did not adjust for maternal characteristics as continuous variables but within large categories only, which would have blunted their effect. They also did not exclude pathological factors such as smoking to allow customised centiles to reflect the growth potential. Furthermore, their analysis focussed on the size of the relative risk, but ignored the fact that the customised method identified overall *more* at-risk pregnancies (SGA) and *more* deaths within each category they examined. In our previous study comparing customised and population centiles,[8] such confounding was avoided by comparing outcome in the same number of babies, defined as the lowest 10% of the population in each group.

Notwithstanding those methodological differences, surely a valid statement on the specific value of adjusting for maternal characteristics has to include an examination of the subgroups of the population for which such adjustment is intended. Our analysis has demonstrated that customising for parity and maternal size results in a weight standard which has a substantially strengthened association with perinatal mortality risk.

We were not able to look at the effects of ethnicity in this cohort, as the non-Nordic group was very heterogeneous and represented only 10.3% of the population. However similar advantages for adjusting by ethnicity are suggested in a recent multi-ethnic Canadian study, which found that an ethnic-specific definition of SGA results in SGA rates which are more congruent with patterns of perinatal mortality than a non-ethnic specific standard derived from the same population.[25]

Our study looked at parity and maternal size variables separately to identify categorical differences. However, individual characteristics are of course interrelated in many possible combinations, which is why we recommend that all available variables are entered into the GROW software program[24] to predict the customised growth potential. Here, we limited our assessment to comparing two models to establish their respective value for determining SGA. Further analyses, using for example logistic regression can quantify the relative associations of these variables with SGA and PMR. We focused on perinatal deaths to allow sufficient numbers within subgroups, but the results are similar when stillbirths are assessed separately (data not shown).

The purpose of defining a baby's estimated fetal weight or birthweight as 'SGA' is to predict pathology and adverse outcome. Our study has shown that adjustment for such maternal characteristics results in significant improvement in identifying the babies who are at risk of perinatal death. Such a metric needs to work for the various groups within a heterogeneous population, and ultimately for the individual mother with her own set of characteristics. It is important for clinicians to use the correct tool to assess fetal size: unadjusted, general population-based limits lead to many cases being falsely considered SGA, while missing a substantial number which are truly at risk.

Conflict of interests

The authors declare that they have no conflict of interest.

Contribution to authorship

JG conceived and designed the study, analysed and interpreted the data, wrote the manuscript and made revisions.

BC provided the data, interpreted the analysis and reviewed the manuscript. AF carried out statistical analysis, interpreted the results and reviewed the manuscript.

Ethics approval
Not required: data fully anonymised.

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