

vascular disease. Three basic processes could generate this association. Firstly, poor social circumstances could lead to both lower birth weight and higher mortality risk. Secondly, maternal health, nutritional, and behavioural profiles could influence both birth weight and cardiovascular mortality. Thirdly, intergenerational factors—such as genomic and epigenetic processes that lead to a positive correlation between the birth weights of mothers and their offspring—could influence cardiovascular risk.

Adjustment for socioeconomic position and marital status had little influence on the findings in either the current study or the previous investigation of this issue,³ rendering a simple explanation in terms of socioeconomic confounding unlikely. In the current study we had no data on health status, but in the earlier study adjustment for a wide range of measures of health and health related behaviours reduced only slightly the association between infants' birth weight and mothers' cardiovascular mortality.³ The magnitude of the association in the current study is too great to be generated plausibly by the known associations between birth weight and maternal smoking, alcohol drinking, or anthropometry.⁵

The marked similarity between the current findings and those from the previous study—which related to an earlier generation of women living in widely different circumstances—suggests that an important influence is being uncovered by our analyses. Possible intergenerational influences on birth weight and cardiovascular risk therefore merit further investigation.

Contributors: GDS formulated the hypothesis regarding infants' birth weight and mothers' mortality. All authors discussed and specified the analyses, which were carried out by SH and MR. The first draft of the paper was written by GDS, with SH and MR contributing to the final draft. GDS is the guarantor of the paper.

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Cohort study of birthweight, mortality, and disability

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A study investigating the relation between fetal growth and subsequent mortality of Swedish residents born in Uppsala during 1915-29 identified slow fetal growth rate rather than small size at birth as the factor associated with increased risk of ischaemic heart disease.¹ The study also showed an expected association between birthweight and infant mortality. Surprisingly, birthweight was also associated with mortality in children aged 1-14 years. This association has not been previously observed, so we sought confirmation of the relation in the 1958 British birth cohort study. Because childhood death is now rare, we also examined whether birthweight had long term effects on disability.

Subjects, methods, and results

The 1958 cohort includes about 17 000 people born on 3-9 March 1958 in England, Scotland, and Wales followed up to 1991.² Birthweight was recorded in ounces and classified into five categories (see table). Birthweight for gestational age was defined as a sex specific Z score for each week of gestation. A total of 423 deaths occurring between the ages of 1 month and 38 years were notified to the NHS central register, with 19% (82) of these occurring in the first year. Disability or chronic illness and social class were ascertained at ages 7, 16, 23, and 33 years. We applied logistic regression to calculate the risk of death and disability associated with birthweight and birthweight for gestational age.

For males, infant mortality was inversely associated with birthweight (odds ratio 2.13 (95% confidence interval 1.48 to 3.07) per 1000 g reduction in

birthweight). For females, infant mortality was higher for those with birthweights under 3250 g, but not significantly, possibly because of the small numbers of deaths (table).

We found no clear associations with mortality after age 1 year. Significant inverse associations were found between birthweight and newly identified cases of disability at ages 7 (both sexes), 16 (males), and 23 (females). Risk of disability at ages 23 and 33 was raised for those with birthweights under 2500 g, although not always significantly. Birthweight for gestational age showed similar associations with mortality and disability to those described for birthweight. Confounding by social class did not account for the relations.

Comment

Although we found the well established relation between birthweight and infant mortality, there was no evidence of a relation with childhood mortality, as seen in the Swedish sample born in 1915-29.¹ Fewer deaths occurred in our 1958 cohort, and this might contribute to the discrepant findings. More plausibly, the inconsistency is due to differences in the main causes of death. Causes related to poor intrauterine growth may have been common earlier this century but have become less so as overall death rates have fallen. Unfortunately, we did not have sufficient numbers of deaths to examine separate causes. Nevertheless, our failure to detect a relation suggests that the effect of birthweight on childhood mortality has weakened.

Odds ratios (95% confidence interval) for all cause mortality and disability by birthweight and sex

Age (years)	No of deaths/ with disability*	Birthweight (g)					P value for trend†
		<2500 (n=743)	2500-3249 (n=3503)	3250-3749 (n=5657)	3750-4249 (n=4668)	>4249 (n=2111)	
Mortality							
Males:							
<1	46	8.41 (2.03 to 34.7)	4.51 (1.27 to 34.8)	2.27 (0.63 to 8.18)	1.58 (0.42 to 6.01)	1.00	<0.001
1-15	68	1.15 (0.31 to 4.25)	1.07 (0.47 to 2.43)	0.95 (0.45 to 2.02)	1.01 (0.48 to 2.12)	1.00	0.71
16-38	143	0.45 (0.13 to 1.51)	1.23 (0.74 to 2.04)	0.61 (0.36 to 1.03)	0.77 (0.47 to 1.26)	1.00	0.94
Females:							
<1	36	2.01 (0.27 to 14.9)	2.94 (0.65 to 13.4)	1.37 (0.29 to 6.44)	1.62 (0.33 to 7.89)	1.00	0.12
1-15	51	2.02 (0.39 to 10.4)	2.11 (0.59 to 7.50)	1.18 (0.33 to 4.27)	2.31 (0.66 to 8.09)	1.00	0.52
16-38	79	1.45 (0.45 to 4.70)	1.09 (0.44 to 2.66)	1.09 (0.47 to 2.56)	1.22 (0.51 to 2.94)	1.00	0.91
Disability*							
Males:							
7	403 (7005)	1.78 (0.94 to 3.37)	1.42 (1.00 to 2.01)	1.24 (0.90 to 1.70)	0.96 (0.69 to 1.34)	1.00	0.02
16	541 (5746)	1.51 (0.81 to 2.80)	1.79 (1.32 to 2.42)	1.18 (0.88 to 1.57)	1.03 (0.76 to 1.38)	1.00	<0.001
23	197 (5017)	3.65 (1.77 to 7.49)	1.33 (0.80 to 2.22)	1.03 (0.65 to 1.64)	1.20 (0.76 to 1.89)	1.00	0.09
33	212 (4387)	2.32 (0.97 to 5.55)	1.19 (0.72 to 1.98)	1.30 (0.84 to 2.01)	1.14 (0.73 to 1.78)	1.00	0.15
Females:							
7	274 (6672)	2.35 (1.19 to 4.62)	1.44 (0.88 to 2.37)	1.32 (0.82 to 2.13)	1.07 (0.64 to 1.77)	1.00	0.001
16	392 (5545)	0.71 (0.35 to 1.42)	0.82 (0.56 to 1.19)	0.90 (0.63 to 1.27)	0.74 (0.51 to 1.08)	1.00	0.41
23	158 (5267)	2.24 (0.93 to 5.37)	1.49 (0.80 to 2.75)	1.01 (0.55 to 1.87)	1.00 (0.52 to 1.89)	1.00	0.02
33	242 (4741)	2.06 (0.94 to 4.51)	1.37 (0.80 to 2.36)	1.28 (0.76 to 2.14)	1.55 (0.91 to 2.63)	1.00	0.17

*Newly identified cases from school doctor and health visitor assessments of disabling "abnormal conditions" ages 7-16, and from individual reports of a limiting longstanding illness ages 23 and 33; the denominator is all those with data minus subjects with disability at any previous age.

†Trend tests were performed with continuous measures of birthweight. The P values show the significance of linear trend except for disability at 7 for males, which has a quadratic trend.

The risk of disability in low birthweight infants has been demonstrated previously, notably for growth, neuromotor, and sensory impairments.³⁻⁵ Our study suggests that the association is graded, with risk of disability at age 7 years increasing as birthweight falls, mirroring the trend for infant mortality. Moreover, effects of birthweight seem to persist, as suggested by associations with incident disability at later ages (16 for men and 23 for women). Risk of later disability among those weighing < 2500 g at birth is raised at most ages, highlighting the vulnerability of this group. Improvements in neonatal care since the 1960s⁴ may have reduced the consequences of low birthweight in recent cohorts, but our study suggests that contemporary adults manifest long term sequelae related to their intrauterine growth.

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One hundred years ago Count sponges and instruments

Abdominal surgery involves special anxieties, and one of the worst is the fear that a forceps, sponge, or other foreign body has been left behind in the peritoneum after closure of the abdominal wound. Dr. Neugebauer of Warsaw has published a monograph on this terrible accident which will not reassure us. He classifies 101 cases, so that many operators are laudably candid, whilst necropsies tell terrible tales. In 38 cases the foreign body was only found at the *post-mortem* examination. In this grim list it is not surprising to find that in 19, or precisely half the cases, the object left behind was a sponge. In 14 cases the foreign body was spontaneously discharged through the anus. This occurrence must imply grave risk to the patient. In 12 cases the body was discharged through an abscess opening through the parietes or into the vagina. In 3 cases the body was missed, searched for and found, just before closure of the abdominal wound. In 7 it was missed just after closure of the wound, the wound was opened and the body removed. There is reason to believe that such an accident is so frequent that to these 10 cases several hundreds might be added. The remaining cases include later operations for

removal of foreign bodies, also one in which several years after laparotomy Douglas's pouch was opened through the vagina and the body—a signet ring—removed, and 2 in which the missing body was left in the peritoneum in the hopes that it would be discharged through an abscess. These 3 cases were not treated according to the generally-accepted rules of surgical practice. To calculate the proportion of fatal cases in which the body was detected or came away before a necropsy would be of no value, so different were circumstances in different cases. The operator should remember that sponges seem very deadly, forceps nearly as dangerous, whilst gauze pads more readily tend to come away by the bowel. But the successor failure of secondary operations must depend in part on the severity of the laparotomy itself. Neugebauer notes 2 cases where the abdomen was reopened on a false alarm, nothing having been left behind. Such an occurrence might turn the scale against a severe case. This monograph of Neugebauer's is grave reading, but it must be read. (BMJ 1900;ii:1047.)