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Effects of size at birth on health, growth and developmental outcomes in children up to age 18: an umbrella review

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ABSTRACT

Background Size at birth, an indicator of intrauterine growth, has been studied extensively in relation to subsequent health, growth and developmental outcomes. Our umbrella review synthesises evidence from systematic reviews and meta-analyses on the effects of size at birth on subsequent health, growth and development in children and adolescents up to age 18, and identifies gaps.

Methods We searched five databases from inception to mid-July 2021 to identify eligible systematic reviews and meta-analyses. For each meta-analysis, we extracted data on the exposures and outcomes measured and the strength of the association.

Findings We screened 16 641 articles and identified 302 systematic reviews. The literature operationalised size at birth (birth weight and/or gestation) in 12 ways. There were 1041 meta-analyses of associations between size at birth and 67 outcomes. Thirteen outcomes had no meta-analysis.

Small size at birth was examined for 50 outcomes and was associated with over half of these (32 of 50); continuous/post-term/large size at birth was examined for 35 outcomes and was consistently associated with 11 of the 35 outcomes. Seventy-three meta-analyses (in 11 reviews) compared risks by size for gestational age (GA), stratified by preterm and term. Prematurity mechanisms were the key aetiologies linked to mortality and cognitive development, while intrauterine growth restriction (IUGR), manifesting as small for GA, was primarily linked to underweight and stunting.

Interpretation Future reviews should use methodologically sound comparators to further understand aetiological mechanisms linking IUGR and prematurity to subsequent outcomes. Future research should focus on understudied exposures (large size at birth and size at birth stratified by gestation), gaps in outcomes (specifically those without reviews or meta-analysis and stratified by age group of children) and neglected populations.

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WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ A search in PubMed returns nearly half a million articles - an unwieldy and unmanageable field to navigate.
⇒ Eight previous umbrella reviews focused on specific subtopics; none was comprehensive in examining different risk factors or a broad range of outcomes.

WHAT THIS STUDY ADDS

⇒ It provides a comprehensive overview of reviews on the effects of size and gestation at birth on all subsequent health, growth and developmental outcomes in children.
⇒ It identifies outcomes with no meta-analyses and topics where there is a large, conclusive literature, and areas needing further or more conclusive research.

INTRODUCTION

Size at birth is affected both by in utero growth and by length of gestation. Researchers have been quantifying the relationship between size at birth and subsequent outcomes for over a century, resulting in a vast, nearly unmanageable, literature.[1–3] A quick PubMed search on size at birth generates almost half-a-million articles (online supplemental material 1), shaped by contemporaneous topics or theories of interest and by prevailing measurement capabilities.

The observation that small neonates were at substantially higher risk of dying than larger babies was quantified by early studies which defined 'prematurity' as low birth weight (LBW).[1 2] By the 1950s, prematurity was redefined using gestational age (GA) cut-offs; table 1 shows these and other definitions used as risk factors in our review. Research expanded from mortality outcomes to other potential consequences of being born with immature lung, neurological or immune-system development. At the other end of the size spectrum, macrosomia or high birth weight (HBW) was explored as a predictor of traumatic delivery or adverse growth outcomes. By the mid-1960, LBW, prematurity and intrauterine growth restriction (IUGR) were being distinguished, and modellers began looking at distributional components and developing population-specific and custom birthweight curves (late 1960s–1990s). The 1990s also saw the 'developmental origins of disease' theory, which suggested that small size at birth, quantified as LBW, increased disease risks in later life. This led to a burgeoning literature examining in utero shocks

Table 1 Measurements and threshold used for size-at-birth definitions

Risk factors (exposures)	Measurement units and thresholds used in definitions
Continuous measures	
Gestational age (GA)*	The duration of gestation is usually reported in completed weeks with additional days, or in completed days.
Birth weight (BW)†	Weight at birth measured in gram or kg. Reported using birth weight thresholds below or as mean birth weight with standard deviation
Small size at birth	
Extremely preterm (EPT)	<28 gestational weeks
Very preterm (VPT)	<32 gestational weeks
Preterm (PT)	<37 gestational weeks
Extremely low birth weight (ELBW)	<1000 g
Very low birth weight (VLBW)	<1500 g
Low birth weight (LBW)	<2500 g
Small for gestational age (SGA)	<10th percentile of birth weight for GA
Intrauterine growth restriction (IUGR)	Defined in the footnotes of online supplemental material 3 tables 1 a-g
Large size at birth/post term	
Post term	>41 gestational weeks
High birth weight (HBW)/ macrosomia	>4000 g
Large for gestational age (LGA)	>90th percentile of weight for GA

*GA is counted in calendar days from the first day of gestation, with the number of completed weeks calculated as the number of days divided by 7, presented as a whole integer plus a remainder, for example, day 258 is 36+6. Methods used to assess GA vary by study, which can affect reliability and comparability between studies. Methods using ultrasound assessment in the first trimester are most accurate.
†Birth weight is the first weight of the fetus or neonate obtained after birth. For live births, birth weight should preferably be measured within the first hour of life before significant postnatal weight loss has occurred.
GA, gestational age.

and their effects on cardiovascular and metabolic outcomes in adults and on early markers of these diseases in young children.[1 2] Starting in 2013, the International Fetal and Newborn Growth Consortium for the 21st Century (INTERGROWTH-21) used eight geographically diverse populations to develop global standard curves for fetal growth by sex and by GA.[3]

Despite a large literature and eight previous umbrella reviews,[4–11] there is no comprehensive summary of the main associations between size at birth and health, growth and developmental (including motor, cognitive and educational) outcomes, or of the literature gaps. Previous umbrella reviews (1) do not examine the full size-at-birth spectrum (neglecting larger neonates)[4 5 7–10]; (2) focus primarily on specific associations, for example, on the effects of LBW on mortality or chronic diseases[11] or of preterm birth on developmental outcomes[4 5]; (3) limit reviews to young children or adults and neglecting older children; and most importantly, to our knowledge, only one umbrella review (4) examines size for GA stratified by gestation, making it difficult to elucidate the relative importance of IUGR versus prematurity.

Our umbrella review aims to serve as a primary source of up-to-date compiled evidence on the effect of the full range of size-at-birth measures on a wide range of subsequent child and adolescent well-being outcomes.

Our umbrella review objectives are to (1) identify systematic reviews on the effects of size at birth on health (including mortality, acute ill health, lung-related ill health, chronic ill health and mental health), growth, developmental outcomes in children and adolescents; (2) map the evidence from reviews with meta-analyses, highlighting the magnitude, direction and consistency of the associations; (3) indicate evidence gaps; in addition, (4) we will suggest approaches needed for future empirical studies and meta-analyses.

METHODS

We conducted an umbrella review, gathering information from existing systematic reviews and meta-analyses which examined the effects of size at birth on health, growth and developmental outcomes in children up to 18 years of age.

We systematically searched MEDLINE, Embase, ERIC and Cochrane Library databases for articles published until 15 July 2021, without restricting on date, language or location. The search was limited to peer-reviewed systematic reviews or meta-analyses. Key search concepts included ("birth weight" OR "gestational age" OR "intrauterine growth restriction" OR "prematurity") AND ("systematic review" OR "meta-analysis"). To maximise the eligible reviews, we did not limit the outcomes or the study population. We also hand-searched the reference lists of the eight identified umbrella reviews to ensure we did not miss any reviews. The full search strategy and the steps for data extraction are included in online supplemental material 2.

In Online supplemental material 3 tables 1 a-g, we mapped the evidence on the effects of 12 different size-at-birth risk factors on a wide range of outcomes, grouped in seven themes: mortality and hospitalisation (theme a); neonatal and early childhood acute ill health (theme b); allergies and lung-related ill health (theme c); chronic ill health (theme d); behavioural and mental health (theme e); growth and nutrition (theme f); and developmental (motor, cognitive and educational) (theme g). The 7 themes had 67 subthemes. The subthemes in the behavioural and mental health themes (theme g) were grouped based on *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition (DSM5), classifications.[12]

The direction of the association was indicated using different colours in online supplemental material 3 tables 1 a-g with dark blue denoting a harmful effect, yellow denoting no statistically significant effect, and green denoting a beneficial effect.

RESULTS

We screened 16641 articles and identified 367 systematic reviews, of which 65 focused on outcomes in adults. This left 302 eligible systematic reviews of outcomes in children or in children and adults: 148 without meta-analyses, 141 with meta-analysis and 13 with meta-analyses of primary data (figure 1). Studies were published between 1989 and 2021.

We identified 7 themes and 67 subthemes of outcomes. Of the 67 subthemes, 13 were systematically reviewed without a meta-analysis (via 29 reviews)[13–41] (figure 2). Out of the 141 reviews with meta-analyses, 52 had a high-quality appraisal score, 61 medium and 28 low (online supplemental material 4a). Most of the meta-analyses (100 of 141) assessed publication bias (online supplemental material 4b).

Online supplemental material 3 tables 1 a-g shows the associations grouped by themes and subthemes. A total of 1041 associations were summarised from the 150 studies with meta-analyses

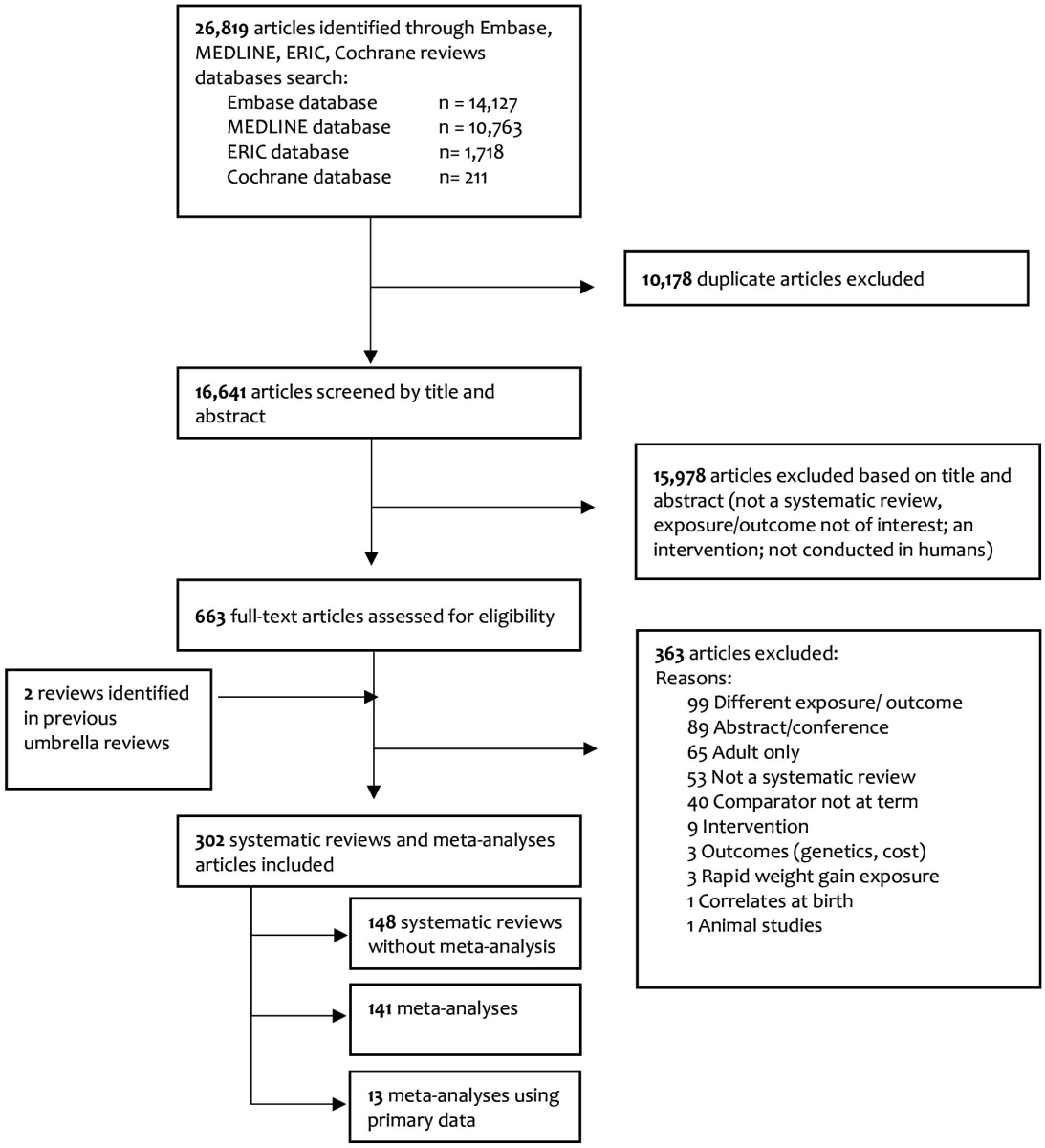

Figure 1 Preferred Reporting Items for Systematic Reviews and Meta-Analyses flowchart for study selection.

(including those with primary data): 772 with small size at birth as risk factor (including extremely preterm, very preterm, preterm, extremely low birth weight (ELBW), very low birth weight (VLBW), LBW and small for gestational age (SGA)), 144 with large size at birth/post-term (including post-term, HBW and large for gestation age (LGA)) and 125 with size as a continuous risk factor (weight and gestation). Only 85 of 1041 associations used SGA or LGA as risk factors. Of the 1041 associations, 225 focused on children under 5, 487 focused on children under 18, and 329 focused on mixed children and adults. The magnitude, direction and consistency of these associations are presented in online supplemental material 3 tables 1 a-g with a detailed narrative summary to explain the results by theme.

The main manuscript contains table 2 as an example of online supplemental table 1 f showing the associations between size at birth and nutrition and growth outcomes. Table 3 shows a subset of seven reviews which measured size for GA stratified by gestation, including four reviews missing from online supplemental material 3 tables 1 a-g because they included only stratified exposures.[42–45]

Figure 3 summarises findings on the direction of the association by subtheme of online supplemental material 3 tables 1 a-g .[46–195] Except for a few subthemes like undernutrition, most studies were conducted in high-income countries (online supplemental material 5).

Small size at birth (extremely preterm, very preterm, preterm, late preterm, ELBW, VLBW, LBW, SGA and IUGR) associations comprised most of the outcomes assessed (32 of 50) (online supplemental material 3 tables 1 a-g and figure 3). Seventeen of the 32 outcomes had been identified previously in eight published umbrella reviews as being associated with size at birth: mortality,[11 46–48 50] dental caries,[8 56–59] infection,[11 50 52 60–63] quality of life,[4 5 65] atopic dermatitis,[5 11 67 68] lung function,[4 5 11 70–73] asthma/wheezing,[11 52 73–80] including hypertension,[4 11 84–88 94] type 2 diabetes type,[9 11 113 114] physical activity,[6 143 144] undernutrition,[11 160] attention-deficit/hyperactivity disorder,[4 5 140–142 149–151] cerebral palsy,[5 170–173] neurodevelopmental,[4 5 164–167] motor development,[4 5 146 147 168] intellectual disabilities[10 11 138 139 141 146 148 151 174 177 179 181–184] and IQ.[10 11 141 142 146 177 181–183 185–189] Unlike most previous umbrella

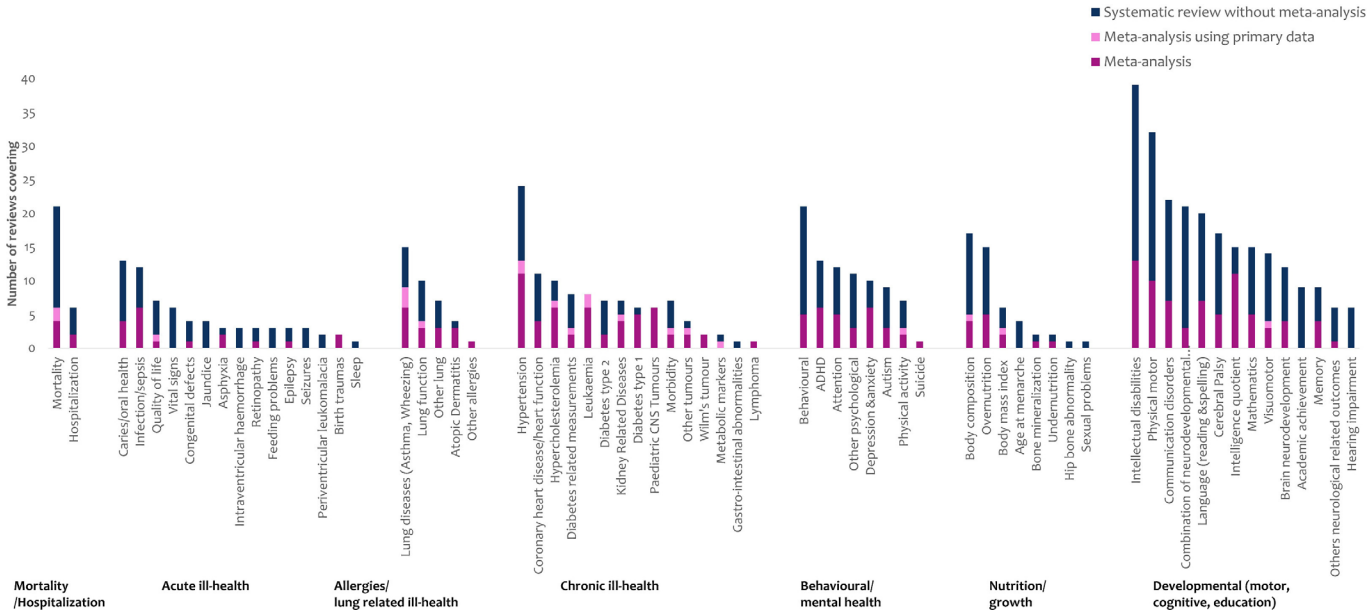

Figure 2 Themes and subthemes identified in 302 reviews.

reviews, we mapped the specific associations between different small size-at-birth risk factors and specific detailed outcomes. We also identified 15 subthemes which were consistently associated with small size at birth that had not been included in previous umbrella reviews of associations with hospitalisation,[52] asphyxia,[54] retinopathy,[55] epilepsy,[64] other lung related measurements,[51 82 83] kidney related diseases,[85 87 105–107] attention,[138 139 146–148] autism spectrum disorder,[140 152 153] body composition,[85 155–158] working memory,[138 141 146 182] communication,[138 148 174 183 190–192] educational outcomes language learning disorder,[138 141 184 190 191 193 194] mathematics learning disorder,[138 141 173 184 193] non-right handedness[195] and combined neurological measurements.[176] We found two subthemes (hypercholesterolaemia[84] and lymphoma[128]) which consistently showed no association. We also identified 16 associations with mixed evidence of association: congenital defects,[53] coronary heart disease heart function,[101 102] type 1 diabetes,[108–111] diabetes-related measurement,[84 115] paediatric central nervous system tumours,[116–120] leukaemia,[121 122 124 126 127] Wilms' tumour,[129] other tumours,[130] metabolic syndrome,[132] depressive/anxiety disorders,[133–138] other psychological,[132 135 139] adverse behaviours,[138 140–142] suicidal behaviour,[154] body mass index,[77 84] overnutrition[156 161 162] and visuomotor.[146 147 168]

Large size at birth/post-term/continuous measurement of birth weight and GA were consistently associated with 11 subthemes: increased risk of hospitalisation,[49] birth trauma,[49] atopic dermatitis,[69] lung function,[70] body composition,[158] overnutrition,[161–163] cerebral palsy,[170] Wilms' tumour,[112 129] intellectual disabilities,[151] and decreased quality of life[66] and working memory.[182] Meta-analyses showed mixed evidence for 24 subthemes.

In table 3, only 11 reviews and 73 meta-analyses within these compared risks by size for GA stratified by gestation. Four reviews[46 48 160 174] (37 meta-analyses) compared term SGA, preterm SGA and preterm- appropriate for gestational age (AGA) to term-AGA babies. These ideal comparisons elucidated the relative magnitude of the effect of SGA matching on preterm/term status and the relative magnitude of the effect of GA matching on AGA status.

DISCUSSION

This umbrella review provides the most recent synthesis of evidence from multiple fields exploring associations of size at birth with a wide range of subsequent health, growth and developmental outcomes in children under 18. This umbrella review summarised 302 reviews and mapped the magnitude and consistency of 1041 meta-analyses (from 150 reviews). The umbrella review also showed 73 meta-analyses (from 11 reviews) which compared risks by size for gestational age, stratified by preterm and term. We revealed gaps in research and an absence of meta-analyses for some exposures and outcomes. We elucidated analytical and measurement approaches which, if replicated, could better reveal the relative importance of preterm and IUGR (SGA) in the aetiology of adverse outcomes in children.

Our findings indicate some of the potential mechanisms underlying the associations. There is a body of theory seeking to distinguish the causes and the consequences of prematurity from those of IUGR.[46 196 197] Prematurity and fetal growth restriction are influenced by some similar factors, many of them maternal, such as weight, height, weight gain during pregnancy, smoking and age among others. Preterm delivery interrupts in utero development of neurological, immunological and lung function.[198 199] By contrast, poor fetal intrauterine growth, reflected in IUGR (SGA), links to subsequent metabolic and growth issues reflected in undernutrition and poorer cognitive development,[200 201] while rapid in utero growth, reflected by LGA, links to subsequent obesity and cancers. Analyses such as those shown in table 3, distinguishing the co-occurrence of preterm and SGA from the occurrence of preterm alone or SGA alone, and comparing these to term AGA babies, enable greater understanding of the relative importance of the prematurity and IUGR (and their respective causes) in the causation of specific adverse outcomes. This review suggests that prematurity mechanisms are the key aetiologies linked to mortality and cognitive development, while IUGR mechanisms are the key ones linked to underweight and stunting. Improved understanding of the relationship of these two different aetiologies to subsequent adverse outcomes will ensure we develop more appropriate interventions to address

Table 2 Associations between size at birth and nutrition and growth outcomes

Ref	Exposures (size at birth)												Population	Outcomes	Effect size (CI), direction of the association
	Small							Cont		Large					
	EPT (<28 weeks)	ELBW (<1000 g)	VPT (<32 weeks)	VLBW (<1500 g)	PT (<37 weeks)	LBW (<2500 g)	SGA (<10th percentile)	BW (cont.)	GA (cont.)	Post term (>41 weeks)	HBW (>4000 g)	LGA (>90th percentile)			
														Body composition	
155					X								Infants	Length (cm)	MD=−3.71 (−4.60 to −2.81)
85		X											11 years	Height (cm)	z-score difference=−0.92 (−0.03), p<0.001
155					X								Infants	Weight (kg)	MD=−0.59 (−0.75 to −0.44)
85		X											11 years	Weight (kg)	z-score difference=−0.61 (0.18), p<0.001
155					X								Infants	Head circumference (cm)	MD=−1.03 (−1.52 to −0.54)
85		X											11 years	Head circumference (cm)	z-score difference=−1.52 (0.44), p<0.001
85		X											11 years	Body surface area	z-score difference=−0.10 (−0.01), p<0.001
155					X								Infants	Total body fat (%)	MD=3.06 (0.25 to 5.88)
156					X								4–7 years	Total body fat (%)	SMD=−3.05 (−8.73 to 2.62)
155					X								Infants	Fat mass (kg)	MD=−0.05 (−0.09 to −0.01)
155					X								Infants	Fat-free mass (kg)	MD=−0.46 (−0.64 to −0.27)
156					X								4–7 years	Fat mass index	SMD=−1.31 (−5.42 to 2.81)
156					X								4–7 years	Childhood Trunk Fat Index	SMD=1.03 (−1.64 to 3.71)
157							**						At birth	Cord blood adiponectin concentrations	SMD=−1.14 (−2.15 to −0.12)
157							*						At birth	Cord blood adiponectin concentrations	SMD=−1.93 (−4.093 to −0.022)
157							X						At birth	Cord blood adiponectin concentrations	SMD=−0.383 (−0.744 to −0.022)
158							X						0.5 hours–11 days	Total body water (%)	MD=4.40 (2.83 to 5.96)
158									X				6 hours–7 days	Total body water (%)	β=−1.44 (−0.63 to −2.24) per week
158												X	0.5 hours–11 days	Total body water (%)	MD=−5.23 (−4.54 to −5.91)
														Bone mineralisation	
159								X					10 years	Bone mass content	β=0.02 (0.01 to 0.04)
159								X					10 years	Bone mass density	β=0.01 (−0.01 to 0.03)
														BMI	
84	X												6–32 years	BMI (kg/m^2)	MD=−0.50 (−1.10 to 0.09)
84			X										5–30 years	BMI (kg/m^2)	MD=−0.30 (−0.54 to −0.05)
84					X								4.5–35.7 years	BMI (kg/m^2)	MD=−0.13 (−0.40 to 0.14)
84					X								<10 years	BMI (kg/m^2)	MD=−0.70(−1.13 to −2.28)
84					X								<19 years	BMI (kg/m^2)	MD=5.20 (−3.82 to 14.21)
84					X								10–19 years	BMI (kg/m^2)	MD=−0.25 (−0.76 to 0.26)
91								X_{GA}					16.0–46.9 years	BMI (kg/m^2)	β=0.52 (0.20 to 0.84)/kg increase
91								GA					16.0–46.9 years	BMI (kg/m^2)	β=0.51 (−0.08 to 1.11)/kg increase
91								X					16.0–46.9 years	BMI (kg/m^2)	β=0.52 (0.17 to 0.86)/kg increase
77					T								0–2 years	BMI trajectory: class 2 (rapid growth to 2 years)	aOR=2.02 (1.49 to 2.74)
77					T								0–6 years	BMI trajectory: class 3 (persistent rapid growth to 6 years)	aOR=1.89 (0.42 to 8.49)
77						◊							0–2 years	BMI trajectory: class 2 (rapid growth)	aOR=1.48 (1.05 to 2.10)
77						◊							0–6 years	BMI trajectory: class 3 (persistent rapid growth)	aOR=0.78 (0.10 to 6.45)
77										X			0–2 years	BMI trajectory: class 2 (rapid growth)	aOR=0.81 (0.68 to 0.96)
77										X			0–6 years	BMI trajectory: class 3 (persistent rapid growth)	aOR=0.48 (0.15 to 1.53)
77											T		0–2 years	BMI trajectory: class 2 (rapid growth)	aOR=0.98 (0.86 to 1.12)
77											T		0–6 years	BMI trajectory: class 3 (persistent rapid growth)	aOR=1.62 (0.88 to 2.99)
														Undernutrition	

Continued

Table 2 Continued

Ref	Exposures (size at birth)												Population	Outcomes	Effect size (CI), direction of the association
	Small							Cont		Large					
	EPT (<28 weeks)	ELBW (<1000 g)	VPT (<32 weeks)	VLBW (<1500 g)	PT (<37 weeks)	LBW (<2500 g)	SGA (<10th percentile)	BW (cont.)	GA (cont.)	Post term (>41 weeks)	HBW (>4000 g)	LGA (>90th percentile)			
160					X								12–60 months	Wasting (weight for length/height for age <2 z-scores)	OR=1.55 (1.21 to 1.97)
160						X							12–60 months	Wasting (weight for length/height for age <2 z-scores)	OR=2.68 (2.23 to 3.21)
160							X						12–60 months	Wasting (weight for length/height for age<2 z-scores)	OR=2.36 (2.14 to 2.60)
160					X								12–60 months	Stunting (length/height for age<2 z-scores)	OR=1.69 (1.48 to 1.93)
160						X							12–60 months	Stunting (length/height for age<2 z-scores)	OR=2.92 (2.56 to 3.33)
160							X						12–60 months	Stunting (length/height for age<2 z-scores)	OR=2.32 (2.12 to 2.54)
160					X								12–60 months	Underweight (weight for age less than 2 z-scores)	OR=1.66 (1.42 to 1.95)
160						X							12c60 months	Underweight (weight for age less than 2 z-scores)	OR=3.48 (3.14 to 3.87)
160							X						12–60 months	Underweight (weight for age less than 2 z-scores)	OR=2.96 (2.61 to 3.36)
														Overnutrition	
161						X							0–18 years	Overweight	OR=0.60 (0.54 to 0.67)
161								X					1–75 years	Overweight	β=0.34 (0.28 to 0.40)/kg increase
161											X		0–18 years	Overweight	OR=1.76 (1.65 to 1.87)
156					X								6–14 years	Obesity	OR=1.19 (1.13 to 1.26)
162						◊							3–18 years	Obesity	OR=0.87 (0.69 to 1.08)
162						X							1–17 years	Obesity	OR=0.61 (0.46 to 0.80)
162						X							<6 years	Obesity	OR=0.61 (0.43 to 0.88)
162						X							6–13 years	Obesity	OR=0.54 (0.32 to 0.90)
162						X							13–17 years	Obesity	OR=0.74 (0.37 to 1.49)
163								X					7–11 years	Obesity	β=0.649/kg increase
162											◊		1–16 years	Obesity	OR=2.23 (1.91 to 2.61)
162											X		0–17 years	Obesity	OR=2.07 (1.91 to 2.24)
162											X		<6 years	Obesity	OR=2.10 (1.93 to 2.29)
162											X		6–13 years	Obesity	OR=1.76 (1.36 to 2.20)
162											X		13–17 years	Obesity	OR=2.58 (1.56 to 4.26)

Exposures: EPT (<28 weeks), ELBW (<1000 g), VPT (<32 weeks), VLBW (<1500 g), PT (<37 weeks), LBW (<2500 g), SGA (<10th percentile), post term (>41 weeks), HBW (>4000 g) and LGA (>90th percentile).
Symbols in exposures: X, as defined in exposure; $X_{a,u}$, adjusted and unadjusted for GA; GA, BW adjusted for GA; **, SGA <3rd, 5th and 10th percentile/value×by SD for GA; *, SGA <3rd percentile/value×by SD for GA; ◊, reference category 2500–4000 g; T, reference category GA 37≤term≤41.
Outcomes: , harmful effect; , no effect; , beneficial effect; *italic*, calculation/post review.
aOR, adjusted OR; BMI, body mass index; BW, birth weight; BW (cont.), birth weight continuous; ELBW, extremely low birth weight; EPT, extremely preterm; GA, gestational age; GA (cont.), gestational age continuous; HBW, high birth weight; LBW, low birth weight; LGA, large for gestational age; MD, mean difference; PT, preterm; SGA, small for gestational age; SMD, standardised mean difference; VLBW, very low birth weight; VPT, very preterm.

Table 3 Association between maturity and SGA/IUGR combinations and different outcomes

			Exposures					Reference			Effect size (CI), direction of association
Ref	Outcomes	Population	PT SGA	PT AGA	T IUGR	T SGA	T LBW	T AGA	T NBW	T	
48	Neonatal mortality	≤28 days	<34					X			OR=56.97 (11.1 to 291.7)
48	Neonatal mortality	≤28 days		<34				X			OR=74.9 (32.6 to 171.7)
48	Neonatal mortality	≤28 days	34–36					X			OR=19.88 (8.3 to 47.5)
48	Neonatal mortality	≤28 days		34–36				X			OR=3.18 (1.0 to 10.7)
48	Neonatal mortality	≤28 days				X		X			OR=2.23 (1.2 to 4.10)
46	Neonatal mortality	<28 days	X					X			RR=15.42 (9.11 to 26.1)
46	Neonatal mortality	<28 days		X				X			RR=8.05 (3.88 to 16.72)
46	Neonatal mortality	<28 days				X		X			RR=2.44 (1.67 to 3.57)
46	Early neonatal mortality	<7 days	X					X			RR=17.19 (9.57 to 30.91)
46	Early neonatal mortality	<7 days		X				X			RR=7.59 (3.38 to 17.08)
46	Early neonatal mortality	<7 days				X		X			RR=2.76 (1.82 to 4.18)
46	Late neonatal mortality	8–28 days	X					X			RR=17.37 (10.27 to 29.37)
46	Late neonatal mortality	8–28 days		X				X			RR=5.60 (2.75 to 11.43)
46	Late neonatal mortality	8–28 days				X		X			RR=2.45 (1.7 to 3.51)
46	Postneonatal mortality	29–365 days	X					X			RR=5.22 (2.8 to 9.64)
46	Postneonatal mortality	29–365 days		X				X			RR=2.72 (1.5 to 4.79)
46	Postneonatal mortality	29–365 days				X		X			RR=1.98 (1.39 to 2.81)
46	Infant mortality	<365 days	X					X			RR=9.24 (4.33 to 19.71)
46	Infant mortality	<365 days		X				X			RR=5.30 (2.39 to 11.76)
46	Infant mortality	<365 days				X		X			RR=2.28 (1.52 to 3.41)
160	Wasting	12–60 months	X					X			aOR=4.19 (2.90 to 6.05)
160	Wasting	12–60 months		X				X			aOR=1.96 (1.46 to 2.63)
160	Wasting	12–60 months				X		X			aOR=2.52 (2.27 to 2.80)
160	Stunting	12–60 months	X					X			aOR=4.51 (3.42 to 5.93)
160	Stunting	12–60 months		X				X			aOR=1.93 (1.71 to 2.18)
160	Stunting	12–60 months				X		X			aOR=2.43 (2.22 to 2.66)
160	Undernutrition	12–60 months	X					X			aOR=5.35 (4.39 to 6.53)
160	Undernutrition	12–60 months		X				X			aOR=2.07 (1.76 to 2.44)
160	Undernutrition	12–60 months				X		X			aOR=3.17 (2.78 to 3.62)
174	Motor	<7 years	X					X			aSMD=−0.15 (−0.40 to 0.09)
174	Motor	<7 years		X				X			aSMD=−0.23 (−0.42 to −0.03)
174	Motor	<7 years				X		X			aSMD=−0.007 (−0.08 to 0.06)
174	Cognitive	<7 years	X					X			aSMD=−0.17 (−0.29 to −0.05)
174	Cognitive	<7 years		X				X			aSMD=−0.14 (−0.24 to −0.05)
174	Cognitive	<7 years				X		X			aSMD=−0.02 (−0.10 to 0.06)
174	Language	<7 years		X				X			aSMD=−0.02 (−0.23 to 0.19)
174	Language	<7 years				X		X			aSMD=−0.03 (−0.12 to 0.06)
172	Cerebral palsy	Neonates	X					X			OR=2.34 (1.43 to 3.82)
42	Neonatal mortality	Neonates				X		X			OR=4.11 (3.70 to 4.56)
42	Non-neurological neonatal morbidity	Neonates				X		X			OR=2.98 (1.58 to 5.61)
42	Neonatal morbidity: neurological	Neonates				X		X			OR=2.12 (1.56 to 2.91)
43	Morbidly composite	1–18 years				X		X			OR=1.49 (1.02 to 2.1)
43	Morbidly composite	1–18 years					X		X		OR=0.98 (0.87 to 1.10)
43	Learning difficulties or learning disabilities	12 months–18 years				X		X			OR=2.03 (1.65 to 2.50)
43	Obesity	2–18 years				X		X			OR=0.94 (0.59 to 1.49)
43	Obesity	6–11 years					X		X		OR=0.90 (0.50 to 1.64)
43	Hypertension	3–16 years					X		X		OR=0.98 (0.8 to 1.12)
44	Neurodevelopmental scores (high scores)	40 weeks–10 years				X		X			Largest SMD=−0.32 (−0.38 to −0.25)
44	Neurodevelopmental scores (low scores)	40 weeks–10y ears				X		X			Smallest SMD=−0.31 (−0.38 to −0.25)
45	Cognitive score	0.16–10.0 years			X	XI		X			SMDH=−0.39 (−0.50 to −0.28)

Continued

Table 3 Continued

Ref	Outcomes	Population	Exposures					Reference			Effect size (CI), direction of association
			PT SGA	PT AGA	T IUGR	T SGA	T LBW	T AGA	T NBW	T	
45	Cognitive score	0.16–10.0 years				X		X			SMDH=−0.34 (−0.45 to −0.22)
45	Cognitive score	2.0–9.5 years			X	I		X			SMDH=−0.58 (−0.82 to −0.35)
45	Borderline intellectual impairment	Child				X		X			OR=1.75 (1.50 to 2.04)
84	Systolic blood pressure	Child/adult	X					X			MD=2.00 (0.21 to 3.78)
84	Systolic blood pressure	Child/adult		X				X			MD=1.46 (0.13 to 2.79)
84	Diastolic blood pressure	Child/adult	X					X			MD=1.39 (0.00 to 2.78)
84	Diastolic blood pressure	Child/adult		X				X			MD=1.22 (0.19 to 2.25)
84	High-density lipoprotein	Child/adult	X					X			MD=0.03 (−0.04 to 0.10)
84	High-density lipoprotein	Child/adult		X				X			MD=0.01 (−0.04 to 0.07)
84	Low-density lipoprotein	Child/adult	X					X			MD=0.67 (0.38 to 0.97)
84	Low-density lipoprotein	Child/adult		X				X			MD=0.13 (−0.03 to 0.29)
84	Triglyceride	Child/adult	X					X			MD=0.00 (−0.07 to 0.06)
84	Triglyceride	Child/adult		X				X			MD=−0.04 (−0.09 to 0.02)
84	Insulin	Child/adult	X					X			MD=−1.65 (−3.39 to 0.10)
84	Insulin	Child/adult		X				X			MD=−1.07 (−2.29 to 0.15)
84	BMI	Child/adult	X					X			MD=−0.38 (−0.98 to 0.22)
84	BMI	Child/adult		X				X			MD=0.06 (−0.34 to 0.46)
87	Systolic blood pressure	11.3–41.3 years	X							X	SMD=0.41 (0.12 to 0.70)
87	Systolic blood pressure	11.3–41.3 years		X						X	SMD=0.31 (−0.33 to 0.95)
87	Diastolic blood pressure	11.3–41.3 years	X							X	SMD=0.28 (0.05 to 0.51)
87	Diastolic blood pressure	11.3–41.3 years		X						X	SMD=0.09 (−0.08 to 0.26)
87	Serum creatinine	17.6–22.9 years	X							X	SMD=0.18 (−0.24 to 0.59)
87	Serum creatinine	17.6–22.9 years		X						X	SMD=0.02 (−0.32 to 0.35)

, harmful effect from high to lower risks; , no effect high to lower risk.
Symbols inexposures: X, as defined in exposure; XI, SGA and IUGR (defined in reference 45); I, IUGR (defined in reference 45).
(45) IUGR is defined as antenatal evidence of growth restriction by abnormal middle cerebral artery pulsatility index and umbilical artery pulsatility index, or late onset verified by ultrasound or clinically, or ultrasound and clinical evaluation, or third trimester serial ultrasound.
AGA, appropriate for gestational age; BMI, body mass index; IUGR, intrauterine growth restriction; LBW, low birth weight; MD, mean difference; NBW, normal body weight; PT, preterm; RR, relative risk; SGA, small for gestational age; SMD, standardised mean difference; SMDH, standardized mean difference for heteroscedastic population variances; T, term.

these risk factors and are better able to track intervention impacts.

It was not feasible in this discussion to explore all the potential reasons why mixed or contradictory effects were observed for each of the subthemes. Key reasons for why mixed estimates of effect were seen could include the number of included studies, the search strategy and inclusion/exclusion criteria, the constituent study designs and heterogeneity. Other potential reasons for inconsistent associations include the population used for the exposure (grouping extremely preterm with preterm), the comparator used (grouping normal birth weight with HBW as a comparator for LBW), the age of the child at assessment (allowing more or less time for a disease, such as type 2 diabetes, to develop), measurement practices in older versus newer reviews, and whether or not sex or other variables were adjusted for (female babies are appropriate for GA at a lower birth weights than male babies and could be misclassified if sex was not adjusted for).

By way of example of how the results have varied by review, we unpacked meta-analysis of the association between LBW and type 1 diabetes. The earliest review, by Harder and colleagues, included eight papers and suggested a protective effect (0.82), but had a confidence interval (CI) that overlapped 1 (95% CI 0.54 to 1.23).[109] However, this review compared LBW to babies born at 2500+ g, including HBW infants. The next review, by Cardwell and colleagues, used a more appropriate normal (2500–4000 g) comparator and included many more studies (29 studies of which five were cohorts).[111] They showed no association (OR=0.98, 95% CI 0.84 to 1.13), with high heterogeneity observed, although a meta-analysis of the cohorts showed a protective effect (OR=0.79, 95% CI 0.67 to 0.92).[111] The most recent meta-analysis by Haiyan Wang and colleagues, focused only on six cohort studies and by virtue of having less heterogeneity and a larger sample size, they established that LBW appears to protect against type 1 diabetes compared with normal birth weight (HR 0.78, 95% CI 0.69 to 0.88).[110] By contrast there was only one systematic review of the effects of prematurity (Li and colleagues[108]) which included 18 studies and showed prematurity increased the risk of type 1 diabetes (OR=1.17, 95% CI 1.10 to 1.25) for high-quality studies.

Although we assessed review quality, we aimed to be comprehensive and so extracted data regardless of quality. This meant we included 28 reviews with low critical appraisal scores which might explain some of the mixed direction of effects observed. Thus, when exploring the association presented, it is important to consider the quality of the meta-analysis. For example, low-quality review on extremely preterm and ELBW and mortality showed very small neonates had a reduced prevalence of

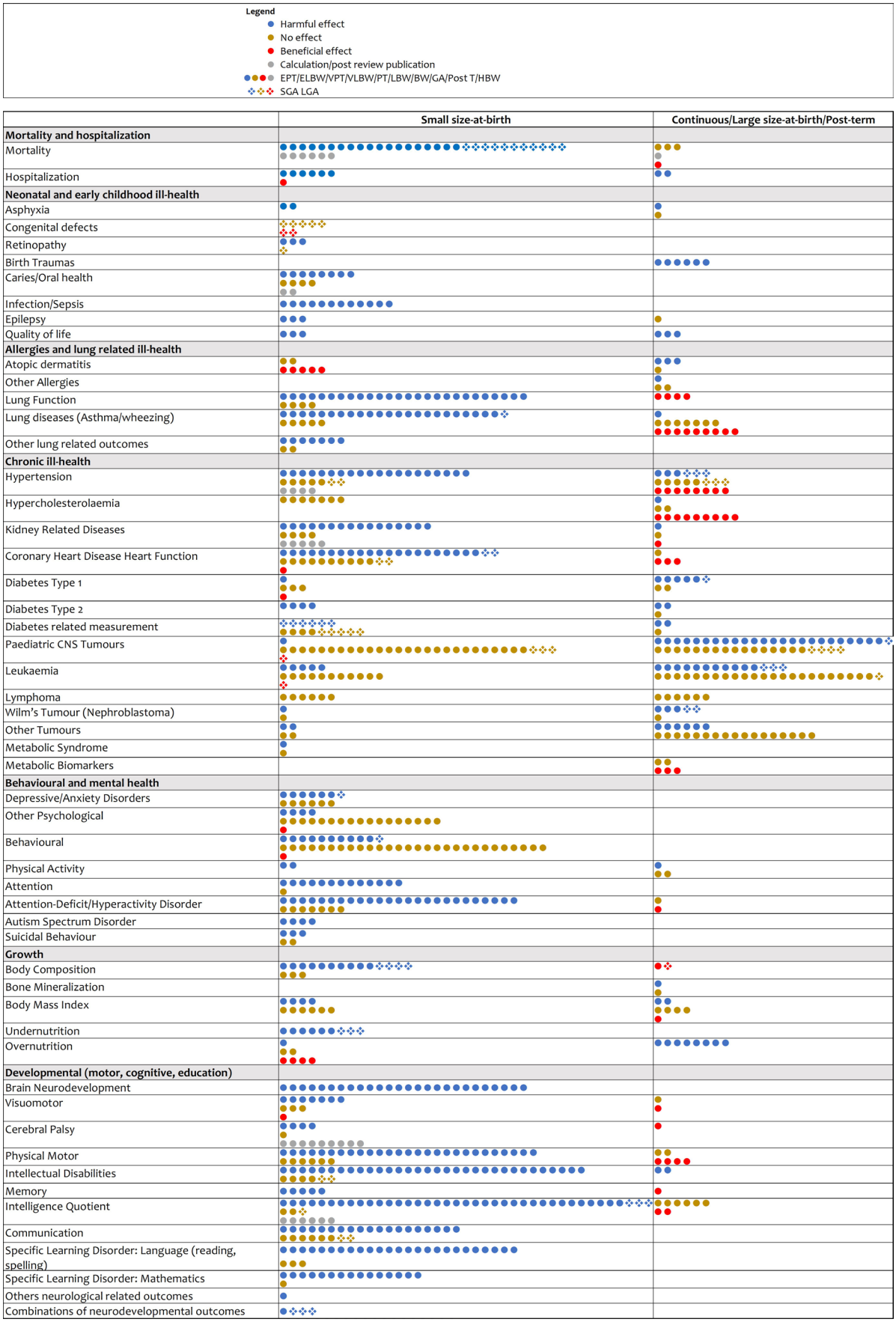

Figure 3 Summary of the associations presented in online supplemental table 1a–g. BW, birth weight; ELBW, extremely low birth weight; EPT, extremely preterm; GA, gestational age; HBW, high birth weight; LBW, low birth weight; LGA, large for gestational age; PT, preterm; SGA, small for gestational age; VLBW, very low birth weight; VPT, very preterm.

mortality compared with larger babies,[47] an anomalous finding which probably stemmed from selection and publication bias favouring reports of very small surviving babies.

The evolution of our understanding of the relationships between size at birth and various outcomes in children is inextricably linked to improvements in measurement and in theory, as well as to disease burden and priority health topics. For example, literature on effects of small size at birth on adult health burgeoned after the 'developmental origins of disease' theory.[1 2] Our review identified several gaps in relation to the risk factors, outcomes and populations studied. Very few meta-analyses examined outcomes linked to the effect of LGA and SGA or of the different combinations of gestation and size for GA at birth. For some subtheme outcomes (cognitive and motor), very small size at birth was the exposure measured rather than LBW or prematurity. Most of the systematic reviews were from high-income countries, reflecting a general bias in research.[202] We also identified 14 subtheme outcomes missing meta-analyses. Older age children are rarely a priority population for studies of mortality or acute ill health, but this neglect may be because they generally have fewer ill-health outcomes and so are more difficult to study.

Strengths and limitations

Our review synthesised an enormous literature and was comprehensive, not restricting on outcome, year or language. It assessed methodological quality using a critical appraisal tool, showed gaps and focused on children up to 18, thereby bridging a gap between studies focused on young children and those focused on adults. Its limitations are its reliance on published systematic reviews, particularly those with meta-analyses. Our approach missed single studies not included in previous reviews and topics without systematic reviews. We did not do additional meta-analyses nor did we recalculate effect sizes, so we include three reviews with inconsistent data presented in abstract, figures and results.[87 124 159] Moreover, while we did not restrict on language, we used English search terms and did not search non-English databases, for example, Chinese literature. As part of the umbrella review, we did not assess methods of the selected papers. In meta-analyses where we did not detect an association, we did not conduct further examination by assessing the confidence intervals.

RECOMMENDATIONS/CONCLUSION

Our umbrella review compiled evidence from 1041 associations and showed the strength of evidence. It also alluded to potential mechanisms, enabling us to identify areas where we can appropriately target or track interventions aimed at improving outcomes in LBW/preterm or HBW children.

To improve future research and evidence on the mechanisms involved, we highlight the need to

- Address gaps in the range of risk factors explored by including the whole spectrum of size and maturity where possible, including (1) splitting preterm into subgroups based on maturity, for example, extremely preterm, very preterm and moderate or late preterm; (2) considering all the combinations of size for GA (adjusted for preterm/term/post-term, specifically focusing on SGA and LGA); and (3) excluding HBW, post-term and LGA from the comparator when examining small size at birth (LBW, preterm and AGA). The latter recommendation is made because when the comparator is 'anyone not SGA', then the relative risk of SGA may be underestimated because the comparator lumps low-risk AGA babies with higher-risk LGA ones.
- Conduct further research on understudied exposures (ie, large size at birth/post-term) or outcomes (eg, current research on LGA is largely limited to outcomes of growth, diabetes or cancer) and on inconclusive areas (for small size these include coronary heart disease and heart function indicators, congenital defects, overweight, leukaemia, paediatric central nervous system tumours, type 1 diabetes, and adverse behavioural and visuomotor outcomes). For large size at birth, there are numerous areas with inconclusive results. There is also a need to conduct meta-analyses on the 14 subthemes without one.
- Address gaps in populations studied by further examining associations by different age groups and by sex, and by conducting additional research in low-income and middle-income countries for specific subtopics, particularly where risks may differ because of differences in access to treatment and preventive measures, or to differing epigenetic and environmental exposures.
- Conduct theme-based meta-analyses starting with subthemes that are inconsistent in the literature and with meta-analysis that have low-quality scores. Considering the different reasons for inconsistency indicated in the discussion, future research would benefit from subanalysis of the associations stratified by age at the occurrence of the outcome and by the sex of the child.

Acknowledging that both small and large size at birth contribute to multiple burdens of diseases, this study gives further evidence on the importance of correctly measuring size at birth in order to be able to intervene properly. Compiling this evidence allows researchers and policymakers to understand potential pathways for child survival and to further explore pathways for children to attain their full thriving potential. This study provides guidance to funders and researchers to help prioritise understanding of inconsistent evidence in the literature and to inform and prioritise points of interventions that contribute the most to disability-adjusted life years.

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