



Low birth weight: epidemiology, risk factors, and prevention strategies. A narrative review

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ABSTRACT

Introduction: Low birth weight, defined as a weight below 2500 grams in live newborns, is a fundamental indicator of maternal and child health and a predictor of neonatal morbidity and mortality with consequences throughout the entire life course. **Objective:** To comprehensively describe the current state of knowledge on low birth weight, covering its epidemiology, pathophysiological mechanisms, maternal risk factors, and strategies for detection, management, and prevention. **Methods:** A narrative literature review was conducted through searches in PubMed/MEDLINE, SciELO, LILACS, and Google Scholar, using the Health Sciences Descriptors (DeCS) and Medical Subject Headings (MeSH) "low birth weight infant," "premature birth," "fetal growth retardation," and "risk factors," complemented with clinical practice guidelines and technical documents from the World Health Organization. **Development:** Low birth weight affects 14.7% of live births worldwide and arises from two pathophysiological pathways—prematurity and intrauterine growth restriction—driven by largely modifiable maternal nutritional, metabolic, toxic, and infectious determinants. Antenatal ultrasound and Doppler surveillance, together with standardized neonatal care protocols, help mitigate its immediate and long-term complications. **Conclusions:** Low birth weight is a critical factor for neonatal survival and human development. Translating the accumulated scientific knowledge into scalable preventive strategies is necessary to achieve the reduction targets set by the World Health Organization.

Keywords: Infant, Low Birth Weight; Premature Birth; Fetal Growth Retardation; Risk Factors; Public Health.

RESUMEN

Introducción: El bajo peso al nacer, definido como un peso inferior a 2500 gramos en recién nacidos vivos, constituye un indicador fundamental de la salud materna-infantil y un predictor de morbilidad neonatal con repercusiones a lo largo de todo el curso de vida. **Objetivo:** Describir de manera integral el estado actual del conocimiento sobre el bajo peso al nacer, abarcando su epidemiología, sus mecanismos fisiopatológicos, los factores de riesgo maternos y las estrategias de detección, manejo y prevención. **Métodos:** Se realizó una revisión narrativa de la literatura mediante búsqueda en PubMed/MEDLINE, SciELO, LILACS y Google Scholar, con los descriptores DeCS/MeSH «recién nacido de bajo peso», «nacimiento prematuro», «retardo del crecimiento fetal» y «factores de riesgo», complementada con guías de práctica clínica y documentos técnicos de la Organización Mundial de la Salud. **Desarrollo:** El bajo peso al nacer afecta al 14,7 % de los nacimientos vivos a nivel mundial y se origina en dos vías fisiopatológicas —la prematuridad y la restricción del crecimiento intrauterino— condicionadas por determinantes maternos nutricionales, metabólicos, tóxicos e infecciosos, en su mayoría modificables. La vigilancia ecográfica y Doppler antenatal, junto con protocolos estandarizados de cuidados neonatales, permiten mitigar sus complicaciones inmediatas y a largo plazo. **Conclusiones:** El bajo peso al nacer es un factor crítico para la supervivencia neonatal y el desarrollo humano. Resulta necesario transformar el conocimiento científico acumulado en estrategias preventivas escalables que permitan alcanzar las metas de reducción establecidas por la Organización Mundial de la Salud.

Palabras clave: Recién Nacido de Bajo Peso; Nacimiento Prematuro; Retardo del Crecimiento Fetal; Factores de Riesgo; Salud Pública.

INTRODUCTION

Low birth weight is technically defined by the World Health Organization as a weight below 2500 grams in live newborns, regardless of gestational age. This definition was formalized approximately a century ago by the Finnish pediatrician Arvo Ylppö, while working in Germany, and was officially codified by the American Academy of Pediatrics and the World Health Organization between 1935 and 1948. The threshold of 2500 grams initially lacked specific biological justification but was selected as a round figure that encompassed approximately 5 % of newborns at that time. In 1961, a committee of experts from the World Health Organization emphasized the importance of distinguishing between premature infants and those born at term but small, consolidating the term “low birth weight” as the international standard(1).

The epidemiological magnitude of low birth weight represents a global challenge of considerable proportions. Recent estimates indicate that approximately 19.8 million newborns had low birth weight in 2020, representing 14.7% of all live births worldwide. Although this figure represents an absolute reduction of 1.9 percentage points since 2000, it reflects insufficient progress toward the established international targets. The geographic distribution of the problem is markedly unequal: nearly three quarters of low-birth-weight births occur in South Asia (44.5%) and sub-Saharan Africa (27.1%), regions that account for 55.5% of global births. The average annual rate of reduction between 2012 and 2020 was only 0.3% worldwide, far below the 3.31% needed to meet the goal set for 2030(1).

The theoretical framework underpinning the current understanding of low birth weight rests on 2 main conceptual pillars. The first recognizes that low birth weight can result from 2 distinct but frequently overlapping pathophysiological mechanisms: preterm birth (before 37 weeks of gestation) and intrauterine growth restriction, manifested as small for gestational age. The second theoretical pillar, known as the Barker hypothesis or the “developmental origins of health and disease” (DOHaD, its English acronym), proposes that adverse conditions during fetal development permanently program the structure, function, and metabolism of the organism through epigenetic modifications. This fetal programming predisposes to chronic noncommunicable diseases in adult life, including type 2 diabetes, hypertension, cardiovascular disease, and metabolic syndrome (2,3).

The consequences of low birth weight transcend the immediate neonatal period and extend throughout the entire life course. In the short term, low-birth-weight newborns face a significantly elevated risk of neonatal mortality, accounting for 40 % of neonatal deaths among preterm infants and 28 % among small-for-gestational-age infants born at term. During childhood, these children are at greater risk of growth retardation, wasting, neurodevelopmental impairment, cerebral palsy, epilepsy, reduced brain volume, respiratory disorders such as asthma, and morphological and functional cardiac alterations. During adolescence, impaired lung function, arterial hypertension, cognitive impairment, depression, anxiety, school problems, and psychiatric disorders persist. In adult life, individuals born with low birth weight have a higher risk of premature mortality, impaired renal function, metabolic syndrome, diabetes, coronary heart disease, stroke, depression, lower likelihood of completing higher education, lower employment, and reduced quality of life(4).

From a public health perspective, low birth weight represents a critical indicator of maternal and child health and a determinant of the human capital of populations, with a substantial economic burden in healthcare costs and lost productivity. Accumulated scientific evidence indicates that many of its risk factors are modifiable and preventable through interventions aimed at improving women’s nutrition, health, and medical care before and during pregnancy. However, despite decades of research and the establishment of specific global targets, progress in reducing its prevalence has been insufficient, particularly in South Asia and sub-Saharan Africa, a gap aggravated by the complexity of the underlying social, economic, and cultural determinants and by limitations in the quality of administrative data available in low- and middle-income countries(5,6).

The urgency of addressing this problem is reinforced by its direct link to multiple United Nations Sustainable Development Goals, particularly those related to neonatal and infant survival, the elimination of malnutrition, and the reduction of health inequalities. Low birth weight represents a critical point for intervention in the life cycle, where preventive actions can have multiplier effects on the health and well-being of present and future generations. Emerging evidence on the transgenerational effects of epigenetic modifications associated with low birth weight further underscores the importance of breaking this cycle through effective interventions (1,7). Therefore, a comprehensive and up-to-date understanding of low birth weight, its determinants, its consequences, and its prevention strategies is essential to inform evidence-based public health policies and programs. This article aims to comprehensively describe the current state of knowledge on low birth weight, including its global epidemiology, underlying pathophysiological mechanisms,

modifiable and non-modifiable risk factors, short- and long-term consequences for health and development, and evidence-based strategies for its prevention and management.

METHODS

A narrative review of the scientific literature on low birth weight was conducted, aimed at synthesizing the available evidence regarding its definition, epidemiology, pathophysiological mechanisms, maternal risk factors, prenatal detection strategies, neonatal management, and short- and long-term complications.

The literature search was conducted in the PubMed/MEDLINE, SciELO, LILACS, and Google Scholar databases, complemented by a review of clinical practice bulletins from the American College of Obstetricians and Gynecologists and technical documents from the World Health Organization. The Health Sciences Descriptors (DeCS) and Medical Subject Headings (MeSH) descriptors used were “low birth weight,” “premature birth,” “fetal growth retardation,” “risk factors,” and “public health,” combined using the Boolean operators AND and OR.

Original articles, systematic reviews, meta-analyses, cohort studies, bulletins, and clinical practice guidelines published preferably between 2000 and 2025, in Spanish or English, that addressed the epidemiology, pathophysiological mechanisms, risk factors, or strategies for the detection, management, and prevention of low birth weight were included. Letters to the editor, conference abstracts without full text available, and publications that had not undergone peer review were excluded.

The selection of studies was carried out by reading the title and abstract of the identified records, followed by full-text review of the documents considered relevant. The information obtained was synthesized narratively, organizing it into the following thematic axes: epidemiological magnitude and classification of low birth weight, pathophysiological differentiation between prematurity and intrauterine growth restriction, maternal factors associated with birth weight, strategies for risk detection before delivery, care protocol in the Neonatal Intensive Care Unit, and short- and long-term complications. Because this was a narrative and non-systematic review, no formal assessment of the risk of bias of the included studies was performed, nor was there one prior registration of the search protocol.

DEVELOPMENT

Magnitude and classification of low birth weight

Birth weight subcategories allow for more precise risk stratification and planning of specific interventions. Low birth weight (2000-2499 grams) represents the largest category, while very low birth weight is defined as a weight of 1500 grams or less, and extremely low birth weight corresponds to 1000 grams or less. Recent estimates from regions with high-quality data, such as North America, Australia, New Zealand, Central Asia, and Europe, show that approximately 65.6% of low-birth-weight newborns fall into the 2000-2499 grams category, 19.4% weigh between 1500-1999 grams, 9.0% between 1000-1499 grams, and only 5.8% weigh less than 1000 grams. Although this last group represents the smallest proportion, they are the most vulnerable newborns and at highest risk of mortality, since most are extremely premature(1,8).

The statistical relevance of low birth weight in neonatal mortality is extraordinarily significant globally. It is estimated that approximately 80 % of all neonatal deaths worldwide occur in infants with low birth weight, of whom approximately two thirds are preterm and a third are small for gestational age. In 2020, more than half of neonatal deaths (1,4 million) were attributable to small vulnerable newborns, with 73,4 % of these deaths related to prematurity or to the combination of prematurity and small for gestational age. Newborns who are both preterm and small for gestational age, although they represent only 1,1 % of births globally, have the highest mortality risk of all groups. In 2021, 81,01 % of global neonatal deaths were linked to low birth weight and short gestation, equivalent to 1,48 million deaths (8).

Prematurity and intrauterine growth restriction (RCIU)

The fundamental difference between a premature infant and an infant with intrauterine growth restriction lies in the underlying pathophysiological mechanisms that lead to low birth weight. Premature birth is characterized by a short gestation (less than 37 completed weeks), in which the infant is born too early but may have grown appropriately for gestational age. In contrast,

intrauterine growth restriction represents a pathological process in which the fetus fails to achieve its optimal growth potential due to fetal, maternal, or placental factors that limit uteroplacental perfusion and, consequently, fetal nutrition. This latter group of infants is born too small for gestational age, regardless of whether they are born preterm or at term(2,9).

This distinction is crucial because not all babies with low birth weight are premature. Low birth weight results from two underlying pathways that can occur independently or simultaneously: short gestation and fetal growth restriction, the latter identified by the classification of small for gestational age. A baby can be born with low birth weight while being premature, but with growth appropriate for gestational age, or can be born at term, but small for gestational age due to growth restriction. There is also a third group, premature and small for gestational age at the same time, which has the highest mortality risk of all (10).

The terminology used to classify these infants reflects this fundamental conceptual difference. The term “fetal growth restriction” (formerly called intrauterine growth restriction) is used during the prenatal period to describe fetuses with an estimated fetal weight or abdominal circumference below the 10th percentile for gestational age, according to the guidelines of the American College of Obstetricians and Gynecologists. This term indicates a pathological process that reflects fetal distress and prevents the fetus from reaching its growth potential. In contrast, the term “small for gestational age” applies exclusively to newborns whose birth weight is below the 10th percentile for their gestational age and sex, compared with international standards. It is important to note that, although most fetuses with intrauterine growth restriction are born small for gestational age, not all small-for-gestational-age infants had intrauterine growth restriction; some may be constitutionally small, physiologically normal, and have reached their growth potential(6,11).

The importance of understanding this distinction is grounded in multiple clinical and epidemiological dimensions. Gestational age is more predictive of the risk of mortality and morbidity than birth weight alone, and various risk prediction models identify it as the most important determinant of viability. Furthermore, the dichotomous classification at 2500 grams is not sufficiently granular to capture continuous risk gradients, and historical thresholds are increasingly less relevant given advances in neonatal intensive care, with neonates of barely 23 weeks surviving today.

Short- and long-term consequences also differ according to the underlying mechanism. Premature infants without growth restriction face risks linked to organ immaturity: intraventricular hemorrhage, hypoxic-ischemic encephalopathy, necrotizing enterocolitis, bronchopulmonary dysplasia, and sepsis. Those born at term but small for gestational age more frequently present perinatal asphyxia, hypothermia, hypoglycemia, and polycythemia. Both groups experience growth retardation and neurocognitive deficits, although the specific mechanisms may differ; infants with the dual condition—preterm and small for gestational age—accumulate the most adverse risk of all (1,8).

From the perspective of public health and prevention, the distinction between prematurity and intrauterine growth restriction is fundamental because risk factors and preventive interventions differ substantially between both conditions. Prematurity is more frequently associated with factors such as intrauterine infections, cervical incompetence, multiple pregnancies, acute obstetric complications, and socioeconomic and maternal stress factors. On the other hand, intrauterine growth restriction is more closely linked to placental insufficiency, maternal hypertension, preeclampsia, chronic maternal malnutrition, specific nutritional deficiencies—particularly of protein, energy, and micronutrients—, smoking, alcohol consumption, and chronic maternal medical conditions(2). This differentiation allows for the design of more effective targeted interventions: nutritional and micronutrient supplementation to prevent growth restriction, versus progesterone, cervical cerclage, or corticosteroids to prevent preterm birth.

Accurate clinical assessment of gestational age is essential to distinguish between these two conditions, but it represents a significant challenge in many resource-limited settings. Gestational age can be determined by early pregnancy ultrasound (ideally before 20 weeks), reliable last menstrual period, or neonatal physical examination using scales such as the Ballard score or the New Ballard Score. However, in low- and middle-income countries, where the greatest burden of low birth weight occurs, early ultrasound is often unavailable, menstrual dates may be uncertain, and neonatal clinical assessment requires trained personnel(12). This limitation in accurately determining gestational age hinders appropriate classification of infants and may lead to underestimation of the true prevalence of intrauterine growth restriction in these populations.

The long-term prognostic implications also differ according to the underlying mechanism of low birth weight. Studies that have examined developmental consequences suggest that infants with intrauterine growth restriction may experience distinct patterns of metabolic and epigenetic programming compared with preterm infants. Fetal growth restriction is particularly associated

with metabolic adaptations that predispose to metabolic syndrome, type 2 diabetes, hypertension, and cardiovascular disease in adult life, following Barker's hypothesis on the developmental origins of health and disease. These adaptations include the redistribution of blood flow that favors vital organs such as the brain at the expense of other organs—a phenomenon known as brain sparing—, a reduction in the number of renal nephrons, alterations in insulin sensitivity, and epigenetic modifications that affect gene expression throughout life(4).

In contrast, preterm infants without growth restriction may face consequences more directly related to organic immaturity, including increased risk of chronic respiratory problems (bronchopulmonary dysplasia), retinopathy of prematurity, neurocognitive deficits related to cerebral white matter injury, and neurodevelopmental problems. Although there is considerable overlap in long-term outcomes between both groups, understanding the specific mechanisms allows better anticipation of follow-up needs and secondary preventive interventions. For example, infants with intrauterine growth restriction may particularly benefit from long-term metabolic and cardiovascular monitoring, while preterm infants require more intensive ophthalmological, audiological, and neurodevelopmental follow-up(9).

It is important to recognize that vulnerability exists on a continuous spectrum rather than in discrete categories. Infants may present with varying degrees of prematurity and growth restriction, and approximately 30-40% of preterm infants are also small for gestational age, experiencing both mechanisms simultaneously. This “double vulnerability” group has the poorest outcomes and requires the most intensive care. Recognizing that low birth weight is not a single entity but the final result of multiple pathophysiological pathways is essential for designing effective preventive strategies and appropriate clinical management(13).

Maternal factors influencing infant weight

Multiple interrelated maternal factors influence infant weight at birth, including demographic characteristics, nutritional status, chronic diseases, and modifiable habits. Maternal age, body mass index (BMI), smoking, chronic diseases—such as diabetes and hypertension—and substance use represent the most significant maternal determinants of birth weight.

Maternal age

Maternal age exhibits a complex relationship with birth weight. Mothers older than 30 years have a higher risk of having preterm infants, whereas advanced age is positively associated with birth weight only in women of White European and South Asian origin without gestational diabetes. Pregnant adolescents tend to respond more favorably to nutritional supplementation, reducing the risk of small and vulnerable newborns; overall, maternal age has significant effects, although of small magnitude compared with other risk factors(14,15).

Maternal nutritional status

Maternal nutritional status before and during pregnancy exerts a profound influence on birth weight. Maternal BMI is positively associated with birth weight across all racial groups, regardless of glycemic status, and each unit increase in BMI is associated with an increase of approximately 14.9 grams in birth weight. Underweight women are at increased risk of having low-birth-weight newborns, whereas maternal overweight and obesity ($\text{BMI} \geq 30 \text{ kg/m}^2$) show a protective effect against low birth weight. However, maternal obesity is causally associated with higher birth weight, probably through maternal and fetal dysregulation of glucose, insulin, lipid, and amino acid metabolism(16).

Gestational weight gain is a critical predictor of birth weight. Each kilogram increase in total weight gain results in an increase of approximately 30 grams in birth weight. Women with a weight gain $\leq 4 \text{ kg}$ during pregnancy have a significantly elevated risk of low birth weight, whereas those with a gain $> 18 \text{ kg}$ have a higher risk of having large-for-gestational-age infants. The transition from an overweight state between adolescence and early pregnancy is associated with significantly higher birth weight (144.6 grams) compared with mothers who maintain normal weight at both time points. Maternal diet quality, expressed by the NAR index, is significantly correlated with birth weight, and its inclusion improves prediction equations by 6-8% (17).

Maternal anemia is a significant risk factor for low birth weight, and micronutrient deficiencies—particularly iron, zinc, and calcium—compromise optimal fetal growth. Interestingly, some regression models show a negative relationship between iron and protein intake and birth weight, which likely reflects targeted supplementation among women with deficiencies rather than a true adverse effect of these nutrients(18).

Diabetes and metabolic disorders

Maternal diabetes, both pregestational and gestational, is a major determinant of birth weight. Infants of mothers with gestational diabetes are significantly heavier than those of mothers without diabetes across all racial groups, with the greatest effect in Black and South Asian women, and gestational diabetes accentuates the effect of maternal BMI on birth weight, especially in non-White populations. Mothers with pregestational diabetes, in contrast, have a higher risk of low birth weight, possibly because hyperglycemia elevates maternal and fetal circulating insulin—a fetal growth hormone—with potential effects on long-term body weight(9).

Arterial hypertension

Maternal hypertension is among the strongest risk factors for low birth weight. Mothers with hypertension have a markedly elevated risk of having low-birth-weight infants, and pregnancy-induced hypertension is significantly associated with low birth weight. Higher diastolic blood pressure is significantly associated with lower birth weight z-scores. The underlying mechanisms include placental insufficiency and intrauterine growth restriction secondary to impaired uteroplacental perfusion(12).

Smoking

Maternal smoking during pregnancy is among the most significant modifiable risk factors for low birth weight. Current smokers have an increased risk of having preterm and low-birth-weight infants. Women who smoke more than 10 cigarettes per day have a particularly high risk of having small-for-gestational-age infants (19).

Babies of smoking mothers weigh approximately 180.5 grams less than those of non-smoking mothers, and after adjusting for maternal age, height, education, and socioeconomic status, the difference remains 224 grams. Mechanisms include increased oxidative stress and limited oxygen delivery to the placenta, since smoking interferes with oxygen binding to maternal hemoglobin. Quitting smoking during pregnancy significantly reduces the risk of low birth weight, and countries that have banned smoking in enclosed public spaces have recorded a substantial reduction in its prevalence (6,19).

Alcohol and other substance use

Alcohol consumption during pregnancy is also associated with low birth weight. The use of crack or cocaine is among the most potent risk factors across all substances, with a nearly three times higher risk, through placental vasoconstriction, reduced uteroplacental flow, and direct toxic effects on the fetus. Caffeine consumption shows a similar association, although of considerably lower magnitude than tobacco and illicit drugs(12).

Detection of the risk of low birth weight before delivery

Initial clinical assessment through measurement of uterine height represents a low-cost, relatively reliable, and easy-to-implement method for detecting fetal growth abnormalities in most pregnant women. When a growth abnormality is clinically suspected, or a medical or obstetric condition that increases risk is present, ultrasound is the modality of choice for identifying abnormal fetal growth. A significant discrepancy between uterine height and expected gestational age should prompt additional ultrasound evaluation(20).

Obstetric ultrasound and fetal biometry allow estimation of fetal weight using four standard parameters: biparietal diameter, head circumference, abdominal circumference, and femur length. These morphological parameters are converted into fetal weight estimates using published formulas, although the variability of the estimate can reach up to 20% in 95% of cases. If the estimated fetal weight or abdominal circumference is below the 10th percentile for gestational age, additional evaluation should be considered, including assessment of amniotic fluid and Doppler studies of the umbilical artery. Because fetuses with growth restriction have a high incidence of structural and genetic anomalies, specialized ultrasound examination of fetal anatomy is recommended if it has not been performed previously (21).

Umbilical artery Doppler constitutes an essential component of fetal surveillance in growth restriction, since it assesses flow impedance in the fetoplacental unit and differentiates the fetus with hypoxic restriction from the nonhypoxic small fetus. Absent or reversed diastolic flow is associated with a higher risk of perinatal mortality, and the incorporation of umbilical Doppler into the management of high-risk pregnancies reduces perinatal death, induction of labor, and cesarean

section, with a decrease of up to 29% in perinatal mortality. Emerging evidence suggests that umbilical vein Doppler—particularly the venous diameter at the free loop—improves risk stratification in small fetuses with normal arterial Doppler, helping to identify high-risk pregnancies and to plan delivery(22).

Surveillance protocols vary according to the severity of the findings. When the estimated fetal weight is below the 10th percentile, fetal surveillance is recommended, which may begin from 26-28 weeks, with serial ultrasound examinations every 3-4 weeks. In the presence of arterial Doppler abnormalities, weekly follow-up is usually sufficient if antegrade diastolic flow persists; when oligohydramnios or absent or reversed flow are associated, surveillance should be intensified to 2-3 times per week, complemented by cardiotocography or weekly biophysical profile(23).

Care protocol in the Neonatal Intensive Care Unit for newborns with low birth weight

Newborns with low birth weight require specialized care in facilities with trained personnel and appropriate resources according to the severity of their condition. The care protocol in the Neonatal Intensive Care Unit (UCIN) is structured into multiple interrelated components, ranging from initial stabilization to preparation for hospital discharge.

Definition and classification according to level of care

Low birth weight is classified into three categories that determine the level of care required: low birth weight (2000-2499 g), very low birth weight (≤ 1500 g), and extremely low birth weight (≤ 1000 g). Level II units care for infants of 32 weeks or more, or ≥ 1500 g, with mild to moderate conditions that do not require urgent subspecialty care or more than CPAP or brief conventional ventilation. Level III units care for infants of any gestational age and weight with critical conditions requiring sustained life support, high-frequency ventilation, inhaled nitric oxide, or therapeutic hypothermia. Level IV units offer the full spectrum of care, including surgery for complex congenital or acquired conditions, extracorporeal membrane oxygenation (ECMO), and access to pediatric subspecialists(24,25).

Initial stabilization and immediate care

Initial stabilization requires meticulous attention to thermoregulation, resuscitation, and monitoring. Core temperature should be maintained above 36.4 °C, since hypothermia increases morbidity and mortality; the use of plastic wrap can help maintain it. Newborns with tachypnea greater than 60 breaths per minute should receive intravenous fluids instead of enteral feeding to prevent aspiration, and hypoglycemia—which may be asymptomatic in large-for-gestational-age infants and infants of diabetic mothers—requires systematic monitoring(24).

Prevention and management of infections

The risk of neonatal sepsis increases with prolonged rupture of membranes and maternal colonization by group B Streptococcus, so low-birth-weight newborns require systematic evaluation and empiric antibiotic treatment when indicated. In settings where hospitalization is not feasible, community provision of antibiotics for possible serious bacterial infection can reduce neonatal mortality(26).

Nutrition and Food Support

Mother's own milk, appropriately fortified, is the optimal source of nutrition for infants with very low birth weight who are hospitalized in the NICU, and confers multiple short- and long-term health benefits, including lower rates of necrotizing enterocolitis. The family of each infant with very low birth weight should receive information about the critical importance of mother's milk for their infant's health, as well as intensive support throughout the entire hospitalization to achieve their lactation goals. When mother's own milk is unavailable, insufficient in volume, or contraindicated, pasteurized donor human milk is recommended, optimally continued until the infant's risk of developing necrotizing enterocolitis is low (approximately 34-36 weeks postmenstrual age). Preterm infant formula is recommended when mother's milk is unavailable and donor milk is also unavailable, or when the family declines its use(26).

Short- and long-term complications

Immediate risks in the neonatal period

Hypothermia is among the most frequent morbidities, affecting 75,26 % of low-birth-weight preterm newborns. This thermal vulnerability is due to immaturity of the thermoregulatory system, a greater relative body surface area, scant subcutaneous adipose tissue, and a limited capacity to generate heat. Sepsis is another critical risk, present in 69,07 % of these neonates, and is associated with double the risk of mortality. The mechanisms include immaturity of the immune system, a greater need for invasive procedures, and prolonged exposure to the hospital environment(27).

Respiratory distress affects 49,83 % of low-birth-weight preterm newborns and is among the strongest predictors of mortality, with a risk of death 4,6 times greater. This complication results from pulmonary immaturity, surfactant deficiency, and incomplete alveolar development. Hypoglycemia, although less frequent, is associated with an almost 4 times greater risk of mortality. Low-birth-weight infants have limited reserves of hepatic glycogen and body fat, along with a relatively higher metabolic demand, which predisposes them to hypoglycemic episodes that can cause permanent neurological damage if not adequately treated(27).

Other frequent neonatal complications include jaundice (13.4%), congenital anomalies (8.25%), intraventricular hemorrhage, hypoxic-ischemic encephalopathy, necrotizing enterocolitis, and bronchopulmonary dysplasia (8).

Long-term consequences in childhood

During childhood, children born with low birth weight have an increased risk of mortality, growth retardation (stunting), and emaciation (wasting). Neurological complications include a higher risk of cerebral palsy and epilepsy in preterm infants, reduced brain volume, and delayed and impaired neurodevelopment in both preterm infants and those small for gestational age. Motor development problems are common in preterm infants and in infants with low birth weight(28).

Cognitive performance is significantly affected, with a reduction in intelligence quotient (IQ) and cognitive performance in preterm infants, those small for gestational age, and those with low birth weight. Children with extremely low birth weight, assessed at 8-9 years, showed that 24 per 100 had an IQ below 85, 38 more received special medical or educational services, and 43 more had some functional limitation, compared with children of normal birth weight. Approximately a third of children with extremely low birth weight, without neurosensory abnormalities at discharge, had an IQ below 85, learning problems or poor motor skills, and two thirds had behavioral problems, a proportion 2 to 3 times higher than that of controls(29).

Academic problems are common in preterm infants, with difficulties in reading, spelling, and mathematics, reduced language skills, and increased risk of dysphonia; school performance is impaired in both preterm infants and those born with low birth weight. Neurodevelopmental disorders include increased risk of ADHD and autism spectrum disorders in preterm infants, those born with low birth weight, and those small for gestational age. One Korean population study of 830 806 births showed that children aged 5-7 years with low birth weight at term had significant associations with motor and cognitive developmental delay, autism spectrum disorders, ADHD, and epileptic and febrile seizures(29).

DISCUSSION

The reviewed evidence confirms that low birth weight does not constitute a single clinical entity, but rather the final expression of two distinct pathophysiological pathways — prematurity and intrauterine growth restriction — which frequently coexist and require differentiated preventive approaches. Recognizing this distinction has direct implications for clinical practice, given that gestational age continues to be the most robust predictor of neonatal morbidity and mortality, whereas fetal growth restriction is more specifically linked to metabolic programming and chronic noncommunicable diseases in adult life, in accordance with Barker's hypothesis on the developmental origins of health and disease (2,3,4,7).

From a prevention perspective, most of the identified maternal determinants—nutritional status, diabetes and other metabolic disorders, hypertension, smoking, and substance use—are modifiable through interventions before and during pregnancy. This reinforces that low birth weight constitutes a critical intervention point in the life course, where cost-effective strategies such as nutritional supplementation, smoking cessation, and strict blood pressure control can translate into substantial reductions in its incidence (12,16,17,18,19).

Antenatal surveillance by ultrasound and Doppler of the umbilical artery and vein, integrated into serial follow-up protocols, makes it possible to differentiate the fetus with growth restriction of hypoxic-placental origin from the constitutionally healthy small fetus, guiding decisions on the optimal timing of delivery. Similarly, classification of neonatal units by levels of complexity and standardization of immediate care—thermoregulation, hypoglycemia screening, sepsis prevention, and prioritization of breastfeeding—constitute interventions with demonstrated impact on the mortality and morbidity of low-birth-weight newborns(22,23,25,26,27).

This review has limitations inherent to its narrative design. Because it is not a systematic review, no exhaustive and reproducible search or formal assessment of the methodological quality or risk of bias of the included studies was performed; thus, the selection of evidence may have been subject to some degree of subjectivity. Likewise, much of the available evidence comes from high- and middle-income countries, which could limit the direct extrapolation of some findings to contexts such as those of Latin America and the Caribbean, where social determinants and access to maternal and child health services differ substantially. Local primary studies are required to characterize the magnitude and determinants of low birth weight in these populations, in order to adapt preventive strategies to their epidemiological reality.

Conclusions

Low birth weight should not be considered solely as an immediate statistical figure, but as a phenomenon with transgenerational repercussions that shapes individuals' metabolic health and long-term socioeconomic development potential. Understanding that its origin lies in two distinct pathophysiological mechanisms—prematurity and intrauterine growth restriction—is decisive for effectively guiding clinical interventions and preventive policies according to the regional context. Limited progress in reducing global incidence demonstrates a critical disconnect between accumulated scientific evidence on modifiable factors and the implementation of public health programs that address underlying social and nutritional inequalities. In addition, local studies are required to adapt these strategies to the epidemiological reality of Latin America and the Caribbean.

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CONFLICT OF INTEREST

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