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ANAEMIA AND IRON DEFICIENCY ANAEMIA: A GLOBAL REVIEW OF EPIDEMIOLOGY, DETERMINANTS AND THE PUBLIC HEALTH RESPONSE

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ABSTRACT

Background: Anaemia affects roughly one in four people worldwide, and iron deficiency is its most common single cause. The two conditions are often conflated in the literature, which distorts burden estimates and policy messages. This narrative review separates all-cause anaemia, iron deficiency and iron deficiency anaemia (IDA), and reviews their epidemiology, determinants, consequences and the public health response.

Methods: We searched PubMed/MEDLINE and Google Scholar and the WHO and Global Burden of Disease (GBD) repositories for English-language sources published between January 2021 and November 2026, supplemented by landmark guidelines and systematic reviews. Estimates are reported with their source, reference year and outcome definition..

Findings: WHO estimates for 2023 put anaemia prevalence at 30.7% in women aged 15–49 years (about 605 million women), 35.5% in pregnant women and 30.5% in non-pregnant women; the latest WHO child estimate (2019) is 39.8% in children aged 6–59 months. GBD 2021 estimated 1.92 billion anaemia cases of all ages, of which 66.2% were attributed to dietary iron deficiency. Prevalence among women of reproductive age is highest in the WHO South-East Asia (46.4%), African (36.5%) and Eastern Mediterranean (35.8%) regions and has not fallen since 2012. Only one of 195 countries and territories is on track for the 2030 target of a 50% reduction from the 2012 baseline. A GBD-based model projects that iron deficiency cases will rise from 1.27 billion in 2021 to 1.44 billion by 2050, although age-standardised rates change little. Causes beyond iron deficiency (inflammation, infection, chronic kidney disease, haemoglobinopathies, blood loss, vitamin B12 and folate deficiency) account for a large share of anaemia in many settings, and iron-deficiency diagnosis is confounded by inflammation. Supplementation and fortification are effective under good delivery conditions, but coverage, adherence and quality limit population impact, as seen in India's Anemia Mukt Bharat programme, whose operational guidelines were revised in June 2026.

Conclusions: Progress requires better-defined indicators (haemoglobin plus inflammation-adjusted ferritin), cause-specific programming, and stronger evaluation of delivery rather than distribution alone.

KEYWORDS Anaemia; iron deficiency; iron deficiency anaemia; global health; Global Burden of Disease; hepcidin; Anemia Mukt Bharat; Sustainable Development Goals

1. INTRODUCTION

1.1 Anaemia, iron deficiency and iron deficiency anaemia: terminology and scope

Anaemia is a reduction in haemoglobin (Hb) concentration below an age-, sex- and pregnancy-specific threshold. It is a syndrome with many causes. Iron deficiency is a state of depleted body iron, usually defined biochemically by low serum ferritin, and can exist with normal Hb. Iron deficiency anaemia (IDA) is the subset of anaemia caused by iron deficiency; strictly, it requires both reduced Hb and laboratory evidence of iron deficiency. These three terms are not interchangeable (Table 1), and the available global data measure different things: WHO estimates are for all-cause anaemia, defined by Hb alone; GBD estimates anaemia by cause, attributing a share to dietary iron deficiency; and no global survey series reports biomarker-confirmed IDA prevalence at comparable scale.

This review therefore uses the following convention throughout. "Anaemia" means all-cause anaemia defined

by Hb. "Iron deficiency" refers to depleted iron stores or, where stated, the GBD category "dietary iron deficiency". "IDA" is used only when both low Hb and iron deficiency are documented. Where a cited source uses a different term, we report its own outcome and say so.

Table 1. Terminology and the indicators typically used to measure each condition

Term	Definition	Usual measurement	Main global data source
Anaemia (all-cause)	Hb below age-, sex- and pregnancy-specific threshold, whatever the cause	Hb (venous or capillary blood), adjusted for altitude and smoking	WHO global anaemia estimates; GBD
Iron deficiency	Depleted iron stores, with or without anaemia	Serum ferritin (inflammation-adjusted); transferrin saturation; soluble transferrin receptor	Biomarker surveys (e.g. BRINDA); GBD "dietary iron deficiency" (modelled)
Iron deficiency anaemia (IDA)	Anaemia caused by iron deficiency	Low Hb plus low ferritin or low transferrin saturation	No standardised global series; model-based attribution only

Hb, haemoglobin; BRINDA, Biomarkers Reflecting Inflammation and Nutritional Determinants of Anemia.
Source: definitions synthesised from [1–4].

1.2 Why the burden deserves continued attention

Anaemia was the third-leading cause of years lived with disability (YLDs) worldwide in 2021 [3]. Unlike child stunting, for which global prevalence has fallen steadily, anaemia in women of reproductive age has been essentially flat for two decades [1]. WHO's 2025 estimates show that prevalence in non-pregnant women has risen since 2012 and that the severity of the public health problem has increased in most countries [1]. Because women and young children have both the highest iron requirements and the slowest progress, the consequences extend across generations.

1.3 Objectives

This review aims to (i) clarify what global estimates do and do not say about anaemia, iron deficiency and IDA; (ii) describe prevalence, regional patterns and trends, with source and year for every estimate; (iii) synthesise diagnostic principles and the full range of causes of anaemia, including non-nutritional causes; (iv) appraise the evidence on consequences, distinguishing association from causation; (v) critically evaluate supplementation, fortification and national programmes, with India's Anemia Mukt Bharat as a case study; and (vi) set out research and policy priorities.

2. REVIEW METHODS

This is a narrative review. We did not register a protocol and did not pool data; the methods below describe how sources were identified and selected so that the process can be reproduced.

2.1 Sources and search strategy

We searched PubMed/MEDLINE and Google Scholar from 1 January 2021 to 8 November 2026, using combinations of controlled vocabulary and free-text terms for anaemia ("anemia" OR "anaemia"), iron deficiency ("iron deficiency" OR ferritin OR hepcidin), global or regional scope ("global burden" OR prevalence OR epidemiology) and response (supplementation OR fortification OR "Anemia Mukt Bharat"). An example PubMed string was: (anaemia[Title/Abstract] OR anemia[Title/Abstract]) AND ("iron deficiency"[Title/Abstract] OR ferritin[Title/Abstract] OR hepcidin[Title/Abstract]) AND (global[Title/Abstract] OR prevalence[Title/Abstract] OR "burden of disease"[Title/Abstract] OR supplementation[Title/Abstract] OR fortification[Title/Abstract]), limited to 2021–2026. We also searched the WHO Global Health Observatory and Vitamin and Mineral Nutrition Information System, the IHME GBD Results tool, the WHO Institutional Repository for guidelines, and the websites of the Government of India's Ministry of Health and Family Welfare and the International Institute for Population Sciences. Reference lists of key reviews were hand-searched, and foundational guidelines and Cochrane reviews published before 2021 were included where they define current recommendations. Box 1 lists each source, the terms or filters used and the rule applied when selecting from it. For every quantitative estimate we retrieved the primary source and checked the figure reported in the text, tables and figures against it; estimates that could not be traced to a primary source were not used.

Box 1. Sources searched, search terms and source-selection rules

Source	What was searched	Terms, filters and period	How it was used
PubMed/MEDLINE	Journal literature	String in Section 2.1 (Title/Abstract fields); 1 January 2021 to 8 November 2026; English	Title/abstract screen, then full text; reviews, trials and large population studies retained

Source	What was searched	Terms, filters and period	How it was used
Google Scholar	Journal and grey literature	Same concepts as free-text queries; same period and language	Used to find sources not indexed in PubMed; same eligibility rules
WHO GHO, VMNIS and IRIS	Prevalence estimates; WHO guidelines	Anaemia in women and children; guideline titles on iron, haemoglobin and ferritin	Primary source for every WHO figure; latest edition used
IHME GBD Results tool and GBD publications	Modelled anaemia burden by cause	GBD 2021 anaemia and dietary iron deficiency	Reported with the GBD region system; never combined with WHO estimates
MoHFW (PIB, NHM) and IIPS	India programme documents and survey reports	Anemia Mukt Bharat; NFHS-5 and NFHS-6	Official documents preferred; news reports used only where no official document was available
Hand-searching	Reference lists of key reviews; pre-2021 guidelines and Cochrane reviews	Not date-limited	Included where they define current recommendations (Table 5)

GHO, Global Health Observatory; VMNIS, Vitamin and Mineral Nutrition Information System; IRIS, WHO Institutional Repository for Information Sharing; IHME, Institute for Health Metrics and Evaluation; GBD, Global Burden of Disease; MoHFW, Ministry of Health and Family Welfare; PIB, Press Information Bureau; NHM, National Health Mission; IIPS, International Institute for Population Sciences; NFHS, National Family Health Survey.

2.2 Eligibility criteria

We included (a) official global or regional estimates from WHO, GBD or national surveys; (b) systematic reviews, meta-analyses, randomised trials and large population-based studies reporting prevalence, determinants, consequences or intervention effects; and (c) WHO guidelines and national programme documents. We excluded case reports, conference abstracts without full data, non-English articles, and articles that did not define their anaemia or iron deficiency outcome. Where several sources reported the same estimate, we preferred the primary source (WHO or the GBD publication) over secondary reports.

2.3 Selection and data extraction

Titles and abstracts were screened by the author, and full texts were read for sources that appeared to meet the criteria; because this is a narrative review, no record counts or flow diagram are reported, and sources were chosen for relevance, methodological quality and recency using the following order of preference: (i) official or primary sources (WHO, GBD publications, national survey reports and government programme documents); (ii) WHO guidelines and systematic reviews or meta-analyses; (iii) randomised trials and large population-based studies; and (iv) news reports, used only where no official document was available (for example, for the content of the NFHS-6 fact sheets). Screening and extraction were done by one reviewer without a second check (Section 2.5). For each source we extracted the population, year, outcome definition (Hb alone, ferritin-based, or GBD cause-attributed), geographic classification and measurement method. Estimates that used different outcome definitions are never combined in this review.

2.4 Geographic classification

Two regional systems appear in the literature and are kept distinct here. WHO estimates are reported for the six WHO regions (African, Americas, European, South-East Asia, Eastern Mediterranean, Western Pacific) and for United Nations/SDG regions. GBD estimates use the 21 GBD regions grouped into seven super-regions. We state the system used for every regional figure.

2.5 Limitations of the approach

Narrative reviews are subject to selection bias, and the restriction to English-language sources and to publications since 2021 may omit relevant earlier or regional work. Global estimates are modelled and carry wide uncertainty intervals, which we report where possible. We did not formally appraise study quality; instead, we flag observational designs and cross-sectional associations explicitly in the text. Screening and data extraction were performed by a single reviewer, no protocol was registered, and the search of Google Scholar cannot be fully reproduced because its ranking changes over time.

3. DEFINITION, PATHOPHYSIOLOGY AND DIAGNOSTIC ASSESSMENT

3.1 Iron homeostasis and hepcidin

Body iron is conserved tightly; humans have no regulated excretory pathway, so balance is set mainly by absorption in the proximal small intestine and by recycling from senescent red cells. The hepatic peptide hormone hepcidin is the master regulator. Hepcidin binds the iron exporter ferroportin on enterocytes and macrophages and causes its degradation, which reduces dietary iron absorption and traps iron in stores [5]. Hepcidin is suppressed by iron deficiency, hypoxia and increased

erythropoietic demand, and is induced by iron repletion and, strongly, by inflammation through interleukin-6. Two clinical consequences follow. First, in inflammation or infection, hepcidin-mediated iron sequestration lowers serum iron and causes anaemia of inflammation even when stores are adequate [6]. Second, a large oral iron dose raises hepcidin for roughly a day, so fractional absorption of subsequent doses falls; alternate-day dosing increased fractional absorption in iron-depleted women in a stable-isotope study [7].

3.2 Stages of iron deficiency

Iron deficiency develops in stages: (i) depletion of storage iron (low ferritin) with normal Hb; (ii) iron-deficient erythropoiesis, in which transferrin saturation falls and red-cell indices begin to change without overt anaemia; and (iii) IDA, with microcytic hypochromic red cells and reduced Hb [8,9]. Because many people are iron deficient without being anaemic, Hb-based prevalence understates the burden of iron deficiency, while in settings with heavy infection or haemoglobinopathy burden it overstates the share of anaemia that is due to iron.

3.3 Haemoglobin thresholds

WHO defines anaemia by Hb thresholds that vary by age, sex and pregnancy status, with adjustment for altitude and smoking. In 2024 WHO replaced the 2011 thresholds with a revised guideline: the threshold for children aged 6–23 months was lowered to 105 g/L, and pregnancy thresholds became trimester-specific, with 105 g/L in the second trimester [10]. Table 2 shows the current cut-offs. The WHO 2025 global estimates for women still used the earlier thresholds (110 g/L in pregnancy and 120 g/L in non-pregnant women) and note that updating to the 2024 criteria is planned [1]. Measurement choices matter: an analysis of 406,106 children in 48 countries comparing CDC 1989 with WHO 2024 altitude adjustments found that most national estimates changed little, but three countries (Uganda, Rwanda and Jordan) shifted by 10 percentage points or more [11]. Hb measured on capillary blood with portable photometers is also more variable than venous measurement, and recent WHO estimates explicitly model device and sample type [1].

Table 2. WHO 2024 haemoglobin thresholds defining anaemia (sea level, non-smokers)

Population	Hb threshold (g/L)	Change from 2011 criteria
Children 6–23 months	< 105	Lowered (was < 110)
Children 24–59 months	< 110	None
Children 5–11 years	< 115	None
Children 12–14 years	< 120	None
Non-pregnant women ≥ 15 years	< 120	None
Men ≥ 15 years	< 130	None
Pregnancy, first trimester	< 110	None
Pregnancy, second trimester	< 105	Lowered (was < 110)
Pregnancy, third trimester	< 110	None

Source: WHO guideline on haemoglobin cutoffs, 2024 [10]. Altitude and smoking adjustments apply. Severity grades (mild, moderate, severe) are defined in the guideline.

3.4 Laboratory assessment of iron status

Hb cannot distinguish IDA from other anaemias. Iron status is assessed with serum ferritin, which reflects storage iron when no inflammation is present. WHO's 2020 guideline defines iron deficiency as ferritin below 15 µg/L in individuals aged 5 years and older and below 12 µg/L in children under 5 years in apparently healthy people; in the presence of infection or inflammation, higher cut-offs (below 70 µg/L and below 30 µg/L respectively) are recommended [2]. Ferritin is an acute-phase protein, so concentrations rise with inflammation, obesity-related inflammation, liver disease and malignancy and can mask true deficiency. For population surveys, ferritin should be interpreted with C-reactive protein and α1-acid glycoprotein, using regression-based adjustment such as the BRINDA approach [4]. Transferrin saturation (serum iron divided by total iron-binding capacity) is a complementary measure; values below about 16–20% suggest iron-restricted erythropoiesis, but it is also low in anaemia of inflammation and varies diurnally [8,9]. Soluble transferrin receptor is less affected by inflammation but is not widely available. In practice, a low ferritin with low Hb supports IDA, whereas a normal or high ferritin with low transferrin saturation and raised inflammatory markers suggests anaemia of inflammation, possibly with coexisting iron deficiency. Red-cell indices (low mean corpuscular volume) support iron deficiency but are also reduced in thalassaemia trait.

3.5 Clinical features

Symptoms are non-specific and include fatigue, reduced exercise tolerance, pallor, dyspnoea and palpitations on exertion, headache and impaired concentration. Koilonychia and glossitis are uncommon signs of long-standing deficiency. In children, iron deficiency is associated with irritability, impaired growth and delayed developmental milestones. Because clinical signs are insensitive, laboratory confirmation is needed for diagnosis.

4. EPIDEMIOLOGY

4.1 Prevalence by population group

Table 3 and Figure 1 summarise the most recent WHO estimates. All figures refer to all-cause anaemia defined by Hb, not to IDA. WHO updates its women's estimates every three to five years; the 2025 edition covers women aged 15–49 years for 2000–2023 and the most recent child estimates are from the 2019 edition [1]. In 2023, 30.7% of women aged 15–49 years (604.8 million) were anaemic: 35.5% of pregnant women (29.2 million) and 30.5% of non-pregnant women (575.6 million). In 2019, 39.8% of children aged 6–59 months were anaemic [1,12].

GBD 2021 provides complementary estimates for all ages. Anaemia affected 1.92 billion people in 2021 (31.2% of women and 17.5% of men). Among those aged 15–49 years, prevalence was 33.7% in women and 11.3% in men, a threefold difference [3]. GBD and WHO figures differ in data, covariates, thresholds and modelling, and should not be mixed within a single comparison or interpreted as time trends against each other.

Table 3. Global anaemia prevalence by population group (all-cause anaemia, Hb-defined)

Population group	Source	Reference year	Prevalence (%)	Persons affected / note
Children 6–59 months	WHO	2019	39.8	Latest WHO child estimate; not updated in the 2025 edition
Pregnant women 15–49 years	WHO	2023	35.5	29.2 million
Non-pregnant women 15–49 years	WHO	2023	30.5	575.6 million
All women 15–49 years	WHO	2023	30.7	604.8 million (equals the pregnant plus non-pregnant rows; do not add it to them)
Women 15–49 years	GBD 2021	2021	33.7	GBD estimate; different method from WHO
Men 15–49 years	GBD 2021	2021	11.3	Comparator for the sex gap
All ages, both sexes	GBD 2021	2021	—	1.92 billion cases

Rows for pregnant and non-pregnant women are mutually exclusive subgroups of all women aged 15–49 years; the "all women" row is their weighted total and must not be added to them. Sources: WHO global anaemia estimates 2025 [1]; Stevens et al. 2022 [12]; Gardner et al. 2023 [3].

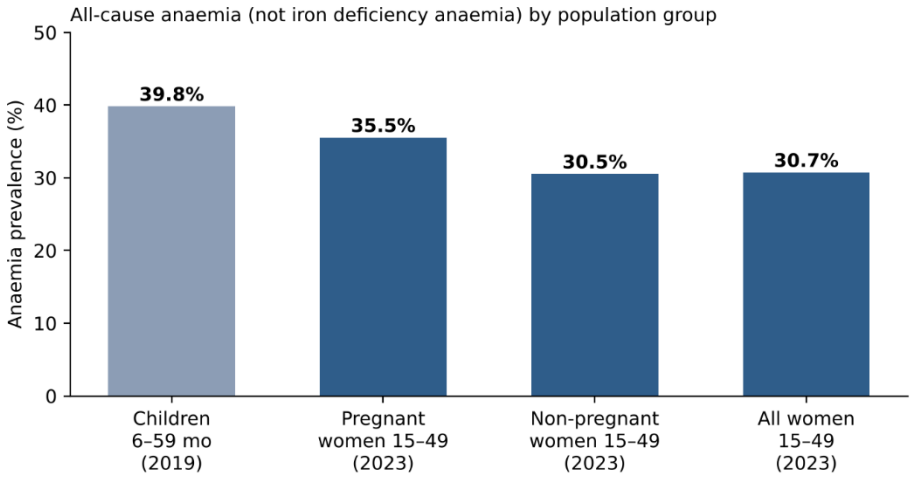


Figure 1. All-cause anaemia prevalence in children (2019) and women aged 15–49 years (2023). Source: WHO global anaemia estimates, 2025 [1]. These are Hb-based estimates and do not measure iron deficiency anaemia

4.2 Regional distribution

Using WHO regions, prevalence in women aged 15–49 years in 2023 was highest in South-East Asia (46.4%), followed by the African (36.5%) and Eastern Mediterranean (35.8%) regions, and lowest in the Western Pacific (16.5%) and Americas (18.3%) regions (Figure 2) [1]. In absolute numbers, the African region (110.0 million) and South-East Asia region (256.6 million) together account for more than 60% of all anaemic women of reproductive age [1]. At sub-regional level, Southern Asia had the highest prevalence (49.3%),

higher than any African sub-region (Middle Africa 41.7%, Western Africa 41.9%) [1]. By income group, prevalence was 35.7% in low-income and 43.8% in lower-middle-income countries versus 17.4% in high-income countries [1].

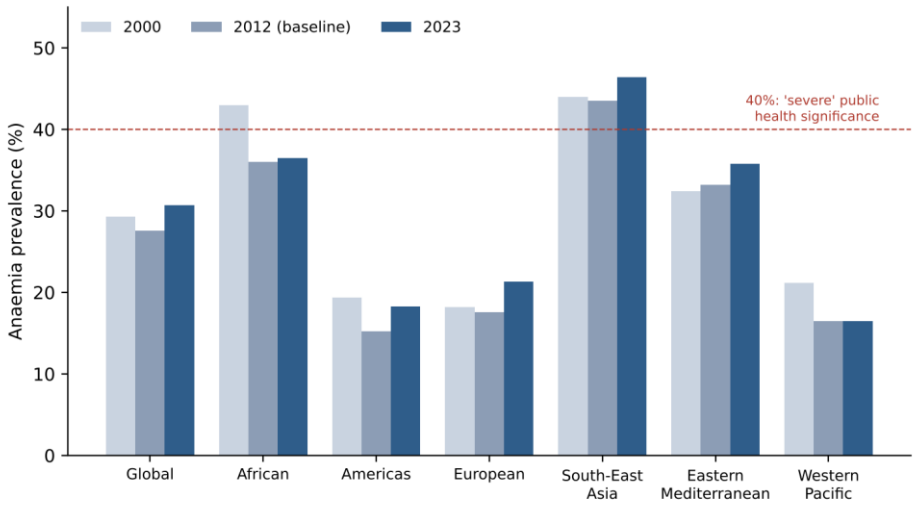


Figure 2. Anaemia prevalence in women aged 15–49 years by WHO region, 2000, 2012 and 2023. Source: WHO global anaemia estimates, 2025 (Table 1) [1]. WHO regions are used here; the dashed line marks the 40% threshold for a severe public health problem.

GBD 2021 uses a different geographic classification (21 GBD regions) and reports the highest prevalence in Western sub-Saharan Africa (47.4%), South Asia (35.7%) and Central sub-Saharan Africa (35.7%) and the lowest in Australasia (5.7%), Western Europe (6.0%) and North America (6.8%) [3]. These GBD figures are not directly comparable with the WHO women-specific estimates above because the population stratum and method differ. The roughly eight-fold gap between highest and lowest GBD regions is therefore a statement about GBD regional estimates, not about women of reproductive age specifically.

4.3 From anaemia to iron deficiency and IDA: what the global data show

No standardised global dataset reports biomarker-confirmed IDA. Two approaches approximate it. First, GBD attributes anaemia to 37 underlying causes and estimates that dietary iron deficiency accounted for 66.2% of anaemia cases in 2021, or about 1.27 billion cases (825 million women and 444 million men) [3]. Second, national surveys that measure both Hb and ferritin or inflammation markers can estimate the share of anaemia associated with iron deficiency, and this share varies widely. In a recent study of young children in Puno, Peru, only 27.5% of anaemia was associated with iron deficiency, while 45.9% was associated with inflammation [13]. That study used a convenience sample of 310 children living at high altitude and defined inflammation by interleukin-6 rather than C-reactive protein and α1-acid glycoprotein, so it illustrates context specificity rather than providing a generalisable estimate. Direct comparisons show the difference matters: Young and colleagues found that GBD 2021 estimates of the share of anaemia associated with iron deficiency (for example 69% in women of reproductive age) were substantially higher than those from nutrition surveys that measured inflammation-adjusted ferritin (median 45% in non-pregnant women and 42% in preschool children), because GBD treats iron deficiency as a modelled residual cause rather than a measured exposure [14]. WHO and other reviews likewise emphasise that this proportion is context-specific and should be measured locally rather than assumed [15,16]. A meta-analysis of studies in pregnant women reported a pooled IDA prevalence of 18.98%, but with heterogeneous diagnostic criteria across the included studies [17]. Accordingly, statements that "iron deficiency causes two-thirds of anaemia" should be read as a GBD modelled attribution, not as a measured global IDA prevalence.

4.4 Long-term trends and projections, 1990–2050

Wang and colleagues applied an extreme gradient boosting (XGBoost) model to GBD 2021 data to project the burden of iron deficiency to 2050 [18]. The outcome in that study is the GBD category "iron deficiency", not IDA and not ferritin-defined iron deficiency. The reported global prevalent cases (984.61 million in 1990 and 1,270.64 million in 2021) match the number of anaemia cases that GBD attributes to dietary iron deficiency (66.2% of 1.92 billion is approximately 1.27 billion) [3,18,19], and the age-standardised prevalence rate in 2021 (about 16,430 per 100,000) is the same as that reported by the GBD 2021 Dietary Iron Deficiency Collaborators [19]. This is consistent with GBD's modelling of iron deficiency as a residual cause of anaemia [14]. The 1.27 billion figure should therefore be read as a modelled estimate of anaemia attributable to iron deficiency, not as a count of people with low ferritin and not as WHO-defined IDA.

Model-based projections for 2050 are 1,439.99 million cases [18]. The age-standardised prevalence rate was 18,204 per 100,000 in 1990 and 16,433 per 100,000 in 2021 (estimated annual percentage change, EAPC, −0.36%) and is projected to reach 15,922 per 100,000 by 2050 (EAPC −0.09% between 2021 and 2050) [18]. Absolute numbers rise because the population grows and

ages while the rate barely changes. These are projections from historical patterns and demographic inputs; they do not model future policy, and the authors note that the approach can be biased when trends reverse or are non-linear [18].

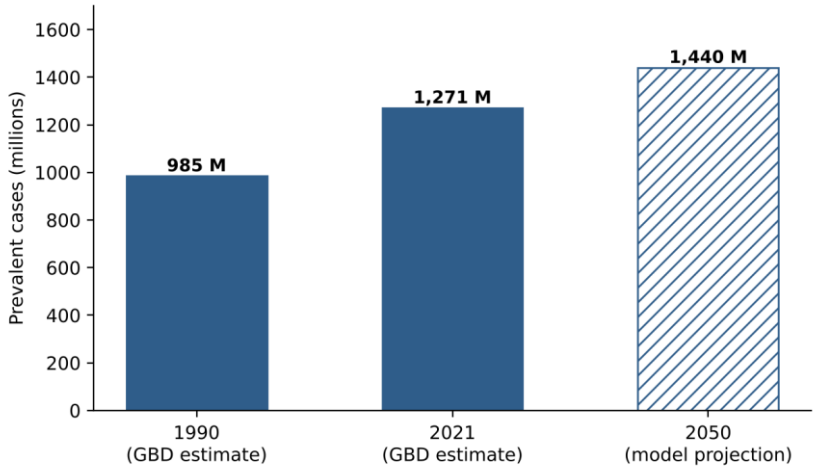


Figure 3. Global prevalent cases of iron deficiency (GBD category "dietary iron deficiency"): 1990 and 2021 estimates and the 2050 model-based projection (hatched). The outcome is not iron deficiency anaemia as confirmed by biomarkers. Source: Wang et al., Nutrients 2024 (GBD 2021, XGBoost projection) [18].

4.5 Progress towards global targets

In 2012 the World Health Assembly (Resolution 65.6) set six global nutrition targets for 2025, including a 50% reduction in anaemia among women of reproductive age from a 2012 baseline. The targets were extended to 2030 in alignment with the Sustainable Development Goals; WHO is custodian of SDG indicator 2.2.3, the prevalence of anaemia in women aged 15–49 years by pregnancy status [1]. Meeting the 2030 target requires an average annual rate of reduction of 3.78% from the 2012 baseline. WHO's 2025 assessment found global prevalence in women of reproductive age was 27.6% in 2012 and 30.7% in 2023 [1]. Of 195 countries and territories, one is on track, 18 show some progress, 50 show no progress, 125 are worsening and one could not be assessed [1]. Earlier analyses of 2000–2019 data had already concluded that progress was insufficient to halve anaemia by 2030 [12]. The Lancet Haematology Commission has further argued that the 50% target does not account for country context and focuses on prevalence rather than disease burden [20].

4.6 Disparities within high-income countries

National prevalence is lower in high-income countries (17.4% among women in 2023 on WHO's income grouping) [1], but disparities persist. In the United States, the National Health and Nutrition Examination Survey (NHANES) for August 2021–August 2023 found anaemia in 13.0% of females and 5.5% of males aged 2 years and older. Among females of this age, prevalence was 31.4% in Black, 15.9% in Asian, 15.0% in Hispanic and 8.3% in White non-Hispanic females [21]. These are cross-sectional descriptive estimates of Hb-defined anaemia and do not indicate its cause. They are consistent with differences in menstrual blood loss, diet, income, access to care and chronic disease burden, but surveys cannot show which mechanisms operate or how much of the gap is causal. Differences in the prevalence of inherited haemoglobin variants (such as α-thalassaemia trait) may also contribute and should be tested rather than assumed.

5. AETIOLOGY AND DETERMINANTS

Anaemia is multifactorial, and in individual patients and populations several causes frequently coexist [15,16]. GBD 2021 modelled 37 underlying causes, and its estimates show substantial regional variation in which causes dominate [3]. Table 4 summarises the main groups; the sections below discuss them.

Table 4. Principal causes of anaemia, mechanisms and diagnostic clues

Cause	Mechanism	Typical laboratory pattern	Where it matters most
Dietary iron deficiency	Intake or absorption below requirement	Low ferritin, low transferrin saturation, microcytosis	Young children, adolescent girls, women of reproductive age, pregnancy
Chronic blood loss: heavy menstrual bleeding, gastrointestinal bleeding, hookworm	Iron loss exceeds intake	As above; may need endoscopy or stool testing in adults	Women with heavy periods; older adults; endemic helminth areas

Cause	Mechanism	Typical laboratory pattern	Where it matters most
Anaemia of inflammation	Hepcidin-mediated iron sequestration; blunted erythropoietin response	Normal or high ferritin, low transferrin saturation, raised CRP	Chronic infection, autoimmune disease, malignancy, obesity
Chronic kidney disease	Reduced erythropoietin; inflammation; iron restriction	Normocytic anaemia; ferritin and transferrin saturation interpreted with eGFR	Adults with CKD, diabetes, hypertension
Vitamin B12 and folate deficiency	Impaired DNA synthesis	Macrocytosis; low B12, folate or raised methylmalonic acid	Vegetarian diets, older adults, malabsorption, poor diet
Haemoglobinopathies	Reduced or abnormal globin synthesis; haemolysis	Microcytosis with normal or high ferritin (thalassaemia); sickle cells	South Asia, Mediterranean, Middle East, sub-Saharan Africa
Malaria, HIV and other infections	Haemolysis, marrow suppression, inflammation, blood loss	Variable; low reticulocyte response; raised CRP	Endemic regions

CRP, C-reactive protein; eGFR, estimated glomerular filtration rate. Compiled from [3,6,8,9,15,22–24].

5.1 Nutritional determinants

Iron requirements and supply. Requirements are highest in infancy and early childhood, adolescence, pregnancy and among menstruating women. Dietary iron has two forms. Haem iron, from meat, poultry and fish, is absorbed at a fractional rate of roughly 15–35% and is little affected by other dietary components. Non-haem iron, from cereals, legumes, vegetables and fortificants, is absorbed at roughly 2–20% depending on iron status and meal composition [25]. Where diets are dominated by unrefined cereals and legumes and animal-source foods are costly, non-haem iron predominates and total absorbed iron may fall short of requirements.

Enhancers and inhibitors. Ascorbic acid reduces ferric to ferrous iron and forms an absorbable chelate, which can counteract phytate in a meal. Muscle tissue appears to enhance non-haem iron absorption (the “meat factor”). Phytate in whole grains and legumes, polyphenols in tea, coffee and some vegetables, and calcium inhibit non-haem iron absorption, and phytate and polyphenols have dose-dependent effects at the same meal [25]. Food-based advice (separating tea from meals, adding vitamin C sources, soaking or fermenting cereals to reduce phytate) is biologically plausible, but trial evidence that such advice alone reduces anaemia at population scale is limited.

Hepcidin and regulation. Because hepcidin rises with inflammation and with recently absorbed iron, absorption can be low even when diet or supplement intake is adequate, as in children with recurrent infection [5,6]. This partly explains why anaemia programmes based only on iron intake often yield smaller Hb gains than expected in high-infection settings.

5.2 Life-stage and physiological determinants

Women of reproductive age face menstrual losses and the demands of pregnancy and lactation, which accounts for much of the sex gap in prevalence (33.7% versus 11.3% in GBD women and men aged 15–49 years) [3]. In pregnancy, plasma volume expansion, fetal growth and placental needs increase iron requirements several-fold in the second and third trimesters. In infants, iron stores laid down in utero are depleted by 4–6 months and complementary foods are often low in bioavailable iron; adolescents have rapid growth plus, in girls, menstruation. These observations explain why prevalence is highest in children aged 6–59 months and pregnant women.

5.3 Blood loss

Heavy menstrual bleeding is a common and under-recognised cause of iron deficiency in menstruating women; GBD analyses identify gynaecological disorders and maternal haemorrhage as important contributors to anaemia among women of reproductive age [3]. In men and post-menopausal women, gastrointestinal blood loss from peptic ulcer, colorectal cancer, angiodysplasia, inflammatory bowel disease, and use of non-steroidal anti-inflammatory drugs or anticoagulants should be sought before iron deficiency is attributed to diet. Coeliac disease and Helicobacter pylori infection impair iron absorption. Hookworm and other soil-transmitted helminths cause chronic intestinal blood loss, most importantly in school-age children and in women in rural, warm, poorly sewered settings [9,15].

5.4 Infection, inflammation and chronic disease

Malaria causes haemolysis, dyserythropoiesis and inflammation and is a leading non-nutritional contributor to anaemia in sub-Saharan Africa; in GBD 2021 it ranked among the largest contributors in Central, Eastern and Western sub-Saharan Africa [3]. HIV infection is the second-largest contributor to anaemia YLDs in Southern sub-Saharan Africa [3]. Tuberculosis, other chronic infections, autoimmune disease and malignancy cause anaemia of inflammation. Chronic kidney disease reduces erythropoietin production and, with inflammation, restricts iron availability; GBD identifies it as an important cause in adults [3]. Obesity is associated with low-grade inflammation, higher hepcidin and lower iron absorption. These conditions often coexist with iron deficiency, so a patient or population may have more than one mechanism.

5.5 Vitamin B12, folate and other micronutrients

Vitamin B12 and folate deficiencies cause megaloblastic anaemia, often with macrocytosis, but can coexist with iron deficiency so that mean corpuscular volume is normal. B12 deficiency results from low animal-source intake (including vegetarian diets), pernicious anaemia, atrophic gastritis, bariatric surgery and metformin or proton pump inhibitor use [24]. Folate deficiency follows poor diet, alcohol misuse, malabsorption and increased demand in pregnancy and haemolysis. Vitamin A, riboflavin and copper deficiencies also impair iron mobilisation or erythropoiesis. This matters programmatically: folic acid given with iron will correct folate-responsive anaemia but may mask B12 deficiency.

5.6 Inherited red-cell disorders

Thalassaemias, sickle cell disease and trait, G6PD deficiency and other haemoglobinopathies contribute importantly to anaemia in South and South-East Asia, the Middle East, the Mediterranean and sub-Saharan Africa [3,23]. Thalassaemia trait produces microcytosis with normal or high ferritin and is easily misdiagnosed as IDA, with the risk that iron is given unnecessarily; iron overload is a particular concern in transfusion-dependent thalassaemia. WHO guidance calls for screening and management of inherited red-cell disorders where they are prevalent [1].

5.7 Socioeconomic and structural determinants, and the limits of inference

Cross-country comparisons show that anaemia prevalence is higher where the Human Development Index is lower, and multi-country Demographic and Health Survey analyses in sub-Saharan Africa associate anaemia in women with lower education, lower household wealth, rural residence and unimproved water and sanitation [26,27]. These are observational associations from cross-sectional surveys. Wealth, education and sanitation are correlated with each other and with diet quality, infection exposure, health service access and fertility, so the independent contribution of each cannot be separated, and reverse causation or residual confounding is possible. Such analyses support targeting and hypothesis generation, not causal claims that improving a single factor (for example, water supply) will reduce anaemia by a particular amount. Evidence on the effect of non-nutritional interventions, such as water, sanitation and hygiene packages, on haemoglobin is mixed, which is a further reason not to equate associations with intervention effects.

6. HEALTH, SOCIAL AND ECONOMIC CONSEQUENCES

Most evidence linking anaemia to outcomes comes from observational studies. Haemoglobin and iron status are correlated with poverty, infection, diet quality and access to care, so associations may be partly or wholly due to confounding, and anaemia may also be a marker of the underlying illness. Where randomised evidence exists, it is cited separately.

6.1 Maternal and neonatal outcomes

Meta-analyses of cohort studies find that low maternal Hb is associated with higher risks of preterm birth, low birthweight, small-for-gestational-age birth, stillbirth and maternal mortality, with a U-shaped relationship in some analyses in which very high Hb is also associated with adverse outcomes [28,29]. Confounding by socioeconomic status, malaria, haemoconcentration in pre-eclampsia and the underlying cause of anaemia is likely, and these data are not proof that raising Hb by supplementation will lower these risks by the same amount. Randomised trials of daily iron in pregnancy show clear reductions in maternal anaemia and iron deficiency at term and a modest reduction in low birthweight, but little evidence of benefit for perinatal or maternal mortality, and trials are generally too small to show effects on rare outcomes [30].

6.2 Child development

Iron deficiency in infancy and early childhood is associated with poorer cognitive, motor and socio-emotional development, and some deficits may persist despite later iron repletion [9]. The strongest evidence concerns iron deficiency itself rather than anaemia of any cause; the developmental consequences of anaemia due to other causes are less clear. Whether trial-based iron supplementation improves long-term neurodevelopment depends on baseline status, age at intervention and follow-up duration, and trial findings are mixed.

6.3 Disability burden and productivity

Anaemia is the third-leading cause of YLDs globally [3]. Between 1990 and 2021 the global distribution shifted towards milder anaemia and prevalence fell most in adult men; progress was slowest in women of reproductive age and children under five [3]. Iron deficiency is associated with reduced aerobic work capacity, but economic cost estimates rest on modelling assumptions and vary widely, and we did not identify a global estimate that is based on measured IDA.

6.4 Intergenerational effects

Maternal iron deficiency and anaemia are associated with lower infant iron stores at birth, and early iron deficiency in girls tracks into adult reproductive life. Together with poverty and gender inequity, this could contribute to an intergenerational cycle, but this is a plausible model supported mainly by observational data rather than a demonstrated causal chain, and the relative importance of biological and social transmission is unknown.

7. THE PUBLIC HEALTH RESPONSE

7.1 Global targets and WHO recommendations

The global anaemia target (50% reduction in women of reproductive age from 2012 to 2030) and SDG indicator 2.2.3 anchor accountability [1]. WHO's 2023 framework, "Accelerating anaemia reduction", emphasises a multi-causal and multisectoral approach combining nutrition, infection control, reproductive health, health systems and social determinants [16]. Table 5 summarises the core recommendations and their key limits.

Table 5. Selected WHO-recommended interventions, evidence and principal limitations

Population	Recommended intervention	Evidence of effect	Key limits
Pregnant women	Daily oral iron (30–60 mg elemental iron) with 400 µg folic acid in antenatal care [31]	Reduces maternal anaemia and iron deficiency at term and modestly reduces low birthweight [30]	Gastrointestinal side effects; late first contact; adherence; stock-outs
Menstruating women (non-pregnant): weekly regimen where anaemia prevalence is ≥ 20%; daily regimen where ≥ 40%	Weekly: 60 mg elemental iron with 2.8 mg folic acid once a week for 3 months, then 3 months off [32]. Daily: 30–60 mg elemental iron for 3 consecutive months in a year [33]	Intermittent vs no iron: higher Hb (about 5 g/L) and less anaemia (RR 0.65). Intermittent vs daily: similar Hb, lower ferritin, fewer side effects. Certainty low to moderate; no data on IDA [34]. Daily iron reduces anaemia and iron deficiency [35]	Low awareness; weak delivery platform outside school and workplace
Children 6–23 months and older children in high-prevalence settings	Daily iron drops or syrup where anaemia prevalence is ≥ 40% [36]; or multiple micronutrient powders [37]	Improves Hb and reduces anaemia and iron deficiency [36,37]	Safety concerns in malaria-endemic areas without malaria control [38]; acceptability and coverage
Staple-consuming populations	Large-scale fortification of wheat flour, maize flour, rice or salt	Population-level improvement in iron status and anaemia in some programmes [39,40]	Compliance and monitoring; iron compound; variable effect size; does not reach non-consumers of fortified foods
Malaria-endemic populations	Integrated malaria prevention and case management with nutrition [38]	Iron does not increase clinical malaria where malaria services are in place [38]	Weak health systems; bed-net and chemoprevention coverage

Evidence statements are summarised from the cited systematic reviews and WHO guidelines and should be read with their original certainty ratings. Hb, haemoglobin; IDA, iron deficiency anaemia; RR, risk ratio. Sources: WHO guidelines [31–33,36,37]; Cochrane reviews [30,34,35,38–40].

7.2 Iron supplementation: effectiveness, adherence and coverage

Iron supplementation is the most thoroughly tested intervention. Cochrane reviews of randomised trials show that daily iron raises Hb and reduces anaemia and iron deficiency in pregnant women and menstruating women [30,35]. Efficacy in trials, however, differs from effectiveness in programmes, for three reasons. Coverage: supplies reach only part of the target group, often late in pregnancy. Adherence: side effects (nausea, constipation, metallic taste), forgetfulness, low perceived need and poor counselling reduce intake, and the number of tablets actually taken is frequently below what is distributed. Delivery: supply-chain breaks, irregular school or community delivery and poor Hb testing weaken programmes. Hepcidin physiology suggests that lower or alternate-day dosing may improve fractional absorption and tolerability, although programme-scale evidence on clinical outcomes is still developing [7]. For menstruating women, WHO recommends a weekly regimen (60 mg elemental iron with 2.8 mg folic acid, taken for three months and then stopped for three months) where anaemia prevalence is 20% or higher [32], and daily iron (30–60 mg for three consecutive months in a year) where it is 40% or higher [33]. A Cochrane review found that intermittent regimens raised Hb and reduced anaemia compared with no supplementation, gave similar Hb but lower ferritin than daily dosing, and caused fewer side effects, with low-to-moderate certainty and no data on iron deficiency anaemia [34]. In malaria-endemic areas, iron does not increase clinical malaria where malaria prevention and treatment are in place [38], but safe delivery requires that these services exist. Iron is also contraindicated or must be individualised in thalassaemia major and iron overload, which should be screened for where haemoglobinopathies are prevalent.

7.3 Food fortification: promise and constraints

Fortification of staple foods is attractive because it does not require daily individual behaviour change and can reach large populations at low per-person cost. Systematic reviews report improvements in iron status and anaemia in several national flour-fortification programmes, but effects are heterogeneous and depend on the iron compound used, fortification level, coverage of the fortified vehicle, concomitant intake of inhibitors and the quality of regulation and monitoring [39]. A Cochrane review of fortification with multiple micronutrients found reductions in anaemia and iron deficiency, with limits in certainty and applicability [40]. Practical challenges include low-bioavailability iron compounds that are used to avoid colour or taste changes, small-scale or informal milling that is not captured by mandatory fortification, rural populations who purchase unfortified foods, lack of routine compliance monitoring, and uncertain benefit where inflammation or haemoglobinopathies drive anaemia. Cost-effectiveness claims are valid only when fortified foods reach the target population at effective doses; they should not be generalised to all vehicles and settings.

7.4 Complementary strategies

Other measures target specific causes: deworming and treatment of schistosomiasis where endemic; malaria prevention and chemoprevention; HIV and tuberculosis care; delayed umbilical cord clamping, which improves infant iron stores [16]; management of heavy menstrual bleeding and postpartum haemorrhage; family planning and birth spacing; screening for haemoglobinopathies; and, where oral iron fails or is not tolerated, intravenous iron in pregnancy and in selected patients. Dietary diversification and behaviour-change communication are widely used but have limited trial evidence on anaemia reduction when delivered alone.

7.5 India's Anemia Mukt Bharat programme

India has among the largest absolute anaemia burdens, with Southern Asia the highest-prevalence sub-region globally [1]. The National Family Health Survey 2019–21 (NFHS-5) reported anaemia in 67.1% of children aged 6–59 months (up from 58.6% in NFHS-4), 57% of women aged 15–49 years, 59% of adolescent girls and 52.2% of pregnant women [41–43]. Anemia Mukt Bharat (AMB), launched in 2018 under the POSHAN Abhiyaan, built on the Intensified National Iron Plus Initiative and set a target of reducing anaemia by three percentage points per year [44]. In June 2026 the Ministry of Health and Family Welfare released revised operational guidelines and renamed the programme Anemia Mukt Bharat Abhiyaan [45–47]; the original 2018 design is described first, followed by the 2026 revisions.

The original 2018 strategy was "6×6×6": six target groups (children aged 6–59 months, children aged 5–9 years, adolescents aged 10–19 years, pregnant women, lactating women, and women of reproductive age); six interventions; and six institutional mechanisms. The interventions were (1) prophylactic iron and folic acid supplementation, with scheduled weekly or daily dosing by group; (2) periodic deworming; (3) intensified year-round behaviour-change communication, including promotion of delayed cord clamping and iron-rich diets; (4) testing and treatment of anaemia using digital point-of-care haemoglobin methods with treatment protocols; (5) mandatory provision of iron-fortified foods in government-funded schemes; and (6) intensified action on non-nutritional causes, including malaria, haemoglobinopathies and fluorosis. Institutional mechanisms included inter-ministerial coordination, a national centre of excellence and a monitoring dashboard [44]. Under this schedule the weekly tablet for women of reproductive age contained 60 mg elemental iron with 500 µg folic acid [42], which differs from the 2.8 mg folic acid dose in WHO's weekly schedule [32].

The 2026 guidelines expand this framework to "7×7×7" by adding a seventh beneficiary group (low-birth-weight infants aged 0–6 months), a seventh intervention ("Eating Right", promoting regular consumption of iron-rich and diverse diets) and a seventh institutional mechanism (a strengthened monitoring and evaluation framework with digital tracking) [45,47]. The earlier "Test, Treat, Talk" (T3) approach becomes "Test, Treat, Talk, Track" (T4), with intensified haemoglobin testing, treatment of iron deficiency anaemia according to national management protocols, and systematic tracking of beneficiaries for referral and follow-up [45]. For severe anaemia and for non-responders among pregnant and lactating women, intravenous ferric carboxymaltose and iron sucrose are included as clinical interventions [45,47]. Haemoglobin testing records for pregnant women are to be mapped through the JANANI portal and those for children through the RBSK and U-WIN portals, converging into a unified AMB Abhiyaan portal, and a nationwide community mobilisation campaign (Jan Bhagidari) aims to present anaemia as a preventable and treatable condition [45,47]. Taken together, the revisions move the programme from supplementation towards testing, therapeutic management and tracking. Some of the gaps discussed below (limited diagnosis and follow-up, data systems that record coverage rather than quality) are directly targeted, but we did not identify any published evaluation of the revised programme.

Evidence on performance is mixed. Programme data show that iron and folic acid supplementation coverage rose from 35.5% in 2018–19 to 57.6% in 2022–23 on the AMB Index, which averages coverage across five target groups using health management information system (HMIS) data, with substantial inter-state variation; supply chain and reporting quality were important determinants [42]. Coverage reported in HMIS reflects distribution, not consumption, and compliance is below 80% in many settings, including in India among pregnant women, where counselling gaps, forgetfulness, side effects and inadequate forecasting and inventory management have been described [42]. Programme studies identify trained health workers, buffer stocks and accessibility as facilitators, and irregular administration, forgetfulness, side effects, low awareness, weak screening and disrupted school-based delivery as barriers [42]. At the population level, NFHS-5 showed higher anaemia prevalence than NFHS-4 in children and women [41,43], so the programme did not meet its three-percentage-point annual target in that period, although the survey interval coincided with the COVID-19 pandemic. The NFHS-6 (2023–24) fact sheets, released on 29 May 2026, did not include anaemia indicators; the Ministry had indicated that anaemia would instead be measured through the Indian Council of Medical Research's Diet and Biomarkers Survey in India (DABS-I) [43]. We did not identify a published national anaemia estimate from that survey by 8 November 2026, so no post-NFHS-5 national estimate is available to assess AMB or its 2026 revisions. These data limit any causal attribution of changes to AMB, and independent evaluation using prospective haemoglobin and ferritin measurement would be valuable.

Key implementation challenges, therefore, are (i) the gap between distributed and consumed tablets; (ii) data systems that measure coverage but not quality; (iii) limited diagnosis and follow-up of moderate and severe anaemia (which the T4 approach and intravenous iron guidance aim to address but which remain unevaluated); (iv) the large share of anaemia not due to dietary iron deficiency (haemoglobinopathies, inflammation, helminths, fluorosis), which the supplementation-centred model addresses only partly; (v) uneven fortified-food reach; and (vi) uncertainty about the validity of capillary Hb and altitude and smoking adjustment in routine surveys, which affects tracking.

7.6 Why progress has stalled

Four themes recur in global assessments. Interventions have been poorly matched to cause, with iron-centred

packages applied where inflammation, infection or inherited disorders dominate. Delivery coverage and adherence have been weak, particularly for adolescents and women outside antenatal care. Surveillance and evaluation have been limited, with few countries monitoring iron biomarkers or inflammation. Finally, health sector programmes have rarely been coordinated with social protection, water and sanitation, education or gender equity efforts. WHO's assessment that only one country is on track for the 2030 target suggests that incremental extension of existing approaches will not suffice [1,16].

8. MEASUREMENT AND DATA CHALLENGES

Sample type and device. Most surveys use capillary blood with portable photometers, which show greater variability than venous measurement. WHO's 2025 estimates now model sample type and device, include venous individual-level data, and exclude surveys that lack method detail, which may change comparability with earlier editions; WHO cautions that differences from earlier editions should not be interpreted as time trends [1].

Thresholds. The change to 2024 Hb thresholds and altitude adjustment alters prevalence in some countries [10,11]. Trend analysis requires consistent application of one set of criteria. A GBD-based modelling study that applied the 2024 elevation adjustment to the same survey inputs estimated all-age, both-sex global anaemia prevalence in 2023 at 26.4%, 2.4 percentage points higher than with the 2001 method, which would move anaemia from the third to the second largest cause of disability [48]; the GBD 2021 figures used in this review predate that update.

Data sparsity and uncertainty. WHO's 2025 estimates draw on 197 countries and territories and rely on Bayesian hierarchical models that borrow strength across countries and years, so estimates for data-poor countries are pulled towards regional means and carry wide uncertainty intervals [1]. Child estimates have not been updated since 2019. Global prevalence of 30.7% in 2023, for example, has an uncertainty interval of 26.6–34.9%, so small year-to-year changes should be interpreted cautiously [1].

Absence of biomarker-linked data. Without ferritin and inflammation markers in representative surveys, it is not possible to say which share of anaemia is due to iron deficiency in a given country, so programme design relies on assumption. Standardised biomarker surveys such as BRINDA-style analyses should be expanded [4].

9. RESEARCH PRIORITIES AND RECOMMENDATIONS

9.1 Research priorities

• Population-representative surveys that measure Hb together with inflammation-adjusted ferritin (and, where feasible, haemoglobinopathy and B12/folate status) to estimate country-specific causes of anaemia and the true prevalence of IDA.

• Implementation trials comparing dosing regimens (daily versus alternate-day or weekly), iron formulations and delivery platforms, with effectiveness and adherence rather than Hb alone as outcomes.

• Evaluation of the effectiveness of fortification vehicles and iron compounds under real-world conditions, including compliance monitoring and reach to rural and poor populations.

• Quasi-experimental or stepped-wedge evaluation of national programmes such as AMB, with independent biomarker endpoints.

• Causal analysis of social determinants using longitudinal and trial designs, including cash transfer and sanitation interventions, rather than cross-sectional associations.

• Safety and efficacy of intravenous iron in pregnancy and in low- and middle-income settings, and effects of iron on the infant microbiome and infection in high-burden areas.

9.2 Policy and programme recommendations

• Measure what is being targeted. Use Hb for anaemia and add ferritin with CRP and AGP for iron deficiency; report the outcome definition with every estimate.

• Match interventions to cause. Combine iron programmes with malaria, helminth, HIV, tuberculosis, haemoglobinopathy and menstrual-health services where these drive anaemia.

• Move from distribution to effective coverage. Track consumed doses, side-effect management, stock availability and treatment of moderate and severe anaemia, not only tablets distributed.

• Strengthen fortification governance. Mandate effective iron compounds and levels, regular compliance testing and inclusion of small mills and social-protection channels.

• Target within-country inequities. Disaggregate national data by wealth, rurality, ethnicity and age, and fund services for underserved groups.

• Sustain financing and accountability for the 2030 target through national nutrition plans and regular independent reporting on SDG indicator 2.2.3.

10. CONCLUSION

Global anaemia remains stubbornly high, and women of reproductive age and young children carry the greatest burden. Iron deficiency is the largest single cause, but the available global data measure anaemia, not IDA, and many cases arise from inflammation, infection, blood loss, nutrient deficiencies or inherited disorders. Existing supplementation and fortification approaches work when well delivered, but weak coverage, adherence and monitoring have limited their population impact. Accelerating progress will require clearer definitions, biomarker-based diagnosis, cause-specific programming and rigorous evaluation of delivery.

11. LIMITATIONS OF THIS REVIEW

As a narrative review, this work is subject to selection bias and does not provide pooled estimates. Several figures are modelled with wide uncertainty intervals, and WHO and GBD estimates use different methods and cannot be directly compared. Projections to 2050 depend on assumptions that exclude future policy changes. Some

programme evidence, including that for India, derives from administrative or small-scale studies of variable quality.

12. DECLARATIONS

12.1. Ethical approval and consent to participate

Not applicable.

12.2. Conflict of interest

No potential conflict of interest was reported by the authors.

12.3. Financial support

This research was self-funded.

12.4. Author's contributions

The sole author was responsible for the conceptualization, literature search, data collection, interpretation, writing the original draft, and critical revision of the manuscript. The author has read and approved the final version.

12.5. Artificial intelligence statement

The authors used an artificial intelligence (AI)-assisted language tools to improve the readability and language of the manuscript, as well as to format the tables and generate the illustrative figure. All scientific content, study design, data collection, data analysis, interpretation of the findings, and final editorial decisions were performed entirely by the authors. The authors have reviewed and approved the final manuscript and accept full responsibility for their accuracy and integrity

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