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REVIEW ARTICLE

A Critical Review of Iron Deficiency Anaemia

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Abstract

Iron deficiency anaemia (IDA) is the most common nutritional deficiency and the leading cause of anaemia worldwide, affecting an estimated 1.2 billion people, while iron deficiency contributes to nearly 50% of all anaemia cases globally. The burden is disproportionately higher in low- and middle-income countries, particularly among children under five years, adolescent girls, women of reproductive age, and pregnant women, where prevalence frequently exceeds 30–40%. This critical review synthesizes empirical evidence from published studies to evaluate the epidemiology, aetiology, pathophysiology, clinical manifestations, diagnosis, treatment, and prevention of IDA. Relevant peer-reviewed articles, clinical practice guidelines, and systematic reviews were critically appraised using evidence-based review methods. The findings identify inadequate dietary iron intake, chronic blood loss, parasitic infections, pregnancy, rapid growth, and malabsorption disorders as the predominant determinants of IDA across different populations. Evidence indicates that serum ferritin remains the most sensitive indicator of depleted iron stores, although inflammatory conditions may reduce its diagnostic specificity, necessitating complementary biomarkers such as transferrin saturation and soluble transferrin receptor. Oral iron supplementation remains the cornerstone of treatment, whereas intravenous iron therapy is recommended for patients with severe deficiency, intolerance to oral preparations, or impaired absorption. Preventive strategies, including food fortification, iron supplementation programmes, dietary diversification, deworming, malaria control, and routine screening of high-risk groups, have demonstrated significant reductions in disease burden. strengthening evidence-based clinical management alongside population-level public health interventions is essential for reducing the global prevalence of iron deficiency anaemia and improving health outcomes.

Keywords: Iron Deficiency Anaemia, Iron Metabolism, Epidemiology, Iron Supplementation, Public Health.

1. Introduction

World Health Organization (WHO) has recognised iron deficiency anaemia (IDA) as the most common nutritional deficiency in the world, with 30% of the population being affected with this condition (Kumar *et.al*., 2022). WHO defines anaemia in children aged under 5 years and pregnant women as a haemoglobin concentration <110 g/L at sea level, and anaemia in

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non-pregnant women as a haemoglobin concentration <120 g/L (WHO, 2025), remains a significant global public health issue, particularly affecting young adults in low- and middle-income countries (Khatun *et.al*., 2025). Anaemia is estimated to affect half a billion women 15–49 years of age and 269 million children 6–59 months of age worldwide (WHO, 2025). In 2019, 30% (539 million) of non-pregnant women and 37% (32 million) of pregnant women aged 15–49 years were affected by anaemia (WHO, 2025).The condition is often multifactorial, with nutritional deficiencies, chronic infections, and menstrual blood loss being common causes among adolescents and young adults (Gosdin *et.al*., 2021). Iron deficiency anemia (IDA) is the most common form, accounting for nearly 50% of anemia cases worldwide (Bhanderi *et.al*., 2025). In the context of undergraduate students, academic stress, irregular eating patterns, and sedentary lifestyles may further contribute to the risk of developing anemia (Bhanderi *et.al*., 2025).

IDA is characterized by a lower concentration of hemoglobin in blood because there are either fewer red blood cells (RBCs) than normal or little hemoglobin in each cell. ID arises when the body's iron stores, particularly those in macrophages and hepatocytes, are depleted (Kolarš *et.al*., 2025). Because most iron approximately 25 mg daily is used for hemoglobin (Hb) synthesis to support the production of around 200 billion red blood cells each day, IDA is the most evident consequence of iron deficiency (Bathla & Arora, 2022). IDA is often a chronic and progressive condition that remains largely undiagnosed and lacks adequate treatment options (Stahl-Gugger *et.al*., 2022). Those afflicted with IDA experience severe repercussions, including impaired growth and cognitive development, diminished mental and physical capabilities, reduced work capacity, and an overall decline in quality of life (Channar *et.al*., 2023). Inadequate intake of dietary iron and iron-enhancing foods such as meat, fish, liver, tempeh, dark green vegetables, nuts and fruits, among other factors, contributes to anemia in most developing countries (Wiafe *et.al*., 2021).

A healthy diet refers to diet that meets the need for all nutritious elements in adequate amounts taking the age, gender and physical condition of an individual into account (Cena & Calder, 2020; Ndidi *et.al*., 2024). It is important to have a well- balanced diet in order to lead a healthy and quality life (Ndidi *et.al*., 2024). Young adults starting independent life are particularly vulnerable to developing unhealthy eating behaviors that can lead to eating disorders (Avram *et.al*., 2024) and nutritional deficiency (Kiani *et.al*., 2022). Diet quality can be measured by comparing an individual's dietary intake with current recommendations using the Diet Quality Indicators (DQI) (Hernández-Ruiz *et.al*., 2021). Although diet quality usually improves with age, a deterioration in diet quality is often observed during adolescence into adulthood.

University education can be considered a transitional period between adolescence and adulthood, during which young adults acquire new health-related behaviors which have influences later in adult life. Many factors come into play as they transition through undergraduate life. These undergraduate students are exposed to numerous academic chores such as continuous assessment, written and oral examinations, tight deadlines and interpersonal conflicts just to mention a few examples of the many events that may induce high levels of stress in students (Najm *et.al*., 2024). While stress around the time of learning is thought to enhance memory formation, stress markedly impairs memory retrieval, bearing for instance the risk of underachieving at the end of each academic session and the final grade at graduation. Due to a lack of time or resources, many students tend to skip meals which can cause energy deficits and nutritional shortages with detrimental effects on academic performance (Almoraie *et.al*., 2024).

Previous research has reported varying anemia prevalence rates among medical students, ranging from 20% to 60%, with a notably higher incidence in female students due to menstruation-related iron loss (Najm *et.al*., 2024). Despite their knowledge of disease pathology and prevention, medical students may neglect their own health, making them a vulnerable yet overlooked group (Bhanderi *et.al*., 2025). Identifying the relationship between iron deficiency anaemia and dietary habitants in undergraduate is essential to design targeted interventions and promote health-conscious behavior among future graduates.

2. Anaemia

Anaemia is a condition when a person's red blood cell count and haemoglobin (Hb) levels are below normal, meaning they are insufficient to support their body's physiological needs (Suprapti *et.al.,* 2025). Anaemia can be caused by poor nutrition, infections, chronic diseases, heavy menstruation, pregnancy issues and family history In many low and lower middle income settings (WHO, 2025). The most commonly recognized causes of anaemia are iron deficiency and malaria (WHO, 2025). Anemia may produce clinical symptoms, such as fatigue, palpitations, headache, and shortness of breath, and signs, such as conjunctival and

palmar pallor, which in spite of having low sensitivity and moderate specificity to diagnose anemia, are still useful when laboratory assessment is limited (Garcia-Casal *et.al.*, 2023). In clinical practice and in public health, the measurement of Hb concentration is the most common indicator and assessment method used to define anemia (Garcia-Casal *et.al.*, 2023). The prevalence of anaemia is particularly elevated in low- and middle-income nations, primarily affecting children under five, pregnant and breastfeeding mothers, and the elderly. Iron deficiency anaemia has a wide range of causes. This illustrates how distal variables influence its determinants, including food insecurity, access to clean water, and sanitation, ultimately leading to the most immediate causes of anaemia, such as nutritional deficiencies, diseases, inflammation, and haemoglobin abnormalities (Suprapti *et.al.*, 2025).

2.1 Components and Functions of Blood

Blood is a biological fluid found in the circulatory systems of humans and other vertebrates. It takes metabolic waste products out from the cells while also giving critical materials like nutrition and oxygen to the cells. Blood consists of two components, namely a liquid component called plasma and a solid component, namely blood cells (Yuyen, 2022).

Floating in blood plasma are the blood cells that make up blood. Plasma, which accounts for 55% of blood fluid, contains proteins, carbohydrates, mineral ions, hormones, CO2 (plasma is the major medium for excretory product transfer), and blood cells (Fdil, 2022). Plasma is 92% water by volume and contains other substances as well. The primary protein in plasma, albumin, regulates the blood's colloidal osmotic pressure. The majority of blood cells are made up of red blood cells (RBCs or erythrocytes), white blood cells (WBCs or leukocytes), and platelets (also known as thrombocytes) (Fdil, 2022). Red blood cells are the most prevalent cells in the blood of vertebrates. For the human body, erythrocytes play a crucial role. The red blood cell (erythrocytes) primary job is to carry carbon dioxide and oxygen from the lungs to the tissues (Luhulima *et.al.,* 2024).

2.2 Hemoglobin Structure and Function

The protein molecule found in red blood cells called haemoglobin is a complex protein that gives the red cells its colour and carries oxygen from the lungs to the body's tissues and returns carbon dioxide from the tissues to the lungs (Noreen *et.al.,* 2020). Heme is a prosthetic group consisting of iron atoms, while globin is a protein that is broken down into amino acids. Four protein molecules called globulin chains are joined to form haemoglobin and a typical adult haemoglobin is composed of two alpha-globulin chains and two beta-globulin chains (Farid *et.al.*, 2023) (Figure 1). Carboxy haemoglobin, which is dark red in colour, is created when haemoglobin and carbon dioxide combine (Folorunso, 2025). Each individual should have a blood count of roughly five million red blood cells per millimetre of blood and approximately 15 kilos of haemoglobin per 100 millilitres of blood.Red blood cells contain haemoglobin (Jain *et.al.*, 2022).

Variations in Hb concentration are influenced by genetic and environmental factors, as well as age, sex, pregnancy, and health status (Lopez de Romaña *et.al.*, 2023). Normal haemoglobin concentrations in newborns range from 170 to 210 g/L (Garcia-Casal *et.al.*, 2023). They decrease during the first few months of life to a value of about 100 g/L for infants aged 6 to 9 months (Fentaw *et.al.*, 2023), before rising once more in childhood to roughly 110 g/L up to 59 months, 115 g/L up to 11 years, and 120 g/L for adult females and 130 g/L for adult males (Garcia-Casal *et.al.*, 2023).

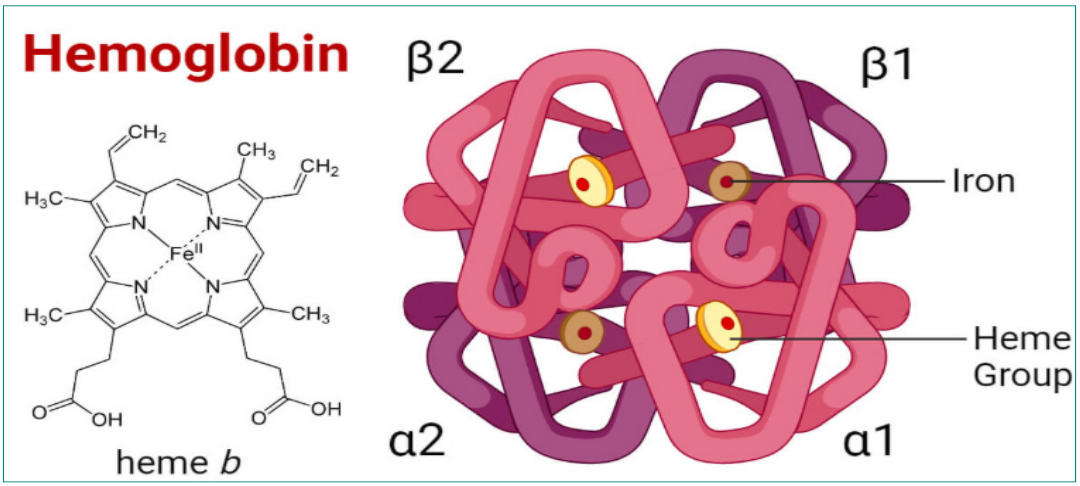

Figure 1. *Hemoglobin structure (Dahal, 2025)*

2.3 Classification of Anemia

Anemia can be classified based on morphology (MCV based classification) (Moorthi *et.al.*, 2023), etiology and severity (Muliya *et.al.*, 2023). The morphological classifications help in the differentiation of anaemia varieties, including microcytic, normocytic, and macrocytic anaemia, which are each associated with unique causes, such as iron deficiency, chronic disease, or vitamin B12 and folic acid deficiencies (Suprapti *et.al.*, 2025). Based on the morphology of RBCs and blood cell indices, anaemia is classified into normocytic normochromic, microcytic hypochromic, macrocytic and dimorphic anaemia (Abusharib, 2019). The level of hemoglobin and severity of anemia can be assessed by the hemoglobin levels of an individual and have been recommended by WHO according to age and severity (Table 2) (Aggarwal *et.al.*, 2020).

A number of physiological, environmental, clinical, and genetic variables can result in iron deficiency (ID) and consequent iron deficiency anaemia (IDA) (Table 1). Iron deficiency, with or without anaemia, can occur alone, as a result of an underlying disease, or in conjunction with a variety of clinical situations (e.g., in the elderly).

The etiology of anemia is characterised based on the amount and shape of RBC using the reticulocyte index (RI) and the mean corpuscular volume (MCV). The major anemia classes comprise hyperproliferative (RI > 2.5) and hypoproliferative (RI < 2.5) anemia. Hypoproliferative anemias divide into microcytic (MCV < 80 femtoliters (fL)), normocytic (MCV 80–100 fL), and macrocytic (MCV > 100 fL) anemia types (Figure 2 & 3) (Kumar *et.al.*, 2022). Anemia is not an illness, rather a presentation of an underlying issue (Fonseca *et.al.*, 2021). Dietary deficits play a causative role in the development of several kinds of anemia. Numerous minerals, including iron, cobalt, magnesium, and micronutrients, including vitamin A, folate, B6, B12, and other B vitamins are directly necessary for the creation of RBC, Hb synthesis, and iron absorption, as well as antioxidant defense and cellular energetics (Kumar *et.al.*, 2022). The nutritional etiology of anemia is multifaceted; yet, iron plays a vital role in oxygen transport by Hb in RBC.

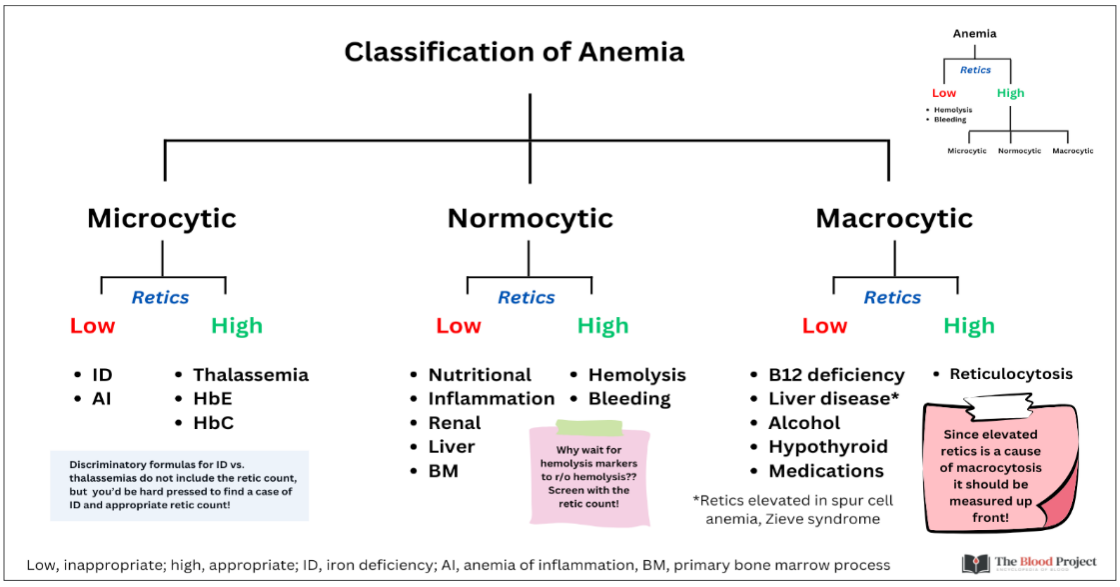

Figure 2. *Classification of anemia (William, 2023)*

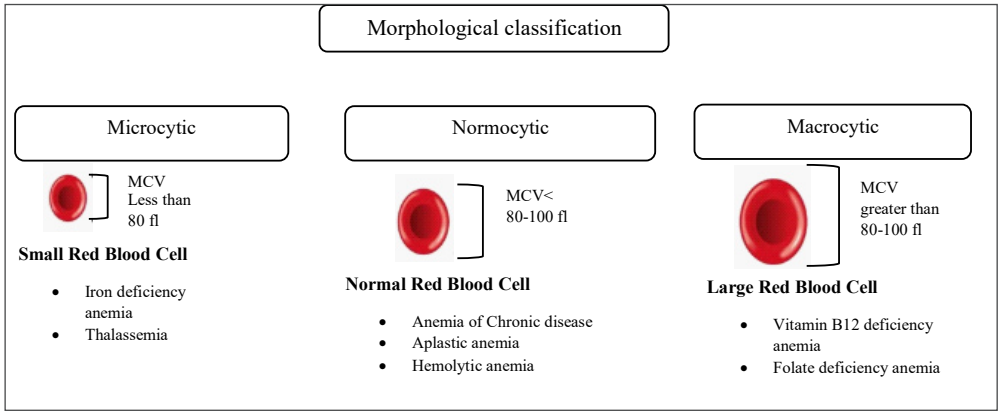

Figure 3. *Morphology classification of anemia (Marly Drug, 2026)*

Table 1. *Classification of anemia based on etiology (Muliya et.al., 2023)*

Type of Anemia	Predisposing factors/Etiology of Anemia
Iron deficiency Anemia	Poor dietary intake
	Worm infestation
	Pica
Megaloblastic Anemia	VitB12 deficiency
	Folic acid deficiency
Hemolytic Anemia	G6PD deficiency
	Malaria
	Thalassemia
	Sickle cell anemia
Anemia of Chronic Disease	HIV
	Liver abscess
	Extrapulmonary TB
	Giardia
	Ulcerative colitis
	Leukemia (ALL)
	Acute myeloid disease
Blood Loss Anemia	Acute blood loss (trauma)
	Chronic blood loss (bleeding from gastrointestinal tract or genitourinary tract)

Table 2. *WHO Classification of anemia according to age and severity (Aggarwal et.al., 2020)*

Population	Non-Anemia	Mild Anemia	Moderate Anemia	Severe Anemia
6-59 months of age	≥11	10-10.9	7-9.9	<7
5-11 years of age	≥11.5	11-11.4	8-10.9	<8
12-14 years of age	≥12	11-11.9	8-10.9	<8
Non-pregnant women (≥ 15 years)	≥12	11-11.9	8-10.9	<8
Pregnant women	≥11	10-10.9	7-9.9	<7
Men (≥15 years)	≥13	11-12.9	8-10.9	<8

2.4 Iron deficiency Anemia

Iron deficiency refers to the loss of iron reserves that precedes overt iron deficiency anemia or persists without development (Yang *et.al.*, 2023). Low iron levels are linked to anaemia and the existence of microcytic hypochromic red blood cells in iron-deficiency anaemia, a more serious illness. No matter how full the stores are, iron-restricted erythropoiesis shows that the transfer of iron to erythroid precursors is compromised (Nehar *et.al.*, 2024). In situations of anaemia of chronic inflammation, which is seen in individuals with autoimmune disorders, cancer, infections, and chronic kidney diseases, stores may be normal or even augmented due to iron sequestration. Elderly persons and those with chronic kidney illness may have both iron deficiency and anaemia of chronic diseases (Wiafe *et.al.*, 2021). Six Nonetheless, a significant portion of the anaemia common in older people happens without an iron shortage or high hepcidin levels.

Iron deficiency occurs when the body's demand for iron exceeds the absorption from dietary sources (Kolarš *et.al.*, 2025). Iron deficiency anaemia (IDA) occurs when the body's iron reserves diminish to insufficient levels for normal red blood cell (RBC) synthesis (Leung *et.al.*, 2024). Iron is essential for biological functions such as respiration, energy production, DNA synthesis, and cell proliferation (Malik *et.al.*, 2025). The human body has adapted to save iron through mechanisms such as recycling iron following the degradation of red blood cells and retaining iron due to the lack of an excretion system (Malik *et.al.*, 2025). Excessive iron levels can be hazardous; therefore, absorption is restricted to 1 to 2 mg daily, while the majority of the daily iron requirement (about 25 mg) is met through recycling by macrophages that phagocytises senescent erythrocytes (Gattermann *et.al.*, 2021). The two systems are regulated by the hormone hepcidin, which sustains total-body iron at normal levels, preventing both deficiency and excess.

2.4.1 Causes and Risk Factors of Iron Deficiency Anemia

Iron deficiency anemia is usually caused by one of three basic mechanisms: low dietary iron intake, poor absorption of iron, or loss of blood (Figure 4) (Hamzah & Nehar, 2025). Of these, blood loss specifically from gastrointestinal sources is a most prominent cause in men and postmenopausal women (Hamzah & Nehar, 2025). Added risk factors for women are menstruation, pregnancy and breast-feeding (Kolarš *et.al.*, 2025). Pregnant women are at heightened risk due to augmented iron demands for foetal development, whereas babies and young children require adequate iron for growth and cognitive advancement. The incidence of iron deficiency anaemia (IDA) escalates among patients with chronic illnesses, such as gastrointestinal problems that impair iron absorption, and in elderly populations due to inadequate nutritional consumption.

Whereas the diagnosis of IDA was traditionally uncomplicated and could be diagnosed by routine laboratory analysis employed in most healthcare settings, interpretation of such results requires to be strictly interpreted due to confounders (Khatun *et.al.*, 2025). A diagnosis of IDA is an indicator that work-up should be undertaken to discover the underlying cause; IDA can be a sign of a life-threatening condition that should be treated. Iron homeostasis in the body is meticulously managed to ensure adequate absorption of iron to offset physiological losses. Consequently, the majority of patients with iron deficiency anaemia seeking medical consultation exhibit insufficient food intake, compromised absorption, or physiological blood loss, particularly in menstrual women (Alkdede *et.al.*, 2020). Nutritional requirements significantly escalate during adolescence to support growth and muscle development, leading to a rise in blood volume.

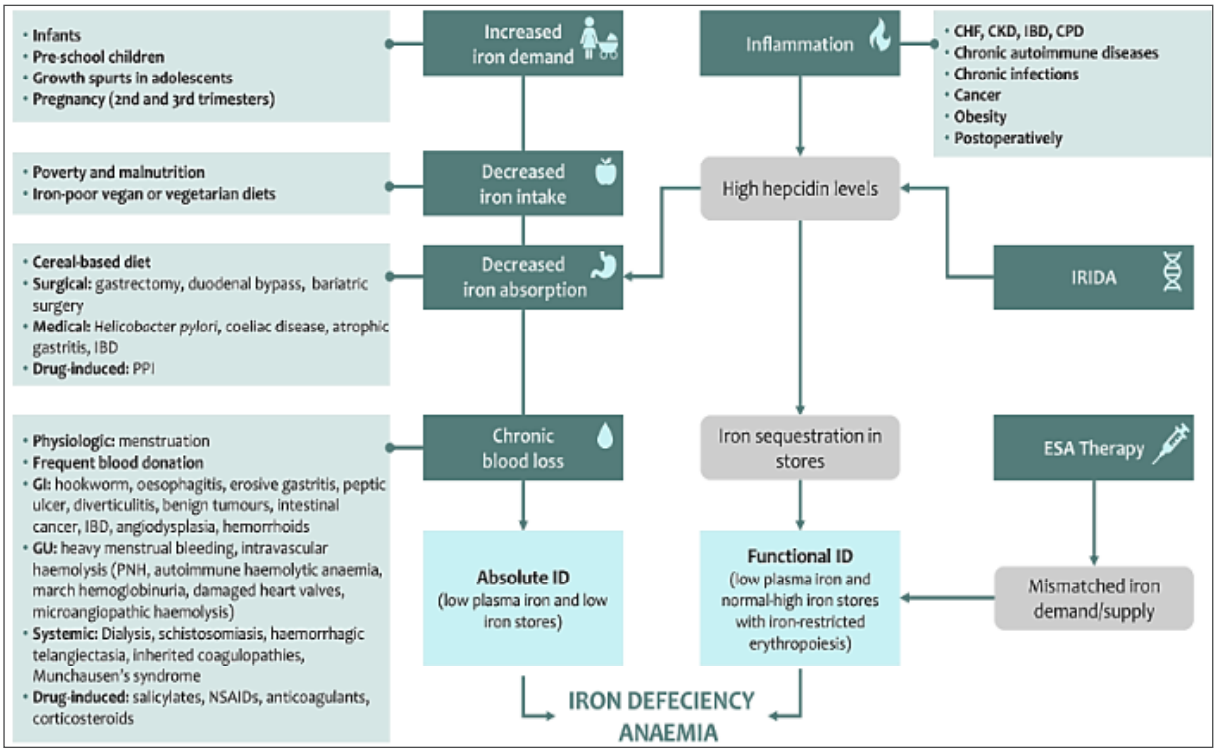

Figure 4. *Diverse etiology of Iron deficiency anemia (Nehar et.al., 2024)*

2.4.2 Clinical Features of Iron Deficiency Anemia

Iron deficiency (ID) can cause serious symptoms in certain people, but it can also be asymptomatic in others. Fatigue, irritability, dyspnoea, headache, hair loss, pica, restless legs syndrome, and changes in epithelial cells, such as dry mouth, cheilitis, atrophic glossitis, Plummer-Vinson pharyngeal webs, and diminished physical performance, are all signs of iron deficiency (Nehar *et.al.*, 2024). Iron deficiency anaemia has few and often undetectable clinical signs and symptoms (Nehar *et.al.*, 2024).

The pallor of anaemia was linked to weakness and fatigue even before its aetiology was understood. It is now acknowledged that mild to severe iron shortage can have detrimental functional effects, even in the absence of anaemia. Iron deficiency negatively impacts cognitive performance, behaviour, and physical growth in infants, preschoolers, and school-aged children; immune function and infection-related morbidity across all age groups; and energy utilisation by muscles, thereby affecting physical capacity and work performance in adolescents and adults of all ages (Leung *et.al.*, 2024). Iron deficiency anaemia during

pregnancy elevates perinatal hazards for mothers and neonates, as well as overall infant mortality (Nehar *et.al.*, 2024). Additionally, iron-deficient animals and humans exhibit compromised gastrointestinal functioning and modified hormone synthesis and metabolic patterns. The latter encompasses neurotransmitters and thyroid hormones linked to neurological, muscular, and thermoregulatory changes that restrict the ability of individuals exposed to cold to sustain their body temperature. Moreover, DNA replication and repair necessitate iron-dependent enzymes (Bardestani *et.al.*, 2021).

2.4.3 Diagnosis of Iron Deficiency Anemia

Iron deficiency anaemia (IDA) necessitates a comprehensive clinical assessment, laboratory analysis, and further studies to ascertain the underlying aetiology. Key diagnostic components include complete blood count (CBC), iron tests, and reticulocyte count. Red blood cells (RBCs), haemoglobin, haematocrit, and red blood cell indices are all thoroughly analysed by a complete blood count (CBC). In IDA, hemoglobin levels are frequently low due to insufficient iron for hemoglobin production (Nyambura, 2024). Hematocrit quantifies the proportion of blood volume filled by red blood cells. Mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), mean corpuscular haemoglobin concentration (MCHC), and red blood cell distribution width (RDW) are indices of red blood cells. Iron studies are crucial for confirming iron insufficiency and understanding the body's iron metabolism. Essential tests include serum iron, ferritin, total iron-binding capacity (TIBC), and transferrin saturation (Nehar *et.al.*, 2024). Reticulocyte count assesses the bone marrow's response to anemia. The reticulocyte count is frequently low or abnormally normal in IDA, which reflects the bone marrow's diminished ability to make new red blood cells as a result of iron shortage (Alkdede *et.al.*, 2020). Differential diagnosis is required for proper therapy. Some kinds of anemia may show with similar laboratory findings but have different underlying causes and therapeutic approaches (Nyambura, 2024). Key differentials include Anemia of Chronic Disease (ACD), Thalassemia, Sideroblastic Anemia, and Vitamin B12 and Folate Deficiency Anemia. A thorough evaluation is crucial for identifying the underlying cause of iron deficiency, whether it be dietary, due to blood loss, or related to malabsorption, which leads appropriate management and prevents recurrence (Nyambura, 2024).

2.4.4 Iron metabolism and absorption

Iron in our body is an essential mineral for many fundamental processes such as oxygen transport and mitochondrial function. Additionally, iron functions as a co-factor in enzymatic processes such as RNA transcription, protein translation, DNA replication, and myelin production (Bardestani *et.al.*, 2021). Males contain about 4,000 mg of iron, of which 2,500 mg is within erythrocytes; 1,000 mg is stored in splenic and hepatic macrophages, and the rest is distributed in various proteins such as myoglobin, cytochromes or other ferroproteins (Waldvogel-Abramowski *et.al.*, 2014). About 1–2 mg of iron is lost every day, through skin and enteric desquamation and minor blood losses and this loss is balanced by intestinal absorption (Waldvogel-Abramowski *et.al.*, 2014). Therefore, iron recycling accounts for most of the iron homeostasis in human. The situation is different in menstruating women where there are discussions about iron stores, ferritin and hemoglobin level.

The human body has roughly 3–4 g of iron which may be lost up to 0.1% daily under physiological and pathological situations that are normally compensated by daily nutritional consumption (Bardestani *et.al.*, 2021). Both iron deficiency and iron excess can alter the development and function of the brain from fetal to maturity (Correnti *et.al.*, 2024). There are two kinds of iron in daily diet: heme iron with absorbable ferrous ion (Fe 2+) that appears in red meat and seafood, and non-heme iron with ferric ion (Fe 3+) that exists in plant-based meals (Cappellini *et.al.*, 2022). Iron absorption can be modulated through body iron levels and different iron regulatory agents. On the apical membranes of intestinal absorptive cells called enterocytes, duodenal cytochrome B (Dcytb), an ascorbate-dependent plasma transmembrane ferrireductase, converts Fe3+ to Fe 2+ (Waldvogel-Abramowski *et.al.*, 2014). Iron enters the cell through metal transporters (Seyid *et.al.*, 2023). Divalent metal transporter 1 (DMT1) and heme carrier protein 1 (HCP1) are the primary non-heme and heme iron transporters, respectively (Figure 4). They can transport Fe2+ and heme from the intestinal lumen into the enterocytes (Dutt *et.al.*, 2022). HCP1 is preferably the high-affinity obligatory folate transporter. Fe 2+ from non-heme and heme iron that has been broken down by heme oxygenase-1 (HO-1) then enters the labile iron pool (LIP), a temporary intracellular iron pool (Kalman *et.al.*, 2025). In the basolateral membrane of enterocytes, iron exporter ferroportin releases most of this Fe2+. Its surplus is transported to a cytosolic iron-storage protein called

ferritin. The ferroxidase activity of intestinal ferritin's H subunit, which reoxidises Fe2+ to Fe3+, makes it an efficient factor in iron absorption (Kuma & Clark, 2020).

On other hand, the iron liberated from the enterocytes is re-oxidized to Fe3+ by ferroxidases (i.e., membrane bound multicopper hephaestin and soluble and/ membrane-bound multicopper ceruloplasmin), which are engaged in the iron export by ferroportin (Kumar *et.al.*, 2022). Iron oxidation is needed for iron transfer by plasma iron-free transferrin, so-called apotransferrin (Apo-Tf). Trapping and holding Fe 3+ by iron-storage proteins such as ferritin and transferrin reduces Fe 3+ reactivity and free radical production. Apo-Tf binds to two ferric ions at normal alkaline pH (7.4) of the plasma to form holo-transferrin (Holo-Tf). This iron-loaded glycoprotein as a plasma iron pool supplies iron to the target tissues such as bone marrow, liver, and brain (Bardestani *et.al.*, 2021). Hepatocytes and macrophages are responsible for iron storage and iron recycling, respectively. Under normal settings, virtually the whole of the extracellular iron penetrates the target cell in the form of transferrin-bound. However, transferrin saturation due to iron overload hinders iron binding to transferrin and leads to non-transferrin bound iron (NTBI) uptake (Bardestani *et.al.*, 2021).

Holo-Tf binds to transferrin receptor (TfR) on the surface of most cells and through clathrin-coated vesicles and adaptor protein 2 (AP2), the Holo-Tf-TfR complex is internalised to the cell during the endocytosis cycle known as clathrin-mediated endocytosis (CME) (Nemeth & Ganz, 2021). The endocytic vesicles merge with the endosome membrane after losing their clathrin covering. While transferrin stayed bound to TfR and changed back into Apo-Tf, Fe3+ is liberated from a transferrin-TfR complex in the acidic pH (5.5–6.0) of late endosomes. Besides, endosomal ferrireductase such as 6-transmembrane epithelial antigen of the prostate (Steap) converts insoluble Fe3+ to soluble Fe 2+ that is transferred from the endosomal lumen into the cytosol by DMT1. At a pH of 7.4, apo-Tf bound to TfR dissociates from the receptor and is recycled to the cell surface (Bardestani *et.al.*, 2021). Here, TfR is ready to bound the next Holo-Tf and initiating recycling. Cytosolic iron faces a number of challenges, including; involvement in biological processes through embedding in metalloproteins, involvement in mitochondrial energy transduction, and storage as ferritin. Besides, lysosomal breakdown of ferritin leads to the formation of an iron-storage complex, namely, haemosiderin, that is related to pathological conditions (e.g., iron overload) and engaged in reactive free radical generation (Bardestani *et.al.*, 2021). Figure 5 below shows the iron absorption pathways.

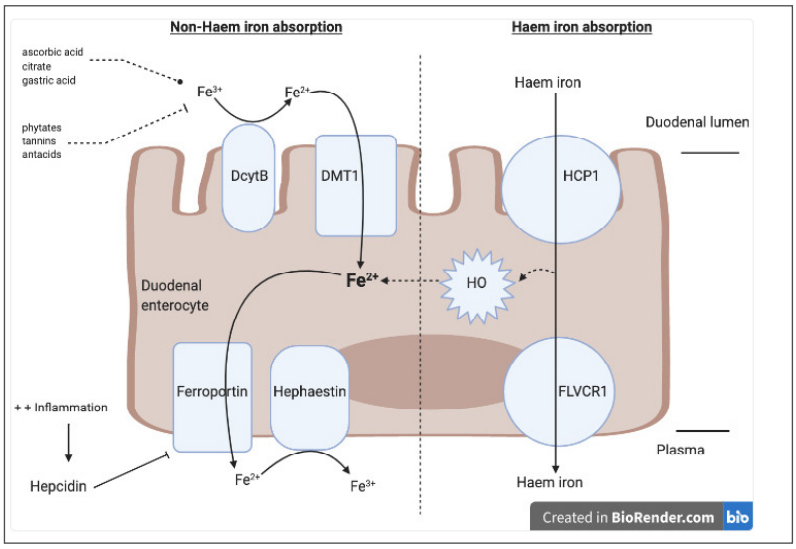

Figure 5. *Iron absorption pathways (Kumar et.al., 2022)*

2.4.5 Systemic Regulation of Iron Absorption

Hepcidin is a liver-derived hormone produced by the (Hepcidin Antimicrobial Peptide) HAMP gene and is the key systemic regulator of iron homeostasis (Nehar *et.al.*, 2024). Hepcidin works as a repressor of cellular iron export, and performs this role by covalently binding to FPN on the cell surface (Nemeth & Ganz, 2021). This leads to the polyubiquitination of lysine residues inside the intracellular loops of FPN, a process controlled by ubiquitin like modifier activating enzyme 6 (UBA6), the adaptor protein Nedd4 family interacting protein 1 (NDFIP1) and the ubiquitin ligase ring finger protein 217 (RNF217) (Qiu *et.al.*, 2025). Following ubiquitination, the hepcidin-FPN complex is destroyed in the lysosome, thus limiting

iron export. The binding affinity of hepcidin for FPN increases 80-fold without iron (Billesbølle *et.al.*, 2020), suggesting that binding of Fe2+ to FPN can regulate hepcidin activity, which potentially allows hepcidin to more readily target FPN in enterocytes and macrophages which are actively exporting iron, although there is no direct evidence for this in vivo (Qiu *et.al.*, 2025).

Iron homeostasis is regulated by various variables, including the hepcidin hormone and the iron-regulatory proteins (IRP1 and IRP2) together with the iron-responsive element (IRE) signalling pathway (Nemeth & Ganz, 2021). Hepcidin, synthesised by the liver, serves as a crucial systemic regulator. When iron is plentiful, hepcidin attaches to enterocyte ferroportin, inhibiting the export of iron from the cell (Bardestani *et.al.*, 2021). The IRP/IRE signalling pathway modulates iron homeostasis at the cellular level, contingent upon the body's iron concentrations. In iron shortage, IRP attaches to the IRE motif in the 5′ untranslated region (5′ UTR) of ferroportin and ferritin mRNAs to inhibit their translation. The binding of IRPs to the IRE motif in the 3′-UTR of TfR and DMT1 transcripts stabilises their mRNAs, hence enhancing translation. These mechanisms lead to decreased plasma iron and increased cellular iron for use in the metabolic processes (Nemeth & Ganz, 2021). On the contrary, when the iron is abun- dant, IRP cannot bind to the IRE motif at 5'UTR of both ferroportin and ferritin transcripts and enhances translation of their mRNAs as well as IRP cannot bind to the IRE motif at 3′-UTR of TfR and DMT1 transcripts and destabilizes mRNAs to suppress translation (Bardestani *et.al.*, 2021).

While the processes involved in the control of dietary iron absorption remain similar throughout maturity and into old age, hepcidin synthesis can be altered by age-dependent increases in inflammation, hormonal changes, and chronic diseases such as renal disease (Longo & Camaschella, 2015). The subsequent elevation of hepcidin production inhibits FPN-mediated iron export from enterocytes, therefore lowering intestinal iron absorption

2.4.6 Iron Transportation Mechanism

Transferrin (Tf) is a single chain glycoprotein that binds and transfers iron to tissues. Each Tf molecule can transport 2 ferrous iron molecules. Conformation of the binding site is appropriate with ferric iron in a sensitive manner. Transferrin attaches to one of the transferrin receptor (TfR) on cell membrane; TfR1 or TfR2 (Guo *et.al.*, 2025). Transferrin receptor 1 is expressed in all tissues except mature erythrocytes. It is a key chemical in embryogenesis. Transferrin receptor 1-knockout embryos do not survive due to poor erythropoiesis and cerebral maldevelopment. However, other tissues in body are not affected in the absence of TfR1 which shows other mechanism(s) for iron transport in these cells. Transferrin receptor 2 is predominantly expressed in the liver. Although protein structures of TfR1 and TfR2 have a significant degree of similarity, their activities and regulation are not the same (Özbek, 2010). Expression of TfR1 is strictly regulated by cellular iron levels through HFE (hereditary hemochromathosis) protein. However, cellular iron levels have little effect on the control of TfR2 and TfR2 recognises the body iron status by sensing the transferrin saturation and regulates hepcidin expression correctly (Özbek, 2010).

After binding of diferric Tf to TfR, Tf/TfR complex on the clathrin-coated cell membrane is absorbed by a receptor-mediated endocytosis. Iron and Tf are separated within endosomes by an acidification mechanism that uses an ATPase proton pump (pH 5.5–6) (Guo *et.al.*, 2025). In erythroid precursor cells, a protein known as STEAP3 (Six-Transmembrane Epithelial Antigen of Prostate 3) has been demonstrated to change Fe+3 into Fe+2 (12). Since DMT1 only permits divalent metals to go from endosomes to cytoplasm, as in enterocytes, this conversion is required. After iron is released, the endosome that contains Tf and TfR fuses back to the plasma membrane. Hepatocytes are the only cells that can produce non-transferrin-bound iron from plasma.

2.4.7 Iron and the Erythrocyte

Transferrin-bound iron travel to the bone marrow, where growing erythroid cells use receptor TfR1 to absorb almost all of it. Depending on the cell's state of maturation, erythroid cells exhibit the largest concentration of TfR1, with several hundred thousand receptors per cell. The iron content of transferrin determines how much iron is taken up by TfR1 [the affinity of diferric transferrin for TfR1 is several times more than that of monoferric transferrin and a thousand times greater than that of apotransferrin] (Correnti *et.al.*, 2024). Through clathrin-coated pits, the transferrin iron-TfR1 complex penetrates the erythroid cell and forms an endosome. When a hydrogen pump brings H+ into the endosome, local ferrireductase causes transferrin and iron to separate, converting Fe3+ to Fe2+ (Bardestani *et.al.*, 2021).

Fe2+ iron is transported by DMT1 into the cytosol, where it is absorbed by the mitochondria and integrated

into the porphyrin ring by ferrochetolase to produce heme (Qiu *et.al.*, 2025). Haemoglobin is created when four globin molecules join with haem. Ferritin binds any iron that isn't utilised to make haemoglobin. The TfR1 protein and transferrin are returned to the cell surface and circulation, where they are efficiently recycled to resume their functions. The patient's iron level and overall erythropoietic activity are reflected in the amount of TfR1 protein in the blood. After 120 days, macrophages in the liver and spleen break down the erythrocyte into its constituent parts (Correnti *et.al.,* 2024). Ferroportin carries 80–90% of erythrocyte iron to the cell membrane, where it is discharged into the blood and bonded to transferrin iron. The macrophages retain and store 10–20% as ferritin (Bardestani *et.al.*, 2021).

2.4.8 Pathophysiology of Iron Deficiency Anemia

Adult men contain 35 to 45 mg of iron per kilogram of body weight in a physiologically normal

state, whereas premenopausal women have lower iron stores because of regular blood loss from menstruation (Yang *et.al.*, 2023). More than two-thirds of body iron is contained in hemoglobin of erythrocytes and erythroblasts in bone marrow, circulation, and reticuloendothelial macrophages (Dutt *et.al.*, 2022). Most of the remaining iron is stored in the liver as ferritin, and the rest is found in myoglobin and enzymes (Yang *et.al.*, 2022). Plasma contains only 0.1% of total body iron, which is bound to transferrin. In this form, iron can be transferred to various tissues via binding to the transferrin receptor. Iron deficiency can be caused by either an insufficient amount of absorbable dietary iron or an excessive loss of body iron (Nehar *et.al.*, 2024). Insufficient intake of dietary iron in an absorbable form is typically the cause of diminished absorption. The most frequent cause of high iron loss in the body is bleeding, however intravascular haemolysis can also result in haemoglobinuria. Malabsorption of iron is comparatively uncommon in the absence of small bowel disease (sprue, coeliac disease, regional enteritis) or prior gastrointestinal surgery (Correnti *et.al.*, 2024).

One reason for the lack of appropriate iron absorption is that upon exposure to oxygen, iron produces highly insoluble oxides, which are inaccessible for absorption in the human gastrointestinal tract (First *et.al.*, 2023). Human enterocytes contain apical membrane-bound enzymes whose activity may be controlled and which function to convert insoluble ferric (Fe3+) to absorbable ferrous (Fe2+) ions (First *et.al.*, 2023).

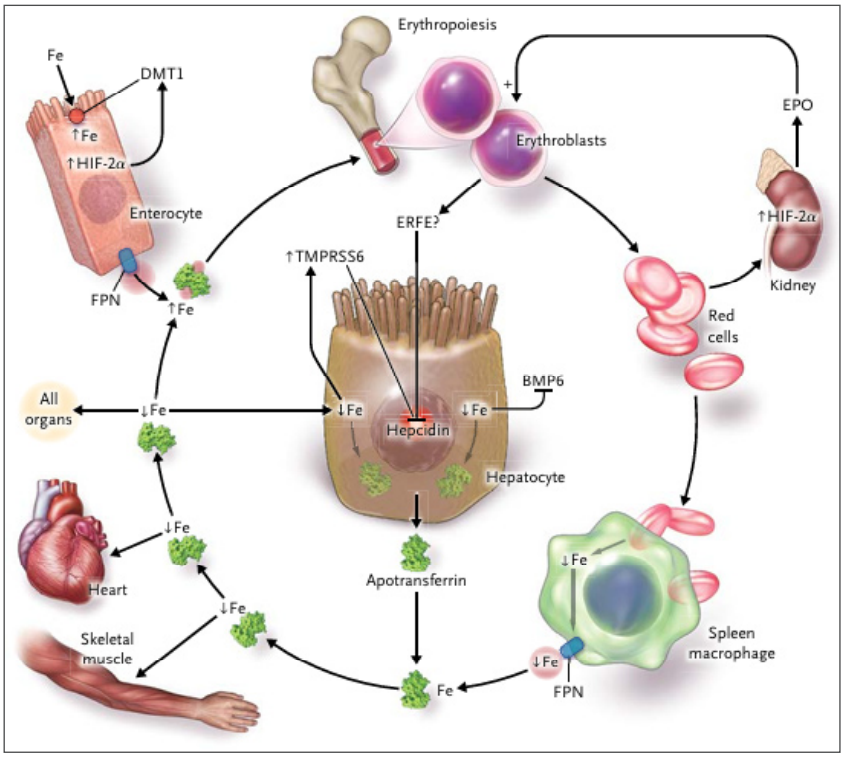

Figure 6. *The iron cycle; Mechanism of adaptation to iron deficiency anemia (Longo & Camaschella, 2015)*

The mechanisms of adaptation to iron shortage are concentrated on the reduction of the hepatic hormone hepcidin and the tissue hypoxia that develops consequent to anemia. Hepcidin (encoded by *HAMP*), a small peptide derived from hepatocytes, is the central regulator of systemic iron homoeostasis (Yang *et.al.*, 2023). The production of erythropoietin (EPO) by the kidney increases in response to elevated levels of hypoxia-inducible factor 2α (HIF-2α). As a consequence of the stimulation of erythropoietin,

erythropoiesis is enhanced, and hypochromic microcytic red cells are generated owing to the poor availability of iron which eventually leads to anemia (Nehar *et.al.*, 2024). Because most iron approximately 25 mg daily is necessary for hemoglobin (Hb) synthesis to enable the creation of over 200 billion red blood cells each day, IDA is the most visible consequence of ID (Kolarš *et.al.*, 2025). This sometimes leads to the false idea that ID and IDA are the same. However, ID is a larger disorder that can precede the beginning of IDA or harm other tissues beyond those involved in red blood cell synthesis (Kolarš *et.al.*, 2025).

Senescent red cells are killed by macrophages, and their iron is recycled. The increase in erythropoiesis decreases the formation of hepcidin. Erythroferrone (ERFE), a hormone produced by erythroblasts in response to EPO, can mediate hepcidin suppression during stress erythropoiesis (Yang *et.al.*, 2023). In cases of extreme iron deficit, the diminished synthesis of hepcidin results in elevated Ferroportin (FPN) expression in erythroblasts and erythrocytes, facilitating the release of iron into the blood through FPN to mitigate serum iron depletion and safeguard erythrocytes from oxidative stress (Zhang *et.al.*, 2018). HIF-2α enhances the expression of the duodenal divalent metal transporter 1 (DMT1) on the apical surface of enterocytes to promote the transfer of dietary iron from the lumen to enterocytes (Qiu *et.al.*, 2025). Hepcidin levels are depressed in response to a reduction in the physiologic signals that maintain its production (e.g., increases in levels of iron-bound transferrin and in the iron content of the liver), to the increased activity of the inhibitor transmembrane protease, serine 6 (TMPRSS6), to the reduction in levels of the activator bone morphogenetic protein 6 (BMP6), and to increased inhibition from erythropoietin-stimulated erythropoiesis (Figure 6) (Longo, & Camaschella, 2015). FPN, which is no longer being degraded because of the low levels of hepcidin, exports the available iron across the enterocyte basal membrane and from macrophage stores to the circulation. Once reserves are expended, levels of circulating iron drop, even if absorption from the lumen is increased. Reduced levels of iron in the liver cause increases in the manufacture of the iron carrier transferrin (referred to as apotransferrin when not bound to iron), further decreasing levels of iron-bound transferrin, the ligand of the transferrin receptor. Consequently, the uptake of iron from transferrin receptors by all cells and organs (e.g., skeletal muscles and the heart) is diminished.

2.5 Dietary Habits and Anaemia

All living creatures depend on food to survive. Food is necessary for our bodies to produce energy, survive, and maintain their strength. A balanced diet is necessary for optimal health; this entails eating a variety of foods to ensure your body gets all the nutrients it needs. A healthy, balanced diet high in protein, energy, and vitamins will improve an individual's health (WHO, 2026). A healthy diet is one that meets an individual's needs for all nutrients in sufficient quantities while considering their age, gender, and physical state into consideration (Omage & Omuemu, 2018). Over the course of a person's life, a healthy diet is essential to their wellbeing. It encourages a healthy body weight, maintains appropriate growth and development, and reduces the risk of chronic illnesses. By reducing non-communicable diseases (NCDs) like diabetes, heart disease, obesity, and some types of cancer, proper eating habits not only improve short-term health outcomes but also contribute to long-term wellness (WHO, 2026). Consuming fruits, vegetables, legumes, whole grains, and lean proteins while limiting processed and high-fat foods is the focus of healthy eating habits. On the other hand, poor eating practices make people more vulnerable to malnutrition in all its manifestations, including undernutrition and overnutrition, which can have negative effects on immunity, productivity, and quality of life (Muchelo *et.al.,* 2025).

Malnutrition occurs in early life when individuals do not ingest or assimilate the essential macronutrients and micronutrients required for growth and physiological activities. The World Health Organization (WHO) states that undernutrition causes around 45% of mortality which occur in children younger than five years in low-and middle-income countries (LMICs) that endure food shortages and financial hardship and have restricted medical care (Tan, 2026). The 2020 Global Nutrition Report stated that more than 149 million children under five years of age experience stunting while 45 million children endure wasting which represents the two types of malnutrition (Tan, 2026). The conditions make people more vulnerable to infectious diseases because they impair their immune systems and reduce their ability to think which creates a cycle of bad health along with economic difficulty (Kiely *et.al.*, 2025).

Dietary patterns have also changed, especially among young individuals, as a result of increased access to convenience and processed foods and rapid urbanisation. Students frequently eat meals heavy in lipids, free sugars (carbonated soft drinks), salt, and sodium but low in fruits, vegetables, and other foods high in fibre, such whole grains. Because they are away

from home and typically preoccupied with academic pursuits, university students may be exposed to poor eating habits. Poor nutrition is also a result of the high expense of healthful meals and the ease with which fast food may be obtained. University students have been found to frequently miss breakfast and to be an independent predictor of obesity.

As they move through undergraduate life, a number of factors are at play. These undergraduate students are subjected to a variety of academic responsibilities, including ongoing evaluation, written and oral exams, strict deadlines, and interpersonal disputes, to name a few instances of the countless situations that can cause high levels of stress in students. Stress is believed to improve memory formation during learning, but it significantly hinders memory retrieval, which increases the likelihood of underachieving at the end of each academic session and the final grade upon graduation, for example. According to a survey conducted among some undergraduate students at the University of Benin in Nigeria, the most prevalent health factor influencing academic progress was perceived stress (Aihie and Ohanaka, 2019). Stress can cause persistent annoyance and a pessimistic outlook on school and the person's skills (Vogel and Schwabe, 2016). Researching the degree of perceived stress and the causes of it can provide insights into how stress can be controlled to enhance students' performance.

Additionally, during their undergraduate years, students typically develop or alter their food and exercise habits. Because these behaviours frequently persist into adulthood and can be very challenging to modify once established, it is crucial to adopt healthy eating habits during this time (Brown et al., 2014). Both short-term and long-term physical activity sessions enhance brain health and cognitive function (Mandolesi et al., 2018).

2.5.1 Prevalence of Iron Deficiency Anemia

Globally, over 1.93 billion people suffer from anemia, with over 89% living in developing countries and poses a significant health challenge (Yusuf *et.al.*, 2025). IDA is the most is the most widespread cause of anemia globally. It affects up to 40% of home-dwelling elderly individuals, 5% of children and adolescents, 10% of premenopausal women, and 1% of men (Almbsuot *et.al.*, 2025). This was a minor to moderately significant public health issue among undergraduates in countries with lower incomes. The prevalence among university students varies greatly, with 34% affected in Yemen, 55.3% in Bangladesh, and 67.35% among Saudi Arabian female students, respectively (Al-Alimi *et.al.*, 2018; Hamali *et.al.*, 2020). These figures show a worrying burden, especially for female students, which is probably caused by menstrual blood loss and nutritional deficiencies.

A recent study from Saudi Arabia stated that the majority of female Saudi students who attended colleges have anemia. Iron deficiency anemia is common, indicating that these students get this sickness very often with a prevalence ranging from 20% to 39.9% (Almasmoum *et.al.*, 2023). Numerous factors, such as age, dietary habits, socioeconomic status, ethnic makeup, and diagnostic criteria, affect the prevalence of iron-deficiency anaemia. Anaemia affected 41.7% of children under five, 40.1% of pregnant women, and 32.5% of non-pregnant women worldwide in 2016 (Nehar *et.al.*, 2024). The prevalence rates vary by country; it affects 2.4 million children in the United States, 5.4% of children in Spain, 14.0% of children in Estonia, and 30.8% of children in Brazil (Benson *et.al.*, 2022). Another study from university students in Libya revealed that the overall prevalence of anemia was 29% and this was due to unhealthy eating habits and lacking sufficient intake of iron-rich foods (Almbsuot *et.al.*, 2025).

In Bangladesh, nutritional anaemia has long been identified as a serious public-health problem and the national vitamin A survey conducted in 1997-1998 showed that nearly 50% of pregnant women in rural Bangladesh had anaemia (Shill *et.al.*, 2014). Moreover, many surveys conducted in the past stated that anaemia is a severe problem among all across age, population and geographic groups in Bangladesh. In 2004, another survey conducted by Nutritional Surveillance Project of Helen Keller International in collaboration with the Institute of Public Health Nutrition showed that 68% of under-five children were anaemic (Shill *et.al.*, 2014). The survey also suggested that 40% of adolescent girls and 31% adolescent boys as well as 46% of non-pregnant and 39% of pregnant women were affected by anaemia (Shill *et.al.*, 2014).

According to the Global impact of Disease (GBD) Study 2021, iron deficiency and its effects, notably anaemia, demonstrate a region-specific impact that varies dramatically across different regions, underlining the need for more regional assessments (Gardner *et.al.*, 2023). Globally, iron deficiency has been connected to a number of social and economic difficulties, including poverty, level of education, and access to medical care (Haematology, 2023). The Middle East and North Africa (MENA) region, home to approximately 400 million people, has long

suffered from pervasive micronutrient malnutrition, notably iron deficiency (Safiri *et.al.*, 2025). This region is defined by political and economic variety, with both undernutrition and overnutrition prevalent. Many MENA nations are witnessing changing nutritional trends, known as the nutrition transition, as diets become less healthful, more processed, higher in calories, and lower in micronutrient density (Safiri *et.al.*, 2025). This alteration in dietary structure, combined with the region's distinctive age-group composition, has considerably contributed to the burden of iron deficiency. Certain populations, notably women of reproductive age, children, and the underprivileged, are particularly vulnerable due to restricted access to iron-rich foods and inadequate healthcare services (Doumat *et.al.*, 2024; Safiri *et.al.*, 2025).

2.5.2 Distribution of Iron Deficiency Anemia

Sex distribution

In high-income nations, iron deficiency anaemia is a significant issue, particularly among women of reproductive age and specific subpopulations. A study in the United States revealed that 5-10% of premenopausal women and 2-5% of men experience iron deficiency anaemia (Manish, 2025). Women are particularly affected, with incidence rates up to four times higher than those in men (Kolarš *et.al.*, 2025). Women are at elevated risk of iron shortage due to iron loss from heavy menstrual bleeding, post-partum hemorrhage and pregnancy (Figure 7). Approximately one-third of women of reproductive age worldwide are anaemic, with iron deficiency accounting for about 50% of these instances. Recent studies indicate a substantial correlation between maternal haemoglobin levels and unfavourable maternal and newborn outcomes, underscoring the necessity of managing iron deficiency during reproductive years (Ohuma *et.al.*, 2023). Iron deficiency in children can hinder cognitive development, leading to enduring learning and educational challenges, which ultimately impact a nation's human capital and economic development. Additionally, iron deficiency anaemia (IDA) during pregnancy constitutes a considerable public health concern, since it elevates the likelihood of maternal and neonatal problems, such as preterm delivery, low birth weight, and developmental delays in newborns (Manish, 2025).

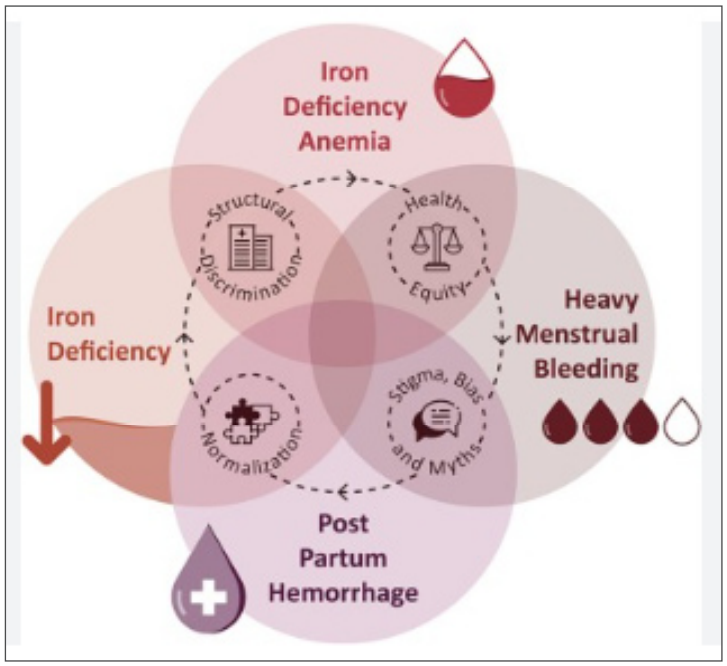

Figure 7. *Causes of anemia in women (Tang, & Sholzberg, 2024)*

Socioeconomic Distribution

Although ID and IDA are frequently considered as problems isolated to low- and middle-income regions, recent research demonstrates their persistent relevance and growing impact in high-income countries. Population-based studies in Europe have indicated a steady increase in IDA incidence from the early 2000s to the early 2010s, with certain countries exhibiting rates above 12 per 1000 person-years. Women are disproportionately impacted, with incidence rates up to four times higher than those in men. Prevalence data reveal a similar pattern, with overall rates nearing 3% and much higher levels in females (Kolarš *et.al.*, 2025). In the United States, data from the National Health and Nutrition Examination Survey (NHANES) between 1999 and 2018 reveal that 7.5% of pregnant women are anemic, whereas nearly 20% had ID based on serum ferritin levels. Among young

children, anemia affects 4.7%, and ID reaches up to 30.5% when using physiologically reasonable cutoffs (Jefferds *et.al.*, 2022). These findings underline that ID and IDA remain under recognized public health concerns in wealthy countries, disproportionately affecting women of reproductive age, young children, and certain ethnic minorities. Contributing causes may include demographic aging, increased rates of obesity and chronic disease, pharmaceutical use, and underdiagnosed gastrointestinal (GI) or gynecological problems (Jefferds *et.al.*, 2022; Kolarš *et.al.*, 2025).

In developing countries, anemia affects 20–39.9% of women of reproductive age in several regions, with certain countries such as Papua New Guinea and parts of Indonesia reporting severe anemia prevalence exceeding 70% (Kolarš *et.al.*, 2025). Specific regional data show to anemia prevalence rates as high as 72.9% among women of reproductive age in Indonesia and up to 89.7% in Papua New Guinea, with a considerable proportion attributed to ID and aggravated by diseases such as malaria and helminthiasis (Kolarš *et.al.*, 2025).

2.5.3 Age Distribution

In the least developed countries, particularly in sub-Saharan Africa, the prevalence of anemia among children aged 6–59 months generally surpasses 60%, with some countries reporting rates as high as 86%. Data suggest that IDA affects up to 72.8% of children in certain places (e.g., Ethiopian Somali region), while ID alone may affect over 90% in extremely sensitive subpopulations (Gardner *et.al.*, 2023). While national estimates for IDA typically range from 12% to 46%, local studies in high-risk groups (e.g., breastfed infants or those exposed to illnesses and food hardship) indicate extraordinarily high rates, often exceeding 60–70% (Lemoine, & Tounian, 2020). Anemia during childhood affects approximately 39% of children between the ages of 6 to 59 months who live in various parts of the world with sub-Saharan Africa and South Asia showing the highest prevalence rates (Tan, 2026).

In children, a review of 44 research conducted across 19 European nations found that 2–25% of newborns aged 6–12 months were iron deficient, with greater rates among those from poorer socioeconomic backgrounds and those who drank cow's milk throughout their first year. For children aged 12–36 months, ID prevalence ranged from 3% to 48%, whereas the rate of IDA was as high as 50% in Eastern Europe, but less than 5% in Western Europe (Kolarš *et.al.*, 2025).

In the elderly, approximately one-third of patients have a nutritional deficiency as the cause of anemia, such as iron, folate, and vitamin B12 insufficiency. In another one-third of patients, there is evidence of renal failure or chronic inflammation. New-onset anemia, especially in people over 55 years of age needs research and should be considered malignancy unless proven otherwise. This is especially true in males of any age who come with anemia (Aparna *et.al.*, 2022). Pregnant women with anemia can go into premature labor and give birth to kids with low birth weight. Anemia during pregnancy also increases the risk of anemia in the foetus and increased blood loss throughout pregnancy (Aparna *et.al.*, 2022).

2.6 Nutritional Factors Affecting Iron Status

2.6.1 Enhancers of Iron Absorption

The hydrochloric acid contained in gastrointestinal secretions turns ferric iron onto ferrous iron, which has a higher absorption rate. In achlorhydria iron absorption was considerably reduced while injecting hydrochloric acid enhanced non-heme iron absorption by 4 times. However, heme iron absorption was unaltered by hydrochloric acid (Malik *et.al.*, 2025). Ascorbic acid is also demonstrated to boost non-heme iron absorption by building a chelate with iron in the small intestine. The ferric form of iron interacts with ascorbic acid in the acidic pH of the small intestine and the chelate produced remains stable in the duodenum's alkaline pH (Skolmowska & Głąbska, 2022). Ascorbic acid transforms ferric iron into ferrous iron which has better bioavailability and subsequently binds with Divalent Metal Transporter 1 (DMT1). The ferric iron forms a compound with ascorbic acid (Fe3+– ascorbate complex) which then enters the Caco-2 cells (Malik *et.al.*, 2025). Meat factors contained in animal source diets have a good impact on numerous micronutrients including iron status in humans (Table 3). Meat factors are chemicals found in animal tissues that improve iron and other micronutrient absorption from plant meals. For example, the inclusion of veal or fish meat to a corn based diet dramatically enhanced non-heme iron absorption (Piskin *et.al.*, 2022). Research indicated a 180 and 100% increase in iron absorption from freeze dried beef and poultry consumption. However, ingestion of egg albumin was not related with any improved iron uptake, demonstrating that protein was the main component responsible for boosting iron absorption. This explains why plant based replacements of animal products although having high iron content have poor bioavailability of the nutrient (Malik *et.al.*, 2025).

Table 3. *Enhancers and Inhibitors of Iron Absorption (Malik et.al., 2025)*

	Substances/Elements	Mechanism of actiion
Enhancers	Hydrochloric acid (HCl)	• Converts Fe^{3+} to ferrous Fe^{2+} • Improves non-heme iron absorption.
	Ascorbic acid (Vitamin C)	• Reduces Fe^{3+} to Fe^{2+} • Forms stable chelates • Enhances absorption even in alkaline duodenum • Counteracts phytate inhibition
	Meat factors	• Enhance non-heme iron absorption likely due to specific proteins.
	Divalent Metal Transporter 1 (DMT1)	• Facilitates uptake of Fe^{2+} bound to ascorbate complex into enterocytes.
	Dietary phytase (enzyme)	• Breaks down phytic acid, improving iron bioavailability.
Inhibitors	Phytates (IP6)	• Bind Fe^{2+} and form insoluble complexes • Reduce bioavailability
	Polyphenols	• Bind iron (especially Fe^{3+}) via catechol/galloyl groups • Form redox-active complexes • Reduce iron absorption
	Calcium	• Inhibits both heme and non-heme iron • Interferes with DMT1 and ferroportin • Hinders transport into plasma
	Oxalic acid/Sodium oxalates (SO)	• Bind with Fe and form large complexes, especially with heme iron • Reduce brush border transport.
	Casein (milk protein)	• Forms casein phosphopeptides (CPPs) that chelate iron and reduce its availability.
	Whey protein	• Acts similarly to casein • reduces iron absorption.
	Fish skin/bones protein hydrolysates	• Contain chelating peptides that bind iron strongly and resist digestion, reducing bioavailability.
	Proton pump inhibitors (PPIs)	• Suppress gastric acid; reduce Fe^{3+} to Fe^{2+} conversion • Lower non-heme iron absorption, especially in deficient individuals.

2.6.2 Inhibitors of Iron Absorption

Phytic acid, also known as Myo-Inositol hexakiphosphate (IP_6), is known to reduce element bioavailability of iron, zinc and calcium. It binds with these divalent cations forming insoluble complexes, which remain stable and unabsorbed in the small intestine. An example is legumes, which contain high levels of both phytates and these micronutrients, but have poor bioavailability of these elements. Iron absorption increases by 4 to 5 times when phytate concentration is reduced to <0.01 mg/g of isolate (Zhang *et.al.*, 2022).

Polyphenols bind with iron to exert their antioxidant activities, as iron is involved in free radical generation. Polyphenols bind with the catechol and galloyl groups present in iron, there bv reducing iron levels required to free radical production. Flavonoids, ellagic acid, curcumin, and iso-flavones, all develop redox-active iron complexes which results in iron depletion (Scarano *et.al.*, 2023).

Calcium has the ability to hinder both heme and non-heme iron absorption. The inhibitory action occurs both during iron uptake by the enterocytes, and its transport across the basolateral membrane from enterocyte to plasma. Calcium interferes with the DMT1 receptors and ferroportin protein, both of which are important in iron transportation to small intestine. Calcium supplements hinder non-heme iron absorption, but if given with ascorbic acid food sources such as orange juice, the inhibitory effect of calcium declines significantly (Reddy *et.al.*, 2022). Oxalates act as anti-nutrient compounds for several elements including iron. They have the ability to bind with minerals and form water soluble salts, there by rendering these minerals unavailable for absorption (López-Moreno *et.al.*, 2022).

Animal proteins such as casein have the ability to form casein phosphopeptides (CCPs) which possess chelating properties. The mainly bind with calcium but also play a significant role in inhibiting iron absorption by binding with and forming iron complexes (Wu *et.al.*, 2020). The animal sources derived chelating peptides when bind with iron form stable complexes that are able to withstand gastrointestinal digestion. Even though stomach enzymes such as pepsin and trypsin act on these complexes and break them down while releasing free amino acids, 75% of the complex remains stable and resist luminal iron digestion (Malik *et.al.*, 2025).

2.7 Link between dietary behavior and health outcomes

Dietary choices are among the most major modifiable factors impacting obesity, long-term health consequences, and overall well-being. While physical inactivity, alcohol use, and smoking are known causes of obesity and chronic illnesses, eating habits are more important because they are deeply ingrained in daily life and consistently practiced, making them a major determinant of long-term health status (Jung & Cho, 2025). Low-quality diet is a leading cause of overweight and obesity and an important contributor to recent decreases in life expectancy in the U.S. and worldwide (Tuyizere & Gustafson, 2023).

Maintaining a balanced diet is essential for attaining optimal nutrition and overall well-being. By enhancing physiological and psychological factors, healthy eating practices can have a substantial impact on health outcomes. Better mood management, improved organ function, and a lower chance of chronic diseases including cancer, diabetes, and cardiovascular disease (CVD) are just a few advantages of proper nutrition (Cena & Calder, 2020).

Key nutrients such vitamins, minerals, proteins, and lipids are important for maintaining cellular activities, metabolism, and immune responses. Fibre, which is rich in whole grains, legumes, and vegetables, promotes digestive health and can assist with weight management. Antioxidants, which are present in fruits, vegetables, and nuts, help fight oxidative stress and inflammation, lowering the risk of chronic diseases. A key component of preventing disease is maintaining a healthy weight. Obesity, which is associated with a higher risk of diabetes, heart disease, and joint issues, can be avoided by eating nutrient-dense foods while controlling portion sizes (WHO, 2026). The body absorbs a wide range of nutrients from a varied, well-balanced diet, which promotes maximum health.

The ability of some dietary patterns, such as the Mediterranean diet, to prevent disease has drawn attention. This diet reduces cardiovascular risk and increases longevity by emphasising plant-based meals, lean meats, and healthy fats. In a similar vein, the DASH (Dietary Approaches to Stop Hypertension) diet, which emphasises fruits, vegetables, and low-fat dairy products, aims to reduce blood pressure and avoid hypertension (Al Mutairi *et.al.*, 2022). Disease prevention requires integrating dietary knowledge into daily life. Chronic illness risk can be reduced by limiting processed foods, sugar-filled drinks, and excessive salt. Rather, concentrating on whole, unprocessed foods guarantees a greater consumption of vitamins, minerals, and other health-promoting bioactive substances (Al Mutairi *et.al.,* 2022).

3. Conclusion

Iron deficiency anaemia remains a major global health concern despite substantial advances in its diagnosis, treatment, and prevention. Evidence reviewed indicates that the condition is driven by multiple interacting factors, including inadequate dietary iron intake, chronic blood loss, infections, pregnancy, and malabsorption disorders, with the greatest burden occurring among women, children, and populations in low- and middle-income countries. Early diagnosis through appropriate laboratory investigations, particularly serum ferritin supported by complementary iron indices, is essential for effective management. Although oral iron therapy remains the primary treatment, intravenous iron provides an effective alternative in selected clinical situations. Preventive strategies such as food fortification, nutritional education, routine screening, and control of parasitic and infectious diseases are critical for reducing disease prevalence. Overall, a multidisciplinary approach that combines evidence-based clinical management with comprehensive public health interventions and strengthened health policies is necessary to reduce the global burden of iron deficiency anaemia and improve health outcomes among vulnerable populations.

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