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# Anaemia and Oxidative Stress: Toxicological Perspectives and Herbal Therapeutics

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## ABSTRACT

Anaemia remains a major global health burden with multifactorial aetiologies including nutritional deficiency, chronic disease, genetic disorders, and toxic exposures. Oxidative stress—an imbalance between pro-oxidant species and antioxidant defenses—plays a central and underappreciated role in the pathogenesis of many anaemia types. Reactive oxygen and nitrogen species damage erythrocyte membranes and haem proteins, precipitating hemolysis; they impair bone marrow erythropoiesis via oxidative injury to progenitors; and they dysregulate iron metabolism through ferritin/ferroportin modulation and hepcidin-driven sequestration. Exogenous toxicants (heavy metals, organic solvents, certain pharmaceuticals) amplify oxidative insults and may precipitate or worsen anaemia. Conversely, a growing body of experimental and clinical research suggests that selected herbal therapeutics—rich in polyphenols, flavonoids, alkaloids, and micronutrients—can mitigate oxidative damage, modulate iron homeostasis, and support erythropoiesis. This review synthesizes mechanistic links between oxidative stress and anaemia, surveys relevant toxicological drivers, evaluates evidence for herbal interventions, and highlights safety concerns and research priorities. We argue that phytomedicines, when standardized and deployed with attention to interactions and patient vulnerability (for example G6PD deficiency or iron-overload disorders), may serve as valuable adjuncts to conventional therapy, especially in resource-limited settings. Robust clinical trials with mechanistic endpoints, quality-assured formulations, and integrated safety monitoring are necessary to translate promise into practice.

**Keywords:** anaemia, oxidative stress, hemolysis, herbal therapeutics, toxicology

## INTRODUCTION

Anaemia, a condition defined by a reduction in haemoglobin concentration or total red blood cell mass, remains one of the most prevalent public health challenges worldwide [1]. It compromises oxygen delivery to tissues, resulting in fatigue, reduced work capacity, impaired cognitive development in children, and increased morbidity and mortality across all age groups [1]. The World Health Organization estimates that nearly two billion people suffer from anaemia globally, with a disproportionate burden in low- and middle-income countries [1]. The condition arises from a wide array of causes, including iron and micronutrient deficiencies, chronic infectious or inflammatory diseases, genetic disorders such as sickle cell disease and thalassaemia, toxic exposures, and bone marrow suppression [2]. While iron deficiency has traditionally been highlighted as the dominant cause, it is increasingly recognized that oxidative stress is a common denominator linking many different aetiologies of anaemia [3]. Oxidative mechanisms affect both the lifespan of circulating erythrocytes and the efficiency of red blood cell production in the bone marrow [4]. Reactive oxygen and nitrogen species compromise cell membranes, alter haemoglobin structure, and disrupt iron metabolism, thereby precipitating haemolysis and ineffective erythropoiesis [5]. Toxicological factors such as heavy metals, pesticides, and oxidant drugs further aggravate this imbalance, creating a vicious cycle of oxidative injury and anaemia.

Herbal medicines, long used in traditional medical systems, are gaining renewed attention in this context. Rich in phytochemicals with antioxidant, anti-inflammatory, and iron-regulating properties, these remedies are used both for prevention and treatment of anaemia in diverse cultural settings. For example, polyphenol-rich extracts can scavenge free radicals, while nutrient-dense plants provide bioavailable iron and vitamins essential for erythropoiesis [6]. However, their safety, efficacy, and interaction with conventional therapies require critical evaluation. Understanding the interplay between oxidative stress, toxicological drivers, and herbal therapeutics is therefore essential for developing integrative strategies against anaemia that are evidence-based, safe, and accessible.

## 2. Oxidative stress: a central mediator in anaemia

Oxidative stress arises when the generation of reactive oxygen species (ROS) and reactive nitrogen species (RNS) exceeds the capacity of endogenous antioxidant defenses such as glutathione, catalase, and superoxide dismutase [7]. Erythrocytes are particularly vulnerable to oxidative damage because they transport high oxygen loads and contain haem iron that can catalyze free radical reactions [8]. Their membranes are also rich in polyunsaturated fatty acids, which are prone to lipid peroxidation [9].

One major mechanism involves direct damage to haem and haemoglobin. Oxidation of haem iron from ferrous ( $\text{Fe}^{2+}$ ) to ferric ( $\text{Fe}^{3+}$ ) forms methaemoglobin, incapable of oxygen transport and rapidly targeted for removal [10]. Persistent oxidative stress promotes denaturation of haemoglobin, Heinz body formation, and premature erythrocyte clearance by the spleen [11]. Lipid peroxidation further compromises membrane integrity, decreasing red cell deformability and increasing fragility, which accelerates intravascular hemolysis [12]. In parallel, oxidative modification of structural proteins such as spectrin and ankyrin disrupts cytoskeletal stability, impairing red cell survival [13]. Beyond circulating erythrocytes, oxidative stress also impairs bone marrow function. Hematopoietic stem and progenitor cells are highly sensitive to ROS, and excess oxidative signals lead to apoptosis, defective differentiation, and reduced responsiveness to erythropoietin [14]. The bone marrow microenvironment suffers collateral damage, further compromising erythropoiesis.

Iron metabolism is tightly regulated by oxidative pathways. ROS influence hepcidin expression and ferroportin function, leading to sequestration of iron in storage sites and limiting availability for haem synthesis [15]. Meanwhile, labile iron pools catalyze Fenton reactions, generating additional radicals and perpetuating redox imbalance [16]. Finally, oxidative stress amplifies inflammatory cascades through activation of NF- $\kappa$ B and inflammasomes [17]. The resulting cytokine storm suppresses erythroid activity and exacerbates functional iron deficiency, as seen in anaemia of chronic disease.

## 3. Toxicological drivers of oxidative anaemia

A range of exogenous agents promote oxidative injury and anaemia:

**Heavy metals.** Lead, cadmium, arsenic, and mercury generate ROS directly or disrupt antioxidant enzymes. Lead inhibits  $\delta$ -aminolevulinic acid dehydratase and ferrochelatase, impairing haem synthesis and producing microcytic anaemia [18]. Cadmium and arsenic induce oxidative damage in erythrocytes and bone marrow [19]. Organic solvents and industrial chemicals. Benzene and related compounds produce marrow toxicity and oxidative metabolites that suppress erythropoiesis and provoke aplastic or hypoplastic anaemia [20].

**Pharmaceutical agents.** Certain antimicrobials (dapsone, primaquine), sulfonamides, and nitrofurantoin provoke oxidative haemolysis, particularly in individuals with glucose-6-phosphate dehydrogenase (G6PD) deficiency [21]. Chemotherapeutic agents and high-dose NSAIDs can impair marrow function [22].

**Pesticides and herbicides.** Organophosphates and paraquat produce systemic oxidative stress; paraquat in particular causes widespread ROS generation with multi-organ injury and can contribute to haemolytic processes [23]. Mycotoxins and natural plant toxins. Some plant-derived toxins induce oxidative hemolysis or bone marrow suppression [24]. Environmental and lifestyle factors. Chronic alcohol, smoking, and pollutants augment oxidative burden and expedite red cell turnover [25].

**Recognizing these toxicants is vital in both prevention and management.** Exposure reduction, chelation for metal toxicity, and targeted supportive care (transfusion, erythropoietic agents) remain mainstays; adjunctive antioxidant strategies are under investigation.

## 4. Herbal therapeutics: mechanisms relevant to anaemia and oxidative stress

Herbal medicines may mitigate oxidative anaemia through several mechanisms: Direct antioxidant activity. Polyphenols, flavonoids, and tannins scavenge free radicals and reduce lipid peroxidation in erythrocytes, preserving membrane integrity [26]. Enhancement of endogenous defenses. Activation of transcription factors such as Nrf2 upregulates glutathione synthesis and antioxidant enzymes, improving cellular resilience [27].

Iron modulation. Some herbs facilitate iron absorption or mobilize stores; others chelate labile iron and reduce Fenton-driven ROS [28]. The net effect depends on constituent chemistry and dosing. Support of erythropoiesis. Certain botanicals stimulate erythroid progenitors or enhance erythropoietin signaling indirectly via anti-inflammatory actions or improved nutritional status. Anti-inflammatory effects [29]. By suppressing cytokine-mediated erythropoiesis inhibition, herbs may counteract anaemia of chronic disease. Mitochondrial protection. Phytochemicals that preserve mitochondrial function support the energy needs of erythroid precursors and red cell maintenance [30].

### Representative classes and agents:

**Curcuminoids.** Curcumin exhibits radical-scavenging activity, upregulates Nrf2-dependent genes, and reduces inflammatory cytokines that suppress erythropoiesis [31]. Experimental models show reduced hemolysis and improved marrow parameters. **Flavonoids.** Quercetin and rutin protect erythrocytes from oxidative damage and have been shown to stabilize membranes and reduce lipid peroxidation [32]. **Silymarin.** Silymarin's antioxidant and

membrane-stabilizing effects support red cell survival in toxin-induced hemolysis; it also improves hepatic function, indirectly benefiting iron metabolism [33]. Polysaccharide-rich herbs. Certain seaweeds and mushrooms provide micronutrients and prebiotic effects that enhance iron bioavailability and general nutritional status [34]. Iron-containing botanicals. *Moringa oleifera* and other greens supply bioavailable iron and vitamin C, which together facilitate absorption; however, quantification is essential to avoid iron overload [35]. Erythropoiesis-supporting mixtures. Traditional multi-herb formulas often combine nutrient-rich plants with antioxidant species to address both deficiency and oxidative mechanisms.

## 6. Safety considerations and toxicological risks of herbal use

Herbal remedies are not inherently benign and may pose risks particularly relevant to anaemia and oxidative stress: Pro-oxidant effects at high doses. Some polyphenols exhibit pro-oxidant properties in the presence of transition metals, potentially worsening oxidative haemolysis if not dosed appropriately. Iron absorption interference [36]. Tea, green tea extracts, and certain polyphenol-rich herbs inhibit non-heme iron absorption and can exacerbate iron-deficiency anaemia if consumed with meals [37]. G6PD deficiency and hemolytic risk. Agents such as fava bean derivatives and other aromatic compounds may precipitate acute hemolysis in individuals with enzymatic deficiencies. Contamination and adulteration [38]. Heavy-metal contamination, misidentified plant species, or addition of pharmaceuticals (e.g., iron salts or erythropoiesis-stimulating agents) can cause harm or mask true efficacy. Herb-drug interactions [39]. Phytochemicals modulate cytochrome P450 enzymes and transporters, altering pharmacokinetics of drugs used in anaemia management (e.g., immunosuppressants after transplant) or other comorbid medications [40]. Allergic and idiosyncratic reactions. Immunoallergic hemolytic anaemia has been reported with certain botanicals in rare cases.

These safety signals motivate careful screening (including G6PD testing in at-risk populations), use of quality-assured products, counseling on timing relative to iron-rich meals, and active adverse-event monitoring.

## CONCLUSION

Oxidative stress is a pivotal mechanism linking diverse causes of anaemia through hemolysis, impaired erythropoiesis, and iron dysregulation. Toxicological exposures amplify these processes, necessitating prevention and targeted interventions. Herbal therapeutics offer multi-modal mechanisms-antioxidant action, enhancement of endogenous defenses, iron modulation, and support for erythropoiesis-that can complement conventional care, particularly in settings where access to standard therapies is limited. However, safety considerations, quality assurance, and rigorous clinical validation are essential before broad adoption. With methodical translational research and prudent clinical practice, phytochemicals may become valuable adjuncts in the integrated management of anaemia driven or complicated by oxidative stress.

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