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# Antioxidant Therapeutics as a Dual Strategy Against Anaemia and Oxidative Stress in Metabolic Disorders

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## ABSTRACT

Anaemia and oxidative stress are frequently comorbid in metabolic disorders such as type 2 diabetes mellitus, obesity, metabolic syndrome, and chronic kidney disease. The two processes are bi-directionally linked: oxidative stress perturbs iron handling, erythropoiesis, and red blood cell (RBC) lifespan, while anaemia exacerbates tissue hypoxia and generates further reactive oxygen and nitrogen species (ROS/RNS). This interplay contributes to inflammation, organ dysfunction, reduced exercise capacity, and worsened clinical outcomes. Antioxidant therapeutics-ranging from endogenous pathway activators and mitochondria-targeted compounds to nutraceuticals and adjunctive agents used with iron or erythropoiesis-stimulating therapies-offer a promising dual strategy to simultaneously mitigate oxidative damage and improve hematologic status. This review synthesizes current mechanistic understanding of the anaemia-oxidative stress axis in metabolic disease, evaluates classes of antioxidant interventions with preclinical and clinical evidence, discusses biomarker-guided use and safety considerations, and outlines research priorities to translate antioxidant strategies into improved patient outcomes.

**Keywords:** Anaemia, oxidative stress, metabolic disorders, antioxidants, erythropoiesis

## INTRODUCTION

Metabolic disorders encompass a spectrum of conditions-including type 2 diabetes mellitus, obesity, metabolic syndrome, and chronic kidney disease, characterized by chronic low-grade inflammation, insulin resistance, lipid dysregulation, and progressive organ impairment [1-5]. These disorders not only disrupt glucose and lipid metabolism but also influence hematologic homeostasis, leading to a high prevalence of anaemia [6-9]. Anaemia in metabolic diseases is often under-recognized, yet it significantly contributes to fatigue, reduced exercise capacity, cognitive decline, and worsened cardiovascular and renal outcomes. Its etiology is multifactorial, arising from inflammation-driven iron sequestration, diminished erythropoietin (EPO) synthesis, impaired erythroid progenitor function, nutrient deficiencies (iron, vitamin $B_{12}$, folate), and shortened red blood cell (RBC) lifespan due to oxidative and inflammatory injury [10]. Parallel to these hematologic changes, oxidative stress plays a central pathogenic role in metabolic dysfunction. Oxidative stress results from an imbalance between reactive oxygen and nitrogen species (ROS/RNS) and the body's antioxidant defenses, leading to damage of lipids, proteins, and nucleic acids [11-14]. In metabolic disorders, excess ROS arises from hyperglycaemia, mitochondrial overload, advanced glycation end products (AGEs), and chronic inflammation [15-17]. The resulting oxidative environment exacerbates insulin resistance, endothelial dysfunction, and cellular apoptosis-core events driving disease progression. Importantly, anaemia and oxidative stress are tightly interlinked and mutually reinforcing. Oxidative stress impairs erythropoiesis and damages RBCs, while anaemia-induced hypoxia promotes further ROS generation through mitochondrial and enzymatic pathways [18-21]. Both processes share common molecular drivers, including chronic inflammation, mitochondrial dysfunction, iron dysregulation, and impaired antioxidant defense systems [22-26]. This bidirectional relationship contributes to a self-perpetuating cycle of tissue injury, hypoxia, and metabolic deterioration. Therapeutically, this interconnection provides an opportunity for integrated management strategies. Interventions that target oxidative stress may not only alleviate redox imbalance but also improve erythropoietic function and iron bioavailability. Antioxidant therapeutics-encompassing pharmacologic agents, natural compounds, and lifestyle measures, have emerged as potential dual-action interventions capable of addressing both anaemia and oxidative stress in metabolic diseases [27-32]. Understanding the mechanistic underpinnings of this relationship is therefore essential for developing comprehensive and effective therapies. This review explores the molecular and

physiological links between oxidative stress and anaemia in metabolic disorders, evaluates emerging antioxidant therapeutic strategies, and discusses their translational potential. By examining both preclinical and clinical evidence, the paper highlights how redox modulation could enhance erythropoiesis, protect RBC integrity, and mitigate systemic oxidative injury. Ultimately, such approaches may improve metabolic homeostasis and patient outcomes through the dual targeting of oxidative stress and hematologic dysfunction [33-37].

**2. Pathophysiologic Links Between Anaemia and Oxidative Stress**

The interaction between oxidative stress and anaemia in metabolic disorders involves multiple, overlapping mechanisms that impair erythropoiesis, disrupt iron handling, and damage circulating erythrocytes.

Iron Dysregulation and Hepcidin: Chronic inflammation, a hallmark of metabolic disease, induces hepatic production of hepcidine peptide that binds and degrades the iron exporter ferroportin [38-43]. This leads to sequestration of iron within macrophages and enterocytes, restricting its availability for hemoglobin synthesis. The resultant functional iron deficiency causes anaemia of chronic disease while simultaneously promoting ROS formation via Fenton reactions from labile intracellular iron [43-46]. The generated hydroxyl radicals inflict oxidative injury on erythroid precursors and mature RBCs, further compounding anaemia [47-50].

Erythropoietic Impairment: Excessive ROS disrupts bone marrow microenvironments by damaging stromal and progenitor cells [51-53]. Key transcription factors involved in erythroid differentiation, such as GATA-1 and NF-E2, are sensitive to redox status, and their dysfunction under oxidative conditions impairs EPO responsiveness [54-57]. Consequently, erythroid progenitor proliferation and maturation decline, reducing red cell output. Decreased RBC Lifespan: RBC membranes are rich in polyunsaturated fatty acids, making them particularly vulnerable to lipid peroxidation [59]. Oxidative modification of membrane proteins such as spectrin and band 3 alters RBC deformability and increases susceptibility to splenic clearance [60-63]. Thus, RBC turnover accelerates even when erythropoiesis remains adequate. Mitochondrial Dysfunction: In metabolic disorders, mitochondrial overproduction of superoxide and hydrogen peroxide drives systemic oxidative stress [64-69]. Within erythroid precursors, defective mitochondrial function impairs heme biosynthesis and ATP production, leading to ineffective erythropoiesis and anemia [70-74]. Inflammation and Nitrosative Stress: Pro-inflammatory cytokines (e.g., IL-6, TNF-α) induce inducible nitric oxide synthase (iNOS), generating RNS that nitrosylate proteins involved in iron metabolism and erythropoietin signaling [75-78]. This nitrosative damage further compromises erythropoietic processes. Collectively, these mechanisms form a vicious cycle in which anaemia-induced hypoxia amplifies oxidative stress via xanthine oxidase activation and mitochondrial uncoupling-while oxidative stress perpetuates anaemia through iron mismanagement, progenitor toxicity, and RBC damage. Understanding this interplay forms the foundation for antioxidant-based therapeutic interventions in metabolic disorders.

**3. Rationale for Antioxidant Therapeutics**

Oxidative stress serves as both a cause and a consequence of anaemia in metabolic disorders, forming a reinforcing cycle that worsens tissue hypoxia, inflammation, and metabolic dysfunction [79-81]. Therapeutic approaches that address oxidative imbalance can potentially disrupt this cycle and restore normal erythropoietic and metabolic function. Antioxidants hold promise as dual-acting agents that not only mitigate redox-driven cellular injury but also enhance hematologic recovery [80-83].

The rationale for antioxidant intervention rests on several interconnected mechanisms. First, antioxidants can directly lower ROS- and RNS-mediated damage to erythroid progenitors and red blood cell (RBC) membranes, thereby improving both RBC production and survival [19]. By stabilizing cellular membranes and preventing lipid peroxidation, antioxidants help maintain RBC deformability and reduce premature destruction in the spleen. Second, antioxidants preserve mitochondrial function—a critical determinant of heme biosynthesis and ATP generation necessary for erythropoiesis [20]. Enhanced mitochondrial efficiency supports the differentiation of erythroid precursors and limits the formation of dysfunctional, short-lived RBCs.

Furthermore, antioxidant compounds can modulate inflammatory signaling pathways that elevate hepcidin, the master regulator of systemic iron homeostasis. By attenuating pro-inflammatory cytokine activity, antioxidants may decrease hepcidin expression, thereby improving intestinal iron absorption and mobilization from macrophage stores [21]. This mechanism directly counteracts the functional iron deficiency seen in anaemia of chronic disease.

Another rationale lies in the ability of antioxidants to complement established therapies such as iron supplementation and erythropoiesis-stimulating agents (ESAs) [22]. By reducing oxidative side effects associated with iron therapy and improving responsiveness to erythropoietin, antioxidants can enhance treatment efficacy and reduce required dosages [23]. Collectively, these effects position antioxidant therapy as a promising adjunct or, in some cases, a primary modality in managing anaemia within metabolic disorders.

**4. Classes of Antioxidant Interventions**

**4.1 Endogenous Pathway Activators (Nrf2 and Related Targets)**

Activation of endogenous antioxidant systems represents a central therapeutic strategy. The nuclear factor erythroid 2-related factor 2 (Nrf2) pathway regulates genes encoding detoxifying enzymes and antioxidant proteins such as glutathione peroxidase, heme oxygenase-1, and NAD(P)H quinone oxidoreductase [24]. Phytochemicals,

including sulforaphane, curcumin, and resveratrol, are natural Nrf2 activators that enhance cellular resilience against oxidative injury [25]. In erythroid tissues, Nrf2 activation promotes redox balance, protects progenitor cells from apoptosis, and suppresses inflammation-driven hepcidin elevation [26]. This dual action supports both erythropoiesis and iron mobilization.

**4.2 Thiol Donors and Glutathione Precursors**

N-acetylcysteine (NAC) and similar thiol-based agents act as glutathione precursors, replenishing intracellular antioxidant pools. By restoring reduced glutathione (GSH) levels, these agents improve redox buffering and neutralize free radicals [26]. In experimental models of anaemia and oxidative stress, NAC reduces hemolysis, stabilizes RBC membranes, and enhances mitochondrial function [27]. Early clinical studies suggest potential benefits in metabolic and inflammatory diseases, though large-scale trials are still needed to confirm their therapeutic impact [28].

**4.3 Mitochondria-Targeted Antioxidants**

Because mitochondria are major sources of ROS generation, targeted delivery of antioxidants to these organelles offers a direct means of redox control. Mitochondria-targeted antioxidants, such as mitoQ, mitoTEMPO, and SS-31 peptides, accumulate in mitochondrial membranes via lipophilic cations [29]. These compounds reduce superoxide formation, preserve respiratory chain integrity, and support heme synthesis. Improved mitochondrial function enhances erythroid cell metabolism and may alleviate anaemia associated with metabolic dysfunction or mitochondrial disease.

**4.4 Polyphenols and Nutraceuticals**

Naturally occurring polyphenols-including resveratrol, quercetin, epigallocatechin gallate (EGCG), and curcumin-have demonstrated strong antioxidant, anti-inflammatory, and iron-modulatory properties [30]. They scavenge ROS, inhibit pro-oxidant enzymes, and suppress hepcidin synthesis, thereby facilitating iron release and utilization. Polyphenols also enhance endothelial and mitochondrial health, providing systemic benefits beyond erythropoiesis [31]. Despite promising preclinical data, variability in bioavailability and dosage standardization remains a limitation for consistent clinical outcomes.

**4.5 Iron Formulations with Redox-Sparing Properties**

Traditional iron therapy can transiently increase oxidative stress due to the catalytic nature of free iron in Fenton chemistry [32]. Novel formulations, such as ferric carboxymaltose, iron isomaltoside, and liposomal iron, aim to minimize labile iron release while maintaining efficient iron delivery [33]. When combined with antioxidants like vitamin C in controlled doses, these formulations improve iron utilization while preventing excessive ROS formation [34]. Such redox-sparing strategies may enhance treatment safety and efficacy in anaemic patients with metabolic disease.

**4.6 Combination with Erythropoiesis-Stimulating Agents (ESA)**

Erythropoiesis-stimulating agents (e.g., recombinant EPO) are widely used in anaemia management, particularly in chronic kidney disease. However, oxidative and inflammatory stress can blunt ESA responsiveness [35]. Antioxidants may protect erythroid precursors from ROS-mediated apoptosis and restore sensitivity to EPO signaling [36]. This synergy allows for lower ESA dosages, reducing associated risks such as hypertension and thromboembolic events. In summary, antioxidant therapeutics encompass diverse molecular classes that target oxidative stress at multiple levels-mitochondrial, cytosolic, and systemic. By restoring redox balance, preserving iron metabolism, and supporting erythroid cell integrity, these agents hold significant potential for mitigating both anaemia and oxidative injury in metabolic disorders.

## CONCLUSION

Anaemia and oxidative stress are entwined contributors to morbidity in metabolic disorders. Antioxidant therapeutics-applied thoughtfully and guided by biomarkers-offer a compelling dual strategy to reduce oxidative injury and support erythropoiesis. While preclinical evidence is strong, translation into routine clinical practice requires rigorously designed trials, precision in patient selection, and attention to safety. Integrating antioxidant approaches with established treatments for iron deficiency and inflammation has the potential to improve hematologic outcomes and overall patient health in metabolic disease.

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