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RESEARCH INVENTION JOURNAL OF BIOLOGICAL AND APPLIED SCIENCES 6(1):8-12, 2026

©RIJBAS Publications ISSN: 1597-2879

https://doi.org/10.59298/RIJBAS/2026/61812

Hepatic and Renal Complications in Metabolic Disorders: The Role of Oxidative Stress in Diabetes-Induced Liver Dysfunction and Kidney Nephropathy

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ABSTRACT

Metabolic disorders such as diabetes mellitus are frequently associated with serious hepatic and renal complications that contribute significantly to morbidity and mortality. The underlying mechanisms involve complex interactions among hyperglycemia, dyslipidemia, inflammation, and oxidative stress. Oxidative stress, characterized by an imbalance between reactive oxygen species (ROS) production and antioxidant defenses, plays a central role in the pathogenesis of diabetes-induced liver dysfunction and diabetic nephropathy. Excessive ROS disrupts cellular macromolecules, alters mitochondrial integrity, and activates pro-inflammatory and fibrotic signaling cascades in both hepatic and renal tissues. This review explores the molecular and biochemical basis of oxidative stress in the progression of hepatic and renal complications in diabetes, emphasizing the interplay between metabolic disturbances, redox imbalance, and cellular injury. It further discusses potential therapeutic strategies targeting oxidative stress, including antioxidant supplementation, Nrf2 activation, mitochondrial protection, and the use of bioactive phytochemicals. Understanding these mechanisms provides a foundation for developing targeted interventions to prevent or mitigate organ damage in metabolic diseases.

Keywords: Diabetes mellitus, oxidative stress, liver dysfunction, diabetic nephropathy, antioxidant therapy

INTRODUCTION

Metabolic disorders are among the leading causes of chronic organ dysfunction globally, with diabetes mellitus standing out as a major contributor to hepatic and renal complications. Diabetes is characterized by chronic hyperglycemia resulting from impaired insulin secretion, insulin action, or both [1]. Prolonged exposure to elevated glucose levels initiates a series of biochemical events, including enhanced oxidative stress, low-grade inflammation, and advanced glycation end-product (AGE) formation [2]. These processes collectively disrupt normal cellular metabolism, contributing to tissue injury in vital organs such as the liver and kidneys. The liver and kidneys play pivotal roles in metabolic homeostasis. The liver regulates carbohydrate, lipid, and protein metabolism, while the kidneys maintain electrolyte balance, remove metabolic waste, and regulate blood pressure [3]. In diabetes, sustained oxidative stress impairs both hepatic and renal functions through mitochondrial dysfunction, lipid peroxidation, and activation of pro-fibrotic pathways [4]. The resultant complications, including non-alcoholic fatty liver disease (NAFLD) and diabetic nephropathy (DN), significantly increase the risk of cardiovascular disease and end-stage organ failure. This review presents an integrated overview of how oxidative stress drives diabetes-induced liver dysfunction and kidney nephropathy. It further examines molecular mechanisms, pathological manifestations, and emerging therapeutic strategies aimed at ameliorating oxidative damage.

2. Oxidative Stress: Mechanistic Overview

Oxidative stress arises when the production of reactive oxygen species exceeds the capacity of the antioxidant defense system. Under physiological conditions, ROS such as superoxide anion, hydrogen peroxide, and hydroxyl radicals serve as signaling molecules in metabolic and immune processes [5]. However, in pathological states like diabetes, ROS levels increase excessively due to hyperglycemia, mitochondrial overload, and activation of NADPH oxidase and xanthine oxidase pathways [6]. Antioxidant defense mechanisms include enzymatic systems-superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx)-and non-enzymatic molecules like

glutathione, vitamin C, and vitamin E [7]. In diabetes, these antioxidants become depleted, leading to oxidative modification of proteins, lipids, and DNA. ROS-mediated damage initiates cellular dysfunction, apoptosis, and necrosis [8]. Moreover, oxidative stress activates transcription factors such as nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) and nuclear factor erythroid 2–related factor 2 (Nrf2) [9]. NF-κB promotes inflammatory cytokine release, while Nrf2 regulates antioxidant gene expression. An imbalance between these pathways contributes to chronic inflammation and tissue fibrosis in metabolic organs [10].

3. Diabetes-Induced Hepatic Dysfunction

3.1 Pathophysiology

The liver is a central metabolic organ and a major target of diabetic complications. Chronic hyperglycemia leads to excessive glucose flux through glycolytic and lipogenic pathways, resulting in hepatic steatosis, mitochondrial dysfunction, and oxidative injury [11]. Increased free fatty acid (FFA) delivery from insulin-resistant adipose tissue further exacerbates lipid accumulation in hepatocytes [12]. Oxidative stress amplifies this injury by promoting lipid peroxidation, generating malondialdehyde (MDA) and 4-hydroxynonenal (4-HNE), which modify proteins and impair enzyme function [13]. These lipid peroxidation products also activate Kupffer cells, leading to inflammatory cytokine release such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6). The result is a progressive transition from simple steatosis to non-alcoholic steatohepatitis (NASH), fibrosis, and even cirrhosis [14].

3.2 Role of Mitochondrial Dysfunction

Mitochondria are the primary sites of ROS generation during oxidative phosphorylation. In diabetes, excessive substrate availability leads to mitochondrial overload, electron leakage, and ROS overproduction [15]. This causes mitochondrial DNA (mtDNA) damage, decreased ATP synthesis, and apoptosis of hepatocytes. Impaired mitochondrial biogenesis due to reduced peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC-1α) expression worsens energy imbalance and hepatic injury [16].

3.3 Inflammatory and Fibrotic Responses

Oxidative stress activates NF-κB and c-Jun N-terminal kinase (JNK) pathways, promoting inflammatory cytokine expression and hepatocellular apoptosis [17]. Additionally, hepatic stellate cells are activated through oxidative and inflammatory cues, leading to collagen deposition and fibrosis [18]. Transforming growth factor-beta (TGF-β) and connective tissue growth factor (CTGF) act as major fibrogenic mediators in this process [19].

3.4 Clinical Implications

Clinically, diabetes-associated hepatic dysfunction manifests as elevated liver enzymes, steatosis, and altered lipid profiles [20]. Non-alcoholic fatty liver disease (NAFLD) is now recognized as the hepatic component of metabolic syndrome and affects a significant proportion of diabetic individuals [21]. Advanced stages, including NASH and fibrosis, are closely linked to cardiovascular mortality [22].

4. Oxidative Stress and Diabetic Nephropathy

4.1 Overview of Diabetic Nephropathy

Diabetic nephropathy is a leading cause of end-stage renal disease worldwide. It is characterized by glomerular hypertrophy, mesangial expansion, basement membrane thickening, and progressive proteinuria [23]. Hyperglycemia-induced oxidative stress plays a central role in the initiation and progression of nephropathy [24].

4.2 Molecular Mechanisms

High glucose levels increase ROS production through multiple pathways. Activation of the polyol pathway consumes NADPH, depleting glutathione and impairing antioxidant defense [25]. The formation of AGEs and subsequent activation of the receptor for AGEs (RAGE) triggers NF-κB signaling, resulting in increased cytokine and adhesion molecule expression. Protein kinase C (PKC) activation enhances NADPH oxidase activity, further elevating ROS levels [26]. Mitochondrial dysfunction is another critical factor. Excessive glucose flux increases mitochondrial superoxide production, damaging glomerular endothelial and mesangial cells [27]. Oxidative DNA damage and impaired mitophagy contribute to apoptosis and glomerulosclerosis.

4.3 Inflammatory and Fibrotic Mediators

ROS and pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6 promote infiltration of immune cells into the renal interstitium. This inflammatory milieu activates TGF-β/Smad signaling, driving extracellular matrix deposition and fibrosis [27]. Persistent oxidative stress also upregulates hypoxia-inducible factor-1α (HIF-1α), which enhances fibrogenic gene expression and tubular injury [29].

4.4 Biomarkers and Clinical Manifestations

Markers of oxidative stress, such as MDA, 8-hydroxydeoxyguanosine (8-OHdG), and oxidized low-density lipoprotein (ox-LDL), are elevated in diabetic nephropathy. Clinically, the disease progresses from microalbuminuria to macroalbuminuria and eventually to renal failure [29]. Structural damage includes thickened glomerular basement membranes and loss of podocytes, which compromise filtration capacity.

5. Interconnection Between Hepatic and Renal Complications

The liver and kidneys share reciprocal metabolic and hemodynamic relationships. Oxidative stress originating in one organ can exacerbate dysfunction in the other through systemic inflammatory mediators and circulating ROS. For example, hepatic steatosis and inflammation increase the production of inflammatory cytokines that may impair renal endothelial function [30]. Conversely, reduced renal clearance of metabolic by-products enhances hepatic oxidative stress and lipid dysregulation [31]. This bidirectional interaction is often referred to as the hepatorenal axis. In diabetes, hyperglycemia, dyslipidemia, and insulin resistance disrupt this axis, amplifying redox imbalance and tissue injury [32]. Additionally, shared molecular pathways-such as activation of NF-κB, suppression of Nrf2, and mitochondrial dysfunction-underline the close mechanistic link between diabetic liver and kidney diseases [33].

6. Therapeutic Strategies Targeting Oxidative Stress

6.1 Antioxidant Supplementation

Therapeutic approaches aimed at restoring redox balance have gained increasing attention. Supplementation with antioxidants such as vitamins C and E, alpha-lipoic acid, and coenzyme Q10 has been shown to reduce oxidative damage in diabetic patients [34]. These compounds scavenge free radicals, enhance glutathione levels, and improve endothelial function.

6.2 Nrf2 Activation

The Nrf2 pathway regulates the expression of antioxidant and detoxification enzymes, including heme oxygenase-1 (HO-1) and NAD(P)H quinone oxidoreductase 1 (NQO1) [35]. Pharmacological activation of Nrf2 using agents like bardoxolone methyl, sulforaphane, and curcumin enhances cellular antioxidant capacity and suppresses inflammation [36]. Experimental evidence indicates that Nrf2 activation mitigates hepatic steatosis and attenuates renal fibrosis in diabetic models [37].

6.3 Inhibition of NF-κB and Pro-Inflammatory Pathways

Since oxidative stress and inflammation are interdependent, targeting NF-κB signaling is another therapeutic strategy. Natural compounds such as resveratrol, quercetin, and berberine have been shown to inhibit NF-κB translocation and reduce cytokine release [38]. Synthetic inhibitors and small molecules that modulate NF-κB activity are also under investigation for diabetic complications.

6.4 Mitochondrial Protection

Protecting mitochondrial function is essential for reducing oxidative stress in both liver and kidney tissues. Mitochondria-targeted antioxidants such as MitoQ and SS-31 peptides have demonstrated potential in restoring mitochondrial membrane potential, reducing ROS leakage, and improving energy metabolism [39]. Enhancing mitochondrial biogenesis through PGC-1α activation also supports cellular resilience against oxidative damage [40].

6.5 Phytochemicals and Herbal Interventions

Several plant-derived compounds possess strong antioxidant and anti-inflammatory properties. Curcumin, silymarin, and catechins not only scavenge ROS but also modulate signaling pathways involved in fibrosis and apoptosis [41]. Polyphenolic compounds activate Nrf2, inhibit TGF-β signaling, and reduce lipid peroxidation in both hepatic and renal tissues [42]. Laboratory studies suggest that dietary phytochemicals can complement conventional diabetes management by protecting vital organs [43].

CONCLUSION

Oxidative stress is a central mediator in the development of hepatic and renal complications associated with diabetes mellitus. In the liver, excessive ROS disrupts mitochondrial function, induces lipid peroxidation, and activates inflammatory and fibrotic pathways, leading to steatosis and fibrosis. In the kidneys, oxidative stress promotes glomerular and tubular injury through mitochondrial damage, cytokine release, and extracellular matrix accumulation. These processes are closely interconnected, forming a vicious cycle that exacerbates multi-organ dysfunction. Therapeutic strategies that restore redox balance, inhibit inflammatory signaling, and protect mitochondrial integrity have shown encouraging results in preclinical and clinical settings. The combined targeting of oxidative stress and metabolic dysregulation represents a promising approach to managing diabetes-induced organ complications. Understanding the molecular underpinnings of oxidative injury will continue to guide the development of novel interventions aimed at preserving hepatic and renal health in metabolic disorders.

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CITE AS: Twesigye Davis (2026). Hepatic and Renal Complications in Metabolic Disorders: The Role of Oxidative Stress in Diabetes-Induced Liver Dysfunction and Kidney Nephropathy. RESEARCH INVENTION JOURNAL OF BIOLOGICAL AND APPLIED SCIENCES 6(1):8-12. https://doi.org/10.59298/RIJBAS/2026/61812