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Redox-Driven Multimorbidity: Integrating Diabetes, Autoimmunity, BPH, and Hepatic Injury

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ABSTRACT

Multimorbidity-the coexistence of two or more chronic conditions-is a growing global health burden, often driven by shared underlying biology rather than independent disease progression. Among key biological determinants, redox imbalance (or oxidative stress) stands out as a central nexus connecting metabolic dysfunction (e.g., type 2 diabetes), autoimmunity, benign prostatic hyperplasia (BPH), and hepatic injury. Oxidative stress alters cellular signaling, immune regulation, metabolic pathways, and fibrotic responses. This review dissects how dysregulated redox systems contribute to these seemingly distinct conditions, highlighting shared molecular pathways, clinical interrelationships, and potential therapeutic targets. We explore how hyperglycemia and mitochondrial dysfunction propagate reactive oxygen species (ROS) and chronic inflammation, fueling autoimmunity and tissue remodeling. We examine redox-modulated immune dysregulation in autoimmune disease and how chronic systemic oxidative stress influences prostate growth and liver inflammation. Finally, we discuss how common interventions (antioxidants, lifestyle changes, and targeted pharmacotherapies) may attenuate disease progression across this multimorbidity spectrum. Understanding redox-driven mechanisms invites more integrated clinical approaches and rational biomarker-guided therapies.

Keywords: Oxidative stress, Type 2 diabetes mellitus, Autoimmunity, Benign prostatic hyperplasia, Hepatic injury

INTRODUCTION

Multimorbidity describes the simultaneous presence of multiple chronic diseases, a pattern increasingly observed in aging populations and in individuals with metabolic disorders[1]. Conventional medical frameworks often approach these diseases independently; however, accumulating evidence indicates that common biological mechanisms interconnect diverse pathologies. Among these shared mechanisms, redox imbalance which is defined as an excess of pro-oxidant molecules relative to antioxidant defenses has emerged as a central integrative driver of metabolic, immune, and organ-specific dysfunction. Rather than serving merely as a byproduct of disease, oxidative stress actively shapes cellular behavior, gene expression, and inflammatory signaling networks[2]. The generation of reactive oxygen species (ROS) and the resulting oxidative damage influence multiple tissues simultaneously. In diabetes, persistent hyperglycemia enhances mitochondrial ROS production and disrupts insulin signaling. In autoimmune disorders, redox imbalance alters antigen presentation and immune tolerance, promoting chronic inflammation. Within prostatic tissue, oxidative stress contributes to stromal remodeling and epithelial proliferation, while in the liver it sustains inflammation, lipid peroxidation, and fibrogenesis. These shared redox-driven pathways suggest that seemingly distinct disorders may represent interconnected manifestations of systemic oxidative dysregulation. This review integrates mechanistic and clinical insights to

highlight how redox biology underpins multimorbidity across metabolic, immune, prostatic, and hepatic conditions.

2. Redox Biology: A Fundamental Overview

Redox (reduction–oxidation) reactions are fundamental to cellular metabolism, energy generation, and intracellular signaling[3]. Reactive oxygen species, including superoxide anions (O_2^-), hydroxyl radicals (•OH), and hydrogen peroxide (H_2O_2), are generated primarily in mitochondria during oxidative phosphorylation. At physiological levels, ROS function as signaling mediators that regulate proliferation, differentiation, apoptosis, and host defense. Endogenous antioxidant systems-such as superoxide dismutase, catalase, and glutathione peroxidase—maintain redox homeostasis by neutralizing excess ROS[4]. When ROS production overwhelms antioxidant capacity, oxidative stress develops. This imbalance damages lipids, proteins, and DNA, impairing cellular integrity. Furthermore, oxidative stress activates redox-sensitive transcription factors such as NF-κB and Nrf2, linking oxidative cues to inflammatory, metabolic, and fibrotic gene expression programs. Persistent redox dysregulation therefore represents a unifying mechanism in chronic disease pathogenesis.

3. Redox Imbalance in Diabetes

3.1 Hyperglycemia-Induced Oxidative Stress

In type 2 diabetes, persistent hyperglycemia is a primary driver of oxidative stress. Excess intracellular glucose increases flux through the mitochondrial electron transport chain, resulting in overproduction of reactive oxygen species (ROS) due to impaired oxidative phosphorylation and electron leakage[5]. In parallel, alternative glucose metabolic pathways such as the polyol and hexosamine pathways become overactive. These pathways consume nicotinamide adenine dinucleotide phosphate (NADPH), a critical cofactor required for regenerating reduced glutathione, thereby weakening endogenous antioxidant defenses[6]. Non-enzymatic autoxidation of glucose and the formation of advanced glycation end products further amplify free radical generation. The cumulative effect of these processes is oxidative damage to lipids, proteins, and DNA. Vascular endothelial cells are particularly vulnerable, leading to endothelial dysfunction, impaired nitric oxide bioavailability, and microvascular injury. These mechanisms underpin the development of diabetic complications, including nephropathy, retinopathy, and neuropathy, and contribute to accelerated atherosclerosis.

3.2 Redox and Insulin Resistance

Oxidative stress also directly impairs insulin signaling pathways[7]. Reactive oxygen species modify insulin receptor substrates and disrupt downstream signaling cascades essential for glucose uptake. Activation of stress-sensitive serine kinases such as c-Jun N-terminal kinase promotes inhibitory phosphorylation of insulin receptor substrates, attenuating insulin responsiveness[8]. Additionally, lipid peroxidation products accumulate in adipose tissue, exacerbating adipocyte dysfunction and inflammatory cytokine release. Together, these events create a self-perpetuating cycle in which insulin resistance worsens hyperglycemia, further increasing oxidative stress and metabolic dysfunction.

4. Autoimmunity: Redox and Immune Dysregulation

4.1 Redox Control of Immune Tolerance

Physiological levels of ROS are integral to immune signaling and pathogen defense[9]. However, excessive oxidative stress disrupts immune homeostasis by altering antigen presentation and enhancing pro-inflammatory macrophage activation[10]. Redox imbalance influences T-cell differentiation, favoring inflammatory subsets such as Th17 cells over regulatory T cells. This shift undermines immune tolerance and increases susceptibility to autoimmune responses.

4.2 ROS in Autoimmune Disease Pathogenesis

Many autoimmune disorders, including rheumatoid arthritis and systemic lupus erythematosus, exhibit elevated oxidative stress markers[11]. ROS activate inflammasomes and nuclear factor kappa B, amplifying cytokine production and sustaining chronic inflammation. Oxidative damage to cellular components can generate neoantigens, further stimulating autoreactive immune cells and perpetuating tissue injury[12].

4.3 Overlap with Diabetes

Type 1 diabetes represents a prototypical autoimmune disease in which oxidative stress accelerates pancreatic beta-cell destruction[13]. Even in type 2 diabetes, chronic low-grade inflammation and immune activation contribute to disease progression, illustrating the convergence of metabolic and autoimmune mechanisms under a shared redox framework[14].

5. Benign Prostatic Hyperplasia: Redox Influences

Benign prostatic hyperplasia (BPH) is a non-malignant enlargement of the prostate gland that commonly affects aging men and contributes significantly to lower urinary tract symptoms[15]. Increasing evidence indicates that

BPH is not merely a hormonally driven condition but is also shaped by chronic inflammation and oxidative stress. Redox imbalance appears to influence cellular proliferation, stromal remodeling, and inflammatory signaling within prostatic tissue, linking BPH to systemic metabolic and inflammatory states.

5.1 Oxidative Stress in Prostate Tissue

Prostatic tissue is particularly susceptible to oxidative injury due to its high metabolic activity and exposure to inflammatory mediators[16]. Local infiltration of immune cells promotes reactive oxygen species (ROS) production through respiratory burst mechanisms. Age-related hormonal alterations, particularly changes in androgen and estrogen balance, may affect the expression and activity of endogenous antioxidant enzymes such as superoxide dismutase and glutathione peroxidase[17]. Additionally, metabolic dysfunction in adjacent stromal and epithelial cells can impair mitochondrial function, further increasing ROS generation. Elevated levels of lipid peroxidation products and diminished antioxidant capacity have been detected in BPH tissue samples[18]. Oxidative stress may stimulate growth factor signaling pathways, activate redox-sensitive transcription factors, and enhance cytokine production, collectively promoting epithelial proliferation and stromal expansion. Over time, this redox-driven remodeling contributes to gland enlargement and urinary obstruction.

5.2 Shared Pathways with Metabolic Disease

Obesity and metabolic syndrome are recognized risk factors for BPH and are characterized by systemic oxidative stress and chronic low-grade inflammation[19]. Hyperinsulinemia and insulin resistance increase sympathetic nervous system activity and augment the expression of growth-promoting factors within the prostate[20]. These metabolic disturbances interact with ROS-mediated inflammatory pathways, amplifying tissue remodeling and hyperplastic growth. Thus, BPH may represent a local manifestation of broader metabolic and redox dysregulation.

5.3 Redox and Fibroblast Activation

Fibroblasts within the prostatic stroma are highly responsive to oxidative signals[21]. Excess ROS can activate these cells, stimulating extracellular matrix synthesis and deposition. This process enhances stromal thickening and glandular expansion, key pathological features of BPH[22]. Redox-sensitive pathways also modulate transforming growth factor-beta signaling, further promoting fibrotic remodeling.

6. Hepatic Injury: Redox as a Central Driver

The liver functions as a central metabolic organ responsible for detoxification, lipid metabolism, and glucose regulation[23]. Because of its high oxidative capacity and continuous exposure to metabolic substrates, it is especially vulnerable to redox imbalance[24]. Persistent oxidative stress disrupts hepatocellular integrity and promotes inflammation and fibrosis.

6.1 Non-Alcoholic Fatty Liver Disease (NAFLD)

Non-alcoholic fatty liver disease spans a spectrum from simple steatosis to non-alcoholic steatohepatitis and progressive fibrosis[25]. Excess free fatty acids delivered to the liver undergo beta-oxidation, generating substantial amounts of ROS. Mitochondrial dysfunction further amplifies oxidative stress, while lipid peroxidation products initiate inflammatory cascades[26]. These events convert benign fat accumulation into hepatocellular injury, inflammatory infiltration, and fibrogenic activation.

6.2 Immune and Redox Crosstalk in the Liver

Kupffer cells, the resident macrophages of the liver, respond to oxidative stress by releasing pro-inflammatory cytokines and chemokines[27]. ROS also activate hepatic stellate cells, the principal mediators of liver fibrosis, promoting collagen deposition and scar formation. Sustained redox signaling thus drives progression from inflammation to structural liver damage[28].

6.3 Diabetes and Hepatic Oxidative Stress

In diabetes, chronic hyperglycemia and dyslipidemia intensify hepatic ROS production[29]. Impaired antioxidant defenses and mitochondrial stress accelerate NAFLD progression and increase the risk of advanced fibrosis[30]. This interplay underscores the systemic nature of redox-driven multimorbidity linking metabolic and hepatic disease.

7. Therapeutic Implications

If redox imbalance represents a central mechanism underlying multimorbidity, then interventions that restore redox homeostasis may confer benefits across multiple chronic conditions simultaneously[31]. Rather than focusing on single-organ treatment, a redox-centered strategy emphasizes systemic metabolic and inflammatory regulation[32].

7.1 Antioxidant Strategies

Dietary antioxidants such as vitamins C and E, carotenoids, and plant-derived polyphenols may help neutralize excess reactive oxygen species and reduce lipid peroxidation[33]. Diets rich in fruits, vegetables, whole grains, and healthy fats provide a broad spectrum of redox-supportive compounds that work synergistically rather than as isolated supplements. Endogenous antioxidant support, including N-acetylcysteine and other glutathione precursors, enhances intracellular detoxification capacity and improves cellular resilience against oxidative injury[34]. Although large clinical trials of antioxidant supplementation have yielded mixed results, carefully selected use in early metabolic disease, non-alcoholic fatty liver disease, or high oxidative stress states may offer targeted benefit.

7.2 Redox-Sensitive Pathway Modulators

Pharmacologic agents that influence redox-sensitive signaling pathways may exert pleiotropic effects[35]. For example, drugs that inhibit nuclear factor kappa B reduce inflammation, while agents such as metformin improve mitochondrial efficiency and decrease oxidative stress, benefiting metabolic and hepatic outcomes[36].

7.3 Lifestyle and Metabolic Interventions

Regular physical activity enhances mitochondrial function and upregulates endogenous antioxidant enzymes[37]. Effective glycemic control reduces hyperglycemia-driven reactive oxygen species production, and weight management lowers systemic inflammation. Collectively, these lifestyle strategies address the root drivers of redox dysregulation and improve overall health outcomes across interconnected diseases[38].

CONCLUSION

Redox imbalance is a unifying thread linking diabetes, autoimmunity, benign prostatic hyperplasia, and hepatic injury. Understanding these connections reframes multimorbidity not as a collection of isolated diseases but as manifestations of systemic dysregulation. Targeting redox biology through lifestyle, pharmacological, and metabolic strategies could offer broad therapeutic effects, potentially slowing or preventing the progression of multiple chronic conditions simultaneously. Bridging basic science with clinical practice offers a comprehensive framework for future research and care strategies focused on improving outcomes in complex, interconnected disease systems.

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