ISSN: 2579-079X

International Digital Organization for Scientific Research IDOSR JST/26/1130000
IDOSR JOURNAL OF SCIENCE AND TECHNOLOGY 12(1):1-6, 2026.
https://doi.org/10.59298/IDOSR/JST/26/113.160000

Rheumatoid Factor as a Metabolic Biomarker: Emerging Links Between Autoantibody Biology, Insulin Resistance, and Type 2 Diabetes

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ABSTRACT

Rheumatoid factor (RF), traditionally regarded as a serological marker of rheumatoid arthritis (RA), is increasingly recognized for its broader relevance in systemic inflammatory states. Accumulating evidence suggests that RF positivity may reflect underlying metabolic inflammation, oxidative stress, and immune dysregulation—core mechanisms also implicated in insulin resistance and type 2 diabetes mellitus (T2DM). This review synthesizes current knowledge on the emerging role of RF as a potential metabolic biomarker. It examines biochemical mechanisms through which chronic inflammation, redox imbalance, altered B-cell tolerance, adipokine signaling, and post-translational protein modifications may promote RF generation outside autoimmune disease. Furthermore, we discuss epidemiological observations linking RF positivity with obesity, dyslipidemia, hepatic steatosis, and increased risk of T2DM. We also evaluate how RF-associated immune activation may exacerbate metabolic dysfunction through pathways involving TNF-α, IL-6, NF-κB, and oxidative damage. Lastly, the review outlines clinical implications for screening, risk stratification, and personalized therapeutic strategies targeting inflammation-driven metabolic disease. Understanding RF as a metabolic signal rather than a disease-specific autoantibody may expand its utility in the early identification of individuals at risk of insulin resistance and type 2 diabetes mellitus (T2DM).

Keywords: Rheumatoid factor, autoimmunity, insulin resistance, oxidative stress, type 2 diabetes

INTRODUCTION

Rheumatoid factor (RF) has historically been used as a classical biomarker in the clinical diagnosis and monitoring of rheumatoid arthritis [1]. Defined as autoantibodies predominantly IgM, directed against the Fc fragment of IgG, RF reflects a breakdown in immune tolerance and an amplified inflammatory response. While RF remains a cornerstone diagnostic tool in rheumatology, recent studies have demonstrated that RF positivity extends well beyond RA and is also associated with chronic infections, aging, metabolic disorders, and other inflammatory states [2]. This broader distribution suggests that RF may serve as a systemic indicator of immune activation rather than a disease-specific antibody. Increasingly, attention has turned to the possibility that RF may be relevant in metabolic dysfunction, particularly insulin resistance and type 2 diabetes mellitus (T2DM). Several epidemiological studies have reported increased RF titers among individuals with metabolic syndrome, central obesity, and non-alcoholic fatty liver disease (NAFLD), conditions underpinned by chronic low-grade inflammation and oxidative stress [3]. Because these processes are also central drivers of T2DM pathogenesis, the presence of RF may indicate shared mechanistic pathways linking metabolism and immune dysregulation. This review explores the novel hypothesis that RF can serve as a metabolic biomarker, integrating autoantibody biology with chronic inflammation, redox imbalance, and insulin resistance [4]. By summarizing molecular pathways, translational evidence, and clinical implications, we aim to provide a comprehensive understanding of how RF relates to metabolic disease and how this emerging connection may enhance future diagnostic and therapeutic strategies.

2. Biology of Rheumatoid Factor Production

2.1 Immune Tolerance Breakdown

Rheumatoid factor (RF) arises primarily from the activation of autoreactive B cells following the recognition of altered or immunogenic IgG molecules. Under healthy physiological conditions, central and peripheral B-cell tolerance mechanisms ensure that potentially autoreactive clones are deleted, anergized, or regulated [5]. However, in chronic inflammatory settings, these fail-safe systems become compromised. Persistent exposure to

tissue-derived antigens and inflammatory mediators enhances antigen presentation, particularly of structurally modified IgG. In parallel, pattern-recognition receptor signaling, especially through Toll-like receptors (TLRs), is amplified by pathogen-associated molecular patterns (PAMPs) or damage-associated molecular patterns (DAMPs) released during metabolic stress [6]. TLR activation provides powerful co-stimulatory signals that override tolerance mechanisms and license autoreactive B cells to proliferate [7]. Another key contributor is the increased production of B-cell activating factor (BAFF), a cytokine critical for B-cell maturation and survival. Elevated BAFF levels, frequently observed in metabolic syndrome and type 2 diabetes, rescue autoreactive B cells from apoptosis and promote their expansion [8]. Collectively, these immunological disturbances facilitate the emergence of B-cell populations capable of producing RF, extending autoantibody formation beyond classical autoimmune contexts.

2.2 Contribution of Post-Translational Protein Modifications

Post-translational modifications of IgG represent a central mechanism for RF induction [9]. Under oxidative stress, IgG undergoes glycation, carbamylation, oxidation, and other structural alterations that generate neo-epitopes not normally recognized by the immune system. These modified epitopes behave as neo-antigens capable of breaking self-tolerance and triggering RF production [10]. In metabolic disorders such as diabetes, chronic hyperglycaemia accelerates the formation of advanced glycation end-products (AGEs), which accumulate on immunoglobulins and other serum proteins. AGE-modified IgG exhibits altered tertiary structure and reduced stability, increasing its immunogenicity [11]. These biochemical modifications heighten B-cell receptor engagement by modified IgG, thereby promoting class-switch recombination and enhanced IgM RF secretion. The metabolic environment therefore, creates continuous antigenic pressure that fuels ongoing RF production.

2.3 Cytokine Mediators

Cytokines form another major axis regulating RF generation. Elevated circulating concentrations of pro-inflammatory mediators such as TNF-α, IL-6, and IL-1β, which are hallmark features of type 2 diabetes and obesity, support the survival, proliferation, and differentiation of autoreactive B cells [12]. These cytokines activate redox-sensitive transcription factors, notably NF-κB, which drive transcriptional programs favouring plasmablast formation and autoantibody secretion [13]. In addition, IL-6 promotes germinal centre reactions and facilitates the expansion of antibody-producing cells, thereby intensifying RF output. The convergence of metabolic inflammation and cytokine dysregulation thus sustains an immune environment highly permissive to RF development.

3. Chronic Inflammation and Metabolic Dysfunction: A Converging Pathway

3.1 Inflammation as a Hallmark of Insulin Resistance

Insulin resistance is characterized by persistent low-grade inflammation, particularly within adipose tissue, liver, and skeletal muscle. In obesity and T2DM, adipose tissue macrophages shift toward a pro-inflammatory M1 phenotype and release cytokines that interfere with insulin receptor signaling [14]. Adipokine imbalance, marked by reduced adiponectin and elevated leptin and resistin, further amplifies inflammatory activation. These disturbances activate intracellular signaling cascades such as NF-κB, JNK, and STAT3, which phosphorylate insulin receptor substrates on inhibitory sites and blunt downstream insulin action [15]. Importantly, the inflammatory milieu fosters biochemical and structural alterations of IgG, providing an abundant antigenic substrate for RF induction. The overlap between metabolic inflammation and autoantibody production underscores the shared pathogenic mechanisms linking RF and insulin resistance.

3.2 Oxidative Stress and B-Cell Activation

Oxidative stress is a defining feature of metabolic dysfunction and serves as a potent driver of autoantibody formation [16]. Chronic hyperglycaemia, mitochondrial overload, and lipid peroxidation generate high levels of reactive oxygen and nitrogen species, leading to modification of endogenous proteins, including IgG. These oxidative changes produce neo-antigens that stimulate B-cell activation and clonally expand autoreactive populations [17]. Oxidative stress not only increases IgG glycoxidation but also enhances antigen uptake and presentation, strengthens B-cell receptor signaling, and facilitates class switching to IgM RF. As such, the oxidative environment inherent to type 2 diabetes perpetuates a cycle of inflammation, autoantibody production, and metabolic dysfunction [18].

4. Potential Mechanistic Pathways Linking RF and T2DM

The mechanistic links between rheumatoid factor (RF) production and type 2 diabetes mellitus (T2DM) are increasingly understood within the broader context of chronic inflammation, oxidative stress, and dysregulated immune–metabolic crosstalk [19]. RF formation has traditionally been associated with autoimmune conditions, particularly rheumatoid arthritis, but accumulating evidence suggests that metabolic perturbations characteristic of T2DM may create an immunological environment conducive to RF induction [20]. These relationships arise from converging pathways involving inflammatory signaling networks, impaired antioxidant responses, microbiome-driven immune activation, and altered adipokine secretion.

4.1 NF-κB and JNK Activation

Insulin resistance is strongly driven by chronic low-grade inflammation, which activates intracellular signaling pathways such as NF-κB and JNK [21]. These transcription factors are stimulated by oxidative stress, adipose tissue inflammation, and the engagement of Fc receptors by immune complexes. Once activated, NF-κB and JNK promote the transcription of pro-inflammatory cytokines including TNF-α, IL-1β, and IL-6. These cytokines impair insulin receptor signaling by inhibiting IRS-1 phosphorylation and enhancing serine kinase activity [22]. At the same time, the inflammatory environment increases the likelihood of antibody modification, forming neoepitopes capable of triggering RF production. Thus, RF generation reflects both heightened immune activity and ongoing metabolic inflammatory stress.

4.2 Nrf2 Suppression

The antioxidant response mediated by Nrf2 is essential for maintaining redox balance. In T2DM, Nrf2 activation is frequently compromised, resulting in inadequate expression of antioxidant enzymes such as HO-1, NQO1, and SOD [23]. Reduced Nrf2 activity not only aggravates oxidative stress but also facilitates the oxidation or glycation of IgG molecules. Modified IgG can become immunogenic, promoting the formation of RF against these neoantigens [24]. This suggests that Nrf2 dysfunction contributes both to metabolic deterioration and to the immunological events that elevate RF levels.

4.3 Gut Microbiota and Mucosal Immunity

Dysbiosis is increasingly recognized as a driver of metabolic inflammation [25]. Obesity and T2DM are associated with altered gut microbiota composition, leading to increased intestinal permeability and translocation of bacterial components such as lipopolysaccharides (LPS). These microbial signals activate pattern recognition receptors on macrophages, dendritic cells, and B cells, promoting systemic inflammation and autoantibody production [26]. Heightened exposure to microbial antigens can stimulate B-cell class switching and enhance RF generation, providing a plausible link between gut barrier dysfunction and RF positivity in metabolic disease.

4.4 Adipokine Imbalance

Adipose tissue serves as an active endocrine organ, and disruptions in adipokine secretion contribute directly to immune activation [27]. Low adiponectin levels, commonly observed in T2DM, reduce anti-inflammatory signaling, while elevated resistin promotes B-cell activation and increases pro-inflammatory cytokine production [28]. Together, these changes favor an immune milieu that supports RF induction and sustains systemic metabolic inflammation.

5. Rheumatoid Factor as a Predictive and Diagnostic Metabolic Biomarker

RF is increasingly being explored as a nontraditional biomarker for metabolic dysfunction [29]. Its association with systemic inflammation makes it particularly relevant in identifying individuals undergoing early immunometabolic derangements.

5.1 Screening and Early Identification

RF measurement may help detect individuals with subclinical inflammation and early insulin resistance, especially among populations at elevated metabolic risk, including obese individuals, patients with dyslipidemia, middle-aged adults, and those predisposed to non-alcoholic fatty liver disease (NAFLD) [30]. Incorporating RF into early screening panels may enhance the prediction of metabolic deterioration [31].

5.2 Risk Stratification

RF positivity may complement classical biomarkers such as fasting glucose, HbA1c, liver enzymes, and lipid profiles [32]. Elevated RF titers could refine cardiometabolic risk assessment, improve prediction of hepatic steatosis progression, and offer insights into the extent of systemic inflammatory activation beyond what metabolic markers alone can capture [33].

5.3 Monitoring Therapeutic Response

Because RF reflects inflammatory activity, its reduction may serve as an indicator of therapeutic response to anti-inflammatory strategies, including TNF-α or IL-6 inhibitors, antioxidant therapies, structured lifestyle modification, and weight-loss interventions [34]. Tracking RF levels may therefore provide an immunologic dimension to monitoring metabolic improvement [35].

CONCLUSION

Rheumatoid factor, long regarded as a hallmark of rheumatoid arthritis, possesses significant potential as a biomarker of metabolic dysfunction. Evidence increasingly suggests that RF reflects underlying inflammatory and oxidative processes that also drive insulin resistance and type 2 diabetes. Mechanistic overlaps-such as cytokine activation, redox imbalance, IgG modification, immune complex formation, and hepatic inflammation-create a biologically plausible framework linking RF to metabolic disease. Although further research is required, RF may serve as an early indicator of metabolic inflammation, a predictor of diabetes risk, and a tool for personalized medicine approaches targeting chronic inflammatory-metabolic disorders. Recognizing RF as a metabolic as well as autoimmune biomarker could transform its clinical utility and broaden our understanding of the

immunometabolic axis.

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CITE AS: Odile Patrick Thalia (2026). Rheumatoid Factor as a Metabolic Biomarker: Emerging Links Between Autoantibody Biolo Insulin Resistance, and Type 2 Diabetes. IDOSR JOURNAL OF SCIENCE AND TECHNOLOGY 12(1):1-6. https://doi.org/10.59298/IDOSR/JST/26/113.160000