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Esdras Couto and [Sodré GB Neto](#) *

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Article

Integrated Injectable Therapies in the Modulation of Chronic Inflammation in Obesity, Cancer, and Type 2 Diabetes

Esdras de Oliveira Couto ¹ and Sodré Gonçalves de Brito Neto ^{2,*}

¹ Médico da UDABOL/UNIRG

² Researcher at the Center for Human Genetics (CEGH/ICB) of the Federal University of Goiás (UFG)

* Correspondence: clinicaltrialinbrazil@gmail.com

Abstract

Chronic inflammation is a central factor in the pathogenesis of obesity, cancer, and type 2 diabetes (T2DM). This article explores an integrated therapeutic strategy, administered via injection, that combines dimethyl sulfoxide (DMSO), coenzyme Q10 (CoQ10), alpha-lipoic acid (ALA), curcumin, GSH, and miR-146a mimetics. Injectable administration optimizes the bioavailability and tissue targeting of these agents, which act synergistically through anti-inflammatory, antioxidant, and metabolic modulating mechanisms. The main focus is on the role of miR-146a in regulating the IRAK1 and TRAF6 signaling pathway, crucial for immunometabolic homeostasis. This multidimensional approach has the potential to modulate chronic inflammation, optimize mitochondrial function, and restore metabolic balance, representing a new frontier in the treatment of chronic inflammatory diseases.

Keywords: obesity; chronic inflammation; Type 2 diabetes; cancer; injectable therapies; miR-146a; DMSO; CoQ10; ALA; curcumin; glutathione (GSH)

Introduction

Obesity and overweight constitute a global epidemic, with over 1.9 billion adults overweight and 650 million obese, representing a public health challenge [1]. This increase is driven by dietary changes and sedentary lifestyles, resulting in a positive energy balance and adipose tissue accumulation [2,3]. Obesity is a complex neuroimmunoendocrine condition where adipose tissue acts as a metabolically active and immunologically dynamic organ. Adipose expansion leads to macrophage and lymphocyte infiltration and elevated secretion of pro-inflammatory cytokines (TNF- α , IL-6, leptin), with reduced adiponectin, an anti-inflammatory and insulin-sensitizing molecule [4,5]. This dysregulation establishes a state of low-grade chronic inflammation, central to the link between obesity and various chronic diseases [6].

Clinical and experimental evidence correlates excess adiposity with a higher prevalence of non-communicable chronic diseases (NCDs), such as type 2 diabetes mellitus (T2DM), cardiovascular diseases (CVD), neoplasms, and non-alcoholic steatohepatitis (NASH) [7,8]. In the pathophysiology of T2DM and insulin resistance, systemic inflammation mediated by adipose tissue is determinant [9]. Recent research indicates that the inflammation-obesity axis is regulated by epigenetic mechanisms, including inflammatory microRNAs such as miR-146a and miR-155, which modulate the transcription of immunometabolic signaling molecules [10–12]. Thus, obesity is a central factor in the genesis and progression of NCDs, requiring innovative preventive and therapeutic approaches that consider its inflammatory and systemic nature.

Mechanisms in Inflammation

Low-grade chronic inflammation is a crucial mechanistic link in the etiology and progression of non-communicable diseases such as cancer, obesity, and type 2 diabetes (T2DM) [1]. This persistent inflammatory state dysregulates immunometabolic signaling pathways, such as NF- κ B and JNK [2–4], orchestrating the production of pro-inflammatory cytokines (TNF- α , IL-6, IL-1 β). These cytokines contribute to insulin resistance, tumor progression, and adipose tissue dysfunction [1,5].

MicroRNAs (miRNAs) are essential post-transcriptional regulators in the modulation of inflammatory responses. MiR-146a and miR-155 are notable for their opposing roles: miR-146a attenuates excessive inflammatory signaling, while miR-155 promotes pro-inflammatory responses [6–8]. The dysregulation of these miRNAs is common in chronic inflammation and exacerbates conditions such as cancer, obesity, and T2DM [9–11].

Injectables

Conventional therapeutic approaches face limitations such as low bioavailability and rapid degradation. Injectable formulations offer significant advantages, including greater systemic bioavailability, rapid onset of action, precise dose control, and effective tissue targeting [12,13]. This strategy is promising for sensitive molecules, such as miRNA mimetics.

Integrated therapeutic strategies combining dimethyl sulfoxide (DMSO), coenzyme Q10 (CoQ10), alpha-lipoic acid (ALA), curcumin, and miR-146a mimetics in injectable formulations demonstrate anti-inflammatory, antioxidant, and metabolic modulating effects. Evidence suggests the clinical potential of this integrated strategy in multifactorial chronic inflammatory diseases, although translational and clinical validation is necessary.

Molecular Mechanisms and Role of miR-146a in Inflammation Regulation

MiRNAs (19–25 nucleotides) regulate gene expression post-transcriptionally by binding to 3' UTRs of mRNAs, resulting in degradation or translational repression [14]. This mechanism adjusts cellular processes such as immunity, proliferation, and differentiation.

MiR-146a is a negative regulator of innate immune responses, suppressing components of the TLR and IL-1R signaling pathway. It directly represses IRAK1 and TRAF6 [6,15], adaptor proteins that activate the NF- κ B cascade, leading to the expression of pro-inflammatory cytokines (TNF- α , IL-6, IL-1 β) [16]. Thus, miR-146a establishes a negative feedback loop, essential for preventing excessive inflammation and maintaining immune homeostasis [6,17].

MiR-146a deficiency is associated with increased tumor inflammation, favoring growth and metastasis [18]. In obesity and T2DM, reduced miR-146a correlates with exacerbated systemic inflammation, insulin resistance, and metabolic dysfunction [9,10,19]. Reduced serum miR-146a levels are detected in patients with T2DM [20] and are associated with fibrosis and systemic complications [21]. The therapeutic use of miR-146a mimetics (synthetic oligonucleotides) is promising. Preclinical models have shown that mimetics encapsulated in nanoparticles reduced inflammation and fibrosis in diabetes and cancer [22,23]. Injectable formulations are advantageous, protecting the molecule and optimizing its absorption and tissue targeting [12,24].

Justification for Injectable Formulations

The injectable route overcomes oral administration by avoiding gastrointestinal degradation and first-pass metabolism, resulting in greater systemic bioavailability [12,25]. It provides a rapid onset of pharmacological action, essential for immediate control of inflammation [26,27], and allows precise dose adjustment in multimodal therapies [28,29]. For miRNA mimetics, nanocarriers or injectable exosomes protect the molecules and optimize their absorption and tissue targeting [12,24,30,31].

Selected Injectable Agents

Dimethyl Sulfoxide (DMSO)

Dimethyl sulfoxide (DMSO) is an amphipathic solvent that facilitates the bioavailability of co-administered compounds. It possesses intrinsic anti-inflammatory and antioxidant effects, with clinical use in inflammatory and neurological conditions [32–36]. Its multifaceted action makes it valuable in integrated injectable formulations.

Coenzyme Q10 (CoQ10)

Coenzyme Q10 (CoQ10) is a vital lipid-soluble antioxidant for the mitochondrial electron transport chain, essential for ATP synthesis and neutralization of reactive oxygen species (ROS). In models of obesity and T2DM, CoQ10 reduces systemic inflammation and improves metabolic balance [36–39]. Injectable formulations overcome low oral absorption, ensuring effective therapeutic levels [40,41].

Alpha-Lipoic Acid (ALA)

Alpha-lipoic acid (ALA) is a mitochondrial dithiol compound with broad antioxidant action, regenerating endogenous antioxidants and modulating insulin sensitivity. It inhibits inflammatory pathways such as NF- κ B and JNK, protecting pancreatic β -cells and target tissues in T2DM and obesity [42–48]. Injectable administration optimizes its delivery and efficacy.

Curcumin

Curcumin, a polyphenol from **Curcuma longa**, inhibits NF- κ B activation and induces miR-146a expression, conferring anti-inflammatory and anticarcinogenic effects [49–51]. Its low oral bioavailability is overcome by injectable formulations, which enhance its clinical efficacy [52,53].

Glutathione (GSH)

Glutathione (GSH), a tripeptide and the main intracellular antioxidant, is essential for neutralizing reactive oxygen species and maintaining cellular redox balance [54]. Intravenous GSH administration rapidly restores its levels in deficient tissues. Its role is well established, showing a reduction in inflammatory markers [55]. In T2DM, GSH reduces oxidative stress and improves insulinotropic function, but clinical trials are limited [56]. In cancer, it acts to protect normal cells but may protect tumor cells [57]. The safety of IV GSH is generally good in regulated products [58].

Discussion and Future Perspectives

The relationship between chronic inflammation, cancer, obesity, and T2DM is a clinical challenge. The integrated strategy of DMSO, CoQ10, ALA, curcumin, and miR-146a mimetics, administered via injection, offers multimodal therapeutic synergy. Each agent acts on complementary targets: miR-146a inhibits inflammation; CoQ10 and ALA reinforce antioxidant defense; curcumin suppresses NF- κ B and induces miR-146a; and DMSO enhances bioavailability. This integration suggests greater efficacy than monotherapies.

Clinical translation requires controlled and large-scale trials to evaluate safety, efficacy, and dosage. Patient stratification by circulating miRNA profiles can optimize therapeutic personalization [59] and minimize side effects [60]. Advances in release nanotechnology are crucial for improving tissue-targeted delivery, reducing toxicities. Mechanistic investigation of miRNA modulation is fundamental for new therapeutic pathways.

Conclusion

The interrelationship between chronic inflammation, cancer, obesity, and T2DM demands innovative therapeutic strategies. The integrated approach with DMSO, CoQ10, ALA, curcumin, GSH, and miR-146a mimetics, administered via injection, is promising. This strategy acts on key inflammatory pathways, optimizes mitochondrial function, and restores metabolic homeostasis, offering a comprehensive intervention.

The synergistic actions of these agents—potentiation of delivery and anti-inflammatory effects of DMSO, antioxidant properties of CoQ10 and ALA, anti-inflammatory and miR-146a-inducing effects of curcumin, and immunomodulation of miR-146a mimetics—provide a robust mechanistic and clinical basis.

Rigorous translational and clinical validation is imperative to establish safety, efficacy, and optimal application. Future research should focus on well-designed clinical trials, personalized medicine guided by biomarkers (e.g., miRNA profiles), and advances in nanodelivery. This integrated strategy has great potential to revolutionize the management of chronic inflammatory diseases, improving clinical outcomes and the understanding of immunometabolic health.

Supplementary Materials: The following supporting information can be downloaded at the website of this paper posted on Preprints.org.

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