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Nanocarrier-Mediated Modulation of Macrophage Polarization in Obesity-Driven Chronic Inflammation

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ABSTRACT

Obesity is accompanied by a state of chronic low-grade inflammation that is primarily driven by immune cell dysfunction within adipose tissue. Among these immune cells, macrophages play a central role by undergoing phenotypic polarization that dictates inflammatory outcomes. In lean adipose tissue, macrophages predominantly exhibit an anti-inflammatory M2 phenotype, whereas obesity promotes a shift toward the pro-inflammatory M1 phenotype, sustaining tissue inflammation and metabolic dysfunction. Conventional anti-inflammatory therapies lack the specificity required to selectively reprogram macrophage phenotypes within adipose tissue, limiting their clinical efficacy. Nanocarrier-based drug delivery systems have emerged as powerful tools to overcome these limitations by enabling targeted modulation of macrophage polarization. This review critically examines the mechanisms underlying macrophage polarization in obesity-driven inflammation and highlights recent advances in nanocarrier-mediated strategies to reprogram macrophage phenotypes. We discuss the design principles of nanocarriers, therapeutic payloads, targeting strategies, and biological challenges associated with this approach. Finally, we explore emerging opportunities and translational prospects for nanotechnology-enabled immunometabolic therapies aimed at resolving chronic inflammation in obesity.

Keywords: nanocarriers; macrophage polarization; obesity; chronic inflammation; immunometabolism

INTRODUCTION

Obesity has emerged as one of the most pressing global health challenges, with its prevalence continuing to rise across both developed and developing nations[1–4]. Beyond excessive adipose tissue accumulation, obesity is now widely recognized as a chronic inflammatory disease that underlies the development of insulin resistance, type 2 diabetes mellitus, cardiovascular disease, and other metabolic complications. This persistent inflammatory state is largely orchestrated by immune cells residing within adipose tissue, among which macrophages are the most abundant and functionally significant[5, 6].

Adipose tissue macrophages (ATMs) display remarkable plasticity, allowing them to adopt distinct functional phenotypes in response to microenvironmental cues[7–10]. In lean and metabolically healthy individuals, ATMs predominantly exhibit an alternatively activated M2 phenotype, characterized by the secretion of anti-inflammatory cytokines, tissue remodeling functions, and support of insulin sensitivity. In contrast, obesity induces a dramatic expansion of macrophage populations within adipose tissue, accompanied by a phenotypic shift toward the classically activated M1 state[11–13]. M1 macrophages produce high levels of pro-inflammatory mediators such as tumor necrosis factor-α, interleukin-1β, and interleukin-6, which disrupt insulin signaling and perpetuate metabolic dysfunction[14, 15].

The transition from M2 to M1 macrophage dominance in obesity is driven by multiple pathological stimuli, including adipocyte hypertrophy, hypoxia, lipotoxicity, and oxidative stress. Enlarged adipocytes release free fatty acids and chemokines that recruit circulating monocytes and activate inflammatory signaling pathways[16, 17]. These macrophages often localize around dead or dying adipocytes, forming crown-like structures that are hallmark features of inflamed adipose tissue. The sustained presence of M1 macrophages establishes a feed-forward loop of inflammation that extends beyond adipose tissue to affect systemic metabolic homeostasis[18, 19].

Targeting macrophage-driven inflammation has therefore become an attractive therapeutic strategy for obesity-related metabolic diseases. However, conventional pharmacological approaches face significant limitations[20, 21]. Systemic anti-inflammatory drugs lack cellular specificity and may suppress protective immune responses

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in other tissues. Similarly, broad-spectrum immunomodulators often fail to selectively reprogram macrophage phenotypes within adipose depots, resulting in modest efficacy and undesirable side effects[22–24]. These challenges underscore the need for innovative strategies that can precisely modulate macrophage function in a tissue- and cell-specific manner.

Nanotechnology offers transformative opportunities in this context. Nanocarriers can be engineered to selectively accumulate in inflamed adipose tissue and be preferentially internalized by macrophages due to their innate phagocytic activity. By encapsulating anti-inflammatory drugs, nucleic acids, or metabolic regulators, nanocarriers enable targeted reprogramming of macrophage polarization while minimizing off-target effects[25–27]. Advances in material science, surface functionalization, and biomimetic design have further enhanced the ability of nanocarriers to interact with macrophages in a controlled and predictable manner.

Over the past decade, a growing body of preclinical evidence has demonstrated the potential of nanocarrier-mediated modulation of macrophage polarization to attenuate obesity-driven chronic inflammation and improve metabolic outcomes. Nevertheless, significant challenges remain in optimizing nanocarrier design, ensuring safety, and translating these approaches into clinical practice. This review provides a comprehensive overview of macrophage polarization in obesity and examines how nanocarrier-based strategies can be leveraged to restore immune balance and metabolic health.

2. Macrophage Polarization in Obesity-Driven Inflammation

Macrophage polarization refers to the ability of macrophages to adopt diverse functional states in response to environmental signals[28, 29]. The classical M1/M2 paradigm, although simplified, provides a useful framework for understanding macrophage behavior in obesity. M1 macrophages are induced by pro-inflammatory stimuli such as lipopolysaccharides and interferon-γ, leading to activation of nuclear factor-κB and inflammasome pathways. These cells produce high levels of inflammatory cytokines and reactive oxygen species that contribute to insulin resistance and tissue damage[30, 31].

In contrast, M2 macrophages are activated by interleukin-4 and interleukin-13 and are associated with anti-inflammatory functions, extracellular matrix remodeling, and maintenance of tissue homeostasis[32]. In lean adipose tissue, M2 macrophages support adipocyte function and promote insulin sensitivity through the secretion of anti-inflammatory mediators such as interleukin-10 and transforming growth factor-β. Obesity disrupts this balance by favoring M1 polarization, resulting in a pro-inflammatory adipose tissue microenvironment[32].

Several obesity-associated factors drive macrophage polarization toward the M1 phenotype. Elevated levels of free fatty acids activate Toll-like receptor signaling, while hypoxia induces hypoxia-inducible factor-1α–dependent inflammatory gene expression. Adipokine dysregulation, characterized by increased leptin and decreased adiponectin, further skews macrophage function toward inflammation[33]. Crosstalk between macrophages and other immune cells, including T lymphocytes, amplifies these effects and sustains chronic inflammation[33].

Importantly, macrophage polarization in obesity is not binary but exists along a dynamic spectrum of activation states. This plasticity presents both challenges and opportunities for therapeutic intervention[34]. Strategies that promote repolarization of macrophages from an M1-like to an M2-like phenotype, rather than complete macrophage depletion, are increasingly viewed as more physiologically relevant and potentially safer approaches for resolving chronic inflammation[34].

3. Design Principles of Nanocarriers for Macrophage Targeting

Effective nanocarrier-mediated modulation of macrophage polarization requires careful consideration of physicochemical and biological design parameters. Particle size is a critical determinant of biodistribution and cellular uptake[35]. Nanocarriers in the range of 50–300 nm are preferentially internalized by macrophages through phagocytosis and endocytosis, making them well-suited for macrophage-targeted delivery[36].

Surface charge and composition also influence macrophage interactions. Slightly negative or neutral nanoparticles generally exhibit prolonged circulation times, whereas positively charged particles may enhance cellular uptake but increase nonspecific interactions and toxicity. Biodegradable materials such as poly(lactic-co-glycolic acid), chitosan, and lipid-based formulations are commonly employed to ensure biocompatibility and controlled release of therapeutic payloads[35].

Surface functionalization strategies further enhance macrophage specificity. Nanocarriers can be decorated with ligands that recognize scavenger receptors, mannose receptors, or folate receptors expressed on macrophages. Alternatively, biomimetic approaches, including coating nanoparticles with macrophage or leukocyte cell membranes, enable immune evasion and homing to inflamed tissues[37, 38].

Stimuli-responsive nanocarriers represent an additional layer of sophistication. These systems release their cargo in response to local cues such as acidic pH, elevated reactive oxygen species, or inflammatory enzymes present in obese adipose tissue. Such designs enhance therapeutic precision and reduce off-target effects, making them particularly attractive for immunometabolic applications[39].

4. Nanocarrier-Based Strategies to Reprogram Macrophage Polarization

Nanocarriers have been extensively explored for delivering anti-inflammatory agents that suppress M1 polarization or promote M2 reprogramming[40]. Encapsulation of small-molecule drugs, including

corticosteroids and nuclear receptor agonists, enhances their accumulation within macrophages and reduces systemic toxicity. Natural compounds with anti-inflammatory properties, such as curcumin and resveratrol, have also shown improved efficacy when delivered via nanoparticles[40].

Gene therapy approaches offer powerful tools for directly modulating macrophage signaling pathways. Nanocarriers can deliver small interfering RNA or microRNA mimics that silence pro-inflammatory genes or enhance anti-inflammatory pathways[41]. For example, silencing components of the nuclear factor-κB pathway in macrophages has been shown to reduce cytokine production and improve insulin sensitivity in obese models[41].

Immunometabolic reprogramming is an emerging strategy that targets macrophage metabolism to influence polarization. Nanocarriers delivering metabolic modulators can shift macrophage energy utilization from glycolysis, associated with M1 activation, toward oxidative phosphorylation, characteristic of M2 macrophages[42, 43]. Such approaches highlight the potential of nanotechnology to integrate immune and metabolic regulation.

5. Biological and Translational Challenges

Despite promising preclinical findings, several challenges impede the translation of nanocarrier-mediated macrophage modulation into clinical therapies. Off-target accumulation of nanoparticles in the liver and spleen remains a significant barrier, reducing delivery efficiency to adipose tissue macrophages[44]. Additionally, heterogeneity among macrophage populations and adipose depots complicates targeting strategies[44].

Safety concerns, including immunogenicity, long-term toxicity, and unintended immune modulation, must be rigorously addressed. Chronic administration of nanocarriers raises questions regarding accumulation and clearance, particularly for non-biodegradable materials. Furthermore, large-scale manufacturing, reproducibility, and regulatory approval of complex nanomedicines present substantial technical hurdles[45].

Addressing these challenges will require standardized evaluation frameworks, improved animal models that better recapitulate human obesity, and early integration of translational considerations into nanocarrier design.

6. Emerging Opportunities and Future Perspectives

Rapid advances in nanotechnology are opening new avenues for macrophage-targeted therapies in obesity. Personalized nanomedicine approaches that tailor nanocarrier design to individual inflammatory and metabolic profiles may enhance treatment efficacy. Integration of diagnostic and therapeutic functions into theranostic nanoparticles enables real-time monitoring of macrophage responses and disease progression[46–48].

Biomimetic and biologically derived nanocarriers, including exosomes and cell membrane-coated nanoparticles, offer improved biocompatibility and targeting specificity. The application of artificial intelligence to optimize nanoparticle design and predict biological interactions is expected to accelerate development and reduce trial-and-error approaches[6, 49]. Combining nanocarrier-based immunomodulation with lifestyle interventions and existing metabolic therapies may provide synergistic benefits. Continued interdisciplinary collaboration will be essential for translating these innovations into effective clinical treatments.

CONCLUSION

Nanocarrier-mediated modulation of macrophage polarization represents a promising strategy for addressing obesity-driven chronic inflammation. By exploiting the intrinsic phagocytic nature and plasticity of macrophages, nanotechnology enables targeted reprogramming of inflammatory responses within adipose tissue. Preclinical studies demonstrate that restoring the balance between pro-inflammatory and anti-inflammatory macrophage phenotypes can significantly improve metabolic outcomes. Nevertheless, substantial challenges related to targeting specificity, safety, scalability, and regulatory approval remain. Future progress will depend on advances in nanocarrier design, a deeper understanding of macrophage immunometabolism, and well-designed translational studies. As these barriers are overcome, nanocarrier-based immunomodulatory therapies hold strong potential to transform the management of obesity and its associated metabolic diseases.

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