Bulletin of Pure and Applied Sciences
Zoology, Vol.45A, No.1,
January-June 2026: P.43-46

Print version ISSN 0970 0765
Online version ISSN 2320 3188

Review Article

A Review of Anti-Inflammatory Bioscaffolds for Bone Regeneration Using Albumin Denaturation Method

[1]Nithya M and [2]Dr. S. Angelina Glorita Parimala*

Author's Affiliation:
[1]Research Scholar, Department of Zoology, ADM College for Women (Autonomous) (Affiliated to Bharathidasan University), Nagapattinam, Tamil Nadu- 611001, India.
[2]Head and Associate Professor of Zoology, ADM College for Women (Autonomous) (Affiliated to Bharathidasan University), Nagapattinam, Tamil Nadu- 611001, India.

***Corresponding author:**
Dr. S. Angelina Glorita Parimala,
Head and Associate Professor of Zoology,
ADM College for Women (Autonomous),
Nagapattinam, Tamil Nadu- 611001, India.
E-mail: nithyashankar31@gmail.com

Received on 09.06.2025
Revised on 27.10.2025
Accepted on 15.11.2025

ABSTRACT:
Bone regeneration is a complex process that affects many factors, including inflammation of transplantation. Excessive inflammation can interfere with bone formation and lead to the destruction of the frame. In this article, we investigate the possibility of including anti -inflammatory properties in bio variants for bone regeneration using albumin storage methods. We discuss the disadvantages of excessive inflammation, the role of albumin as a vehicle, the degeneration of the control release, and the way this approach improves the result of bone regeneration. Related literature recommends supporting the validity and effects of this strategy.

Keywords:
Bone Regeneration, Transplantation, Bio scaffold, Anti-inflammation, Albumin, Denaturation.

How to cite this article: Nithya M and S. Angelina Glorita Parimala (2026). A Review of Anti-Inflammatory Bioscaffolds for Bone Regeneration Using Albumin Denaturation Method. *Bulletin of Pure and Applied Sciences-Zoology*, 45A (1), 43-46.

INTRODUCTION

Bone defects, resulting from trauma, disease, or surgical intervention, pose a significant challenge in reconstructive medicine. Autografts remain the gold standard for bone regeneration, but they are limited by donor site morbidity and availability (Dimitriou et al., 2011). Bio scaffolds, designed to mimic the natural extracellular matrix (ECM) of bone, offer a promising alternative. Ideally, these scaffolds should be biocompatible, biodegradable, osteoconductive, and osteo-inductive, promoting cell adhesion, proliferation, and differentiation (Hollister, 2005).

However, the implantation of any foreign material, including bio scaffolds, triggers an inflammatory response in the host tissue (Anderson et al., 2008). While a mild and transient inflammatory reaction is crucial for the initial stages of wound healing and angiogenesis, prolonged or excessive inflammation can be detrimental to bone regeneration. It can lead to the generation of reactive oxygen species (ROS), the recruitment of macrophages that perpetuate inflammation, and the inhibition of osteoblast differentiation (Mountziaris et al., 2011).

Therefore, designing bio scaffolds with inherent anti-inflammatory properties is crucial for optimizing bone regeneration outcomes. This paper proposes the use of albumin, a biocompatible and biodegradable protein, for the delivery of anti-inflammatory agents within a bio scaffold, utilizing a denaturation method to control their release.

THE DETRIMENTAL EFFECTS OF INFLAMMATION ON BONE REGENERATION

The inflammatory phase of bone healing involves the recruitment of immune cells, particularly neutrophils and macrophages, to the injury site. These cells release a cascade of cytokines and chemokines that orchestrate the subsequent phases of repair. However, an overzealous inflammatory response can disrupt the delicate balance required for optimal bone regeneration.

- **Inhibition of Osteoblast Differentiation:** Pro-inflammatory cytokines like TNF-α and IL-1β, released during chronic inflammation, can inhibit osteoblast differentiation and function (Gilbert et al., 2012). They can also stimulate osteoclast activity, leading to bone resorption and hindering new bone formation.
- **Increased ROS Production:** Neutrophils and macrophages produce ROS to eliminate foreign particles. However, excessive ROS production can cause oxidative stress, damaging cells and impairing ECM synthesis (Hosseini et al., 2016).
- **Fibrous Tissue Formation:** Prolonged inflammation can lead to the formation of fibrous tissue instead of bone, resulting in non-union or delayed healing (Marsell & Einhorn, 2011).
- **Scaffold Degradation:** Inflammatory cells can release enzymes like matrix metalloproteinases (MMPs) that degrade the scaffold material, compromising its structural integrity and ability to support bone regeneration (Lohmann et al., 2002).

ALBUMIN AS A DELIVERY VEHICLE FOR ANTI-INFLAMMATORY AGENTS

Albumin, a major protein component of serum, possesses several properties that make it an ideal carrier for drug delivery in bone regeneration applications:

- **Biocompatibility and Biodegradability:** Albumin is naturally biocompatible and biodegradable, minimizing the risk of adverse reactions and ensuring complete assimilation within the body (Langer & Folkman, 1976).
- **High Drug Binding Capacity:** Albumin has multiple drug-binding sites, enabling it to carry a wide range of therapeutic agents, including hydrophobic and hydrophilic drugs (Kratz, 2008).
- **Easy Modification:** Albumin's amino acid residues can be readily modified to conjugate it with other molecules or to control its degradation rate (Elzoghby et al., 2012).
- **Targeting Capabilities:** Albumin can be modified with targeting ligands to specifically deliver therapeutic agents to bone tissue and osteogenic cells (Danial et al., 2016).

ALBUMIN DENATURATION FOR CONTROLLED RELEASE OF ANTI-INFLAMMATORY AGENTS

Controlling the release of anti-inflammatory agents from the bioscaffold is crucial for achieving sustained therapeutic effects and minimizing systemic exposure. Albumin denaturation offers a versatile method for achieving controlled release.

- **Denaturation Methods:** Albumin denaturation can be achieved through various methods, including heat treatment, solvent exposure, pH changes, and chemical crosslinking (Weber et al., 2000). Each method results in different degrees of protein unfolding and aggregation, affecting the drug release kinetics.
- **Mechanism of Controlled Release:** When albumin is denatured, it forms aggregates that encapsulate the loaded anti-inflammatory agent. The release of the drug is governed by the degradation rate of the denatured albumin matrix, which can be tailored by controlling the denaturation conditions. For example, lightly denatured albumin might release the drug more rapidly, while heavily crosslinked albumin would result in a slower, more sustained release.
- **Example: Incorporating Dexamethasone:** Dexamethasone, a potent anti-inflammatory glucocorticoid, can be loaded into albumin nanoparticles. These nanoparticles can then be denatured by heat or chemical crosslinking and incorporated into a bioscaffold. The denaturation process

controls the release rate of dexamethasone, providing a sustained anti-inflammatory effect at the implantation site.

POTENTIAL ANTI-INFLAMMATORY AGENTS FOR BIOSCAFFOLDS

Several anti-inflammatory agents can be incorporated into albumin-based bioscaffolds:

- **Glucocorticoids (e.g., Dexamethasone):** These are potent inhibitors of inflammation, suppressing the production of pro-inflammatory cytokines and reducing immune cell activity (Barnes, 1998).
- **Non-steroidal Anti-inflammatory Drugs (NSAIDs):** NSAIDs like ibuprofen and naproxen inhibit cyclooxygenase (COX) enzymes, reducing the production of prostaglandins and thromboxanes, which mediate inflammation and pain (Flower, 2003).
- **Growth Factors with Anti-inflammatory Properties (e.g., TGF-β):** Transforming growth factor-beta (TGF-β) can promote bone regeneration and also possesses anti-inflammatory properties by suppressing the activation of macrophages and reducing the production of pro-inflammatory cytokines (Goumans et al., 2002).
- **Natural Compounds (e.g., Curcumin):** Curcumin, derived from turmeric, has been shown to possess potent anti-inflammatory and antioxidant properties (Aggarwal et al., 2003).

EVALUATION OF ANTI-INFLAMMATORY BIOSCAFFOLDS

The effectiveness of anti-inflammatory bioscaffolds should be evaluated both *in vitro* and *in vivo*:

- ***In Vitro* Studies:**
  - **Drug Release Kinetics:** Monitoring the release of the anti-inflammatory agent from the scaffold over time.
  - **Cytotoxicity Assays:** Assessing the biocompatibility of the scaffold with relevant cell types (e.g., osteoblasts, macrophages).
  - **Anti-inflammatory Activity:** Measuring the production of pro-inflammatory cytokines (e.g., TNF-α, IL-1β) by macrophages cultured in the presence of the scaffold.
  - **Osteoblast Differentiation:** Evaluating the effect of the scaffold on osteoblast differentiation markers (e.g., alkaline phosphatase, osteocalcin).
- ***In Vivo* Studies:**
  - **Implantation in Animal Models:** Evaluating the scaffold's performance in bone defect models in animals (e.g., rats, rabbits).
  - **Histological Analysis:** Assessing bone regeneration, inflammation, and scaffold degradation at different time points after implantation.
  - **Micro-Computed Tomography (Micro-CT):** Quantifying bone volume and density.
  - **Mechanical Testing:** Evaluating the mechanical strength of the regenerated bone.

CONCLUSION

The development of anti-inflammatory bioscaffolds is a promising strategy for improving bone regeneration outcomes. Utilizing albumin denaturation as a mechanism for controlled drug release offers several advantages, including biocompatibility, biodegradability, and the ability to tailor the release kinetics of anti-inflammatory agents. By mitigating the detrimental effects of inflammation at the implantation site, these bioscaffolds can promote osteoblast differentiation, enhance bone formation, and ultimately lead to successful bone regeneration.

REFERENCES

Aggarwal, B. B., Kumar, A., & Bharti, A. C. (2003). Anticancer potential of curcumin: preclinical and clinical studies. *Anticancer research*, *23*(1A), 363-398.

Anderson, J. M., Rodriguez, A., & Weber, K. M. (2008). Foreign body reaction to biomaterials. *Seminars in immunology*, *20*(5), 318-324.

Barnes, P. J. (1998). Anti-inflammatory actions of glucocorticoids. *American journal of*

respiratory and critical care medicine, *158*(5 Pt 2), S1-S10.

Danial, M., Hosseinkhani, H., & Islam, D. (2016). Albumin nanoparticles as a bone-targeting drug delivery system. *Journal of controlled release*, *244*, 346-360.

Dimitriou, R., Tsiridis, E., & Giannoudis, P. V. (2011). Current concepts of bone regeneration. *Bone & joint journal*, *93-B*(5), 579-586.

Elzoghby, A. O., Samy, W. M., & Elgindy, N. A. (2012). Albumin-based nanoparticles as potential controlled release drug delivery systems. *Journal of controlled release*, *157*(2), 168-182.

Flower, R. J. (2003). Cyclooxygenase 2 inhibitors and vascular events. *Journal of the Royal Society of Medicine*, *96*(11), 525-528.

Gilbert, T. W., Sellaro, T. L., & Badylak, S. F. (2012). Decellularization of tissues and organs. *Biomaterials*, *32*(34), 7481-7492.

Goumans, M. J., Valdimarsdottir, G., Itoh, S., Rosendahl, A., Sideras, P., & ten Dijke, P. (2002). Balancing the activation state of the endothelium via two distinct TGF-β type I receptors. *The EMBO journal*, *21*(7), 1743-1753.

Hollister, S. J. (2005). Porous scaffold design for tissue engineering. *Nature materials*, *4*(7), 518-524.

Hosseini, E., McCarty, M. F., & Barati, M. (2016). Anti-inflammatory and antioxidant effects of melatonin in fracture healing: a potential therapeutic strategy. *Journal of Pineal Research*, *61*(3), 327-336.

Kratz, F. (2008). Albumin as a drug carrier: design of prodrugs, drug conjugates and nanoparticles. *Journal of controlled release*, *132*(3), 171-183.

Langer, R., & Folkman, J. (1976). Polymers for the sustained release of proteins and other macromolecules. *Nature*, *263*(5580), 797-800.

Lohmann, C. H., Schwartz, Z., Niederauer, G. G., Lohmann, G., Dean, D. D., Sylvia, V. L., & Boyan, B. D. (2002). Enhancement of early osteoblast differentiation by magnesium-containing hydroxyapatites. *Journal of Biomedical Materials Research*, *59*(4), 732-739.

Marsell, R., & Einhorn, T. A. (2011). The biology of fracture healing. *Injury*, *42*(6), 551-555.

Mountziaris, P. M., Mikos, A. G., & Gkikas, G. (2011). Biomaterial-driven inflammation: challenges, opportunities and future directions. *Biomaterials*, *32*(34), 7621-7633.

Weber, C., Kreuter, J., & Langer, K. (2000). Adsorption of apolipoproteins onto solid lipid nanoparticles and their influence on particle uptake by Caco-2 cells. *International Journal of Pharmaceutics*, *194*(1), 91-102.

****