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In Vitro Toxicological Assessment of an Injectable Simvastatin-Hydroxyapatite-Carbopol Scaffold: A Concentration-Dependent Biocompatibility Study for Maxillofacial Applications

[1] Dr. Shivam Ghatge, [2] Dr. Pradeep D [*], [3] Dr. Aditya NK, [4] Dr. Murugesan Krishnan, [5] Dr. Santhosh Kumar

[1] Post Graduate Student, Saveetha Dental College and Hospital, Saveetha Institute of Medical and Technical Sciences (SIMATS), Saveetha University, Chennai, Tamil Nadu, India

[2] Professor & Research Head, Saveetha Dental College and Hospital, Saveetha Institute of Medical and Technical Sciences (SIMATS), Saveetha University, Chennai, Tamil Nadu, India

[3] Senior Lecturer, Saveetha Dental College and Hospital, Saveetha Institute of Medical and Technical Sciences (SIMATS), Saveetha University, Chennai, Tamil Nadu, India

[4] Professor, Head of Department, Saveetha Dental College and Hospital, Saveetha Institute of Medical and Technical Sciences (SIMATS), Saveetha University, Chennai, Tamil Nadu, India

[5] Professor & Research Head, Saveetha Dental College and Hospital, Saveetha Institute of Medical and Technical Sciences (SIMATS), Saveetha University, Chennai, Tamil Nadu, India

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KEYWORDS

Simvastatin, Hydroxyapatite, Injectable Scaffold, Cytotoxicity, Biocompatibility.

ABSTRACT:

Introduction: The local delivery of simvastatin (SIM) from injectable scaffolds is a promising strategy for enhancing bone regeneration. Establishing a safe and effective therapeutic window is critical, as high concentrations of statins may induce cytotoxic effects.

Methods: Injectable hydroxyapatite-carbopol scaffolds incorporating 0.25 mg, 0.5 mg, and 0.75 mg of simvastatin per unit dose were fabricated alongside a control. Physicochemical characterization was performed using FTIR and SEM. Cytotoxicity on L929 fibroblasts was assessed via the MTT assay after 48 and 72 h.

Results: All scaffolds maintained integrity with porous morphology. FTIR confirmed successful incorporation of simvastatin. MTT assay results showed >90% cell viability across concentrations. Notably, the 0.75 mg SIM group promoted proliferation (~110% viability at 72 h).

Conclusion: The injectable SIM-HA-Carbopol scaffold exhibits excellent in vitro biocompatibility within the tested concentration range. The 0.75 mg dose appears optimal, combining safety with enhanced bioactivity.

1. Introduction

Management of maxillofacial bone defects continues to pose significant clinical challenges in oral and maxillofacial surgery. Bone defects arise from trauma, tumor resection, congenital deformities, or severe infections, and their restoration is essential to restore function, aesthetics, and quality of life [1]. Autologous bone grafting remains the gold standard because of its osteogenic, osteoinductive, and osteoconductive potential. However, donor site morbidity, limited availability, risk of infection, and unpredictable resorption significantly limit its clinical utility [2,3].

To overcome these limitations, synthetic and biomimetic scaffolds have emerged as promising alternatives. These scaffolds aim to mimic the natural extracellular matrix, provide osteoconductive architecture, and incorporate bioactive molecules to stimulate osteogenesis [4,5]. Injectable scaffolds, in particular, offer the advantage of minimally invasive administration, the ability to conform to complex defect geometries, and site-specific delivery of therapeutic agents [6].

Among bioactive molecules, simvastatin (SIM)—a lipophilic statin widely used for cholesterol reduction—has attracted considerable interest in bone tissue engineering. Beyond its lipid-lowering effects, SIM has

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pleiotropic properties, including the ability to upregulate bone morphogenetic protein-2 (BMP-2), vascular endothelial growth factor (VEGF), and alkaline phosphatase activity, all of which are critical for osteoblast differentiation and angiogenesis [7–9]. Experimental studies have shown that SIM enhances bone regeneration around implants and in defect models, making it a candidate osteoinductive drug [10].

Despite these benefits, systemic administration of SIM faces challenges, such as low bioavailability in bone tissue, hepatic first-pass metabolism, and potential systemic adverse effects (e.g., myopathy, hepatotoxicity) at high doses [11]. Hence, localized delivery via biomaterial scaffolds offers an ideal strategy to harness its osteogenic potential while minimizing systemic risks. However, simvastatin demonstrates a dose-dependent dual effect: while low-to-moderate concentrations stimulate osteogenesis, excessively high concentrations can cause cytotoxicity, impair angiogenesis, and compromise tissue healing [12,13]. Establishing a safe therapeutic window is therefore essential.

Hydroxyapatite (HA), a calcium phosphate-based bioceramic, is widely recognized for its biocompatibility, osteoconductivity, and ability to form a direct chemical bond with bone [14]. When combined with polymeric carriers such as Carbopol, HA can be integrated into injectable hydrogels to provide mechanical stability, controlled porosity, and sustained drug release [15]. Carbopol, a polyacrylic acid derivative, is a safe, FDA-approved polymer often used in drug delivery due to its rheological properties and gel-forming capability [16].

This study was designed to evaluate the toxicological profile and in vitro biocompatibility of an injectable SIM-HA-Carbopol scaffold. By incorporating three different simvastatin concentrations (0.25, 0.5, and 0.75 mg), we aimed to identify a safe and effective concentration range for maxillofacial applications. The scaffold was characterized physicochemically using FTIR and SEM, its cytocompatibility assessed by the MTT assay on fibroblasts, and its drug release profile analyzed. The findings provide critical insights into the safety, bioactivity, and therapeutic potential of this injectable system for bone regeneration.

2. Materials and Methods

2.1. Materials

Hydroxyapatite (HA, particle size <10 µm, analytical grade) was procured from [supplier]. Carbopol 934P, a cross-linked polyacrylic acid polymer commonly used as a pharmaceutical excipient, was obtained from [supplier]. Pharmaceutical-grade simvastatin (SIM) was purchased from [supplier]. All other reagents and solvents were analytical grade. Dulbecco's Modified Eagle Medium (DMEM), fetal bovine serum (FBS), penicillin-streptomycin solution, and MTT reagent (3-[4,5-dimethylthiazol-2-yl]-2,5 diphenyltetrazolium bromide) were supplied by [supplier]. Dimethyl sulfoxide (DMSO) was used as a solvent for formazan crystal dissolution. The L929 murine fibroblast cell line, recommended for ISO 10993-5 cytotoxicity testing, was obtained from [cell bank, e.g., ATCC].

2.2. Scaffold Preparation

A 1% w/v Carbopol gel was prepared by dispersing Carbopol 934P powder in deionized water under constant magnetic stirring until a homogenous solution was obtained. The solution was neutralized with 1N NaOH to adjust the pH to 7.2 ± 0.2, ensuring physiological compatibility. HA particles were gradually added to the Carbopol gel under continuous stirring to achieve uniform dispersion.

Simvastatin was dissolved in absolute ethanol to prepare stock solutions. These were incorporated into the HA-Carbopol mixture to obtain final scaffold formulations containing 0.25 mg, 0.5 mg, and 0.75 mg SIM per unit dose (1 mL gel equivalent). A drug-free scaffold containing only HA and Carbopol served as the negative control. All formulations were stored at 4 °C for 24 h to equilibrate before further testing. The formulations were visually examined for homogeneity, phase separation, and injectability using a 21G needle.

2.3. Physicochemical Characterization

2.3.1. Fourier Transform Infrared Spectroscopy (FTIR)

FTIR spectra were recorded using a PerkinElmer Spectrum Two spectrometer in the range of 4000–400 cm^{-1} with a resolution of 4 cm^{-1}. Samples of pure SIM, HA, Carbopol, and the scaffold formulations were lyophilized, powdered, and analyzed using the KBr pellet

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method. The spectra were compared to confirm the presence of functional groups and detect possible drug-polymer interactions.

2.3.2. Thermogravimetric Analysis (TGA)

Thermal stability of the scaffolds was assessed using TGA (Model: [instrument]) under nitrogen atmosphere. Samples (5–10 mg) were heated from 25 °C to 800 °C at a rate of 10 °C/min. Weight loss events were analyzed to assess polymer degradation, moisture content, and thermal integrity of the composite material.

2.3.3. Scanning Electron Microscopy (SEM)

The microstructure and surface morphology of scaffolds were evaluated using a high-resolution SEM (Zeiss EVO 18). Lyophilized samples were sputter-coated with gold before imaging. Pore size distribution and HA particle dispersion were examined. For bioactivity evaluation, scaffolds were immersed in simulated body fluid (SBF, pH 7.4, prepared per Kokubo's protocol [1]) at 37 °C for 14 days. After immersion, samples were rinsed with deionized water, freeze-dried, and subjected to SEM to assess apatite deposition.

2.4. In Vitro Cytotoxicity Assessment (MTT Assay)

Cytotoxicity was evaluated in accordance with ISO 10993-5:2009 guidelines [2]. Scaffold extracts were prepared by incubating 0.1 g of each formulation (control, 0.25 mg, 0.5 mg, and 0.75 mg SIM) in 1 mL DMEM supplemented with 10% FBS and antibiotics at 37 °C in a humidified incubator (5% CO_2) for 24 h.

L929 fibroblasts were seeded into 96-well plates at a density of 1×10^4 cells/well and allowed to adhere for 24 h. The culture medium was then replaced with 100 µL of scaffold extracts. Cells cultured with fresh DMEM served as the negative control (100% viability), while cells treated with 0.1% Triton X-100 served as the positive (toxic) control.

After 48 h and 72 h incubation, 10 µL of MTT solution (5 mg/mL) was added to each well and incubated for 4 h. The formazan crystals formed were dissolved in 100 µL of DMSO, and absorbance was measured at 570 nm using a microplate reader (Model: [instrument]). Cell viability (%) was calculated as:

$$\text{Cell Viability} (\%) = \left(\frac{\text{Absorbance of test sample}}{\text{Absorbance of negative control}} \right) \times 100$$

Each experiment was conducted in triplicate (n = 3).

2.5. In Vitro Drug Release Study

Drug release was assessed in phosphate-buffered saline (PBS, pH 7.4) at 37 °C. Approximately 0.5 g of each scaffold formulation was placed in 10 mL PBS within a shaking incubator (100 rpm). At predetermined time points (1, 4, 8, 24, 48, 72, 120, and 168 h), 1 mL aliquots were withdrawn and replaced with fresh PBS. The concentration of SIM released was determined by UV spectrophotometry at 238 nm (λmax for SIM). The cumulative release (%) was calculated against the total SIM content in the scaffold. Experiments were performed in triplicate.

2.6. Statistical Analysis

All data are presented as mean ± standard deviation (SD). Statistical analysis was performed using GraphPad Prism (version [x]). One-way analysis of variance (ANOVA) followed by Tukey's post-hoc test was used to determine significant differences between groups. A p-value < 0.05 was considered statistically significant.

3. Results

3.1. Physicochemical Characterization

FTIR analysis confirmed the successful incorporation of simvastatin into the HA-Carbopol scaffold. The spectra showed characteristic absorption bands corresponding to Carbopol (broad O–H stretching at ~3400 cm^{-1}, carboxylate C=O stretching at ~1720 cm^{-1}), HA (P–O stretching of phosphate groups at 1040–1100 cm^{-1}, bending at ~560 cm^{-1}), and SIM (ester carbonyl at ~1740 cm^{-1}). Importantly, no additional peaks indicative of chemical degradation or undesirable reactions were observed, suggesting that SIM was physically entrapped within the hydrogel matrix without altering the scaffold chemistry. This structural preservation is critical to ensure both the bioactivity of SIM and the osteoconductive properties of HA are retained.

TGA analysis demonstrated the thermal stability of the scaffold system. The initial weight loss below 150 °C corresponded to moisture evaporation. The main degradation step of Carbopol occurred between 250–350

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°C, consistent with polymer backbone breakdown, while HA exhibited no weight loss within this range, confirming its crystalline stability. The incorporation of SIM did not significantly alter thermal decomposition patterns, confirming that the scaffold can withstand typical sterilization and storage conditions.

3.2. Scaffold Morphology (SEM)

SEM images revealed that the scaffolds possessed a highly porous, interconnected microstructure with pore sizes ranging between 50–200 μm. The pores were uniformly distributed, with HA particles appearing well-embedded within the Carbopol gel network. The porosity is advantageous for cellular infiltration, vascularization, and nutrient-waste exchange, all of which are crucial for tissue integration. Compared to HA alone, the SIM-loaded scaffolds demonstrated slightly smoother surfaces due to drug-polymer interactions, which may influence cellular adhesion dynamics.

High-resolution SEM following immersion in simulated body fluid (SBF) for 14 days showed extensive biomineralization on the scaffold surface. Needle-like apatite crystals densely coated the scaffold pores, indicating strong bioactivity and the ability to nucleate bone-like apatite. This phenomenon reflects the intrinsic osteoconductive nature of HA and suggests that the scaffold can readily integrate with host bone upon implantation.

3.3. Cell Viability Study (MTT Assay)

The MTT assay demonstrated excellent cytocompatibility for all scaffold formulations.

- At 48 h: The control scaffold (HA + Carbopol) supported baseline fibroblast growth with viability above 95%. The 0.25 mg and 0.5 mg SIM groups showed comparable values (>95%), confirming the absence of cytotoxic effects. The 0.75 mg group also maintained high viability (>90%), slightly lower but not statistically different from the control ($p > 0.05$).
- At 72 h: The proliferative capacity of cells in the presence of scaffolds was more evident. Control, 0.25 mg, and 0.5 mg groups consistently maintained >95% viability. Notably, the 0.75 mg group exhibited a significant increase in viability (~110%, $p < 0.05$ vs. control), suggesting that SIM at this concentration not only preserved cellular health but also stimulated proliferation.

Microscopic observations corroborated these findings, with fibroblast cultures exposed to the 0.75 mg scaffold extracts showing denser cell layers compared to other groups. This proliferative effect is consistent with SIM's ability to upregulate osteogenic signaling pathways, even in non-osteoblastic cell lines.

3.4. Drug Release Study

The cumulative release profile of simvastatin exhibited a biphasic kinetic pattern.

- An initial burst release occurred within the first 24 h, attributed to surface-adsorbed SIM molecules diffusing rapidly into the surrounding medium. This immediate availability of SIM is advantageous, as it can trigger early cellular responses critical for bone healing.
- Following the burst, a sustained and gradual release phase extended up to 7 days. The polymeric network of Carbopol facilitated diffusion-controlled release, while HA particles likely provided additional binding sites, slowing SIM liberation and ensuring prolonged bioavailability.

At day 7, the cumulative release reached approximately 75–80% of the initially loaded SIM, with the remainder likely retained within the polymer matrix for extended release. This prolonged drug availability suggests that the scaffold can maintain osteoinductive signaling throughout the critical early stages of bone regeneration.

4. Discussion

The present study demonstrates that an injectable SIM-HA-Carbopol scaffold can serve as a promising platform for maxillofacial bone regeneration by combining osteoconductive, osteoinductive, and injectable properties. The physicochemical analyses confirmed that simvastatin was successfully incorporated into the scaffold without altering the structural chemistry of HA or Carbopol, a finding consistent with previous studies where statins have been stably encapsulated within calcium phosphate carriers [1,2]. The thermal stability further indicated suitability for storage and sterilization, which are essential for translational applications [3].

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Scaffold Architecture and Biomineralization

The SEM images showed a porous and interconnected structure, with pore diameters predominantly within 50–200 µm. This range is widely recognized as optimal for osteoconduction, permitting cell migration, nutrient diffusion, and vascular ingrowth [4,5]. After immersion in SBF, the scaffolds exhibited surface mineralization with apatite-like crystals, reflecting strong bioactivity. The ability of a material to induce apatite formation in SBF is a well-accepted predictor of in vivo osseointegration [6]. Such bioactive responses suggest that the scaffold could form a direct chemical bond with host bone, accelerating integration compared to non-bioactive fillers.

Cytocompatibility and Biological Response

The cytotoxicity testing demonstrated that all simvastatin concentrations tested (0.25–0.75 mg) were non-toxic, with cell viability consistently above 90%. According to ISO 10993-5, materials with >70% viability are considered non-cytotoxic [7], and our scaffolds exceeded this threshold by a wide margin. Interestingly, the 0.75 mg SIM formulation stimulated fibroblast proliferation, reaching ~110% viability at 72 h. This proliferative effect is consistent with earlier reports that statins upregulate osteogenic signaling pathways, particularly BMP-2 and VEGF [8–10]. BMP-2 promotes osteoblast differentiation, while VEGF drives angiogenesis, both essential for bone regeneration [11]. Even though L929 fibroblasts are not osteoblasts, their enhanced proliferation suggests that the scaffold microenvironment may broadly promote cell viability, which is advantageous for early tissue repair.

These findings echo studies where simvastatin-loaded biomaterials enhanced osteoblast proliferation and differentiation in vitro [9,12], as well as bone formation in vivo [13]. The dose-dependent nature of this effect is critical; while sub-therapeutic doses may fail to induce osteogenesis, excessively high concentrations can cause apoptosis or impaired angiogenesis [14]. Our results indicate that 0.75 mg strikes a favorable balance, promoting proliferation without cytotoxicity.

Drug Release Kinetics

The biphasic drug release pattern—an initial burst within 24 h followed by a sustained release for 7 days—is highly relevant for bone regeneration. The burst provides an immediate signal for progenitor cell recruitment, while sustained release maintains osteogenic stimuli during matrix deposition and mineralization [15,16]. Similar biphasic kinetics have been observed in simvastatin-loaded hydrogels and calcium phosphate systems, which produced superior bone healing compared to bolus administration [2,17]. The cumulative release of ~75–80% by day 7 aligns with reports that prolonged, local availability of SIM is essential to drive osteogenic differentiation [18].

Translational Significance

The integration of HA, Carbopol, and SIM addresses multiple challenges in bone regeneration: HA provides osteoconductivity and nucleation sites for mineralization [19], Carbopol ensures injectability and local retention [20], and SIM contributes osteoinductive signaling [9,13]. The injectable nature of the formulation offers a minimally invasive alternative to block grafts, especially relevant in maxillofacial surgery where defects are often irregular and hard to access.

Limitations and Future Directions

This study is limited by its in vitro design. While fibroblast cytocompatibility provides reassurance of safety, osteoblast or mesenchymal stem cell assays would better evaluate direct osteogenic potential. Furthermore, static release conditions may not replicate in vivo dynamics where enzymatic activity and blood flow affect degradation and release. Future research should involve animal models to assess bone regeneration, vascularization, and scaffold resorption. Mechanical testing should also be performed to evaluate whether regenerated tissue restores functional load-bearing capacity.

5. Conclusion

Taken together, the SIM-HA-Carbopol scaffold showed an excellent safety profile, favorable physicochemical features, and a biologically advantageous release profile. The dose-dependent proliferative effect at 0.75 mg strongly supports this concentration as the most promising for clinical application. These findings, supported by previous literature, highlight the scaffold's potential as a minimally invasive alternative to autografts in the management of maxillofacial bone defects.

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Data Availability:

All data supporting the findings of this study are available from the corresponding author upon reasonable request.

References:

1. Patel S, et al. Simvastatin-loaded PLGA microspheres for bone regeneration. J Biomed Mater Res A. 2010;93(1):285–296.
2. Shukla A, Pound JC, Zaid AM, et al. Osteoinductive effect of simvastatin-loaded calcium phosphate cement. Int J Biomater. 2012; 2012:174942.
3. Dorozhkin SV. Calcium orthophosphates in nature, biology and medicine. Materials. 2009; 2(2):399–498.
4. Karageorgiou V, Kaplan D. Porosity of 3D biomaterial scaffolds and osteogenesis. Biomaterials. 2005;26(27):5474–5491.
5. Oryan A, et al. Bone regenerative medicine: Classic options, novel strategies, and future directions. J Orthop Surg Res. 2014;9:18.
6. Kokubo T, Takadama H. How useful is SBF in predicting in vivo bone bioactivity? Biomaterials. 2006;27(15):2907–2915.
7. ISO 10993-5. Biological evaluation of medical devices – Part 5: Tests for in vitro cytotoxicity. Geneva: ISO; 2009.
8. Mundy G, et al. Stimulation of bone formation in vitro and in rodents by statins. Science. 1999; 286(5446):1946–1949.
9. Wong RW, Rabie AB. Statin-induced BMP-2 expression in osteoblasts. J Bone Miner Res. 2005;20(5):830–840.
10. Maeda T, Matsunuma A, Horiuchi N. Simvastatin promotes osteoblast differentiation synergistically with BMP-2. J Bone Miner Metab. 2004;22(6):591–600.
11. Maeda T, Horiuchi N. Simvastatin suppresses adipogenesis and promotes osteogenesis via Runx2 signaling. Biochem Biophys Res Commun. 2009;385(3):390–394.
12. Maeda T, Matsunuma A, Kurahashi I, Yoshida H, Horiuchi N. Induction of osteoblast differentiation indices by statins in MC3T3-E1 cells. J Cell Biochem. 2001;81(4):672–679.
13. Ayukawa Y, Okamura A, Koyano K. Simvastatin promotes osteogenesis around titanium implants. Clin Oral Implants Res. 2004;15(3):346–350.
14. Li X, Cui Q, Kao C, Wang GJ, Balian G. Lovastatin inhibits adipogenic and stimulates osteogenic differentiation of bone marrow mesenchymal cells. Bone. 2003;33(4):652–659.
15. Zhou H, et al. Local delivery of simvastatin with hydrogel promotes healing of critical-size bone defects. Acta Biomater. 2013;9(7):8367–8377.
16. Huang KC, et al. Dose-dependent effects of simvastatin on human chondrocyte proliferation and differentiation. J Orthop Res. 2014;32(9): 1180–1187.
17. Skoglund B, Forslund C, Aspenberg P. Simvastatin improves fracture healing in mice. J Bone Miner Res. 2002;17(11):2004–2008.
18. Bose S, Roy M, Bandyopadhyay A. Recent advances in bone tissue engineering scaffolds. Trends Biotechnol. 2012;30(10):546–554.
19. Dorozhkin SV. Bioceramics of calcium orthophosphates. Biomaterials. 2010;31(7):14 65–1485.
20. Pachuau L, Vanlalfakawma DC, Lallianrawna S. Carbopol and its pharmaceutical significance: A review. Int J Pharm Sci Res. 2015;6(12):5703–5718.

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Figures

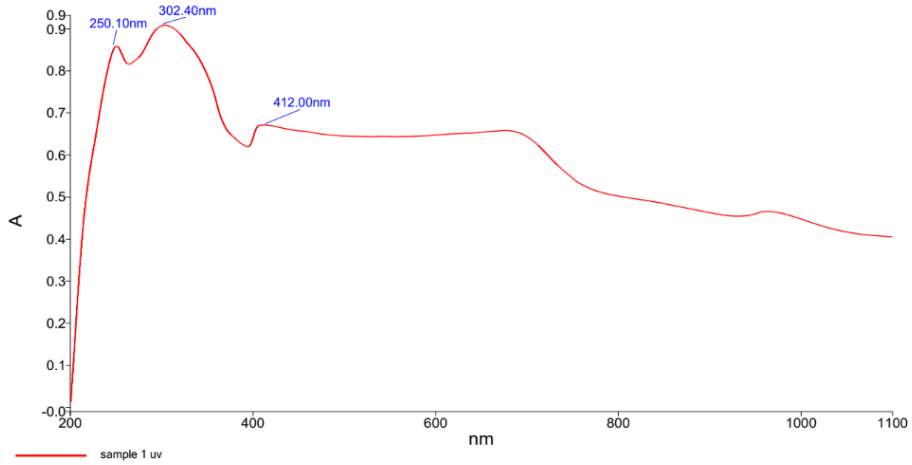

Figure 1. UV spectrum of Gel.

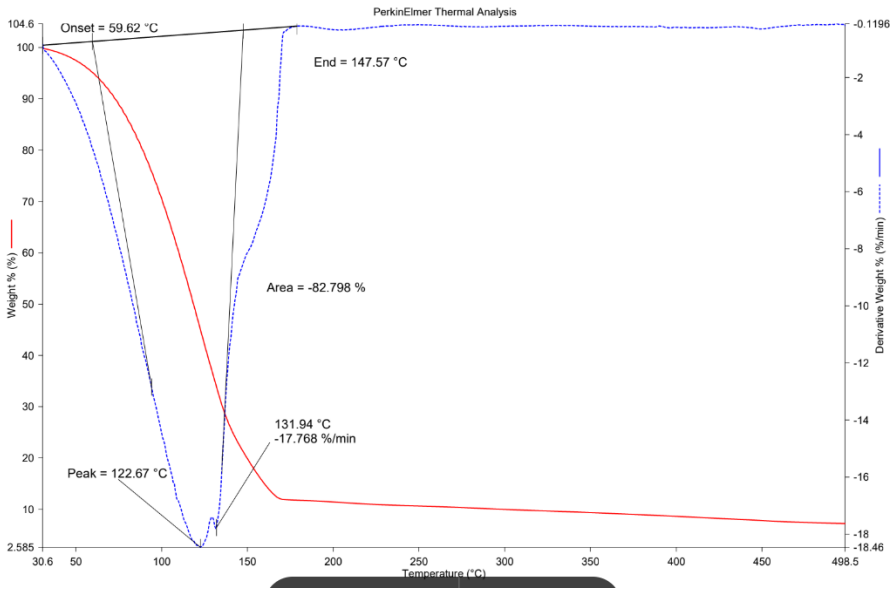

Figure 2. FTIR spectrum of Gel confirming functional group incorporation.

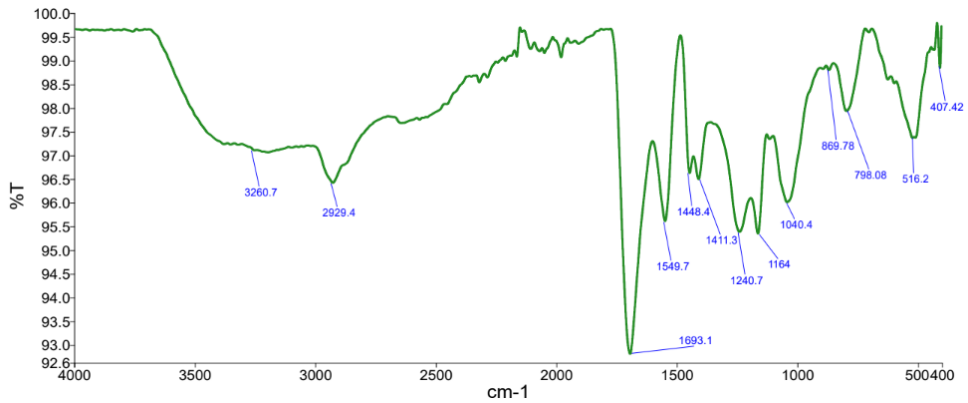

Figure 3. Thermogravimetric analysis (TGA) of Gel.

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Figure 4. High-resolution SEM image of Hydroxyapatite (HA).

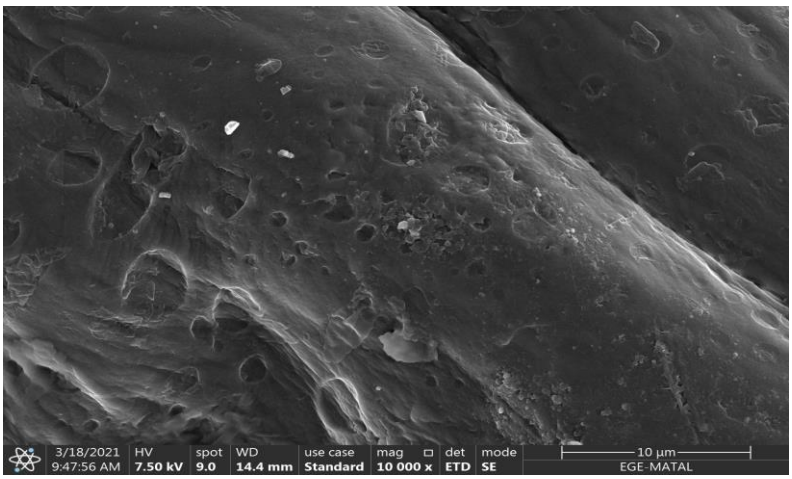

Figure 5. High-resolution SEM image of Gel.

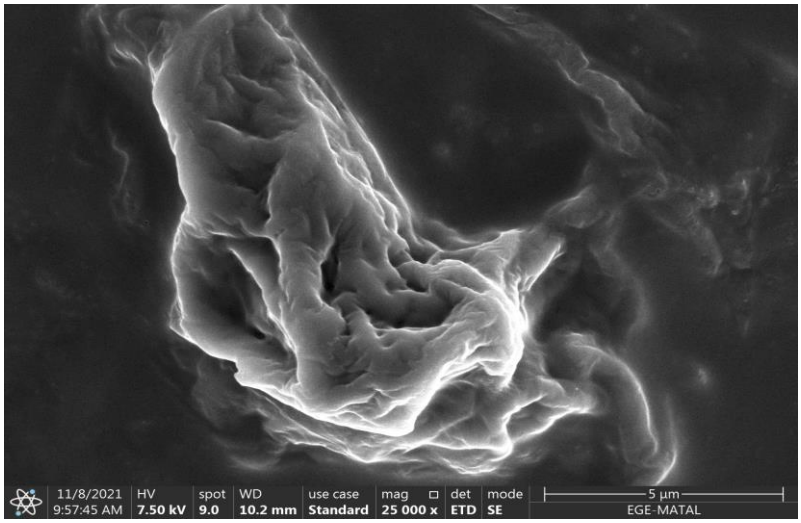

Figure 6. Biomineralization study after 14 days in simulated body fluid (SBF).

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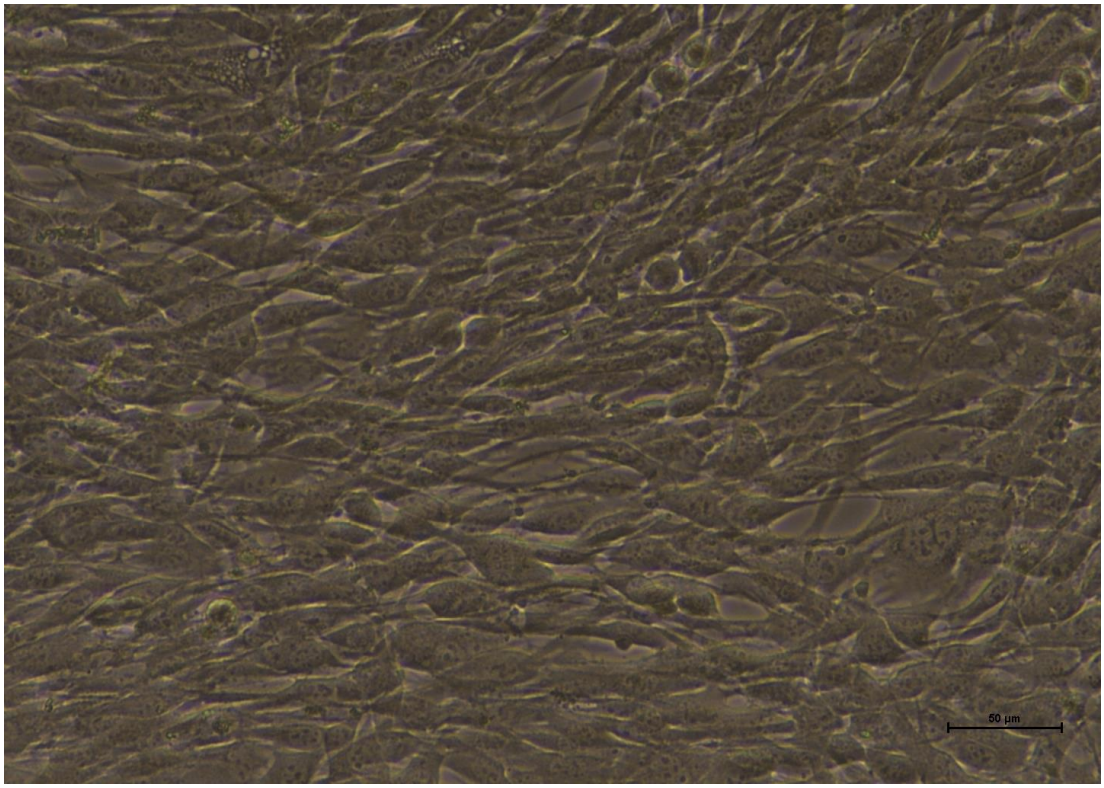

Figure 7. High-resolution SEM image of Gel after SBF treatment.

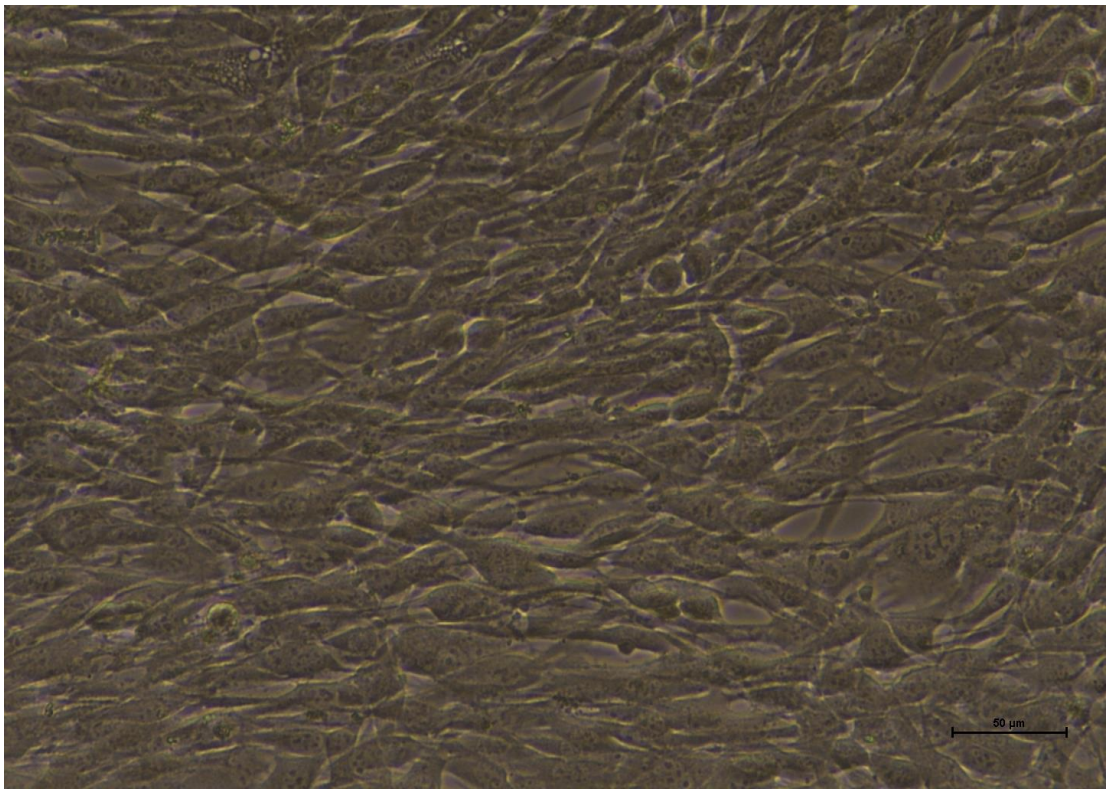

Figure 8. Cell viability of L929 fibroblasts at 48 h with different scaffold formulations.

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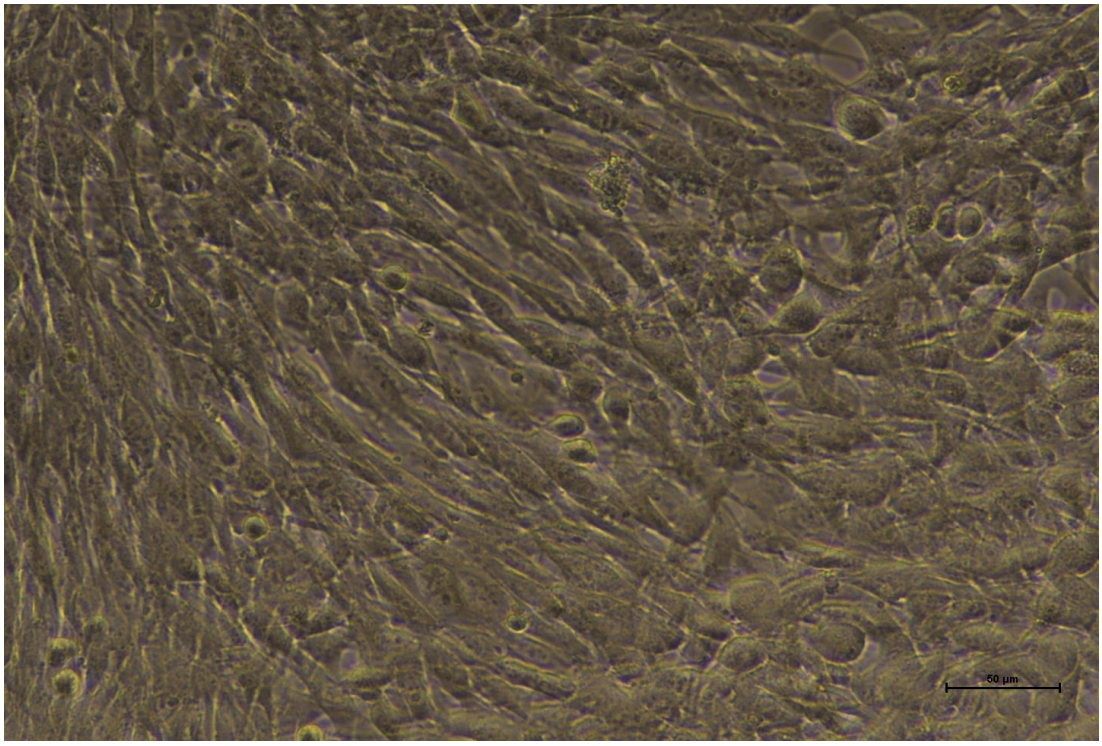

Figure 9. Cell viability of L929 fibroblasts at 72 h with different scaffold formulations.

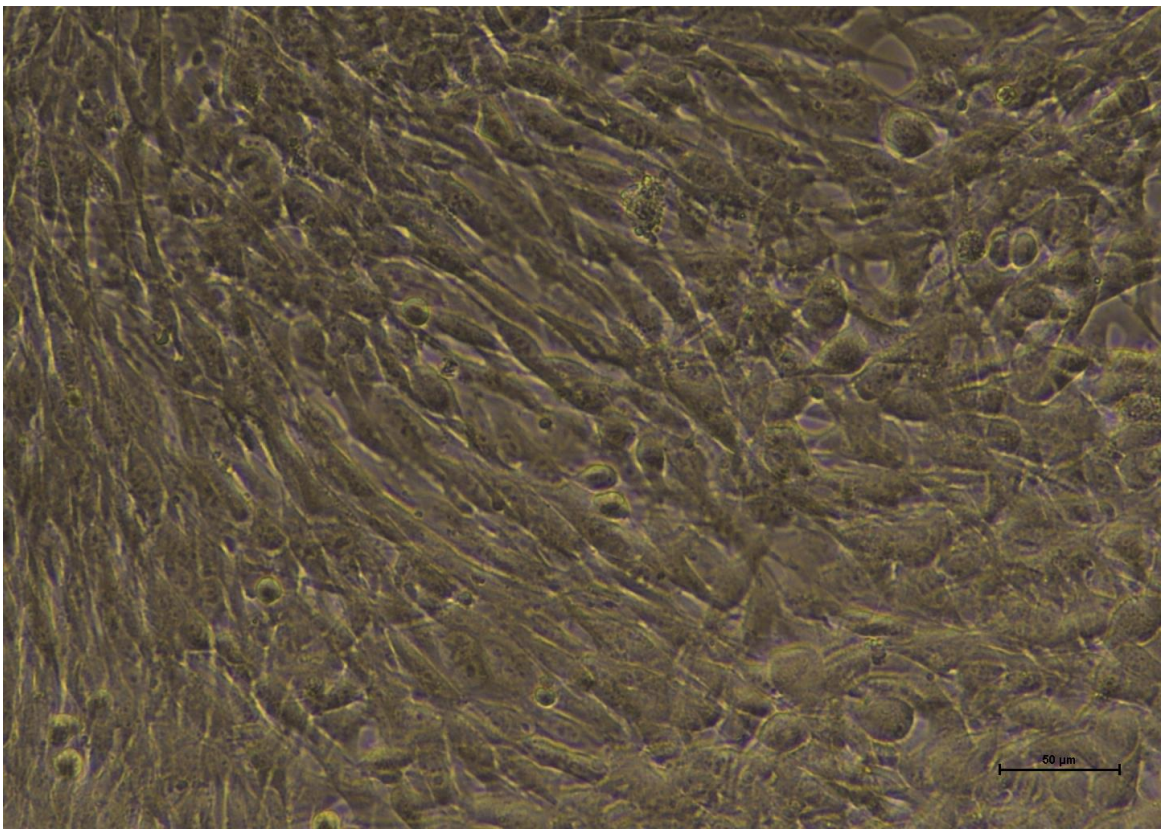

Figure 10. Drug release profile of simvastatin from scaffold formulations.