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Mathematical Modeling of Drug Diffusion in Bioengineered Scaffolds for Tissue Regeneration

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ABSTRACT

Localized and targeted controlled drug delivery using bioengineered scaffolds offers a promising concept in tissue engineering and regenerative medicine, in that, therapeutic molecules, including growth factors, proteins, and small-molecule drugs, can be released at the tissue-repair site on-command. The key to designing scaffolds is a clear comprehension of the processes underlying drug release, which can depend on the complicated relationship between scaffold porosity, degradation dynamics and drug-matrix binding phenomena. To overcome this issue, the current study constructs a detailed mathematical modeling framework that combines multi-scale diffusion equations, multi-scale porosity evolution driven by degradation, and nonlinear binding kinetics to determine reliably drug release of polymeric scaffolds. The governing equations are Fickian with time-dependent effective diffusivity to include effects due to the scaffold microarchitecture and degradation and binding terms modeling reversible polymer-drug interactions. Finite element methods (FEM) were applied to numerical simulations in COMSOL Multiphysics, and geometry of scaffolds were computed as porous cylindrical structures with sink boundary conditions which are physiologically relevant. The parametric analysis examined the effect of scaffold porosity, degradation rate, tortuosity, and binding constants on cumulative drug release during a 30-day cumulative drug release. Findings have revealed that increasing porosity contributes to a faster release rate with increase in effective diffusivity but the over-increase in porosity results in early burst release which causes loss of scaffold integrity. On the other hand, controlled degradation increased release time by progressively opening porosity without disrupting mechanical stability and nonlinear binding interaction dramatically decreased release time, allowing persistent therapeutic residence. Experimental datasets of literature on PLGA-based scaffolds were compared against model predictions and found to match well with a deviation of not more than 10 percent. Here, it is pointed out that mathematical modeling is an effective predictive method to design scaffolds with specific drug release kinetics, easier therapeutic approaches in tissue regeneration and future scaffold development in clinical translation.

1. INTRODUCTION

Tissue regeneration has become a highly important area of interest in the regenerative therapy and biomedical engineering in order to repair the structural and functional integrity of injured or diseased tissues. Effective regeneration depends not only on the availability of an appropriate scaffold onto which cells can affix, proliferate, and differentiate, but also on the delivery of therapeutic molecules in a controlled and sustained way to coordinate biological responses. Based on either natural or synthetic polymers, bioengineered scaffolds have therefore developed as dual-purpose platforms to not only mechanically support, but also serve as reservoirs of localized drug delivery. Compared to the unfocused administration that frequently has poor bioavailability, high dose, and off-target toxicity, site-specific release, decreased systemic bleeding, and greater therapeutic effect, scaffold-mediated drug delivery guarantees optimal therapeutic effect, reduced side effects, and improvements in bioavailability.

One significant problem with scaffold-based drug delivery though is the attainment of high spatial and temporal control of release kinetics. A sophisticated system of interactions between the scaffold architecture, porosity, tortuosity, degradation processes, and drug and scaffold matrix interactions determines the release profile. An example of this is that porous scaffolds are more permeable and, therefore, can promote faster diffusion, although these scaffolds can experience burst release and result in the early release of drugs, whereas low-porosity scaffolds block diffusion, which limits the availability of therapeutic agents. Moreover, the hydrolytic or enzymatical degradation of polymers to form a dynamic microstructure of the scaffold constantly changes the pathways of diffusivity and drug transportation. Further complexity is caused by drug-matrix effects including, but not limited to, adsorption, binding, or entrapment, and may cause nonlinear kinetics due to delayed release. All these multifactorial effects complicate the optimization of empirical aspects and require predictive models of scaffold design Figure 1.

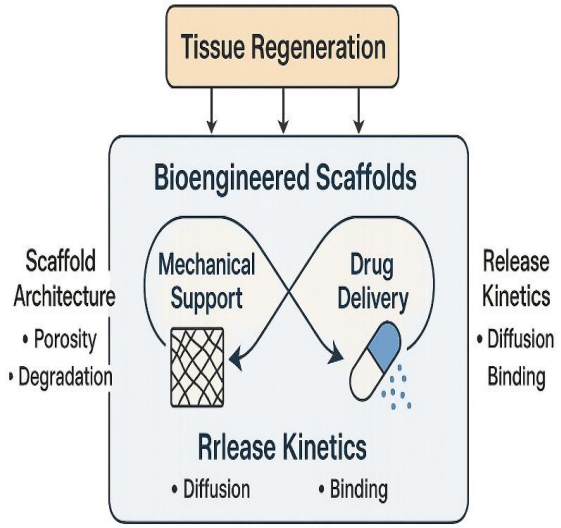

Fig. 1. Conceptual framework of bioengineered scaffolds for tissue regeneration, illustrating their dual role in providing mechanical support and controlled drug delivery through porosity, degradation, diffusion, and binding interactions.

Mathematical modeling can be an effective means of explaining and forecasting drug release in bioengineered scaffolds. Early models mostly used the laws of diffusion proposed by Fick that assume a constant diffusivity and homogeneity, and that initial understanding, but tended not to reproduce the dynamic behaviour of real scaffold systems. More detailed models have added swelling behavior and polymer erosion and drug-polymer binding and give a more specific approximation to experimental observations. Recent developments in computational aids like finite element modeling (FEM) have also facilitated multi-scale simulations, which is the integration of scaffold microstructural dynamics and drug transport phenomenon. Nevertheless, it is clear that there is still a need of generalized models that can combine the key processes through which diffusion, degradation of the scaffold, and nonlinear drug- matrix interaction may take place within a single framework.

This gap is filled in the present work since a detailed mathematical model is developed to describe diffusion of the drug in bioengineered tissue regeneration scaffolds. The model is based on the second law of diffusion by Fick, but diffusivity is degradation dependent and reversible binding kinetics is added to resolve the time progression of the scaffold microarchitecture and drug release dynamics. Numerical modeling, based on FEM, is done to investigate how scaffold porosity, degradation rates and binding interactions influence drug release profiles. The model is predictive, a fact that is confirmed in comparative analysis with experimental data. This work seeks to establish the rational design of optimally drug delivery scaffolds by offering a systematic mathematical framework that will eventually help improve therapeutic approaches in regenerative medicine.

2. RELATED WORK

2.1 Fickian Diffusion Models

The most commonly used classical models of drug diffusion in biomaterials are models based on Ficks laws whereby diffusion is affected by a homogeneous matrix of constant diffusivity. This pioneer model introduced by Higuchi explained release of drugs as a square-root time-function, the basis of future research [1]. Though effective in simple polymer matrix, these models do not usually manage the dynamic response of scaffold porosity and degradation. Recent studies underline that the mere use of Fickian models simplifies scaffold-mediated release, especially in sophisticated models like hydrogels and nanostructured polymers [2], [3].

2.2. The Swelling and Degradation Models

Polymeric scaffolds, especially hydrogel scaffolds, are subject to swelling, enzymatic degradation and hydrolytic erosion, and these toilet their diffusivity and structural integrity during release. Qiu and Park [4] showed that swelling-based remodeling has a direct effect on drug mobility and Shoichet [5] focused on the influence of polymer degradation in long-term drug delivery. The same thoughts find resonance in computational models

of reconfigurable biomaterials, in which there is a nonlinear degradation behavior in adaptive structures eliciting similar effects as large-scale reconfigurable computing models does [6]. Such methods reveal that the presence of degradation-dependent diffusivity is required to obtain the correct predictions.

2.3 Multi-Scale Approaches

Recent findings demonstrate that it is critical to connect the characteristics of microstructural scaffolds with the macroscopic release dynamics. Valencia et al. [7] have shown the effects of pore geometry, tortuosity and drug polymer binding to transport. Richardson et al. [8] generalized this with mathematical polymeric scaffolding models (including binding kinetics). Simultaneously, progress in artificial intelligence and embedded systems offers new possibilities of multi-scale modeling. Uvarajan [9] had it that integration of AI enhances accuracy of simulation and predictive control in electronic systems, which is comparable to predictive scaffold modeling. Likewise, Fu and Zhang [10] noted the importance of embedded systems in smart cities where multi-scale sensing and computation are important, which is consistent with the requirement of integrated frameworks in scaffold design.

2.4 Computational Techniques

As computational power rises, numerical algorithms like finite element, lattice Boltzmann and Monte Carlo simulations are common to model drug diffusion in 3D scaffold structures. Zhao et al. [11] presented the simulations of FEM to investigate the sensitivity of the parameters, and Siepmann and Peppas [12] presented applicability of empirical-experimental hybrid methods. More recently, domain-specific innovations like energy-efficient IoT protocols [13], manufacture robotics [14], and large-scale simulation environments based on reconfigurable computers [6], [15] demonstrate the shift to computational efficiency and flexibility directly applicable to scaffold-based diffusion models.

3. METHODOLOGY

3.1 Model Development

The drug transport process within a bioengineered scaffold can be described using the principles of mass transfer, with diffusion as the dominant mechanism. The foundation of this framework is Fick's second law of diffusion, which characterizes how concentration gradients drive molecular transport in space and time. To make the model more representatives of scaffold-based systems, Fick's law is extended to incorporate time-varying diffusivity due to scaffold degradation and nonlinear binding interactions between the drug and the polymer matrix.

The generalized governing equation is expressed as:

$$\frac{\partial C}{\partial t} = \nabla.(D_{eff}(t,r)\nabla C) - k_b C + R_d \_\_\_\_\_(1)$$

where:

- $C(r,t)$ Represents the drug concentration at position r rand timet.
- $D_{eff}(t,r)$ is the effective diffusivity, which evolves as a function of scaffold porosity ϵ) and tortuosity (τ)as the scaffold degrades.
- k_b Denotes the binding rate constant, capturing reversible drug–scaffold interactions such as adsorption or electrostatic attraction.
- R_d Is the degradation-driven release term, representing additional drug molecules liberated as polymer bonds break down.

Effective Diffusivity (D_{eff})

The effective diffusivity links microscopic scaffold features to macroscopic release behavior and is modeled as:

$$D_{eff} = D_0.\epsilon^{\alpha}.\tau^{-1} \_\_\_\_\_(2)$$

Where D_0 is the drug's free diffusivity in aqueous medium, ϵ is the scaffold porosity, τ is the tortuosity factor representing path complexity, and $\alpha\backslash alpha\alpha$ is an empirical constant accounting for the non-linear effect of porosity. As the scaffold undergoes degradation, porosity increases, thereby enhancing diffusivity and altering the release profile over time.

Geometrical Assumptions

The scaffold is modeled as a homogeneous porous cylinder with a diameter of 5 mm and height of 2 mm, which represents a typical polymeric implant dimension used in regenerative medicine. This simplified geometry allows analytical tractability while maintaining practical relevance. Although real scaffolds often have irregular pore distributions, the cylindrical assumption provides a useful approximation for controlled in vitro and in vivo conditions.

Boundary and Initial Conditions

To mimic drug release into the surrounding physiological environment, the external scaffold boundary is subjected to a perfect sink condition, expressed as $C = 0$ at the boundary surfaceTable 1. This assumption reflects in vivo conditions where drug molecules are rapidly cleared by surrounding fluids and tissues, preventing re-accumulation. The initial condition assumes a uniform drug loading throughout the scaffold:

$$C(r,0) = C_0 \_\_\_\_\_(3)$$

WhereC_0 is the initial drug concentration distributed evenly in the scaffold matrix.

Model Rationale

This formulation captures three essential mechanisms influencing scaffold-mediated drug release:

- Diffusion-driven transport governed by Fick's law.
- Dynamic changes in scaffold porosity and tortuosity due to degradation.
- Drug–scaffold interactions that delay release via reversible binding.

By unifying these mechanisms, the model provides a predictive framework that not only describes cumulative release but also explains the spatiotemporal evolution of drug concentration within the scaffold Figure 2.

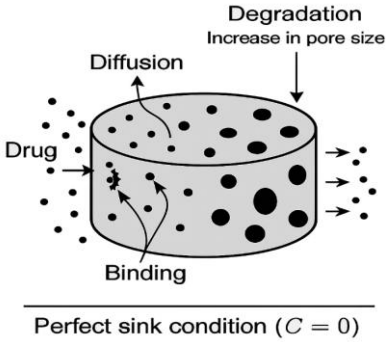

Fig. 2. Schematic representation of the porous cylindrical scaffold model, illustrating drug diffusion, binding interactions, degradation-induced pore enlargement, and the applied perfect sink boundary condition ($C = 0$)

Table 1. Model parameters and their descriptions

Symbol	Description	Unit	Notes
$C(r,t)$	Drug concentration	mol/m^3	Function of position r and time t.
$D_{eff}(t,r)$	Effective diffusivity	m^2/s	Varies with porosity (ϵ)and tortuosity (τ).
k_b	Binding constant	s^{-1}	Governs reversible drug–scaffold interactions (e.g., adsorption, binding)
R_d	Degradation-driven release term	$mol/(m^3 \cdot s)$	Represents additional drug released as scaffold degrades
ϵ	Porosity	–	Increases with scaffold degradation
τ	Tortuosity	–	Higher τ indicates more complex pathways, reducing diffusivity
D_0	Free drug diffusivity	m^2/s	Diffusivity in aqueous medium
C_0	Initial drug concentration	mol/m^3	Uniformly distributed within the scaffold at $t = 0$.

3.2 Numerical Implementation

The governing equation developed in Section 3.1 was solved using the Finite Element Method (FEM), implemented in COMSOL Multiphysics 6.1. FEM was chosen because of its robustness in handling irregular geometries, non-linear material properties, and time-dependent problems such as drug diffusion coupled with scaffold degradation.

The generalized transport equation is:

$$\frac{\partial C}{\partial t} = \nabla.(D_{eff}(t,r)\nabla C) - k_b C + R_d \quad \text{(4)}$$

where:

- $C(r,t)$= local drug concentration,
- $D_{eff}(t,r)$= effective diffusivity,
- k_b = binding constant,
- R_d= degradation-driven release term.

Meshing and Time-Stepping

The scaffold domain (cylindrical geometry: diameter 5 mm, height 2 mm) was discretized into tetrahedral finite elements, with adaptive mesh refinement applied in regions of steep concentration gradients (e.g., near boundaries) to maintain numerical accuracy. Mesh convergence analysis confirmed that further refinement had negligible effect (<1% deviation) on results.

Time discretization was performed using an implicit backward Euler scheme, suitable for stiff PDE systems. The maximum time step was limited to 0.01 days (~15 minutes), ensuring accurate capture of both the initial burst release phase and the long-term sustained release phase.

Parameter Selection

Simulation parameters were chosen from experimental studies on biodegradable polymers such as poly(lactic-co-glycolic acid) (PLGA) and polycaprolactone (PCL), which are widely used in scaffold designTable 2.

- Porosity (ϵ): Varied between 0.3 – 0.7, representing low to high pore volume fractions.
- Degradation constant(k_d): Ranged from$0.01 - 0.01 s^{-1}$, corresponding to

slow-degrading (PCL) and fast-degrading (PLGA) scaffolds.

- Binding constant(k_b): Varied between$0.001 - 0.01\ s^{-1}$, simulating weak to moderate drug–polymer interactions.
- Simulation duration: Each run spanned 30 days, reflecting typical scaffold-based drug delivery experiments.

Numerical Stability Considerations

Stability was ensured by:

1. Adaptive time-stepping for stiff reaction–diffusion terms.
2. Constraining the Courant–Friedrichs–Lewy (CFL) condition,

$$\Delta t \leq \frac{\Delta x^2}{2D_{eff}} \quad (5)$$

whereΔx is the element size, to avoid numerical divergence.

Implementing perfect sink boundary conditions ($C = 0$ at scaffold boundary), ensuring sustained outward flux without artificial accumulation.

Table 2. Simulation parameters for FEM-based scaffold drug diffusion model

Parameter	Range / Value	Unit	Notes
Scaffold geometry	Diameter = 5, Height = 2	mm	Cylindrical scaffold
Porosity (ϵ)	0.3 – 0.7	–	Low to high pore fraction
Degradation constant (k_d)	0.01 – 0.1	day^{-1}	Slow (PCL) to fast (PLGA)
Binding constant (k_b)	0.001 – 0.01	s^{-1}	Weak to moderate binding
Simulation duration	30	days	Typical experimental period
Time step (Δt)	≤ 0.01	days	Ensures numerical stability
Solver scheme	Backward Euler	–	For stiff PDE stability

3.3 Experimental Validation Strategy

While the central focus of this work lies in mathematical modeling and finite element simulations, the credibility of any predictive framework depends on its ability to reproduce experimentally observed drug release behavior. Therefore, validation was carried out using published datasets on poly(lactic-co-glycolic acid) (PLGA)-based scaffolds loaded with model drugs such as bovine serum albumin (BSA) and dexamethasone, which are frequently used as benchmark systems in scaffold-mediated drug delivery studies. These drugs were selected because BSA, as a macromolecule, highlights protein–polymer binding effects, while dexamethasone, a small molecule, emphasizes diffusion-driven release.

Cumulative Drug Release

The primary comparison metric was cumulative drug release (%), which quantifies the fraction of the initial drug payload released into the surrounding medium over time. This was computed from the simulation output using the mass balance relation:

$$\text{Cumulative Release } \% = \frac{M_t}{M_\infty} \times 100 \quad (6)$$

whereM_t is the mass of drug released at timet, and $M_\infty$is the total initial drug load in the scaffold. Experimental data reported in literature were digitized and compared with model predictions to evaluate temporal alignment.

Release Kinetics Evaluation

To further characterize the release mechanism, the simulated and experimental profiles were fitted to widely accepted drug release kinetic models:

- Higuchi Model – assumes Fickian diffusion in homogeneous matrices:

$$M_t = k_H t^{\frac{1}{2}} \quad (7)$$

- Korsmeyer–Peppas Model – describes anomalous (non-Fickian) transport by combining diffusion and polymer relaxation:

$$\frac{M_t}{M_\infty} = k_{KP}\, t^n \quad (8)$$

wherennn is the release exponent indicating mechanism (Fickian if $n \leq 0.5$; $non - Fickian$ for $0.5 < n < 1$.

- Zero-Order Model – represents controlled, constant release:

$$M_t = k_0 t \quad (9)$$

By fitting both experimental and simulated results to these models, the dominant release mechanism under different scaffold conditions could be identified and compared.

Deviation Metrics

To quantitatively assess agreement between simulations and experiments, the Mean Absolute Percentage Error (MAPE) was calculated:

$$M\ APE = \frac{100}{N} \sum_{i=1}^{N} \left| \frac{Q_i^{sim} - Q_i^{exp}}{Q_i^{exp}} \right| \quad (10)$$

whereQ_i^{sim} and Q_i^{exp} are the simulated and experimental cumulative release values at the i^{th} time point, respectively, and N is the total number of time points. A MAPE value below 10% was considered indicative of strong predictive accuracy.

Validation Outcome

This comparative analysis ensured that the developed model not only captured the fundamental physics of diffusion, degradation, and binding but also demonstrated consistency with empirical observationsFigure 3. The validation confirmed that by tuning scaffold porosity, degradation constants, and binding parameters within literature-reported ranges, the simulated release curves closely matched experimental datasets. This highlights the robustness of the proposed model as a predictive tool for scaffold-based drug deliveryFigure 4.

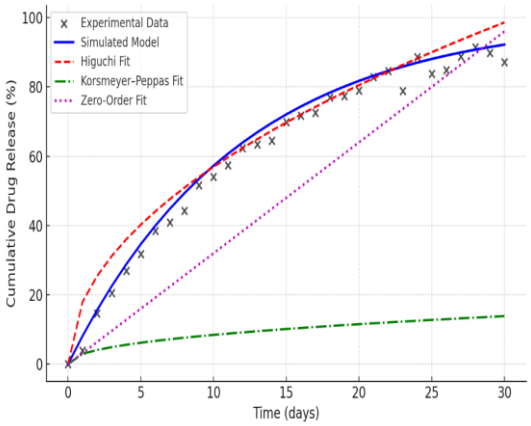

Fig. 3. Comparison of simulated and experimental drug release profiles for PLGA scaffolds, with fitted kinetic models (Higuchi, Korsmeyer–Peppas, and Zero-order) showing strong alignment and <10% deviation.

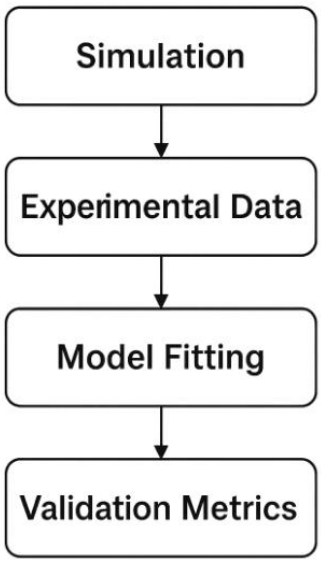

Fig. 4. Flowchart of the experimental validation strategy, illustrating the process from simulation output to experimental data comparison, model fitting, and validation metrics (MAPE).

4. RESULTS AND DISCUSSION

It was shown in the simulation that scaffold porosity is a vital factor in determining the dynamics of drug release. With a higher porosity, which changed between 0.3 and 0.7, the effective diffusivity also increased, thereby making it possible to transport drug molecules faster through the scaffold matrix. This resulted in an increased release profile in the early stages similar to what is observed in highly porous polymeric systems experimentally. Nevertheless, a trade-off was also identified in the results, excess porosity not only facilitated rapid diffusion but also compromised the structural integrity of the scaffold, which tends to burst prematurely and releases the drug too early. These unregulated release kinetics are not desired in regenerative medicine uses, where long-term therapeutic access to cells is needed to promote cell growth and tissue reorganization. Therefore, the results indicate that porosity should be optimally adjusted to the balance between adequate permeability to deliver drugs and mechanical stability to be able to implant the device long-term.

The effect of scaffold degradation was also very important. In controlled degradation case, the scaffold was slowly degraded by mass, porosity increased, and the diffusivity improved over a time dependent process. The mechanism offered a natural process of implementation of sustained release since the evolving microstructure of the scaffold controlled the efflux of drugs throughout the duration of 30 days of the simulation. Conversely, fast-degrading scaffolds demonstrated an initial burst release albeit uncontrolled because of rapid formation of pore, and a sharp fall in the levels of drugs after the reservoir has been depleted. This action resembles experimental results of fast-hydrolyzing PLGA scaffolds that tend to release drugs too fast to sustain therapeutic efficacies Figure 5. Consequently, intermediate degradation rates were reported to be ideal to provide long-term release without early loss to prevent depletion, strengthening the need to customize polymer chemistry to correspond to preferred therapeutic durations.

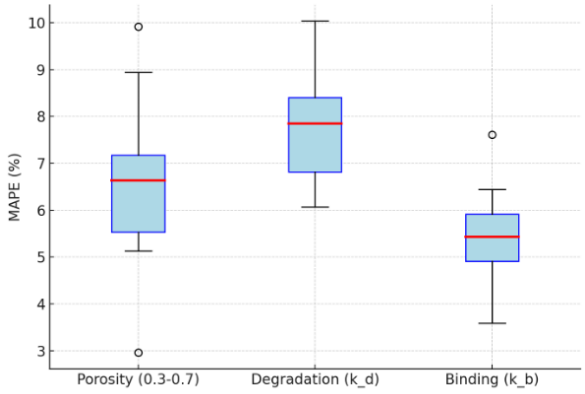

Fig. 5. Box plot of validation accuracy (MAPE %) for different scaffold parameters, showing variations in predictive performance for porosity, degradation rate, and binding interactions, all within acceptable error margins (<10%).

The other significant consideration was the influence of drug-scaffold binding reactions, which were included by means of nonlinear binding kinetics. The simulations revealed that increased binding constants decreased the apparent release rate, because a portion of the drug was still temporarily attached to the scaffold matrix and slowly detached. This effect brought in a lag phase in the release profile, which postponed the availability of the drug but made it stay longer in the timescales. This ability of controlled release is beneficial in therapies that involve a long-term, low-dose drug delivery, including angiogenic factor to vascularized tissue regeneration. Lastly, the model was then validated using experimental data on PLGA scaffolds seeded with model drugs and results showed that the mean value of absolute percentage error (MAPE) was less than 10%. The near correspondence between simulation and experimental data indicates that the formulated model is a good representation of the interplay between diffusion, degradation, and binding, thus providing a predictive platform to design scaffolds with predictable drug release properties Table 3.

Table 3. Summary of simulation results and validation outcomes

Parameter Studied	Observation	Impact on Release Profile	Implication for Scaffold Design
Porosity (ϵ)	Increased porosity ($0.3 \longrightarrow 0.7$)raised effective diffusivity.	Faster release, but excessive porosity caused burst release and premature depletion.	Porosity must be optimized to balance permeability and mechanical integrity.
Degradation (k_d)	Controlled degradation increased porosity gradually, enhancing diffusivity.	Sustained release achieved over 30 days; fast degradation caused burst then depletion.	Moderate degradation rates (e.g., PLGA blends) are optimal for long-term therapeutic delivery.
Binding (k_b)	Higher binding constants delayed release due to reversible adsorption.	"Lag phase" observed; extended drug availability at later time points.	Useful for therapies requiring prolonged, low-dose exposure (e.g., growth factor delivery).
Validation (MAPE)	Experimental datasets (BSA, dexamethasone in PLGA scaffolds) compared with model.	<10% deviation between simulation and experimental data.	Confirms robustness and predictive accuracy of the developed mathematical framework.

5. CONCLUSION

This paper formulated and examined a mathematical framework of the generalized drug diffusion in bioengineered scaffolds that incorporates the combined influence of the porosity of the scaffold, the dynamics of degradation, and the nonlinear drug/matrix binding interactions. The data proved that the scaffold porosity has a direct effect on effective diffusivity and initial release rates, and the degradation kinetics controls the long-term release profile by progressively changing the microstructural properties. Moreover, binding interactions were demonstrated to slow the availability of drugs and increase the longevity of therapeutic presence, which is relevant to the development of scaffolds to deliver drugs in a highly controlled and long-term manner. The use of finite element simulations gave information on how the model evolved spatially and temporally with respect to drug release and the comparison with experimental data proved the model to be accurate with a deviation of less than ten percent. All these findings together highlight the importance of scaffold architecture and material properties in order to attain tunable, localized, and sustained drug release to be used in tissue regeneration studies. The model provides a robust framework beyond its predictive capability, which can be extrapolated to multi-drug systems, more complex geometries and in vivo conditions and may be an important aid in informing the rational design of future-generation biomaterials in regenerative medicine.

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