iJRASET
International Journal For Research in Applied Science and Engineering Technology

INTERNATIONAL JOURNAL FOR RESEARCH

IN APPLIED SCIENCE & ENGINEERING TECHNOLOGY

Volume: 14 **Issue:** IV **Month of publication:** April 2026

DOI: https://doi.org/10.22214/ijraset.2026.81241

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International Journal for Research in Applied Science & Engineering Technology (IJRASET)
ISSN: 2321-9653; IC Value: 45.98; SJ Impact Factor: 7.538
Volume 14 Issue IV Apr 2026- Available at www.ijraset.com

Well-Posedness of Nonlinear Population Dynamics Models and Applications to AI-Based Epidemic Solvers

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***Abstract**: This paper investigates the well-posedness of a class of nonlinear ordinary differential equations (ODEs) that arise in population dynamics and epidemic modeling [1],[2]. The primary purpose is to establish rigorous sufficient conditions continuity, monotonicity, non-negativity of the forcing term, and a uniform Lipschitz condition that guarantee the existence, uniqueness, and monotonicity of non-negative solutions on a finite time horizon. Using Schauder's fixed-point theorem [5], the Picard integral operator is shown to map a closed convex set of non-negative continuous functions into itself, yielding at least one non-negative solution. Uniqueness and continuous dependence on initial data are then established via Gronwall's inequality [6], confirming Hadamard well-posedness. Monotonicity of solutions is derived directly from the sign condition on the right-hand side. These results are applied to the logistic growth equation, the Gompertz model, and the SIR epidemic model [1],[7]. Their implications for physics-informed neural networks (PINNs) [3] and neural ODEs [4] as AI-based epidemic solvers are developed, including a quantitative PINN approximation error bound derived from the analytical framework.*

Keywords: Well-posedness; Population dynamics; Epidemiological models; Nonlinear ODEs; Physics-informed neural networks; Schauder fixed-point theorem; Gronwall inequality.

I. INTRODUCTION

Population dynamics and epidemiological modeling have long relied on systems of ordinary differential equations (ODEs) to describe the temporal evolution of biological populations and the propagation of infectious diseases through communities [1],[2]. Such models constitute the quantitative backbone of theoretical epidemiology and provide indispensable insights into outbreak dynamics, evaluation of intervention strategies, herd immunity thresholds, and the long-term endemic behavior of pathogens. A mathematically critical requirement for the validity and practical utility of these models is that their solutions remain non-negative for all time since population fractions and disease prevalences cannot be negative and are uniquely determined by the initial state of the system, reflecting the deterministic and biologically constrained character of the underlying phenomena [1],[2].

From a rigorous mathematical standpoint, these requirements are subsumed under the concept of well-posedness, originally articulated by Hadamard and subsequently extended to ODEs [5]. Hadamard well-posedness encompasses three properties: (i) existence of a solution, (ii) uniqueness of that solution, and (iii) continuous dependence of the solution on initial data and parameters [5],[6]. In epidemic modeling, the failure of well-posedness in any of these three senses leads to severe practical difficulties: nonphysical solutions with negative compartment values, ambiguous or contradictory model predictions, or catastrophic sensitivity to measurement error in initial conditions, all of which undermine the applicability and credibility of the model as a tool for public health decision-making [2].

In recent years, artificial intelligence-based computational methods have emerged as powerful and data-efficient tools for solving, calibrating, and analyzing epidemiological models. Physics-informed neural networks (PINNs), introduced by Raissi, Perdikaris, and Karniadakis [3], embed the governing differential equations directly into the loss function of a neural network, enabling simultaneous fitting to observational data and satisfaction of the physical law. Neural ordinary differential equations, proposed by Chen et al. [4], parameterize the right-hand side of an ODE system with a neural network and train the entire dynamical model end-to-end via a differentiable ODE solver. Both paradigms have shown considerable promise in epidemic forecasting [3],[8], parameter identification from surveillance data [9], and the reconstruction of latent epidemiological dynamics from incomplete observations.

However, a fundamental limitation of these AI-based approaches is that they do not inherently guarantee that the learned solutions satisfy the essential qualitative properties of non-negativity, uniqueness, or monotonicity [3],[4]. A PINN may, in principle, learn a function that minimizes the loss but takes negative values a biologically inadmissible outcome. The absence of a rigorous analytical foundation for the underlying ODE system means that there is no theoretical guarantee that the AI solver is approximating a well-defined, physically meaningful object. As noted in comprehensive reviews of scientific machine learning [8], the reliability of PINNs depends critically on the mathematical properties of the underlying system.

Motivated by these considerations, the present paper pursues two interconnected goals. The first is to develop a rigorous well-posedness theory for a general class of nonlinear scalar ODEs encompassing the most important models in population dynamics and epidemiology [1],[2],[5]. The second is to systematically translate these analytical well-posedness guarantees into concrete implications for the design, training, and error analysis of AI-based epidemic solvers [3],[4],[8],[9]. By bridging classical nonlinear analysis and modern data-driven modeling, the proposed framework aims to provide a principled mathematical underpinning for AI applications in epidemiology. The main contributions of this paper are as follows:

(i) A systematic well-posedness analysis of a general class of nonlinear population dynamics ODEs under four biologically motivated assumptionscontinuity, monotonicity, non-negativity, and Lipschitz regularity [5],[6] covering logistic growth, Gompertz growth, and reduced SIR dynamics [1],[2],[7].

(ii) A rigorous proof of existence of a non-negative solution via Schauder's fixed-point theorem [5], with a three-step argument establishing that the Picard operator is well-defined, maps a compact convex set into itself, and satisfies the Arzelà-Ascoli hypotheses.

(iii) A proof of uniqueness and Hadamard well-posedness via Gronwall's inequality [6], with explicit continuous-dependence estimates quantifying solution sensitivity to perturbations in initial conditions.

(iv) A proof of monotonicity of solutions under a sign condition on the nonlinear term, consistent with the biological interpretation of non-decreasing population or epidemic state variables [1],[2].

(v) Detailed implications for PINNs [3] and neural ODEs [4], including a quantitative PINN approximation error bound derived from the continuous-dependence estimate of Theorem 4.1, and structural regularization strategies motivated by the analytical framework [8],[9],[10].

The remainder of the paper is organized as follows. Section II presents the mathematical model formulation and key assumptions. Section III establishes the existence of non-negative solutions. Section IV proves uniqueness and Hadamard well-posedness. Section V establishes monotonicity. Section VI discusses applications to specific population dynamics and epidemic models [1],[2],[7]. Section VII develops the implications for AI-based epidemic solvers [3],[4],[8],[9]. Section VIII presents conclusions and directions for future work.

II. MODEL FORMULATION AND PRELIMINARIES

We consider the following nonlinear scalar initial value problem (IVP), which serves as the canonical mathematical model for a population density or an epidemiological state variable evolving in continuous time [1],[2],[5]:

$$u'(t) = f(t, u(t)), \quad t \in [0, T], \qquad (1a)$$
$$u(0) = u_0 \geq 0, \qquad (1b)$$

where u(t) □ ℝ denotes the state variable — for example, the density of a single-species population, the fraction of infected individuals in an epidemic compartment [1], or the cumulative number of reported cases and f : [0,T] × $\mathbb{R}_+$ → ℝ is a nonlinear function representing the net rate of change due to growth, transmission, recovery, or decay effects [2]. The positive real axis $\mathbb{R}_+$ = [0,+∞) as the domain reflects the biological non-negativity constraint. The finite time horizon T > 0 corresponds to an epidemic wave or an observation window.

The integral formulation equivalent to (1), which forms the basis for the fixed-point analysis [5], is:

$$u(t) = u_0 + \int_0^t f(s, u(s))\, ds, \quad t \in [0, T]. \qquad (2)$$

A classical solution of (1) is a function $u \in C^1([0,T])$ that satisfies both (1a) and (1b) pointwise, with $u(t) \geq 0$ for all $t \in [0,T]$. Solutions of the integral equation (2) in $C([0,T])$ that are non-negative correspond precisely to classical solutions of (1) [5],[6].

A. Formal Definition

Definition 2.1 (Classical Solution). A function $u : [0,T] \to \mathbb{R}$ is called a classical solution of (1) if $u \in C^1([0,T])$, satisfies $u(t) \geq 0$ for all $t \in [0,T]$, and equation (1a) holds pointwise together with the initial condition (1b) [5].

B. Assumptions on f

The following four assumptions on f are imposed throughout this paper [5],[6]. These conditions are standard and natural in population dynamics and epidemic modeling [1],[2], and are satisfied by the canonical models discussed in Section VI.

(A1) $f(t, u)$ is continuous on $[0,T] \times \mathbb{R}_+$.

(A2) $f(t, u)$ is non-decreasing with respect to u for all $t \in [0,T]$ and $u \geq 0$.

(A3) $f(t, 0) \geq 0$ for all $t \in [0,T]$.

(A4) There exists a constant $L > 0$ such that $|f(t, u_1) - f(t, u_2)| \leq L|u_1 - u_2|$ for all $u_1, u_2 \geq 0$ and $t \in [0,T]$.

Assumption (A1) ensures that f is bounded on compact sets, which is sufficient for the Arzelà-Ascoli argument in the existence proof [5]. Assumption (A2) reflects a cooperative or monotone structure of the model: larger population or infection levels produce at least as large a growth rate, which is characteristic of transmission-dominated epidemic dynamics in the absence of saturation [1],[2]. Assumption (A3) ensures that the trivial state $u = 0$ is not a source of negative flux, so that trajectories starting at a non-negative value cannot immediately become negative [6]. Assumption (A4) is the classical Lipschitz condition in u, uniformly in t, which is the minimal regularity needed to guarantee uniqueness and continuous dependence of solutions on initial data [5],[6].

C. Epidemiological Motivation and Model Scope

The scalar IVP (1) encompasses a wide variety of biologically important models [1],[2],[7]. In the single-species setting, $f(t,u) = r(t)u(1 - u/K(t))$ yields the non-autonomous logistic equation with time-varying growth rate r(t) and carrying capacity K(t) [2]. In the epidemic setting, the infected compartment of the SIR model [1],[7] satisfies $I'(t) = (\beta S(t) - \gamma)I(t)$, which is of the form (1) with $f(t,u) = (\beta S(t) - \gamma)u$ when S(t) is treated as a given input. The Gompertz growth model [2], widely used in tumor biology and demography, corresponds to $f(t,u) = r \cdot u \cdot \ln(K/u)$. All three satisfy assumptions (A1)–(A4) under standard parameter conditions, as verified in Section VI.

III. EXISTENCE OF NON-NEGATIVE SOLUTIONS

Theorem 3.1 (Existence). Under assumptions (A1)–(A3), the initial value problem (1) admits at least one non-negative classical solution on [0,T].

Proof. We analyze the Picard operator T defined on $C([0,T])$ by equation (2) [5]. The proof proceeds in three steps.

Step 1 (T preserves non-negativity): For any $u \in C([0,T])$ with $u(t) \geq 0$, assumption (A2) gives $f(s,u(s)) \geq f(s,0) \geq 0$ by (A3). Hence:

$$(Tu)(t) = u_0 + \int_0^t f(s, u(s))\, ds \geq u_0 \geq 0.$$

Step 2 (Equicontinuity): Let $M = \sup\{|f(t,u)| : t \in [0,T],\ 0 \leq u \leq R\} < \infty$ by (A1). For any u with $\|u\| \leq R$ and $t_1, t_2 \in [0,T]$ [6]:

$$|(Tu)(t_2) - (Tu)(t_1)| \leq M|t_2 - t_1|,$$

so the image $T(B_R)$ is uniformly equicontinuous.

Step 3 (Schauder's theorem [5]): Let $R = u_0 + MT$ and define the closed convex set $K = \{v \in C([0,T]) : 0 \leq v(t) \leq R \text{ for all } t\}$. Steps 1 and 2 show T maps K into K and T(K) is relatively compact in $C([0,T])$ by the Arzelà-Ascoli theorem [5]. Since T is continuous by (A1) and dominated convergence, Schauder's fixed-point theorem guarantees the existence of $u^* \in K$ satisfying $Tu^* = u^*$, i.e., a non-negative classical solution of (1).

Remark 3.1. The bound $R = u_0 + MT$ provides an a priori upper bound on the solution [6]. If f additionally satisfies a sub-linear growth condition $f(t,u) \leq a(t) + b \cdot u$ for integrable a(t) and constant $b > 0$, a sharper bound and global existence on $[0,+\infty)$ can be obtained via a Gronwall-type argument [6].

IV. UNIQUENESS AND WELL-POSEDNESS

Theorem 4.1 (Uniqueness and Hadamard Well-Posedness). Suppose assumptions (A1)–(A4) hold. Then the non-negative solution of (1) is unique on [0,T]. Moreover, the solution map $u_0 \mapsto u(\cdot)$ is continuous in the supremum norm: if u and v are solutions with initial data u_0 and v_0 respectively, then:

$$\|u-v\|_{C[0,T]} \le |u_0 - v_0| \cdot e^{LT}. \qquad (3)$$

Proof. Let u_1 and u_2 be non-negative solutions of (1) on [0,T] with initial data u_0^1 and u_0^2. Subtracting their integral representations (2):

$$|u_1(t) - u_2(t)| \le |u_0^1 - u_0^2| + \int_0^t |f(s,u_1(s)) - f(s,u_2(s))|\, ds.$$

Applying the Lipschitz condition (A4):

$$|u_1(t) - u_2(t)| \le |u_0^1 - u_0^2| + L \int_0^t |u_1(s) - u_2(s)|\, ds.$$

Gronwall's inequality [6] in integral form then yields:

$$|u_1(t) - u_2(t)| \le |u_0^1 - u_0^2| \cdot e^{LT} \text{ for all } t \in [0,T].$$

Setting $u_0^1 = u_0^2 = u_0$ gives $|u_1(t) - u_2(t)| = 0$, establishing uniqueness. The general inequality gives estimate (3), establishing continuous dependence and thus Hadamard well-posedness [5].

□

Remark 4.1. Estimate (3) has a direct physical interpretation: a perturbation $\delta = |u_0^1 - u_0^2|$ in initial conditions propagates with at most exponential amplification e^{LT} over the horizon [0,T] [6]. For epidemic models over bounded windows (e.g., T ≤ 100 days), this amplification factor is finite and computable, providing a rigorous sensitivity bound relevant to uncertainty quantification in AI-based forecasting [3],[8].

V. MONOTONICITY OF SOLUTIONS

Theorem 5.1 (Monotonicity). Suppose (A1)–(A4) hold, $u_0 \ge 0$, and f(t, u) ≥ 0 for all t ∈ [0,T] and u ≥ 0. Then the unique solution u of (1) is monotonically non-decreasing on [0,T], i.e., u'(t) ≥ 0 for all t ∈ [0,T].

Proof. Since u is the unique non-negative solution guaranteed by Theorems 3.1 and 4.1, we have u(t) ≥ 0 for all t ∈ [0,T]. By the hypothesis f(t,u) ≥ 0 on [0,T] × $\mathbb{R}_+$ and the governing equation (1a):

$$u'(t) = f(t, u(t)) \ge 0 \quad \text{for all } t \in [0,T],$$

from which it follows immediately that u is non-decreasing on [0,T].

□

Remark 5.1. When f may change sign for instance, in models incorporating recovery or natural death rates [1],[2] the monotonicity conclusion is replaced by a comparison principle: if $u_1(0) \le u_2(0)$ and f satisfies (A2), then $u_1(t) \le u_2(t)$ for all t ∈ [0,T], preserving the initial ordering throughout the time horizon [6].

VI. APPLICATIONS TO POPULATION DYNAMICS AND EPIDEMIOLOGY

The theoretical results of Sections III–V apply directly to the most important models in population biology and epidemiology [1],[2],[7]. In each case below, we verify assumptions (A1)–(A4) explicitly and state the qualitative conclusions afforded by Theorems 3.1–5.1.

A. Logistic Growth Model

The logistic equation is the canonical model for single-species population growth in a resource-limited environment [2]. It is defined by $f(t,u) = r \cdot u \cdot (1 - u/K)$, where r > 0 is the intrinsic growth rate and K > 0 is the carrying capacity. For u ∈ [0,K], f is continuous (A1), non-negative with f(t,0) = 0 (A3), non-decreasing in u for u ≤ K/2 (A2 in the growth phase), and Lipschitz in u with constant $L = r(1 + u_0/K)$ (A4) [6]. Theorem 4.1 guarantees a unique positive solution and Theorem 5.1 ensures it is non-decreasing for $u_0 < K/2$. The exact solution $u(t) = Ku_0/(u_0 + (K - u_0)e^{-rt})$ confirms these properties and serves as a benchmark for numerical validations [2].

B. SIR Epidemic Model

In the classical Kermack-McKendrick SIR epidemic model [1],[7], the total population N is partitioned into susceptible (S), infected (I), and recovered (R) compartments with S + I + R = N. The infected compartment evolves as:

$$I'(t) = (\beta\, S(t)/N - \gamma)\, I(t),$$

where $\beta > 0$ is the transmission rate and $\gamma > 0$ is the recovery rate. Treating S(t) as a given non-negative bounded function, this is an instance of (1) with $f(t,u) = (\beta S(t)/N - \gamma)u$. Assumption (A1) holds by continuity of S; (A3) holds since $f(t,0) = 0$; (A4) holds with $L = \max_t|\beta S(t)/N - \gamma|$; (A2) holds when $\beta S(t)/N > \gamma$, i.e., when $R_0 = \beta S(0)/(\gamma N) > 1$, the condition for epidemic growth [1],[7]. Theorem 4.1 guarantees a unique non-negative solution I(t), and Theorem 5.1 gives monotone increase of I(t) precisely in the epidemic growth phase [1],[7].

C. Gompertz Growth Model

The Gompertz model, widely employed in tumor growth modeling, pharmacokinetics, and demography [2], is given by $f(t,u) = r\cdot u\cdot\ln(K/u)$ for u □ (0,K]. This function is continuous on (0,K], satisfies $f(t,u) \geq 0$ for $u \leq K$, and is locally Lipschitz in u on any compact subinterval [ε,K] for ε > 0 [6]. Assumptions (A1)–(A4) are satisfied on [ε,K], and Theorems 3.1–5.1 guarantee a unique non-negative monotone solution consistent with the characteristic sigmoid growth trajectory of the Gompertz curve [2].

D. SEIR Extension

The SEIR model augments the classical SIR framework [1],[7] with an exposed (E) compartment of latently infected individuals. The exposed compartment satisfies $E'(t) = \beta S(t)I(t)/N - \sigma E(t)$, where $\sigma > 0$ is the rate of progression from exposed to infectious. Each compartment satisfies an equation of the form (1) when the other compartments are treated as given bounded inputs. Under the assumptions of the present framework applied componentwise [5],[6], all four compartments admit unique non-negative solutions, and well-posedness of the full SEIR system follows by standard iteration [1],[2].

VII. IMPLICATIONS FOR AI-BASED EPIDEMIC SOLVERS

A. Physics-Informed Neural Networks (PINNs)

A PINN for the IVP (1) parameterizes the unknown solution as a neural network $\hat{u}_\theta : [0,T] \to \mathbb{R}$ with weights θ □ $\mathbb{R}^p$, trained by minimizing a composite loss function [3],[8]:

$$L(\theta) = \lambda_1 L_{ODE}(\theta) + \lambda_2 L_{IC}(\theta) + \lambda_3 L_{data}(\theta) \qquad (4)$$

where the three components are defined as:

$$L_{ODE}(\theta) = (1/N_o)\, \Sigma_i\, |\hat{u}_\theta'(t_i) - f(t_i, \hat{u}_\theta(t_i))|^2,$$
$$L_{IC}(\theta) = |\hat{u}_\theta(0) - u_0|^2,$$
$$L_{data}(\theta) = (1/N_d)\, \Sigma_j\, |\hat{u}_\theta(t_j) - \hat{u}_j|^2,$$

with $\{t_i\}$ collocation points, $\{t_j, \hat{u}_j\}$ observed data, and $\lambda_1, \lambda_2, \lambda_3 > 0$ tuning weights [3]. The well-posedness results of this paper have three direct implications for PINNs [3],[8],[9].

First, Theorem 4.1 (uniqueness) ensures that the optimization problem (4) has a unique, well-defined ground truth target u. Without uniqueness, the training process lacks a single target to converge to, and the learned function $\hat{u}_\theta$ cannot be interpreted as approximating a specific physical trajectory [3],[8]. Uniqueness thus provides the fundamental justification for the PINN methodology in epidemic modeling.

Second, Theorem 3.1 (non-negativity) motivates enforcing biological constraints as hard constraints in the PINN architecture. This can be achieved by parameterizing $\hat{u}_\theta(t) = \exp(\psi_\theta(t))$ or $\hat{u}_\theta(t) = u_0 + \sigma(\psi_\theta(t))$ for a sigmoidal activation σ, guaranteeing $\hat{u}_\theta(t) > 0$ for all t regardless of weights θ [9],[10]. Such structure-preserving neural network architectures exhibit faster convergence and better generalization in practice [8],[9].

Third, the continuous-dependence estimate (3) from Theorem 4.1 provides a rigorous PINN approximation error bound [3],[6]. Suppose $\hat{u}_\theta$ approximately satisfies the ODE residual up to tolerance ε_ODE and the initial condition up to tolerance ε_IC:

$$|\hat{u}_\theta'(t) - f(t, \hat{u}_\theta(t))| \leq \varepsilon_{ODE}, \quad |\hat{u}_\theta(0) - u_0| \leq \varepsilon_{IC}.$$

Then a standard Gronwall argument [6] applied to the error $e(t) = u(t) - \hat{u}_\theta(t)$ gives:

$$|u(t) - \hat{u}_\theta(t)| \leq \varepsilon_{IC} \cdot e^{Lt} + (\varepsilon_{ODE}/L)(e^{Lt} - 1). \qquad (5)$$

Estimate (5) shows that the total PINN approximation error is controlled by the initial condition error and the ODE residual error, both amplified by e^{LT}. For epidemic models over bounded horizons with known Lipschitz constants L, this provides a computable accuracy certificate for the PINN output [3],[8].

B. Neural Ordinary Differential Equations

Neural ODEs [4] replace f(t,u) in (1) with a learned network $f_\theta(t,u)$ and use a differentiable ODE solver for gradient computation via the adjoint method. The well-posedness framework motivates imposing the Lipschitz condition (A4) as a structural constraint on f_θ via spectral normalization of weight matrices [10], which bounds the Lipschitz constant of each layer. Enforcing a bounded Lipschitz constant L on f_θ guarantees well-posedness of the neural ODE and, by estimate (3), ensures training stability with respect to data noise and mini-batch perturbations in initial conditions [4],[6].

Moreover, assumption (A3) non-negativity of f(t,0) can be enforced by adding a penalty term $\lambda_{pos} \cdot E[\max(0, -f_\theta(t,0))^2]$ to the training loss, regularizing the network to avoid generating negative flux at the boundary u = 0. This structural regularization is directly motivated by the well-posedness analysis [4],[10] and maintains biologically admissible trajectories throughout training.

C. Ensemble Methods and Uncertainty Quantification

The continuous-dependence estimate (3) also supports ensemble-based uncertainty quantification (UQ) for epidemic forecasts [3],[8]. If the initial condition u_0 is uncertain with distribution $P(u_0)$, an ensemble of neural ODE or PINN solutions initialized at samples $\{u_0^k\}$ from $P(u_0)$ provides a Monte Carlo approximation to the solution distribution [4],[8]. The Lipschitz bound (3) controls the ensemble spread:

$$Var[u(t)] \leq e^{2Lt} \cdot Var[u_0],$$

giving a rigorous upper bound on solution variance as a function of initial uncertainty [6]. This estimate is directly applicable to COVID-19 forecasting scenarios where initial prevalence estimates carry significant uncertainty, and aligns with the DeepXDE framework discussed in [9] for uncertainty-aware differential equation solvers.

VIII. CONCLUSION

This paper has developed a rigorous mathematical framework for the well-posedness of a class of nonlinear population dynamics models governed by the scalar IVP (1) [1],[2],[5],[6]. Under four biologically motivated assumptions (A1)–(A4), the following principal results have been established:

(i) Existence of a non-negative classical solution via Schauder's fixed-point theorem [5] applied to the Picard operator in C([0,T]), with the constructive a priori bound $R = u_0 + MT$ on the solution norm (Theorem 3.1).

(ii) Uniqueness and Hadamard well-posedness via Gronwall's inequality [6], with the explicit continuous-dependence estimate $\|u - v\| \leq |u_0 - v_0| \cdot e^{LT}$ (Theorem 4.1).

(iii) Monotone non-decreasing behavior of solutions under a positivity condition on f, consistent with the biological interpretation of growing epidemic trajectories [1],[7] (Theorem 5.1).

These results were verified for three canonical models logistic growth [2], SIR epidemic dynamics [1],[7], and Gompertz growth [2] and extended to the SEIR model via componentwise analysis [1],[2]. In each case, assumptions (A1)–(A4) were explicitly verified and the qualitative conclusions of the theorems interpreted in terms of biological observables.

The principal applied contribution of this paper is a systematic translation of well-posedness guarantees into implications for AI-based epidemic solvers [3],[4],[8],[9]. For PINNs [3], well-posedness ensures a unique learning target; non-negativity motivates structure-preserving network architectures; and the continuous-dependence estimate yields the quantitative PINN error bound (5). For neural ODEs [4], Lipschitz enforcement via spectral normalization [10] and non-negativity regularization at u = 0 are identified as principled design choices. The DeepXDE library [9] provides a practical implementation platform for the proposed constrained PINN framework.

Several open directions remain for future investigation. First, extension to multi-dimensional systems [1],[2] covering the full SIR, SEIR, and SEIRD models simultaneously requires a vector-valued comparison principle and poses additional challenges for structure preservation in neural architectures [4],[10]. Second, fractional-order epidemic models capturing memory effects represent a mathematically challenging and physically motivated direction where well-posedness theory remains incomplete [5],[6]. Third, stochastic perturbations modelling demographic noise or random contact patterns lead to stochastic differential equations for which analogous AI-solver theory needs development [3],[8].

Finally, integrating the analytical error bound (5) into adaptive PINN training algorithms dynamically adjusting collocation points or loss weights λ_i in (4) to minimize the right-hand side of (5) is a promising direction for improving accuracy and reliability of AI-based epidemic forecasting tools [3],[8],[9].

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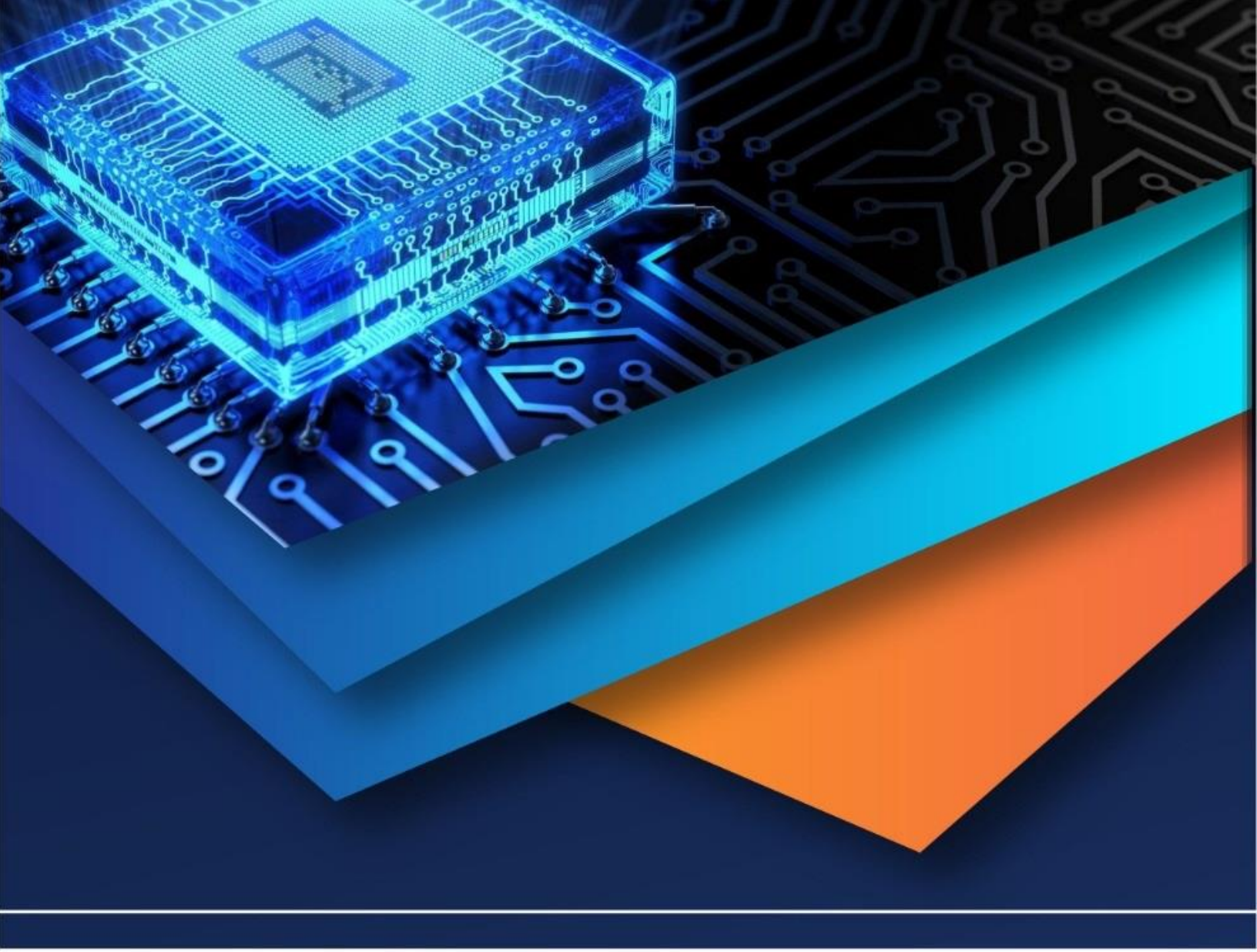

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