

Article

Stochastic Wolbachia Dynamics: Persistence, Invariant Measures, and Probability-Based Release Strategies

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Abstract

We develop a stochastic discrete-time framework for *Wolbachia* invasion in mosquito populations under environmental variability. Starting from a deterministic frequency-dependent difference equation with an Allee-type threshold, we consider the random iteration $X_{n+1} = F_{\Theta_n}(X_n)$, where Θ_n represents fluctuations in fitness cost, cytoplasmic incompatibility, and maternal transmission. We establish existence, uniqueness, positivity, forward invariance of $[0, 1]$, continuity, and pathwise order preservation, and formulate the model as a random dynamical system. Under independent environmental forcing, the process is Markovian; the associated Markov operator is Feller, and compactness of the state space implies the existence of invariant probability measures. Local behavior near extinction is characterized by the stochastic Lyapunov exponent $\lambda = \mathbb{E}[\log((1 - M)(1 - S_f))]$, with $\lambda < 0$ yielding local exponential stability of the *Wolbachia*-free state. We further introduce finite-horizon establishment probabilities and probability-based release thresholds, whose monotonicity follows from the pathwise comparison principle. Numerical simulations show how the magnitude, source, and dependence structure of environmental variability affect invasion probabilities and release requirements. The framework combines nonlinear difference equations, random dynamical systems, Markov operators, invariant measure theory, and stochastic stability in a unified analysis of threshold-dependent *Wolbachia* invasion.

Keywords: random dynamical systems; stochastic difference equations; Markov operators; invariant probability measures; stochastic stability

MSC: 37H10; 37H30; 37H15; 60J05; 37N25

1. Introduction

Mosquito-borne diseases remain among the most significant public health challenges worldwide. Dengue fever alone is responsible for hundreds of millions of infections annually, while Zika virus, chikungunya, and yellow fever continue to threaten populations throughout tropical and subtropical regions. Rapid urbanization, increasing human mobility, climate change, and the widespread evolution of insecticide resistance have substantially reduced the long-term effectiveness of conventional vector-control strategies based on insecticides and habitat elimination. Consequently, considerable attention has shifted toward environmentally sustainable biological control methods capable of reducing pathogen transmission while minimizing ecological disruption [1–5].

Among the most successful of these biological interventions is the use of the intracellular endosymbiotic bacterium *Wolbachia*. Naturally occurring in many insect species but absent from *Aedes aegypti*, *Wolbachia* modifies mosquito reproduction through cytoplasmic



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incompatibility while simultaneously reducing the mosquito's ability to transmit numerous arboviruses. Depending on the release strategy, these mechanisms may either suppress mosquito populations or replace wild mosquito populations with infected mosquitoes that exhibit substantially reduced vector competence. Large-scale field deployments in Australia, Indonesia, Brazil, Colombia, Vietnam, China, and several other countries have demonstrated remarkable reductions in dengue transmission, establishing *Wolbachia* as one of the most promising biological tools currently available for vector control [6–13].

The remarkable biological success of *Wolbachia* has stimulated an equally active mathematical literature devoted to understanding invasion dynamics, persistence, and optimal release strategies. Deterministic frequency models have shown that invasion is governed by the interaction between three principal biological mechanisms: the fitness cost associated with infection, the strength of cytoplasmic incompatibility, and the fidelity of maternal transmission. Under biologically realistic parameter regimes these models frequently exhibit bistability, with an unstable intermediate equilibrium separating extinction from successful establishment. This unstable equilibrium is commonly interpreted as an invasion threshold or Allee-type threshold, representing the minimum infection frequency required for successful population replacement. Numerous extensions have subsequently incorporated age structure, spatial heterogeneity, reaction–diffusion equations, integrodifference equations, traveling waves, metapopulation dynamics, and optimal release protocols [14–17].

Despite these advances, deterministic models necessarily assume that biological parameters remain constant throughout the invasion process. In reality, mosquito populations experience continual environmental fluctuations arising from seasonal temperature variation, rainfall, humidity, larval resource availability, demographic variability, imperfect releases, and heterogeneous habitat conditions. These factors may simultaneously influence the fitness cost of infection, the intensity of cytoplasmic incompatibility, and maternal transmission efficiency. Consequently, two releases initiated with identical infection frequencies may produce substantially different outcomes under different environmental conditions. Such variability suggests that deterministic invasion thresholds should be interpreted as idealized benchmarks rather than exact predictors of field success and motivates the development of stochastic mathematical frameworks capable of quantifying uncertainty in invasion outcomes.

Stochastic population dynamics have been studied extensively in both continuous and discrete time. A large body of work has established rigorous mathematical foundations for stochastic persistence, extinction, invariant probability measures, random dynamical systems, stochastic difference equations, and environmentally forced population models [18–25]. These developments have shown that environmental stochasticity can fundamentally alter long-term population behavior by replacing deterministic equilibria with stationary probability distributions and by transforming deterministic thresholds into probabilistic transition regions. Nevertheless, comparatively little attention has been devoted to stochastic discrete-time *Wolbachia* models, despite the fact that mosquito generations are naturally discrete and that many of the most successful deterministic invasion models are formulated as nonlinear difference equations.

Existing stochastic studies of *Wolbachia* typically focus on particular sources of randomness, most commonly continuous-time stochastic differential equations or specific noise perturbations. While these approaches provide valuable biological insight, they often depend upon particular stochastic assumptions and therefore do not provide a unified mathematical framework capable of accommodating general environmental processes. In particular, a systematic treatment combining random dynamical systems, stochastic evolution operators, Feller–Markov theory, invariant probability measures, stochastic persistence,

and probabilistic release strategies for discrete-time *Wolbachia* invasion models appears to be largely absent from the existing literature.

The primary objective of this paper is to develop such a unified stochastic framework. Rather than prescribing a particular probability distribution or noise mechanism, we replace the deterministic biological parameters by a stationary stochastic environmental process whose realizations represent successive environmental conditions experienced by the mosquito population. This formulation naturally accommodates independent environmental fluctuations, temporally correlated environments, stationary ergodic processes, and simultaneous stochastic variation in multiple biological mechanisms while preserving the biological interpretation of the underlying deterministic model.

From this perspective, the stochastic *Wolbachia* model is naturally interpreted as a random dynamical system generated by a family of environmentally dependent nonlinear maps. This viewpoint provides a mathematically coherent framework that separates the pathwise dynamics from their probabilistic description and allows the Markovian formulation to emerge as a special case when the environmental process is assumed to be independent and identically distributed. Consequently, the paper integrates techniques from nonlinear difference equations, random dynamical systems, Markov processes, and stochastic population dynamics into a single analytical framework.

The principal contributions of this work are as follows.

1. We formulate a general stochastic discrete-time *Wolbachia* invasion model driven by an arbitrary stationary environmental process.
2. We establish the well-posedness of the stochastic model by proving existence, uniqueness, positivity, forward invariance, continuity, and pathwise comparison principles.
3. We develop the associated random dynamical systems framework through the stochastic evolution operator and show how the classical Markov formulation arises naturally under independent environmental forcing.
4. For the Markovian model, we establish the Feller property and prove the existence of invariant probability measures, thereby providing a rigorous description of the long-term statistical behavior of the infection frequency.
5. We investigate stochastic persistence and extinction through invariant probability measures and the local Lyapunov exponent near the disease-free equilibrium, thereby clarifying the role of environmental stochasticity in the long-term dynamics.
6. We introduce a finite-horizon establishment probability together with probability-based release thresholds that directly quantify the likelihood of successful *Wolbachia* establishment under environmental uncertainty.
7. Finally, we perform numerical simulations illustrating how environmental variability influences invasion success, release thresholds, and intervention strategies.

Beyond its immediate application to *Wolbachia*, the mathematical framework developed here provides a general methodology for analyzing nonlinear biological invasion models evolving in random environments. Because the formulation is based on random families of maps rather than a particular noise mechanism, it can be adapted to a wide range of discrete-time ecological and evolutionary systems in which environmental variability plays a fundamental role.

The remainder of the paper is organized as follows. Section 2 briefly reviews the deterministic *Wolbachia* model and summarizes the principal results from the existing literature that motivate our stochastic formulation. Section 3 introduces the stochastic model and establishes the general probabilistic framework. Section 4 develops the associated random dynamical systems theory, while Section 5 studies the induced Markov chain, Feller operator, and invariant probability measures under independent environmental forcing. Section 6 investigates stochastic persistence, extinction, and probability-based

release thresholds. Finally, Section 7 presents numerical simulations illustrating the effects of environmental variability on invasion success and biologically relevant release strategies.

2. Model Formulation and Deterministic Background

Let $x_n \in [0, 1]$ denote the frequency of *Wolbachia*-infected mosquitoes in generation n . We consider a population consisting of infected and uninfected males and females and assume that the infection frequencies are the same in both sexes. Thus,

$$x_n = \frac{I_F(n)}{I_F(n) + U_F(n)} = \frac{I_M(n)}{I_M(n) + U_M(n)},$$

where $I_F(n)$, $U_F(n)$, $I_M(n)$, and $U_M(n)$ denote the numbers of infected females, uninfected females, infected males, and uninfected males, respectively. The dynamics are governed by three biologically meaningful parameters:

$$s_f \in [0, 1), \quad s_h \in [0, 1], \quad \mu \in [0, 1].$$

Here, s_f is the relative fitness cost experienced by infected females, s_h is the intensity of cytoplasmic incompatibility, and μ is the maternal-transmission leakage rate. Consequently, $1 - s_f$ is the relative fitness of infected females, while $1 - \mu$ is the proportion of offspring of infected females that successfully inherit *Wolbachia*. Under random mating, infected offspring are produced at frequency

$$(1 - \mu)(1 - s_f)x_n.$$

Indeed, the three terms have distinct biological origins: $(1 - s_h)(1 - x_n)x_n$ represents viable offspring from an uninfected female mating with an infected male, $(1 - x_n)^2$ represents matings between two uninfected mosquitoes, and $\mu(1 - s_f)x_n$ represents uninfected offspring produced through maternal-transmission failure. Their sum therefore gives the total viable uninfected contribution. Uninfected offspring arise from compatible matings involving uninfected females, from matings between two uninfected mosquitoes, and from maternal-transmission failure. Their total contribution is

$$(1 - s_h)(1 - x_n)x_n + (1 - x_n)^2 + \mu(1 - s_f)x_n.$$

After normalizing by the total number of viable offspring, the infection frequency satisfies the discrete-time model

$$x_{n+1} = F(x_n; s_f, s_h, \mu) = \frac{(1 - \mu)(1 - s_f)x_n}{s_h x_n^2 - (s_h + s_f)x_n + 1}, \quad n \in \mathbb{N}_0. \quad (1)$$

This model was introduced to describe the frequency dynamics generated by fitness costs, cytoplasmic incompatibility, and imperfect maternal transmission; see, for example, ref. [26]. The denominator in (1) represents the total viable offspring produced during one generation, whereas the numerator represents the infected component of that offspring population. For biologically admissible parameter values, the interval $[0, 1]$ is forward invariant. Indeed, for $x \in [0, 1]$,

$$s_h x^2 - (s_h + s_f)x + 1 = (1 - s_f)x + (1 - s_h)(1 - x)x + (1 - x)^2 > 0,$$

and

$$\begin{aligned} s_h x^2 - (s_h + s_f)x + 1 - (1 - \mu)(1 - s_f)x \\ = (1 - s_h)(1 - x)x + (1 - x)^2 + \mu(1 - s_f)x \geq 0. \end{aligned}$$

Therefore,

$$0 \leq F(x; s_f, s_h, \mu) \leq 1,$$

and every trajectory beginning in $[0, 1]$ remains biologically meaningful.

Summary of the Deterministic Dynamics

The deterministic dynamics of model (1) and several closely related frequency models have been investigated extensively in the literature [26–28]. We briefly summarize the properties most relevant to the stochastic analysis developed below.

If $\mu = 0$, then

$$x_{n+1} = \frac{(1 - s_f)x_n}{s_h x_n^2 - (s_h + s_f)x_n + 1}. \quad (2)$$

When

$$0 < s_f < s_h \leq 1,$$

model (2) has three equilibria,

$$0, \quad x_A = \frac{s_f}{s_h}, \quad 1.$$

The condition $s_f < s_h$ means that the fitness disadvantage associated with Wolbachia infection is weaker than the reproductive advantage generated by cytoplasmic incompatibility. It also guarantees $x_A = s_f/s_h \in (0, 1)$ and hence a biologically meaningful interior invasion threshold. If $s_h \leq s_f$, this bistable invasion mechanism is lost.

When $\mu > 0$, the complete-infection state is no longer an equilibrium, since

$$F(1; s_f, s_h, \mu) = 1 - \mu < 1.$$

Nevertheless, positive equilibria may persist. For $x^* > 0$, the equilibrium condition $F(x^*; s_f, s_h, \mu) = x^*$ reduces to

$$s_h(x^*)^2 - (s_h + s_f)x^* + s_f + \mu(1 - s_f) = 0.$$

Consequently, whenever

$$\Delta = (s_h + s_f)^2 - 4s_h[s_f + \mu(1 - s_f)] \geq 0$$

and the resulting roots lie in $(0, 1]$, the positive equilibria are

$$x_{\pm}^* = \frac{s_h + s_f \pm \sqrt{\Delta}}{2s_h}.$$

The lower equilibrium x_-^* plays the role of the deterministic invasion threshold, while x_+^* represents the high-infection equilibrium attainable in the presence of imperfect maternal transmission. These equilibria continuously recover those of the perfect-transmission model as $\mu \rightarrow 0$:

$$x_-^* \longrightarrow \frac{s_f}{s_h} = x_A, \quad x_+^* \longrightarrow 1.$$

Thus, x_-^* is the natural imperfect-transmission analogue of the Allee-type threshold x_A . These quantities are used below as deterministic reference values in the numerical experiments.

For $\mu = 0$, local stability follows from

$$F'(x) = \frac{(1-s_f)(1-s_h x^2)}{[s_h x^2 - (s_h + s_f)x + 1]^2}.$$

Thus,

$$F'(0) = 1 - s_f < 1, \quad F'(1) = \frac{1-s_h}{1-s_f} < 1,$$

whereas

$$F'(x_A) = \frac{s_h - s_f^2}{s_h(1-s_f)} > 1 \quad (s_f < s_h).$$

Hence, 0 and 1 are locally asymptotically stable and x_A is unstable.

For $\mu > 0$, let

$$q(x) = s_h x^2 - (s_h + s_f)x + s_f + \mu(1 - s_f).$$

At a positive equilibrium x^* ,

$$F'(x^*) - 1 = -\frac{x^* q'(x^*)}{(1-\mu)(1-s_f)}.$$

Since $q'(x_-^*) < 0 < q'(x_+^*)$, the lower equilibrium x_-^* is unstable, whereas x_+^* is locally asymptotically stable. Thus x_-^* acts as the invasion threshold and x_+^* as the attainable high-infection state. Returning to the perfect-transmission case $\mu = 0$, x_A separates the basins of attraction of the two stable equilibria:

$$x_0 < x_A \implies x_n \rightarrow 0, \quad x_0 > x_A \implies x_n \rightarrow 1.$$

Thus, the deterministic model is bistable and exhibits a strong Allee-type threshold. In the context of *Wolbachia*, the value

$$x_A = \frac{s_f}{s_h}$$

is interpreted as the minimum infection frequency required for successful population replacement. Increasing the fitness cost s_f raises this threshold, whereas increasing the intensity s_h of cytoplasmic incompatibility lowers it.

If $s_h \leq s_f$, the reproductive advantage generated by cytoplasmic incompatibility is insufficient to compensate for the fitness cost of infection. In this regime, the infection frequency decreases and the *Wolbachia*-free equilibrium is the relevant long-term state.

If $\mu > 0$, complete infection is generally no longer an equilibrium since

$$F(1; s_f, s_h, \mu) = 1 - \mu < 1.$$

Maternal-transmission leakage therefore lowers the attainable infection frequency and may alter or eliminate the bistable structure. Although these deterministic effects are biologically important, our objective is not to repeat the full deterministic analysis. Instead, model (1) will serve as the baseline for studying how random environmental and demographic fluctuations modify the known threshold, persistence, and extinction properties.

3. A General Stochastic Framework

The deterministic threshold provides a useful benchmark, but it assumes that all biological parameters remain constant throughout the duration of the invasion process. In natural mosquito populations, however, these quantities fluctuate over time due to environmental, demographic, and operational factors. Examples include seasonal variation in

temperature, rainfall, humidity, larval resource availability, imperfect releases, genetic variability, fluctuations in maternal transmission, and changes in cytoplasmic incompatibility.

Consequently, identical releases performed under different environmental conditions may lead to substantially different invasion outcomes.

The objective of this section is therefore to formulate a stochastic framework that incorporates these sources of uncertainty while preserving the biological structure of the deterministic model. To incorporate environmental variability, let $(\Omega, \mathcal{F}, \mathbb{P})$ be a complete probability space, where Ω denotes the sample space, $\mathcal{F}$ is the associated σ -algebra, and $\mathbb{P}$ is the underlying probability measure.

For each generation $n \in \mathbb{N}_0$, let

$$\Theta_n : \Omega \longrightarrow [0, 1] \times [0, 1] \times [0, 1]$$

be the random environmental state defined by

$$\Theta_n(\omega) = (S_{f,n}(\omega), S_{h,n}(\omega), M_n(\omega)),$$

where $S_{f,n}$ is the random fitness cost, $S_{h,n}$ is the random cytoplasmic incompatibility intensity, and M_n is the random maternal-transmission leakage rate.

Thus, the deterministic parameter vector (s_f, s_h, μ) is replaced by the stochastic environmental process $\{\Theta_n\}_{n \geq 0}$, whose realizations describe the sequence of environmental conditions experienced by the mosquito population. For every environmental state

$$\theta = (s_f, s_h, m) \in [0, 1] \times [0, 1] \times [0, 1],$$

we define the map

$$F_\theta : [0, 1] \longrightarrow [0, 1]$$

by

$$F_\theta(x) = \frac{(1 - m)(1 - s_f)x}{s_h x^2 - (s_h + s_f)x + 1}.$$

Here m is a realized value of the random maternal-transmission leakage M_n and is the stochastic analogue of the deterministic parameter μ . Consequently, $1 - m$ is the maternal-transmission fidelity during that environmental realization, i.e., the fraction of offspring of infected females that successfully inherit Wolbachia.

The stochastic Wolbachia model is then given by the random iteration

$$X_{n+1} = F_{\Theta_n}(X_n), \quad n \in \mathbb{N}_0, \quad (3)$$

or, equivalently,

$$X_{n+1} = \frac{(1 - M_n)(1 - S_{f,n})X_n}{S_{h,n}X_n^2 - (S_{h,n} + S_{f,n})X_n + 1}. \quad (4)$$

Thus, at each generation, the state X_n evolves according to the map F_{Θ_n} , whose parameters are determined by the environmental state realized during that generation.

Remark 1. Treating the environmental state θ as a vector rather than as independent random parameters allows multiple biological mechanisms to fluctuate simultaneously and permits statistical dependence between these mechanisms. For example, elevated temperatures may simultaneously increase the fitness cost of infection while reducing maternal transmission efficiency. The proposed formulation therefore accommodates correlated environmental fluctuations in a natural way.

3.1. Generality of the Stochastic Framework

Although the present paper is motivated by Wolbachia invasion dynamics, the stochastic framework is not restricted to this particular biological system. Its essential mathematical structure is a random iteration of the form

$$X_{n+1} = F_{\Theta_n}(X_n),$$

where F_θ is a deterministic population map depending on a parameter vector θ , and $\{\Theta_n\}_{n \geq 0}$ represents a random environmental process.

Accordingly, the Wolbachia-specific quantities $S_{f,n}$, $S_{h,n}$, and M_n should be distinguished from the general mathematical construction. In another application, the state variable, parameter vector, and biological interpretation of F_θ may be different, while the same stochastic methodology may still be applicable. In particular, whenever a discrete-time ecological, evolutionary, epidemiological, or biological-invasion model can be written in the form

$$x_{n+1} = F(x_n; \theta),$$

environmental uncertainty may be introduced by replacing the fixed parameter vector θ with a stochastic process Θ_n .

The resulting system can then be studied using the same general tools developed here, including pathwise random dynamics, transition kernels, Markov operators, invariant probability measures, and probability-based establishment criteria, provided the corresponding continuity, measurability, and state-space assumptions are satisfied. Thus, Wolbachia invasion serves as a biologically important case study for a broader class of nonlinear discrete-time systems evolving in random environments, rather than as the only setting in which the framework is applicable.

3.2. Model Assumptions

The following assumptions are imposed throughout the remainder of the paper.

(H1) **Biological admissibility.** For every $n \geq 0$,

$$0 \leq S_{f,n} < 1, \quad 0 \leq S_{h,n} \leq 1, \quad 0 \leq M_n < 1, \quad \text{almost surely.}$$

Consequently, all parameter realizations remain biologically meaningful.

(H2) **Measurability.** The stochastic process $\{\Theta_n\}_{n \geq 0}$ is defined on a complete probability space $(\Omega, \mathcal{F}, \mathbb{P})$, and each random vector Θ_n is $\mathcal{F}$ -measurable.

(H3) **Stationarity.** The sequence $\{\Theta_n\}_{n \geq 0}$ is strictly stationary. Consequently, the joint probability law of $(\Theta_n, \Theta_{n+1}, \dots, \Theta_{n+k})$ does not depend on n .

(H4) **Environmental dependence.** The sequence $\{\Theta_n\}_{n \geq 0}$ may exhibit temporal dependence. In particular, it may consist of

- (i) Independent and identically distributed random vectors;
- (ii) A stationary Markov chain;
- (iii) Any stationary ergodic stochastic process.

Unless otherwise stated, all theoretical results are proved under the weakest assumption required.

4. Random Dynamical System Framework

In this section we establish the basic mathematical properties of the stochastic Wolbachia model introduced in Section 3. We show that the random iteration generates a well-defined stochastic dynamical system on the biologically meaningful state space, identify

conditions under which the process is Markovian, and introduce the associated transition operator that will be used throughout the remainder of the paper. Beyond establishing the general stochastic framework, the analysis below exploits the specific algebraic structure of F_θ to obtain monotonicity, stability, comparison, and long-term probabilistic properties.

Unless otherwise stated, assumptions (H1)–(H4) are assumed to hold throughout this section.

4.1. Existence and Positivity of Solutions

We first establish the well-posedness of the stochastic Wolbachia model. Specifically, we show that the random iteration admits a unique solution for every admissible initial condition and that the biologically relevant state space is forward invariant almost surely.

Theorem 1. *Suppose that assumptions (H1) and (H2) hold. Then, for every initial condition $X_0 = x_0 \in [0, 1]$, the stochastic recursion*

$$X_{n+1} = F_{\Theta_n}(X_n)$$

admits a unique solution $\{X_n\}_{n \geq 0}$ defined recursively for every realization $\omega \in \Omega$. Moreover,

$$0 \leq X_n \leq 1, \quad \text{almost surely for every } n \geq 0.$$

Proof. Let $E = [0, 1]$ and fix an initial condition $X_0 = x_0 \in E$. By assumption (H1), there exists a set $\Omega_0 \in \mathcal{F}$ with

$$\mathbb{P}(\Omega_0) = 1$$

such that, for every $\omega \in \Omega_0$ and every $n \geq 0$,

$$0 \leq S_{f,n}(\omega) < 1, \quad 0 \leq S_{h,n}(\omega) \leq 1, \quad 0 \leq M_n(\omega) < 1.$$

Fix $\omega \in \Omega_0$. For notational simplicity, write

$$s_f = S_{f,n}(\omega), \quad s_h = S_{h,n}(\omega), \quad m = M_n(\omega).$$

For $x \in E$, define

$$D_\theta(x) = s_h x^2 - (s_h + s_f)x + 1, \quad \theta = (s_f, s_h, m).$$

The denominator can be rewritten as

$$D_\theta(x) = (1 - s_f)x + (1 - s_h)(1 - x)x + (1 - x)^2.$$

All terms on the right-hand side are non-negative. Moreover, they cannot vanish simultaneously for $x \in [0, 1]$, because $s_f < 1$. Hence, $D_\theta(x) > 0$ for every $x \in E$. Therefore, $F_\theta(x)$ is well defined and non-negative.

Furthermore,

$$D_\theta(x) - (1 - m)(1 - s_f)x = (1 - s_h)(1 - x)x + (1 - x)^2 + m(1 - s_f)x \geq 0.$$

It follows that

$$0 \leq \frac{(1 - m)(1 - s_f)x}{D_\theta(x)} \leq 1,$$

and consequently,

$$F_\theta(E) \subseteq E.$$

We now construct the solution recursively. Set

$$X_0(\omega) = x_0.$$

For $n \geq 0$, define

$$X_{n+1}(\omega) = F_{\Theta_n(\omega)}(X_n(\omega)).$$

Since every map $F_{\Theta_n(\omega)}$ maps E into itself, induction gives

$$X_n(\omega) \in E \quad \text{for every } n \geq 0.$$

Thus,

$$0 \leq X_n(\omega) \leq 1 \quad \text{for every } n \geq 0 \text{ and every } \omega \in \Omega_0.$$

Since $\mathbb{P}(\Omega_0) = 1$, we conclude that

$$0 \leq X_n \leq 1, \quad \text{almost surely for every } n \geq 0.$$

Existence follows from the recursive construction. Uniqueness is immediate: if $\{X_n\}$ and $\{Y_n\}$ are two solutions with the same initial condition $X_0 = Y_0 = x_0$, then

$$X_1 = F_{\Theta_0}(X_0) = F_{\Theta_0}(Y_0) = Y_1.$$

Repeating this argument inductively yields

$$X_n = Y_n, \quad \text{almost surely for every } n \geq 0.$$

Hence the solution is unique. $\square$

4.2. Random Dynamical System Generated by the Wolbachia Maps

The family of random maps introduced in Section 3 naturally generates a discrete-time random dynamical system on the biologically meaningful state space $E = [0, 1]$. Indeed, each realization of the environmental process $\{\Theta_n(\omega)\}_{n \geq 0}$ determines a sequence of deterministic maps

$$F_{\Theta_0(\omega)}, F_{\Theta_1(\omega)}, F_{\Theta_2(\omega)}, \dots,$$

whose successive compositions describe the evolution of the infection frequency under that particular environmental realization.

For every realization $\omega \in \Omega$, define

$$\Phi(0, \omega, x) = x,$$

and recursively,

$$\Phi(n+1, \omega, x) = F_{\Theta_n(\omega)}(\Phi(n, \omega, x)), \quad n \geq 0.$$

Equivalently,

$$\Phi(n, \omega, x) = F_{\Theta_{n-1}(\omega)} \circ \dots \circ F_{\Theta_0(\omega)}(x), \quad n \geq 1.$$

Here

$$\Theta_j(\omega) = (S_{f,j}(\omega), S_{h,j}(\omega), M_j(\omega)), \quad 0 \leq S_{f,j} < 1, \quad 0 \leq S_{h,j} \leq 1, \quad 0 \leq M_j < 1,$$

and each $F_{\Theta_j(\omega)}$ is the stochastic map given explicitly in (Equation (4)). Thus, $\Phi(n, \omega, x)$ is obtained by successively applying the environmentally realized maps over the first n generations.

The mapping

$$\Phi : \mathbb{N}_0 \times \Omega \times E \longrightarrow E$$

is called the stochastic evolution operator associated with the random Wolbachia model.

Interpretation

For a fixed realization ω , the operator $\Phi(n, \omega, \cdot)$ represents the cumulative effect of the sequence of environmental conditions experienced during the first n generations. Consequently, each realization of the environmental process generates a deterministic population trajectory, while the collection of all realizations describes the stochastic evolution of the mosquito population.

Proposition 1. *Suppose assumptions (H1) and (H2) hold. Then, for every $n \geq 0$,*

$$\Phi(n, \omega, \cdot) : E \longrightarrow E$$

is well defined for almost every $\omega \in \Omega$.

Proof. The conclusion follows inductively from Theorem 1. $\square$

Definition 1. *The pair $(\{\Theta_n\}_{n \geq 0}, \Phi)$ consisting of the environmental process and the stochastic evolution operator defines the random dynamical system associated with the stochastic Wolbachia model.*

4.3. Pathwise Dynamical Properties

The stochastic evolution operator allows the dynamics to be studied separately along each realization of the environmental process. We now establish several properties of the maps $x \mapsto \Phi(n, \omega, x)$ that hold pathwise, without requiring the environmental process to be independent or Markovian. In particular, we show that the stochastic dynamics depend continuously on the initial infection frequency and preserve the natural ordering of initial conditions. For an environmental state $\theta = (s_f, s_h, m)$, recall that

$$F_\theta(x) = \frac{(1-m)(1-s_f)x}{s_h x^2 - (s_h + s_f)x + 1}.$$

The following proposition summarizes the relevant properties of this family of maps.

Proposition 2 (Continuity and order preservation). *Suppose that assumption (H1) holds. Then, for every admissible environmental state θ , $F_\theta : E \longrightarrow E$ is continuous and nondecreasing.*

Consequently, for every $n \geq 0$ and almost every $\omega \in \Omega$, the stochastic evolution map $\Phi(n, \omega, \cdot) : E \longrightarrow E$ is continuous and nondecreasing.

Proof. Let

$$D_\theta(x) = s_h x^2 - (s_h + s_f)x + 1.$$

As shown in Section 2, $D_\theta(x) > 0$ for every $x \in E$. Hence, F_θ is continuous on E . Differentiating with respect to x , we obtain

$$\begin{aligned} F'_\theta(x) &= (1-m)(1-s_f) \frac{D_\theta(x) - x D'_\theta(x)}{D_\theta(x)^2} \\ &= (1-m)(1-s_f) \frac{1 - s_h x^2}{[s_h x^2 - (s_h + s_f)x + 1]^2}. \end{aligned}$$

Since

$$0 \leq s_h \leq 1, \quad 0 \leq x \leq 1,$$

we have

$$1 - s_h x^2 \geq 0.$$

Therefore, $F'_\theta(x) \geq 0$, and F_θ is nondecreasing on E . For a fixed realization ω , the map $\Phi(n, \omega, \cdot)$ is a finite composition of continuous, nondecreasing maps:

$$\Phi(n, \omega, \cdot) = F_{\Theta_{n-1}(\omega)} \circ \cdots \circ F_{\Theta_0(\omega)}.$$

It is therefore continuous and nondecreasing. $\square$

Order Preservation Property

This is a situation when two populations experiencing the same environmental realization retain the ordering of their initial infection frequencies: $x_0 \leq y_0$ implies $\Phi(n, \omega, x_0) \leq \Phi(n, \omega, y_0)$ for all $n \geq 0$.

The order-preserving property yields an immediate pathwise comparison principle.

Corollary 1 (Pathwise comparison principle). *Suppose that*

$$0 \leq x_0 \leq y_0 \leq 1.$$

Then, for every $n \geq 0$,

$$\Phi(n, \omega, x_0) \leq \Phi(n, \omega, y_0), \quad \text{for almost every } \omega \in \Omega.$$

Proof. The conclusion follows directly from the monotonicity of $\Phi(n, \omega, \cdot)$. $\square$

Thus, two mosquito populations exposed to the same sequence of environmental conditions preserve the ordering of their initial infection frequencies. In particular, a population beginning with a larger infected fraction cannot fall below a population beginning with a smaller infected fraction when both are subjected to the same environmental realization. The boundary states also have a direct biological interpretation. For every admissible environmental state $\theta = (s_f, s_h, m)$,

$$F_\theta(0) = 0, \quad \text{and } F_\theta(1) = 1 - m.$$

Consequently,

$$\Phi(n, \omega, 0) = 0$$

for every $n \geq 0$. Thus, the Wolbachia-free state is absorbing: once the infection is absent, environmental variation alone cannot introduce it into the population. In contrast, when maternal transmission is imperfect, the complete-infection state is not invariant, since

$$F_{\Theta_n(\omega)}(1) = 1 - M_n(\omega) < 1, \quad \text{whenever } M_n(\omega) > 0.$$

These pathwise properties provide the foundation for the subsequent analysis of stochastic invasion thresholds. In particular, order preservation allows initial infection frequencies to be compared under a common environmental realization and supports the construction of probabilistic boundaries separating extinction from persistence.

4.4. Transition Probabilities

The pathwise description developed above characterizes the evolution of the system for each individual realization of the environment. We now adopt a probabilistic viewpoint by describing the distribution of the infection frequency over all environmental realizations.

For a given initial state $x \in E$, the stochastic evolution operator induces a probability distribution on the state space after n generations. These transition probabilities play a fundamental role in the analysis of long-term stochastic behavior.

For every $n \geq 0, x \in E$, and every Borel set $A \subseteq E$, define

$$P_n(x, A) = \mathbb{P}(\Phi(n, \omega, x) \in A).$$

The quantity $P_n(x, A)$ represents the probability that a population starting with infection frequency x lies in the set A after n generations.

Proposition 3. *Suppose assumptions (H1)–(H3) hold. Then, for every $n \geq 0$, the mapping*

$$A \mapsto P_n(x, A) \quad \text{defines a probability measure on the Borel subsets of } E.$$

Moreover,

$$x \mapsto P_n(x, A) \quad \text{is measurable for every Borel set } A.$$

Remark 2. *Unlike the pathwise evolution operator, which follows a single realization of the environment, the transition probability $P_n(x, A)$ averages over all possible environmental realizations. Consequently, $P_n(x, \cdot)$ describes the uncertainty associated with the infection frequency after n generations.*

5. Markovian Dynamics and Long-Term Behavior

The random dynamical system developed in the previous section is valid under the general assumptions (H1)–(H4) and therefore accommodates a broad class of environmental processes, including temporally correlated and stationary ergodic environments. In many applications, however, it is reasonable to assume that the environmental conditions experienced in successive generations are independent realizations of a common probability distribution. Under this additional assumption, the stochastic Wolbachia model admits a Markovian description, allowing the use of transition kernels, Markov operators, and invariant probability measures to investigate its long-term behavior. Throughout this section we strengthen assumption (H4) by assuming that $\{\Theta_n\}_{n \geq 0}$ is a sequence of independent and identically distributed random vectors.

5.1. The Markov Property

Under the additional assumption that the environmental states are independent and identically distributed, the future evolution of the infection frequency depends only on its current state and not on its past history. Consequently, the stochastic recursion defines a time-homogeneous Markov chain on the state space $E = [0, 1]$.

Theorem 2. *Suppose assumptions (H1)–(H3) hold and assume further that $\{\Theta_n\}_{n \geq 0}$ is a sequence of independent and identically distributed random vectors. Then the stochastic process $\{X_n\}_{n \geq 0}$ defined by*

$$X_{n+1} = F_{\Theta_n}(X_n)$$

is a time-homogeneous Markov chain on $E = [0, 1]$.

Proof. Recall that for every $n \geq 0$, $X_{n+1} = F_{\Theta_n}(X_n)$, where F_{Θ_n} depends only on the current state X_n and on the random environmental variable Θ_n . Since Θ_n is independent of $(X_0, \dots, X_n)$ and has the same distribution for every n ,

$$\begin{aligned} \mathbb{P}(X_{n+1} \in A \mid X_0, \dots, X_n) &= \mathbb{P}(F_{\Theta_n}(X_n) \in A \mid X_n) \\ &= \mathbb{P}(X_1 \in A \mid X_0 = X_n), \end{aligned}$$

for every Borel set $A \subseteq E$. Hence, $\{X_n\}$ satisfies the Markov property. Since the distribution of Θ_n does not depend on n , the transition probabilities are independent of time. Therefore, $\{X_n\}$ is a time-homogeneous Markov chain. $\square$

5.2. Transition Kernel

Having established that the stochastic Wolbachia model defines a time-homogeneous Markov chain, we now characterize its transition probabilities. These probabilities completely determine the probabilistic evolution of the process and form the basis for the subsequent analysis of long-term stochastic behavior.

Let $\mathcal{B}(E)$ denote the Borel σ -algebra on the state space $E = [0, 1]$. For every $x \in E$, and every Borel set $A \in \mathcal{B}(E)$, the one-step transition kernel is defined by

$$P(x, A) = \mathbb{P}(F_{\Theta}(x) \in A), \quad (5)$$

where $\Theta = (S_f, S_h, M)$ has the common distribution of the environmental process. Equivalently,

$$P(x, A) = \mathbb{P}\left(\frac{(1-M)(1-S_f)x}{S_h x^2 - (S_h + S_f)x + 1} \in A\right).$$

Thus, $P(x, A)$ represents the probability that a mosquito population with infection frequency x evolves into the set A after one generation under a random environmental realization.

Proposition 4. *The mapping $P : E \times \mathcal{B}(E) \rightarrow [0, 1]$ defined by (5) is a Markov transition kernel. That is,*

- (i) *For every fixed $x \in E$, the mapping $A \mapsto P(x, A)$ is a probability measure on $\mathcal{B}(E)$;*
- (ii) *For every Borel set $A \subseteq E$, the mapping $x \mapsto P(x, A)$ is Borel measurable.*

Proof. For each fixed $x \in E$, the random variable $F_{\Theta}(x)$ is measurable by assumption (H2) and the continuity of F_{θ} with respect to x . Therefore, $A \mapsto \mathbb{P}(F_{\Theta}(x) \in A)$ defines the distribution of the random variable $F_{\Theta}(x)$, and is therefore a probability measure on $\mathcal{B}(E)$. For every Borel set A , the mapping $x \mapsto P(x, A)$ is measurable since $F_{\theta}(x)$ is jointly measurable in (x, θ) , and the probability measure is obtained by integrating over the distribution of the environmental random vector Θ . Hence, P is a Markov transition kernel. $\square$

Biological Interpretation

The transition kernel provides a probabilistic description of the infection dynamics over a single mosquito generation. Rather than following one particular realization of the environmental process, it averages over all possible environmental conditions weighted by their probability of occurrence. Consequently, $P(x, A)$ quantifies the likelihood that a population beginning with infection frequency x reaches the range of infection frequencies contained in A after one generation.

5.3. The Markov Operator

The transition kernel introduced in the previous subsection naturally induces a linear operator acting on bounded measurable functions. This operator provides a convenient functional representation of the stochastic dynamics and plays a central role in the study of invariant probability measures and stationary distributions.

Let $B(E)$ denote the space of bounded Borel measurable functions

$$\varphi : E \longrightarrow \mathbb{R}.$$

The Markov operator

$$P : B(E) \longrightarrow B(E)$$

associated with the transition kernel is defined by

$$(P\varphi)(x) = \int_E \varphi(y) P(x, dy), \quad (6)$$

for every $\varphi \in B(E)$ and every $x \in E$.

Equivalently,

$$(P\varphi)(x) = \mathbb{E}[\varphi(F_\Theta(x))],$$

where the expectation is taken with respect to the distribution of the environmental random vector $\Theta = (S_f, S_h, M)$.

Proposition 5. *The Markov operator P satisfies the following properties.*

(i) (Linearity)

$$P(a\varphi + b\psi) = aP\varphi + bP\psi \quad \text{for all } a, b \in \mathbb{R}, \quad \varphi, \psi \in B(E).$$

(ii) (Positivity)

If $\varphi(x) \geq 0$ for every $x \in E$, then $(P\varphi)(x) \geq 0$.

(iii) (Constant preserving)

$$P\mathbf{1} = \mathbf{1}.$$

(iv) (Contraction in the operator-norm sense) *The Markov operator P satisfies*

$$\|P\varphi\|_\infty \leq \|\varphi\|_\infty, \quad \varphi \in C(E).$$

Hence $\|P\| \leq 1$. Since $P\mathbf{1} = \mathbf{1}$, we in fact have

$$\|P\| = 1.$$

Thus, P is a contraction operator in the standard non-strict operator-norm sense, equivalently non-expansive in the supremum norm; no strict contraction with factor $c < 1$ is asserted.

Proof. Properties (i) and (ii) follow immediately from the linearity and positivity of the Lebesgue integral. For the constant function $\mathbf{1}(x) \equiv 1$, we have

$$(P\mathbf{1})(x) = \int_E \mathbf{1} P(x, dy) = P(x, E) = 1,$$

since $P(x, \cdot)$ is a probability measure. Finally,

$$\begin{aligned} |(P\varphi)(x)| &= \left| \int_E \varphi(y) P(x, dy) \right| \\ &\leq \int_E |\varphi(y)| P(x, dy) \\ &\leq \|\varphi\|_\infty. \end{aligned}$$

Taking the supremum over $x \in E$ gives

$$\|P\varphi\|_\infty \leq \|\varphi\|_\infty.$$

This completes the proof. $\square$

Biological Interpretation

For a given observable $\varphi(x)$, the quantity $(P\varphi)(x)$ represents its expected value after one mosquito generation when the current infection frequency is x . Thus, the Markov operator propagates expected population characteristics forward in time while averaging over all possible environmental conditions.

5.4. The Feller Property

The Feller property is one of the fundamental regularity properties of Markov operators. It guarantees that the action of the Markov operator preserves the continuity of observables and plays a central role in the existence theory of invariant probability measures.

Let $C(E)$ denote the Banach space of continuous real-valued functions on $E = [0, 1]$, equipped with the supremum norm

$$\|\varphi\|_\infty = \sup_{x \in E} |\varphi(x)|.$$

We now show that the Markov operator associated with the stochastic Wolbachia model is a Feller operator.

Theorem 3 (Feller property). *Suppose assumptions (H1)–(H3) hold and let*

$$P : C(E) \rightarrow B(E)$$

be the Markov operator defined by

$$(P\varphi)(x) = \mathbb{E}[\varphi(F_\Theta(x))].$$

Then $P(C(E)) \subseteq C(E)$.

Consequently,

$$P : C(E) \longrightarrow C(E)$$

is a Feller operator.

Proof. Let $\varphi \in C(E)$, and let $x_n \rightarrow x$ in E . Since, for every environmental state θ , the map $F_\theta : E \rightarrow E$ is continuous (Proposition 2), we have

$$F_\theta(x_n) \longrightarrow F_\theta(x).$$

Because φ is continuous,

$$\varphi(F_\theta(x_n)) \longrightarrow \varphi(F_\theta(x))$$

for every admissible θ . Moreover,

$$|\varphi(F_\theta(x_n))| \leq \|\varphi\|_\infty,$$

which is integrable with respect to the distribution of Θ . The Dominated Convergence Theorem therefore gives

$$\begin{aligned} (P\varphi)(x_n) &= \mathbb{E}[\varphi(F_\Theta(x_n))] \\ &\longrightarrow \mathbb{E}[\varphi(F_\Theta(x))] \\ &= (P\varphi)(x). \end{aligned}$$

Hence, $P\varphi$ is continuous on E . Therefore,

$$P(C(E)) \subseteq C(E),$$

which proves that P is a Feller operator. $\square$

Remark 3. *The Feller property provides the principal analytical tool for studying the long-term behavior of the stochastic Wolbachia model. In particular, it forms the basis for proving the existence of invariant probability measures and stationary distributions in the following subsection.*

5.5. Invariant Probability Measures and Stationary Distributions

Having established that the Markov operator associated with the stochastic Wolbachia model is Feller, we now investigate its long-term probabilistic behavior. The central objects are invariant probability measures, which describe statistical equilibrium states of the stochastic dynamics and form the foundation for the analysis of persistence and extinction under environmental variability.

Let $\mathcal{P}(E)$ denote the set of Borel probability measures on $E = [0, 1]$.

The Markov operator naturally induces an operator on probability measures, defined by

$$(\mu P)(A) = \int_E P(x, A) \mu(dx), \quad (7)$$

for every $A \in \mathcal{B}(E)$.

A probability measure $\mu \in \mathcal{P}(E)$ is called *invariant* if

$$\mu P = \mu. \quad (8)$$

Equivalently,

$$\int_E \varphi(x) \mu(dx) = \int_E (P\varphi)(x) \mu(dx)$$

for every bounded measurable function $\varphi : E \rightarrow \mathbb{R}$.

An invariant probability measure represents a statistical equilibrium of the stochastic Wolbachia dynamics: if the initial infection frequency is distributed according to μ , then the distribution remains unchanged after one generation.

Theorem 4 (Existence of invariant probability measures). *Suppose assumptions (H1)–(H3) hold and assume that the environmental process is independent and identically distributed.*

Then the Markov operator associated with the stochastic Wolbachia model admits at least one invariant probability measure on $E = [0, 1]$.

Proof. By Theorem 3, the Markov operator

$$P : C(E) \rightarrow C(E)$$

is Feller. Since $E = [0, 1]$ is compact, every family of probability measures on E is tight. The existence of an invariant probability measure therefore follows directly from the Krylov–Bogolyubov theorem for Feller–Markov chains on compact metric spaces. $\square$

Definition 2. A probability measure $\pi \in \mathcal{P}(E)$ is called a stationary distribution if $X_0 \sim \pi$ implies

$$X_n \sim \pi, \quad n \geq 0.$$

Equivalently,

$$\pi P = \pi.$$

Hence, for the stochastic Wolbachia model, invariant probability measures and stationary distributions coincide.

Biological Interpretation

A stationary distribution describes the long-term statistical behavior of the infection frequency under repeated environmental fluctuations. An invariant probability measure describes a possible long-term statistical regime of the infection frequency under repeated environmental fluctuations. Rather than requiring convergence to a single deterministic equilibrium, the stochastic framework allows long-term behavior to be described by a stationary probability distribution.

Consequently, environmental stochasticity may replace deterministic positive equilibria by stationary probability distributions, reflecting the continual influence of random environmental variation.

Remark 4. The existence theorem guarantees the presence of at least one statistical equilibrium but does not address uniqueness. Uniqueness depends on additional properties of the stochastic dynamics, such as irreducibility, strong mixing, or suitable contraction conditions. These questions are left for future investigation.

5.6. Characterization of Stationary Distributions

The preceding results establish the existence of invariant probability measures for the stochastic Wolbachia model. It is natural to ask whether such measures can be characterized more explicitly. Although a closed-form stationary density is generally not expected when all three biological parameters fluctuate, the transition kernel and the corresponding stationary integral equation can be derived by a change-of-variables argument. We first illustrate this construction in a reduced stochastic setting and then indicate its extension to the full model.

Recall that

$$X_{n+1} = F_{\Theta_n}(X_n),$$

where

$$F_{\theta}(x) = \frac{(1-m)(1-s_f)x}{s_h x^2 - (s_h + s_f)x + 1}.$$

To make the derivation transparent, suppose initially that

$$S_{h,n} \equiv a, \quad M_n \equiv m,$$

while $S_{f,n}$ is an i.i.d. random variable having density φ on an admissible interval $I_f \subset (0, 1)$. Set

$$c = 1 - m.$$

Conditioning on $X_n = s > 0$, the next state is

$$X_{n+1} = \frac{c(1 - S_{f,n})s}{as^2 - (a + S_{f,n})s + 1}.$$

For fixed s , write $x = X_{n+1}$ and $y = S_{f,n}$. Then

$$x = \frac{c(1 - y)s}{as^2 - (a + y)s + 1}.$$

Solving for y gives the inverse transformation

$$y = \Psi(s, x) = \frac{as^2x - asx - cs + x}{s(x - c)}.$$

A direct calculation yields

$$\left| \frac{\partial \Psi}{\partial x}(s, x) \right| = \frac{c(1 - s)(1 - as)}{s(x - c)^2},$$

for $s \in (0, 1)$, with the usual interpretation restricted to values of x for which $\Psi(s, x) \in I_f$.

Consequently, the conditional transition density of X_{n+1} given $X_n = s$ is

$$k(x | s) = \varphi(\Psi(s, x)) \frac{c(1 - s)(1 - as)}{s(x - c)^2} \mathbf{1}_{I_f}(\Psi(s, x)).$$

Hence, if f_n denotes the density of X_n on the interior of the state space, its evolution is governed by the Frobenius–Perron equation

$$f_{n+1}(x) = \int_0^1 k(x | s) f_n(s) ds.$$

Any absolutely continuous stationary distribution with density f_+ must therefore satisfy

$$f_+(x) = \int_0^1 \varphi(\Psi(s, x)) \frac{c(1 - s)(1 - as)}{s(x - c)^2} \mathbf{1}_{I_f}(\Psi(s, x)) f_+(s) ds, \quad (9)$$

together with

$$f_+(x) \geq 0, \quad \int_0^1 f_+(x) dx = 1.$$

The normalization is preserved by the Frobenius–Perron operator. Indeed, using Tonelli’s theorem and $\int_0^1 k(x | s) dx = 1$,

$$\int_0^1 f_{n+1}(x) dx = \int_0^1 \left(\int_0^1 k(x | s) dx \right) f_n(s) ds = \int_0^1 f_n(s) ds.$$

Hence a normalized stationary density satisfies $\int_0^1 f_+(x) dx = 1$. Thus, the stationary density is characterized as a normalized non-negative fixed point of the stochastic evolution operator.

The same construction extends to the full stochastic model. Let $\Theta = (S_f, S_h, M)$ have joint probability law ν . The one-step transition kernel is

$$P(s, A) = \int_{\Theta} \mathbf{1}_A(F_{\theta}(s)) \nu(d\theta), \quad A \in \mathcal{B}([0, 1]).$$

An invariant probability measure π therefore satisfies

$$\pi(A) = \int_{[0,1]} P(s, A) \pi(ds), \quad A \in \mathcal{B}([0, 1]). \quad (10)$$

Whenever the interior part of $P(s, \cdot)$ admits a density $k(x | s)$, the absolutely continuous component of an invariant measure satisfies the corresponding integral equation

$$f_+(x) = \int_0^1 k(x | s) f_+(s) ds.$$

For independent scaled-Beta environmental variables, the kernel can be constructed by integrating the appropriate change-of-variables expression over the remaining environmental parameters. Although the resulting equation is generally not expected to possess an elementary closed-form solution, it provides an exact characterization that can be approximated numerically.

An important distinction is required at the extinction state. Since

$$F_\theta(0) = 0$$

for every admissible environmental state θ , the Dirac measure

$$\pi_0 = \delta_0$$

is always an invariant probability measure. Thus, the existence of an invariant measure on the compact state space does not imply that the invariant measure is unique or that it is supported in the interior. Depending on the stochastic dynamics, an additional invariant probability measure π_+ , supported away from extinction, may coexist with δ_0 .

Remark 5. The distinction between the extinction measure δ_0 and a possible positive stationary distribution is consistent with the numerical results presented later. For initial infection frequencies below or near the deterministic invasion threshold, the empirical long-term distribution concentrates at zero, corresponding to the invariant measure δ_0 . In contrast, trajectories initiated sufficiently above the threshold generate an empirical distribution sharply concentrated near the high-infection state. This latter distribution provides numerical evidence for a nontrivial positive invariant regime and may be viewed as a numerical approximation to a solution of the stationary Equation (10), or of (9) in the reduced setting. Below, we provide numerical evidence for a stochastic analogue of deterministic bistability: extinction and a high-frequency infection regime may represent distinct long-term behaviors selected by the initial release frequency.

6. Persistence, Extinction, and Probability-Based Release Thresholds

The mathematical framework developed in the previous sections provides the necessary tools to investigate the long-term biological consequences of environmental stochasticity. While the deterministic model predicts invasion or extinction through a single threshold determined by the parameters s_f , s_h , and μ , random environmental fluctuations fundamentally alter this picture. Instead of deterministic outcomes, one must consider the probabilities of persistence, extinction, and successful invasion under repeated environmental variability.

In this section, we establish conditions under which the infection either dies out or persists and discuss how environmental stochasticity modifies the classical invasion threshold. These results provide the mathematical foundation for interpreting the numerical simulations presented in the following section.

6.1. Extinction

The first question in the study of stochastic Wolbachia invasion is whether the infection can eventually disappear from the mosquito population under repeated environmental fluctuations. We therefore begin by introducing the notion of stochastic extinction and establishing some of its basic properties.

Definition 3 (Almost sure extinction). *The stochastic Wolbachia infection is said to become extinct almost surely if*

$$\lim_{n \rightarrow \infty} X_n = 0$$

with probability one; that is,

$$\mathbb{P}\left(\lim_{n \rightarrow \infty} X_n = 0\right) = 1.$$

The next result shows that the Wolbachia-free equilibrium remains an absorbing state under environmental stochasticity.

Proposition 6 (Absorbing extinction state). *Suppose assumptions (H1)–(H4) hold.*

If $X_0 = 0$, then $X_n = 0$ for every $n \geq 0$ almost surely.

Proof. Since $F_\theta(0) = 0$ for every admissible environmental state $\theta = (s_f, s_h, m)$, it follows that

$$X_1 = F_{\Theta_0}(0) = 0.$$

Assume inductively that $X_n = 0$. Then

$$X_{n+1} = F_{\Theta_n}(X_n) = F_{\Theta_n}(0) = 0.$$

Hence, $X_n = 0$ for every $n \geq 0$. The conclusion therefore holds almost surely. $\square$

Biological Interpretation

The preceding proposition reflects an important biological fact. Environmental stochasticity may alter the success or failure of Wolbachia invasion, but it cannot generate infected mosquitoes once the infection has been completely eliminated. Consequently, the Wolbachia-free state remains absorbing, just as in the deterministic model.

The preceding proposition characterizes extinction once the infection has already been lost. A more challenging question is to determine conditions under which positive initial infection frequencies converge to extinction despite random environmental fluctuations. Addressing this question requires additional information on the average growth of the stochastic dynamics and will be the focus of the subsequent analysis.

6.2. Persistence and the Principal Lyapunov Exponent

While extinction corresponds to the eventual disappearance of the infection, an equally important question is whether the infection can be maintained over long time scales despite continual environmental fluctuations. In stochastic systems, persistence is naturally interpreted in terms of the long-term statistical behavior of the process rather than convergence to a single equilibrium.

Definition 4 (Stochastic persistence). *The stochastic Wolbachia model is said to be stochastically persistent if there exists an invariant probability measure μ satisfying*

$$\mu(\{0\}) = 0.$$

Equivalently, the invariant measure is supported entirely within the positive state space $(0, 1]$.

This definition reflects the fact that, under environmental stochasticity, long-term persistence is described by an invariant probability distribution rather than by convergence to a deterministic equilibrium. In particular, persistence means that the infection remains present with positive frequency in the long run.

Remark 6. The notion of stochastic persistence adopted here is formulated in terms of invariant probability measures. Since the stochastic Wolbachia model defines a Feller–Markov chain on the compact state space $E = [0, 1]$, invariant probability measures exist by Theorem 4. However, this existence result alone does not imply stochastic persistence, since the extinction measure δ_0 is always invariant.

It is therefore important to distinguish between the results established analytically and those suggested by the numerical simulations. The invariance of δ_0 is rigorous, and Theorem 5 below shows that the extinction state is locally exponentially stable under the stated assumptions. By contrast, the numerical results presented later provide evidence for the possible existence of a distinct high-infection invariant regime supported away from extinction. A rigorous proof of the existence of such a nontrivial invariant probability measure, as well as questions concerning its uniqueness and ergodic properties, requires additional assumptions on the environmental process and remains an open analytical problem.

Under suitable additional conditions, such as irreducibility or appropriate ergodicity assumptions, the invariant-measure formulation may be related to the classical notion of stochastic persistence based on long-term occupation probabilities. We use the invariant-measure formulation here because it naturally complements the Markovian framework developed in Section 5 while making explicit the distinction between the rigorously established extinction results and the numerically observed persistent regime.

Biological Interpretation

Unlike deterministic models, where persistence is identified with convergence to a stable positive equilibrium, stochastic persistence is characterized by a stationary probability distribution supported on positive infection frequencies. Consequently, the infection level may fluctuate substantially from one generation to the next, while remaining established in the population over long time scales.

6.3. A Stochastic Invasion Criterion

The deterministic Wolbachia model predicts invasion by examining the local stability of the disease-free equilibrium. In the stochastic setting, local growth is naturally quantified by the expected logarithmic growth rate near the extinction state.

Definition 5 (Principal Lyapunov/stochastic invasion exponent). *The stochastic invasion exponent is defined by*

$$\lambda = \mathbb{E}[\log F'_\Theta(0)].$$

Since

$$F'_\Theta(0) = (1 - m)(1 - s_f),$$

we obtain

$$\lambda = \mathbb{E}[\log((1 - M)(1 - S_f))].$$

Theorem 5 (Local exponential stability of the extinction state). *Suppose that assumptions (H1)–(H3) hold and that the environmental process $\{\Theta_n\}_{n \geq 0}$ is stationary and ergodic. Assume further that*

$$\mathbb{E} \left[\left| \log((1 - M)(1 - S_f)) \right| \right] < \infty.$$

Then

$$\lambda \leq 0.$$

Moreover, if

$$\mathbb{P}((1 - M)(1 - S_f) < 1) > 0,$$

then

$$\lambda < 0,$$

Remark 7. *Note that $\lambda < 0$ implies that the extinction state 0 is locally exponentially stable almost surely. More precisely, there exists a positive random variable $r(\omega)$ such that, whenever $0 < X_0 < r(\omega)$, the corresponding trajectory satisfies*

$$X_n(\omega) \longrightarrow 0 \quad \text{exponentially fast.}$$

In particular,

$$\limsup_{n \rightarrow \infty} \frac{1}{n} \log X_n(\omega) < 0 \quad \text{for almost every } \omega.$$

Proof. For every admissible environmental state $\theta = (s_f, s_h, m)$, we have

$$F'_\theta(0) = (1 - m)(1 - s_f).$$

Since $0 \leq m < 1$, $0 \leq s_f < 1$, it follows that

$$0 < F'_\theta(0) \leq 1.$$

Consequently,

$$\log F'_\theta(0) \leq 0,$$

and therefore

$$\lambda = \mathbb{E}[\log F'_\Theta(0)] \leq 0.$$

If $\mathbb{P}(F'_\Theta(0) < 1) > 0$, then $\log F'_\Theta(0) < 0$ with positive probability. Since it is nonpositive almost surely and integrable, we obtain

$$\lambda < 0.$$

We now establish local exponential stability. Write

$$A_n(\omega) = (1 - M_n(\omega))(1 - S_{f,n}(\omega))$$

and

$$D_n(x, \omega) = S_{h,n}(\omega)x^2 - (S_{h,n}(\omega) + S_{f,n}(\omega))x + 1.$$

For $x > 0$,

$$\frac{F_{\Theta_n(\omega)}(x)}{x} = \frac{A_n(\omega)}{D_n(x, \omega)}.$$

Because

$$0 \leq S_{h,n}(\omega) \leq 1, \quad 0 \leq S_{f,n}(\omega) < 1,$$

we have, for $0 \leq x \leq \delta < 1/2$,

$$\begin{aligned} D_n(x, \omega) &= 1 - (S_{h,n}(\omega) + S_{f,n}(\omega))x + S_{h,n}(\omega)x^2 \\ &\geq 1 - 2x \\ &\geq 1 - 2\delta. \end{aligned}$$

Hence, whenever $0 < X_n \leq \delta$,

$$X_{n+1} \leq \frac{A_n}{1 - 2\delta} X_n.$$

Choose $\delta \in (0, 1/2)$ sufficiently small that

$$\gamma_\delta := \lambda - \log(1 - 2\delta) < 0.$$

Define

$$Y_n = \log A_n - \log(1 - 2\delta).$$

By the ergodic theorem,

$$\frac{1}{n} \sum_{k=0}^{n-1} Y_k \longrightarrow \gamma_\delta < 0 \quad \text{almost surely.}$$

It follows that, for almost every ω ,

$$C(\omega) := \sup_{n \geq 0} \sum_{k=0}^{n-1} Y_k(\omega) < \infty,$$

where the empty sum for $n = 0$ is taken to be zero.

Set

$$r(\omega) = \delta e^{-C(\omega)}.$$

Then $r(\omega) > 0$ almost surely. Suppose that

$$0 < X_0 < r(\omega).$$

We claim that $X_n \leq \delta$ for every n . Indeed, as long as the trajectory remains in $(0, \delta]$,

$$X_n \leq X_0 \exp\left(\sum_{k=0}^{n-1} Y_k\right) \leq X_0 e^{C(\omega)} < \delta.$$

This inequality closes the induction and therefore holds for every $n \geq 0$. Furthermore,

$$\frac{1}{n} \log X_n \leq \frac{1}{n} \log X_0 + \frac{1}{n} \sum_{k=0}^{n-1} Y_k.$$

Taking the upper limit gives

$$\limsup_{n \rightarrow \infty} \frac{1}{n} \log X_n \leq \gamma_\delta < 0.$$

Thus, every sufficiently small initial infection frequency converges to zero exponentially fast almost surely. $\square$

6.4. Release Strategies and Probabilistic Invasion Thresholds

The local Lyapunov exponent at the extinction state satisfies

$$\lambda = \mathbb{E}[\log F'_\Theta(0)] = \mathbb{E}[\log((1-M)(1-S_f))] \leq 0$$

under the biologically admissible parameter assumptions. Thus, an infinitesimally small introduction cannot grow from rarity. Successful establishment must instead result from a finite release capable of overcoming the Allee-type barrier.

In the deterministic model, this barrier is represented by the sharp threshold

$$x_A = \frac{s_f}{s_h}.$$

Under environmental stochasticity, however, the outcome of a release is no longer determined by a single fixed threshold. The same initial infection frequency may lead to different outcomes under different environmental realizations. It is therefore natural to formulate release strategies in terms of probabilities of successful establishment.

Successful Wolbachia establishment is naturally formulated over a finite planning horizon. We therefore introduce a finite-horizon establishment probability that quantifies the likelihood that the infection reaches and remains above a prescribed target infection level.

Definition 6 (Finite-horizon establishment probability). *Let*

$$\eta \in (0, 1), \quad T \in \mathbb{N}, \quad L \in \{1, \dots, T + 1\}.$$

For an initial release frequency $x \in [0, 1]$, define the establishment event

$$\mathcal{E}_{\eta, T, L}(x) = \left\{ \min_{T-L+1 \leq n \leq T} \Phi(n, \omega, x) \geq \eta \right\}.$$

The corresponding finite-horizon establishment probability is

$$p_{\eta, T, L}(x) = \mathbb{P}(\mathcal{E}_{\eta, T, L}(x)).$$

Thus, establishment means that the infection frequency is at least η throughout the final L generations of the planning horizon.

Proposition 7 (Monotonicity of establishment probability). *Suppose assumptions (H1) and (H2) hold. For fixed $\eta \in (0, 1)$, $T \in \mathbb{N}$, and $L \in \{1, \dots, T + 1\}$, the mapping*

$$x \longmapsto p_{\eta, T, L}(x)$$

is nondecreasing on $[0, 1]$.

Proof. Let

$$0 \leq x \leq y \leq 1.$$

Couple the two trajectories by exposing them to the same realization of the environmental process. By the pathwise comparison principle,

$$\Phi(n, \omega, x) \leq \Phi(n, \omega, y)$$

for every $n \geq 0$ and almost every ω . Suppose that

$$\omega \in \mathcal{E}_{\eta,T,L}(x).$$

Then

$$\Phi(n, \omega, x) \geq \eta$$

for every $n = T - L + 1, \dots, T$. The pathwise ordering therefore gives

$$\Phi(n, \omega, y) \geq \Phi(n, \omega, x) \geq \eta$$

for every n in the same final time window. Hence, $\omega \in \mathcal{E}_{\eta,T,L}(y)$. Consequently,

$$\mathcal{E}_{\eta,T,L}(x) \subseteq \mathcal{E}_{\eta,T,L}(y)$$

up to a null set. Taking probabilities yields

$$p_{\eta,T,L}(x) \leq p_{\eta,T,L}(y).$$

Thus, $p_{\eta,T,L}$ is nondecreasing on $[0, 1]$. $\square$

Remark 8 (Importance of monotonicity). *The monotonicity of the finite-horizon establishment probability*

$$x \mapsto p_{\eta,T,L}(x)$$

has both mathematical and biological significance. Mathematically, it ensures that increasing the initial infection frequency cannot reduce the probability of reaching the target establishment level within the prescribed time horizon. Consequently, the superlevel set

$$\{x \in [0, 1] : p_{\eta,T,L}(x) \geq q\}$$

is an upper interval whenever it is nonempty, and the probability-based release threshold

$$x_{q,T,L} = \inf\{x \in [0, 1] : p_{\eta,T,L}(x) \geq q\}$$

is therefore well defined and biologically interpretable.

From an intervention perspective, monotonicity guarantees that a release strategy meeting a prescribed probability of establishment remains sufficient when the initial release frequency is increased. It also permits efficient numerical estimation of $x_{q,T,L}$, since one may search for the threshold using ordered grid refinement or bisection rather than evaluating all possible initial frequencies independently. Thus, monotonicity is essential for turning the establishment probability into a meaningful and operationally useful release criterion.

Definition 7 (Probability-based release threshold). *Let $q \in (0, 1)$ be a prescribed target probability of successful establishment. The q -level release threshold is defined by*

$$x_{q,T,L} = \inf\{x \in [0, 1] : p_{\eta,T,L}(x) \geq q\},$$

where

$$p_{\eta,T,L}(x) = \mathbb{P}_x\left(\min_{T-L+1 \leq n \leq T} X_n \geq \eta\right)$$

is the finite-horizon establishment probability. The quantity $x_{q,T,L}$ is the minimum initial infection frequency required to ensure successful establishment with probability at least q over the planning

horizon T , while maintaining the infection frequency above the target level η throughout the final L generations.

For example,

$$x_{0.50,T,L}, \quad x_{0.75,T,L}, \quad x_{0.90,T,L}$$

represent the minimum release frequencies required to achieve establishment probabilities of at least 50%, 75%, and 90%, respectively.

Remark 9. In the absence of environmental stochasticity and under perfect maternal transmission, the establishment probability reduces to a step function:

$$p(x) = \begin{cases} 0, & x < x_A, \\ 1, & x > x_A, \end{cases} \quad x_A = \frac{s_f}{s_h}.$$

Thus, every probability-based release threshold x_q , with $0 < q < 1$, coincides with the deterministic Allee threshold x_A . Environmental stochasticity replaces this sharp transition by a probabilistic transition region.

7. Numerical Investigation of Release Strategies

The preceding sections developed the analytical structure of the stochastic model, including its pathwise dynamics, Markovian formulation, invariant measures, extinction behavior, and probability-based establishment criteria. We now turn from the theoretical framework to its numerical and biological consequences. The purpose of the simulations is not merely to illustrate individual trajectories, but to determine how the magnitude, source, and dependence structure of environmental variability affect the release frequency required for successful Wolbachia establishment.

We now use numerical simulations to investigate how environmental variability affects Wolbachia establishment and the probability-based release thresholds introduced above. Specifically, we examine stochastic trajectories near the deterministic invasion threshold (Figure 1), establishment probabilities as functions of the initial release frequency (Figure 2), release thresholds under increasing environmental variability (Figure 3), parameter-specific sensitivity (Figure 4), the effects of correlated environmental fluctuations (Figure 5), and long-term empirical distributions (Figure 6). These six numerical experiments provide complementary views of how environmental uncertainty modifies Wolbachia establishment and release requirements. The complete simulation design and numerical implementation are provided in Appendix A.

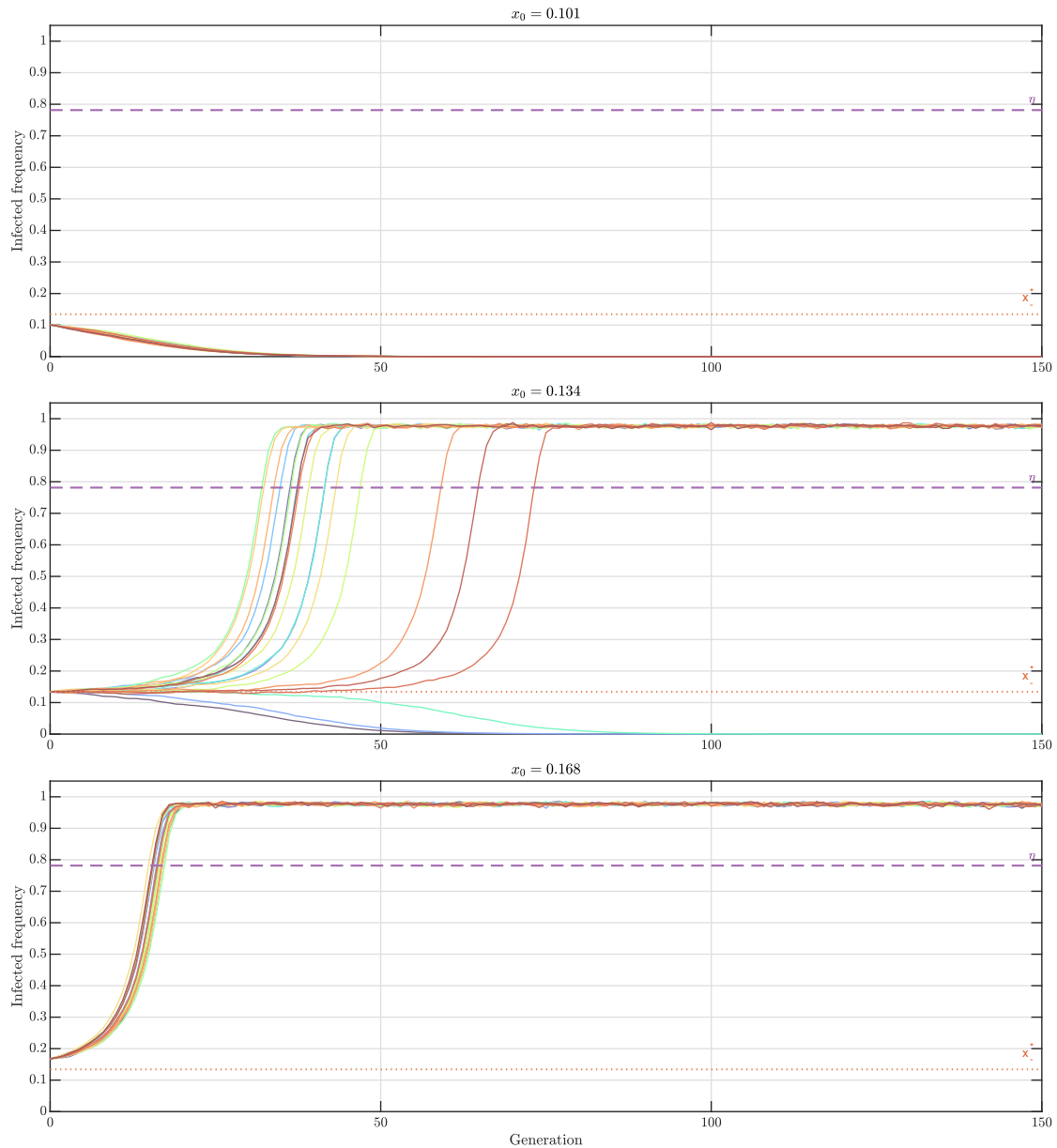


Figure 1. Sample paths of the stochastic Wolbachia model for initial infection frequencies below ($x_0 = 0.75x_-^*$), at ($x_0 = x_-^*$), and above ($x_0 = 1.25x_-^*$) the deterministic invasion threshold. Twenty independent realizations are shown in different colors, with independent fluctuations in (S_f, S_h, M) and $\kappa = 100$; the colors distinguish individual stochastic trajectories and do not represent different parameter values. The dotted and dashed lines denote x_-^* and the establishment target $\eta = 0.8x_+^*$, respectively. Stochasticity produces variable outcomes near the deterministic threshold.

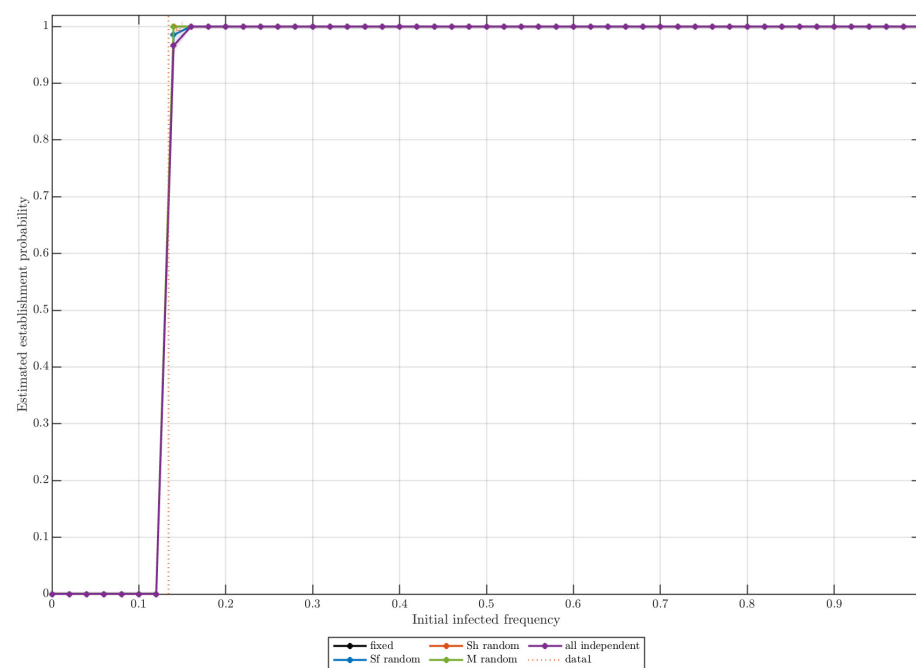


Figure 2. Estimated finite-horizon establishment probability as a function of the initial Wolbachia infection frequency under fixed parameters and different sources of environmental variability ($\kappa = 100$). The curves correspond to the fixed-parameter case, individual fluctuations in S_f , S_h , or M , and simultaneous independent fluctuations in all three parameters. Several curves are nearly coincident and therefore overlap, causing some colors—in particular the black and orange curves—to be partially or completely masked by other curves. Establishment is defined by $\min\{X_n : T - L + 1 \leq n \leq T\} \geq \eta$, with $T = 300$ and $L = 50$. The vertical dotted line denotes the deterministic invasion threshold x_* . Environmental stochasticity transforms the sharp deterministic threshold into a narrow probabilistic transition region.

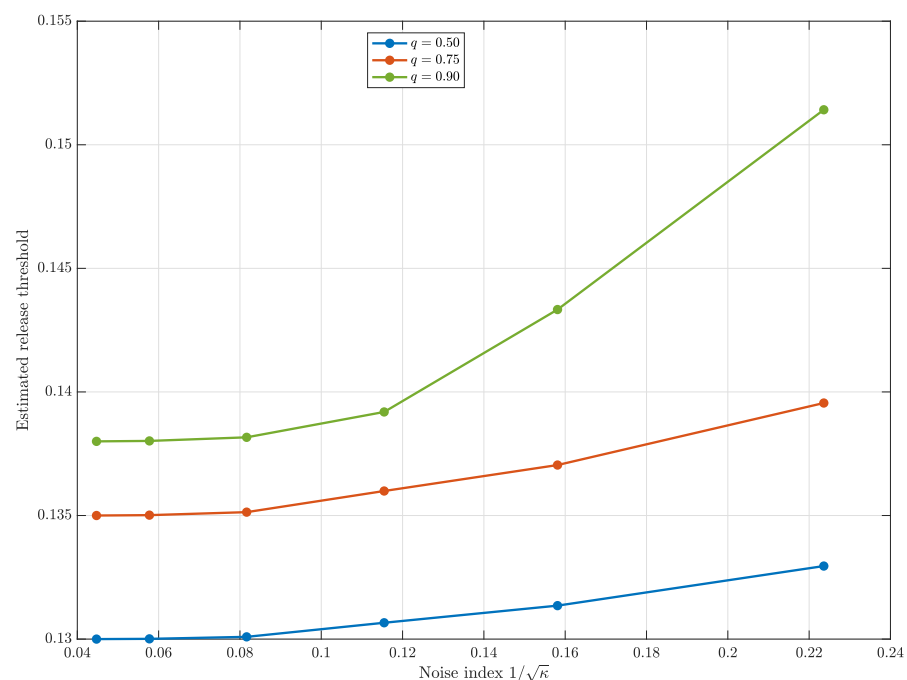


Figure 3. Effect of increasing environmental variability on the probability-based Wolbachia release thresholds for target establishment probabilities $q = 0.50, 0.75$, and 0.90 . Environmental variability is measured by the noise index $\nu = 1/\sqrt{\kappa}$, so that larger values correspond to stronger variability. The required release frequency increases with environmental variability, with the effect becoming more pronounced for higher target establishment probabilities.

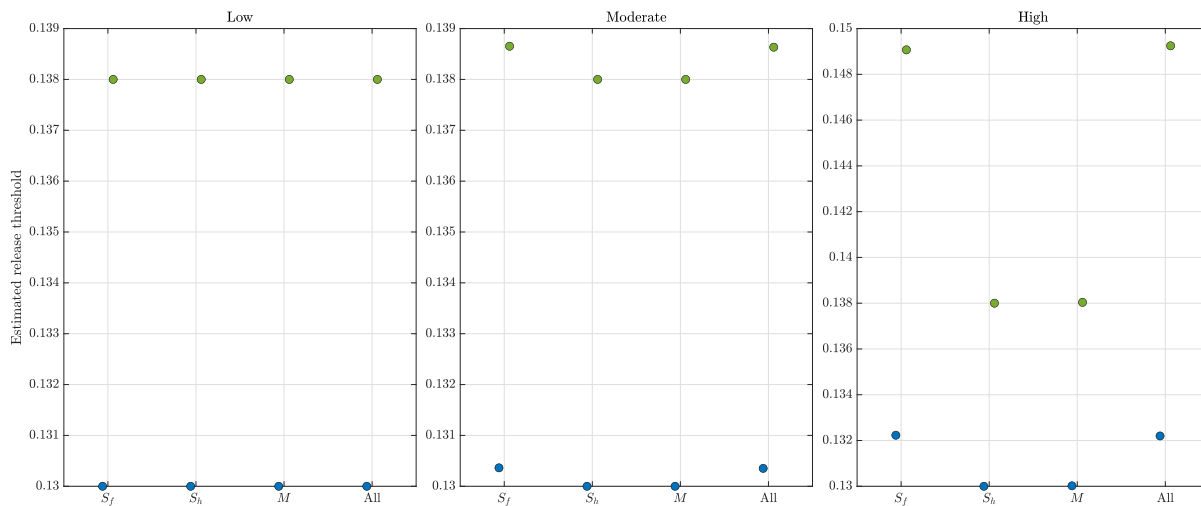


Figure 4. Parameter-specific sensitivity of the probability-based Wolbachia release thresholds under low ($\kappa = 500$), moderate ($\kappa = 100$), and high ($\kappa = 25$) environmental variability. The green dot denotes the deterministic release threshold used as the baseline reference. Differences are negligible at low variability but become more pronounced as variability increases. Among the individual parameters, fluctuations in the fitness cost S_f have the strongest influence, whereas fluctuations in S_h and M have comparatively little effect. Simultaneous variation of all three parameters produces the largest overall effect.

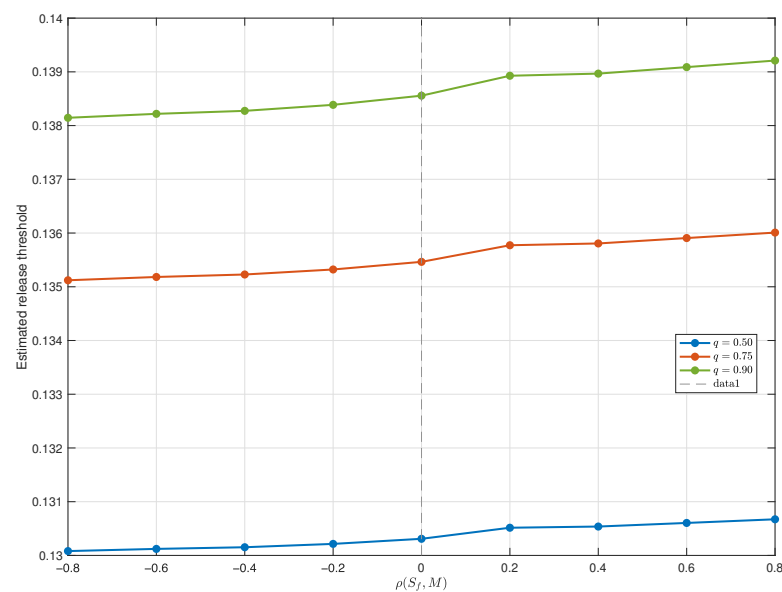


Figure 5. Effect of correlation between the fitness cost S_f and maternal-transmission leakage M on the probability-based Wolbachia release thresholds for target establishment probabilities $q = 0.50$, 0.75 , and 0.90 . The thresholds increase gradually as $\rho(S_f, M)$ increases, indicating that stronger positive dependence between these two sources of environmental variability raises the initial infection frequency required for a prescribed probability of successful establishment.

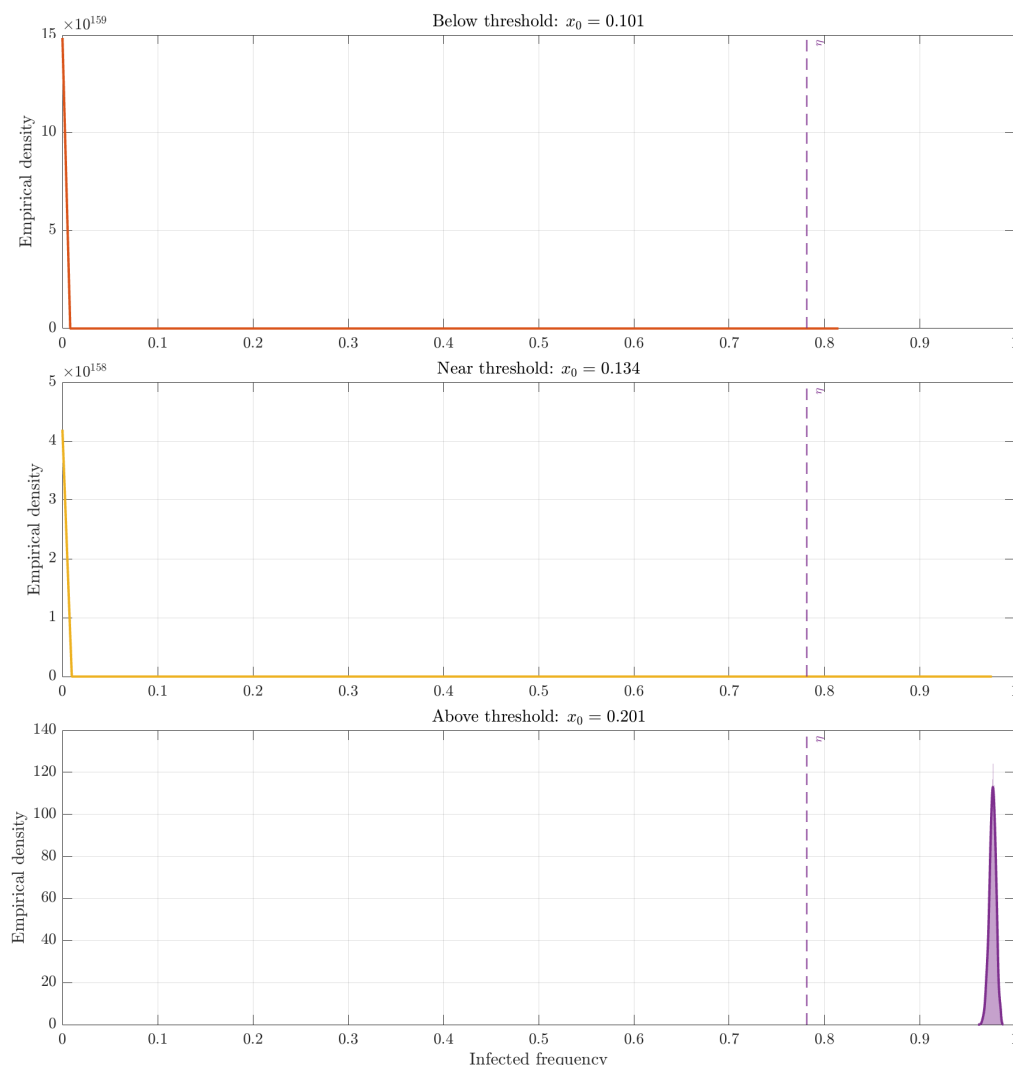


Figure 6. Long-term distributions of the Wolbachia infection frequency for initial conditions below ($x_0 = 0.101$), near ($x_0 = 0.134$), and above ($x_0 = 0.201$) the deterministic invasion threshold. For initial conditions below or near the threshold, the distribution concentrates at zero, indicating extinction, whereas an initial condition above the threshold produces a distribution strongly concentrated near the high-infection state. The dashed vertical line denotes the establishment target η . The results illustrate the threshold-dependent transition between extinction and a numerically observed high-frequency infection regime under environmental stochasticity.

Synthesis of the Numerical Results

The numerical experiments reinforce three main conclusions. First, environmental stochasticity replaces the sharp deterministic invasion threshold by a probabilistic transition region, with releases near the deterministic threshold being most sensitive to environmental fluctuations. The probability-based threshold $x_q(T, \eta)$ therefore provides a natural measure of the release required to achieve a prescribed establishment probability.

Second, release requirements depend on both the magnitude and source of environmental variability. Increasing variability generally raises the required release frequency, with fluctuations in the fitness cost S_f having the strongest individual effect under the baseline parameter regime. Positive dependence between S_f and maternal-transmission leakage M further increases the release threshold, showing that the dependence structure of environmental parameters also matters.

Finally, the long-term simulations suggest a stochastic analogue of deterministic bistability: smaller releases tend toward extinction, whereas sufficiently large releases

concentrate near a high-infection regime. Consistent with Remark 6, this provides numerical evidence, rather than a proof, for a nontrivial positive invariant regime. Overall, the simulations support the theoretical conclusion that Wolbachia establishment under environmental uncertainty is fundamentally a finite-release problem.

8. Discussion and Conclusions

This work develops a stochastic framework for studying the establishment and long-term behavior of Wolbachia infections when key biological parameters are subject to environmental variability. In contrast with the deterministic model, where invasion is organized around a sharp threshold, the stochastic model naturally leads to a probabilistic description of establishment. This distinction is particularly important from the perspective of release design: an initial infection frequency above the deterministic threshold does not by itself specify the probability of successful establishment in a fluctuating environment.

The analytical results establish the basic random dynamical and Markov structure of the model, including positivity, order preservation, the Feller property, and existence of invariant probability measures. The extinction state remains locally attracting under biologically admissible parameter values, emphasizing that successful Wolbachia introduction is fundamentally a finite-release problem rather than an invasion-from-rarity phenomenon. This motivates the finite-horizon establishment probability and the associated q -level release threshold introduced in this paper.

The stochastic formulation is motivated by experimental evidence that the biological mechanisms governing Wolbachia establishment are sensitive to environmental conditions. In particular, heat stress can reduce Wolbachia density, weaken cytoplasmic incompatibility, and decrease maternal-transmission fidelity. For example, field experiments with wMel-infected *Aedes aegypti* have shown substantial reductions in Wolbachia density and cytoplasmic incompatibility under high-temperature conditions [29], while laboratory experiments have demonstrated temperature-dependent reductions in maternal-transmission fidelity [30]. These observations provide biological support for treating S_f , S_h , and M as environmentally varying quantities rather than as fixed parameters. The present simulations are not fitted to a particular field trial; instead, they examine the consequences of such variability for establishment and release thresholds. Direct estimation of the environmental parameter distributions from longitudinal field data is an important direction for future work.

The present framework should also be distinguished from classical SIR/SIRS models, which describe horizontal pathogen transmission among epidemiological compartments. Wolbachia invasion instead involves the intergenerational frequency of a maternally transmitted endosymbiont driven by fitness effects, cytoplasmic incompatibility, and maternal transmission. Similarly, spatial models of bacterial colonies and biofilm formation may incorporate cell proliferation, quorum sensing, signalling, and spatial heterogeneity [31]. Although these models represent different biological mechanisms, they share the broader objective of understanding how variability and heterogeneity modify thresholds, persistence, and collective population behavior. Thus, the stochastic methodology developed here may be transferable to other discrete-time biological systems, but the underlying map must be chosen to reflect the mechanisms of the particular application.

The numerical results reinforce this interpretation. Environmental stochasticity replaces the sharp deterministic threshold by a probabilistic transition region, with releases near the deterministic threshold displaying the greatest sensitivity to environmental fluctuations. The simulations also show that the magnitude and source of variability matter: uncertainty in the fitness cost S_f has a stronger influence on the required release frequency than comparable variability in S_h or M , while simultaneous fluctuations can further in-

crease the release requirement. Moreover, positive dependence between fitness cost and maternal-transmission leakage increases the probability-based release threshold. These observations suggest that release strategies should account not only for mean biological parameter values but also for their variability and dependence structure.

Although the recurrence studied here is specific to Wolbachia, the underlying stochastic methodology is not. More generally, a discrete-time biological model

$$x_{n+1} = F(x_n; \theta)$$

can be placed in a random environment by replacing the fixed parameter vector θ with a stochastic process Θ_n , yielding

$$X_{n+1} = F(X_n; \Theta_n).$$

The resulting framework may therefore be adapted to other discrete-generation invasion, population-replacement, or frequency-dependent ecological and evolutionary systems subject to environmental variability. The biological mechanisms must, however, be represented by an appropriate system-specific map F : the present Wolbachia recurrence describes maternal transmission and cytoplasmic incompatibility and should not be interpreted as a general model of horizontal bacterial transmission. Thus, what is transferable is the random-environment framework rather than the Wolbachia-specific recurrence. The present model deliberately focuses on temporal environmental variability in a nonspatial population. Several extensions are therefore natural, including temporally correlated environments, alternative dependence structures, spatial dispersal, repeated or adaptive releases, and stochastic integrodifference or lattice models. Such extensions would allow the probability-based threshold framework developed here to be connected more directly with operational Wolbachia deployment strategies and spatial invasion dynamics. Overall, the results indicate that environmental stochasticity does not simply perturb the deterministic invasion threshold; it changes the relevant management question from whether a release exceeds a fixed threshold to how large a release is required to achieve a prescribed probability of successful establishment.

The present framework should also be viewed as complementary to existing spatial models of Wolbachia and biological invasion. Reaction–diffusion, integrodifference, and related spatial models are primarily designed to describe the propagation of an established infection through space, including invasion fronts, traveling waves, and their associated spreading speeds [16,17]. By contrast, the model developed here is deliberately nonspatial and focuses on uncertainty in the establishment stage arising from temporal fluctuations in fitness cost, cytoplasmic incompatibility, and maternal transmission. Its principal contribution in this direction is an explicit finite-horizon establishment probability and the associated probability-based release threshold, which quantify how release requirements depend on the magnitude, source, and dependence structure of environmental variability. Thus, the present results address uncertainty in whether establishment occurs, rather than the subsequent spatial rate at which an established infection spreads. Incorporating both mechanisms within stochastic reaction–diffusion, integrodifference, or lattice models would provide a natural extension of the present work.

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Appendix A. Numerical Implementation and Simulation Design

The numerical experiments are based on the stochastic difference equation

$$X_{n+1} = \frac{(1 - M_n)(1 - S_{f,n})X_n}{S_{h,n}X_n^2 - (S_{h,n} + S_{f,n})X_n + 1}.$$

Unless otherwise stated, the deterministic reference parameters are

$$\bar{s}_f = 0.10, \quad \bar{s}_h = 0.90, \quad \bar{m} = 0.02.$$

The positive equilibria of the corresponding deterministic system satisfy

$$s_h x^2 - (s_h + s_f)x + s_f + m(1 - s_f) = 0.$$

For the baseline values,

$$x_-^* \approx 0.1342, \quad x_+^* \approx 0.9769,$$

and the establishment target is chosen as

$$\eta = 0.8x_+^* \approx 0.7815.$$

Environmental variability is generated with scaled Beta distributions. If $\Theta \in [a, b]$ has prescribed mean $\bar{\theta}$, we write

$$\Theta = a + (b - a)Z, \quad Z \sim \text{Beta}(\alpha, \beta),$$

where

$$r = \frac{\bar{\theta} - a}{b - a}, \quad \alpha = \kappa r, \quad \beta = \kappa(1 - r).$$

Thus, large κ corresponds to weak environmental variability and small κ to stronger variability. We use

$$\nu = \frac{1}{\sqrt{\kappa}}$$

as a monotone noise index. This is a convenient index rather than the exact standard deviation, since the latter also depends on the support and mean of the scaled Beta distribution. The supports are

$$S_f \in [0.02, 0.25], \quad S_h \in [0.60, 1], \quad M \in [0, 0.08].$$

Unless otherwise specified, the simulations use $T = 300$, $L = 50$, and N_{MC} independent Monte Carlo realizations for each initial condition. A realization is classified as established if

$$\min \{X_n : T - L + 1 \leq n \leq T\} \geq \eta.$$

The Monte Carlo estimator is

$$\hat{p}_{\eta,T,L}(x) = \frac{1}{N_{\text{MC}}} \sum_{j=1}^{N_{\text{MC}}} \mathbf{1}_{\left\{ \min_{T-L+1 \leq n \leq T} X_n^{(j)} \geq \eta \right\}}.$$

Pointwise 95% confidence intervals are computed with the Wilson binomial interval. For a target success probability q , the release threshold is estimated from

$$x_{q,T,L} = \inf\{x \in [0, 1] : \hat{p}_{\eta,T,L}(x) \geq q\},$$

with linear interpolation between adjacent grid points when necessary.

Appendix B. Brief Guide to the Stochastic Concepts Used in the Paper

For additional background on random dynamical systems, Markov chains, Feller operators, invariant measures, and stochastic persistence, we refer to [18–20,22,32,33].

Appendix B.1. Random Dynamical System

The deterministic model has the form

$$x_{n+1} = F(x_n; \theta),$$

with fixed parameter vector θ . In the stochastic model, θ is replaced by a random environmental process Θ_n , giving

$$X_{n+1} = F_{\Theta_n}(X_n).$$

For a fixed environmental realization

$$\Theta_0(\omega), \Theta_1(\omega), \dots,$$

the system generates a deterministic trajectory. The collection of such trajectories over all environmental realizations constitutes the random dynamical system considered in Section 4.

Appendix B.2. Markov Property

When the environmental variables Θ_n are independent and identically distributed, the future evolution of X_n depends on the present infection frequency but not on its complete past history. More precisely,

$$\mathbb{P}(X_{n+1} \in A \mid X_0, \dots, X_n) = \mathbb{P}(X_{n+1} \in A \mid X_n).$$

Thus, under i.i.d. environmental forcing, $\{X_n\}$ is a time-homogeneous Markov chain on $E = [0, 1]$.

Its one-step transition kernel is

$$P(x, A) = \mathbb{P}(F_{\Theta}(x) \in A),$$

which represents the probability that a population currently at infection frequency x moves into the set A after one generation.

Appendix B.3. Feller Property

The associated Markov operator is

$$(P\varphi)(x) = \mathbb{E}[\varphi(F_{\Theta}(x))].$$

It is called Feller if

$$\varphi \in C(E) \implies P\varphi \in C(E).$$

Intuitively, this means that small changes in the current infection frequency produce small changes in expected future observables. In the present model, continuity of F_θ implies the Feller property. Since $E = [0, 1]$ is compact, this regularity allows the use of standard Krylov–Bogolyubov arguments to establish the existence of invariant probability measures.

Appendix B.4. Invariant Probability Measures

A probability measure π is invariant if

$$\pi P = \pi.$$

Equivalently, if

$$X_0 \sim \pi,$$

then

$$X_n \sim \pi \quad \text{for every } n \geq 0.$$

Thus, an invariant measure describes a statistical equilibrium of the stochastic system. Unlike a deterministic equilibrium, the infection frequency may continue to fluctuate even though its probability distribution remains unchanged. Since

$$F_\theta(0) = 0,$$

the extinction measure δ_0 is always invariant.

Appendix B.5. Stochastic Lyapunov Exponent

The local behavior near extinction is characterized by

$$\lambda = \mathbb{E}[\log F'_\Theta(0)].$$

For the present model,

$$F'_\Theta(0) = (1 - M)(1 - S_f),$$

and therefore,

$$\lambda = \mathbb{E}[\log((1 - M)(1 - S_f))].$$

A negative value of λ indicates average exponential contraction near the extinction state. Since

$$(1 - M)(1 - S_f) \leq 1$$

under the biologically admissible assumptions, successful establishment is therefore a finite-release phenomenon rather than invasion from an arbitrarily small initial frequency.

In summary, the random dynamical system describes individual trajectories under specified environmental realizations, while the Markov kernel and Markov operator describe the corresponding probabilistic evolution. The Feller property provides the regularity needed for the existence of invariant measures, and the stochastic Lyapunov exponent characterizes local behavior near extinction.

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